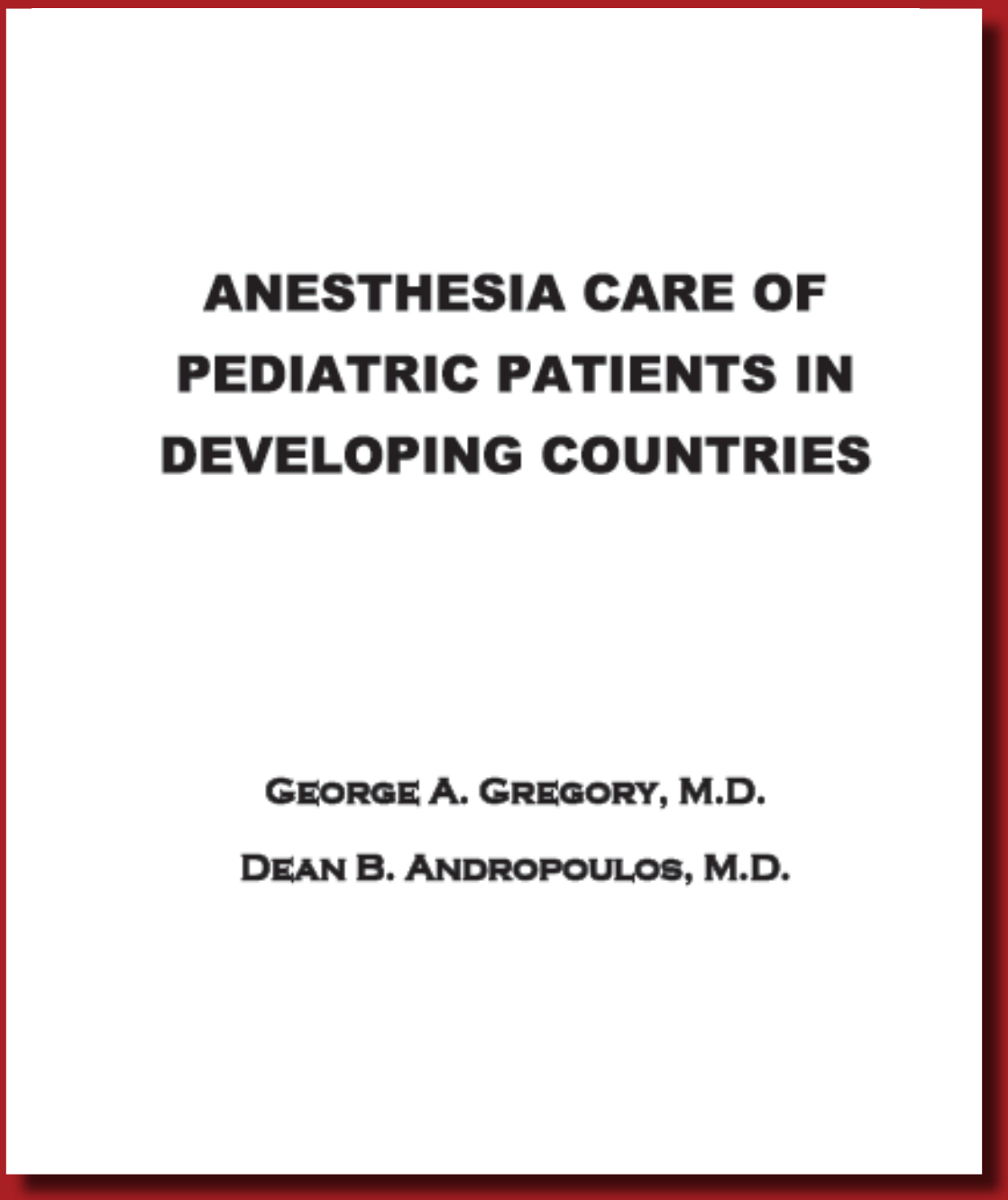
Anesthesia Care Of Pediatric Patients In Developing Countries



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ANESTHESIA CARE OF PEDIATRIC PATIENTS IN DEVELOPING COUNTRIES

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Dedication:

This book is dedicated to the children of the world and to those who provide anesthesia care for them.

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Preface

George A. Gregory, MD and Dean Andropoulos, MD

Approximately 35 percent of the world's population is < 18 years of age. Many of these children require surgery each day. As a result, about 10 percent of all surgeries performed throughout the world each year are performed in this age group, which requires someone to provide anesthesia. For anesthesia to be safe, the anesthetist must understand the many physiologic and pharmacologic differences between children and adults. Providing anesthesia by a formula (giving a specific amount of a drug by some formula, e.g., mg/kg), as is often done, is dangerous and can be disastrous for some patients. Not all patients are the same and what works for one patient often does not work for another. One reason for this is that there are genetic differences among patients that make their responses to drugs and drug metabolism quite different. An example is codeine. About 85% of people effectively metabolize codeine to morphine and get pain relief from the drug. But for genetic reasons, some people cannot metabolize codeine to morphine and get no pain relief at all from the drug beyond that due to other drugs with which it is mixed (e.g., acetaminophen). Some people, especially those in West Africa, convert twice-as-much codeine to morphine, which can produce respiratory depression or respiratory arrest, even with normal doses of codeine. Failure to understand these differences either proves to be disastrous or to result in inadequate pain relief for some people.

It is the authors' belief and experience that administration of anesthesia must be given based on physiologic principles, not by a formula that is supposed to fit every patient. Where data are available, it is clear that the mortality of surgical patients is much higher when given based on formula than when it is given based on sound principles of physiology and pharmacology. Therefore, wherever possible, the multiple authors of this book have based their statements and recommendations on physiology and pharmacology.

Some of the topics and material presented in this book may be difficult to do or use in low-income settings, but the concepts in these chapters can be applied to the care of many patients. Therefore, it is important for the reader to read and understand each chapter and to determine how to apply the information and concepts to their patients.

The purpose of this book is to provide information for anesthetists in developing countries who must anesthetize children but have limited access to pediatric anesthesia textbooks. All that is

needed to access the contents of this book is access to a computer, and the Internet. This book is available through several anesthesia society websites and commercial e-book companies. There is no charge for downloading the book from the website, and no purchase of the book from a third party is allowed. In many instances it may be appropriate for one person or a group of people to download the book and to reproduce it for distribution to other anesthetists. If this is done, please let us know how many copies of the book were made and distributed. This will help us know how widely the book is distributed. We encourage readers to translate parts of or the entire book into their native languages.

Our goal is to update this material periodically, especially after feedback from people who use the book. Contact the authors at the following email addresses: George Gregory--gregoryg@anesthesia.ucsf.edu; Dean Andropoulos—dra@bcm.edu with questions and suggestions. These questions will be answered as promptly as possible. Also, if there are other subjects you would like to have covered in the book, please let us know at the same email addresses, and we will add them if possible.

George A. Gregory and Dean B. Andropoulos, Editors.

September 2014

Chapter 1: PREOPERATIVE EVALUATION

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Case Study: –A five-day-old male infant has had abdominal distention and vomiting since birth. He was born at term in a small village by a midwife and had Apgar scores of 8 and 9 at 1 and 5 minutes of life. He never had a bowel movement. After 24 hours of life he refused feedings and began vomiting green bilious material. Further attempts to feed him were unsuccessful. After a 16-hour trip to the hospital, he was found to be dehydrated and near death and thought to have a bowl obstruction. The following is a discussion of the preoperative evaluation of this and all patients.

Preoperative evaluation is the most important part of any anesthetic! Failure to properly evaluate patients before anesthesia and surgery and to clearly understand any preoperative problems and potential intraoperative complications increases the incidence of intra and postoperative complications, including cardiac arrest and death. Information gained from the patient's history and from the physical and laboratory examinations allows the anesthetist and surgeon to correct preoperative problems, appropriately plan the anesthetic, and take steps to avoid complications. It also allows for proper planning for postoperative relief of any pain the patient will have. It only takes a few minutes to do an effective preoperative evaluation, especially if the history is obtained while the physical examination is being done.

Obtaining Information

There are multiple sources of patient information available to the anesthetist, including chart notes and discussions with the nurses and the physicians. Discussions with the patient's nurse(s) are very important because they spend most of their day with the patient and often understand the patient's problems better than anyone else. The patient's nurses often is aware of such things as the effects of changes in body position on arterial blood pressure and heart rate (e.g., does the patient become hypotensive and/or tachycardic with changing his position?) and of problems associated with drug administration (e.g., hypotension, tachycardia, allergic reactions)? After discussions with the nurse(s), information is sought from the physician(s) caring for the patient. The anesthetist must be aware of and understand the effects of all drugs being administered to the child and the interaction(s) of these drugs with anesthetics and other drugs administered during surgery.

Chapter 1: PREOPERATIVE EVAUATION

Evaluation of the Hospital Chart

The purpose of the hospital chart (record) is to store patient information that is both readable and readily available to other people who will care for the patient. Consequently, written notes should be clear, concise, and informative. There should be no doubt about the meaning of information written in the chart. Information obtained from the chart preoperatively should include the birth history, especially prenatal or neonatal complications that occurred. Hypoxemia or asphyxia before (e.g., fetal bradycardia, decreased fetal movement), during, or immediately after birth (e.g., cyanosis, apnea, delayed onset of breathing, bradycardia, and/or hypotension) can be associated with intracranial hemorrhage, tricuspid valve insufficiency, myocardial damage, decreased cardiac output, renal failure, and necrotizing enterocolitis. These abnormalities may persist for days-to-months and occasionally may be permanent. It is important to determine if there was pre- or intra-partum bleeding due to premature placental separation. If there was, the patient may well be hypovolemic and anemic and require a blood transfusion prior to anesthesia and surgery to prevent her/him from becoming severely hypotensive and suffer a cardiac arrest with the induction of anesthesia. Finally, was the umbilical cord wound so tightly around the fetus' neck or body at the time of birth that it was necessary to cut the cord to deliver the baby? Having to do so can leave 25-40 percent of the fetus' blood volume in the placenta, causing severe neonatal hypovolemia. **Appendix 1** provides the normal heart rates, arterial blood pressures, and central venous pressures of infants and children.

Hydration

Babies live in a sac of water for nine months before birth. Consequently, they have excess total body and of lung water at birth. The kidneys excrete this excess water over the first 3 - 4 days after birth, which causes a 10% - 15% decrease in body weight. Babies born by cesarean section often have more excess water (especially lung water) than babies born vaginally. Thus, *transient tachypnea of the newborn,* which consists of tachypnea and oxygen desaturation (SpO_2 <90% when breathing room air), develops more often in babies delivered by cesarean section than in vaginally delivered babies and often persists for several days. The combination of the fluid loss and insufficient fluid intake during the first few days of life can lead to volume depletion (dehydration). Babies born in a hospital are often more likely to be dehydrated because physicians caring for them often limit their fluid intake to 65ml/kg/day (rather than 120-150ml/kg/day) to help the babies loose their excess fluid more quickly. If such babies require surgery, they are likely to develop hypotension with the induction of anesthesia. In hot climates they may be more dehydrated. Infants born outside a hospital may be very dehydrated upon arrival at hospital if fluid intake was inadequate (as it was in the case described above). Thus, determination of fluid intake (oral and I.V.) and output (urine, blood, vomiting, diarrhea), the state of hydration, and the need to replace all volume deficits is important to prevent hypotension and decreased tissue perfusion during the induction of anesthesia.

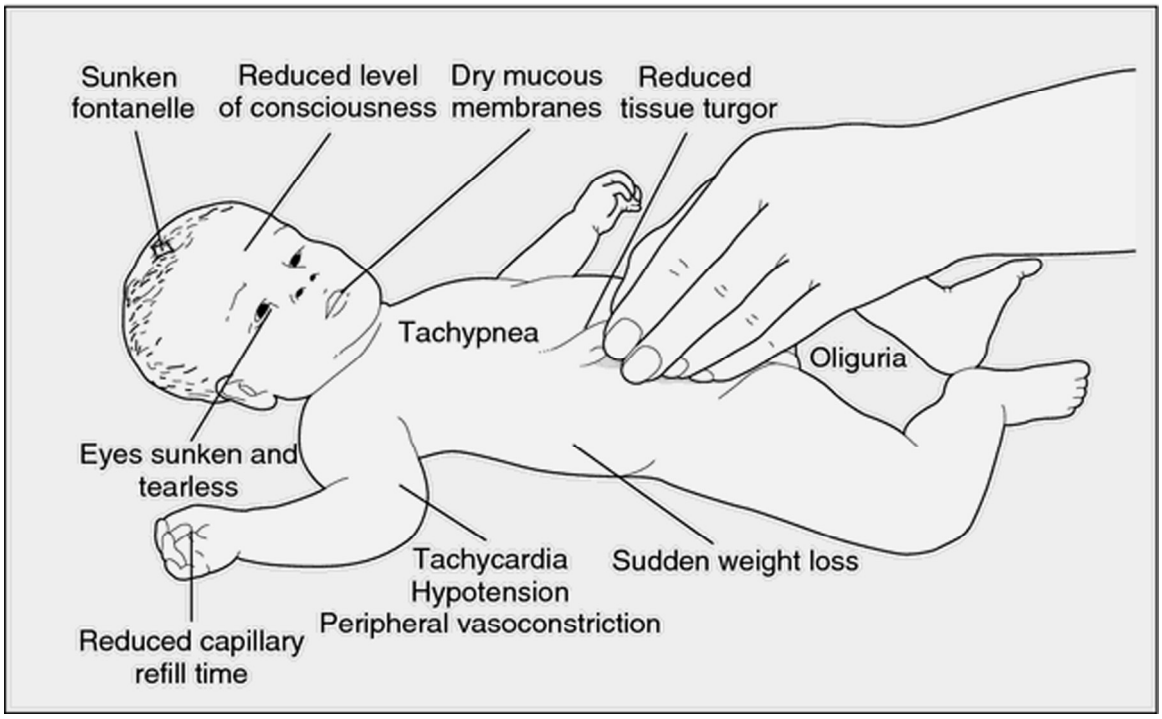
The volume status of patients can usually be determined from the history and physical examination. Changes in the child's weight are important, especially if they occurred over a short period of time. For instance, if a baby weighed 5kg three days ago and now weighs 4.5 kg, this weight loss is almost certainly due to fluid loss and dehydration. But it must be remembered that some patients can have excessive loss of fluid into tissues (third space losses) and still have inadequate intravascular volume without a change in body weight. This occurs when fluids are sequestered in the bowel, peritoneum, plural cavities, or in the extremities (edema). In this instance the weight will not have changed, but the *intravascular* volume may be inadequate.

Urine output is another useful sign of hydration. Most infants and young children produce at least 0.75 ml/kg/hr. of urine; failure to urinate during the previous 4 hours is associated with hypotension (>30% decrease in systolic blood pressure from preoperative values) in approximately 70% of neonates during the induction of anesthesia. Because neonates cannot concentrate their urine for the first weeks of life, their urine specific gravity is often 1.005 or less. The concentrating ability of the kidneys increases to near adult levels by about two years of age. The urine pH is usually equal to or greater than five, due to loss of bicarbonate in the urine. The concentrating ability of the kidneys increases when the child's protein intake increases. If the urine specific gravity is exceeds 1.011, 70% of infants will also become hypotensive when anesthesia is induced. Older children with a preoperative urine specific gravity ≥1.035 are usually dehydrated and often develop hypotension during the induction of anesthesia.

If the child has a fontanel, the skin position over the fontanel is a useful guide to his or her volume status. Skin covering the fontanel of a well-hydrated baby is even with the bone of the outer skull. If the fontanel is sunken to the inner level of the skull bone, the intravascular volume is reduced by 5%. If the fontanel is below the inner level of the skull, the volume deficit is at least 10%. Crying or excessive ventilation pressures cause the fontanel to bulge outward and invalidates the use of fontanel skin position as an aid in determining intravascular volume. *Since, the head and fontanel are usually in front of the anesthetist during surgery, the anesthetist can use the position of the skin over the fontanel to evaluate intravascular volume during surgery.*

Decreased skin turgor and slow capillary refill are additional signs of dehydration. The skin of normally hydrated infants returns immediately to its normal flat position when it is lifted and released. However, the skin of a hypovolemic baby remains " tented up" for several seconds before returning to its normal position (**Figure 1**). After the skin is blanched, the capillaries of normally hydrated babies and young children refills with blood in less than three seconds. That of older normally, hydrated patients refills in less than two second. The capillary filling times of severely dehydrated infants and children often exceeds five seconds. The mucus membranes of the mouth should be moist. If they are dry, this too is a sign of dehydration.

Figure 1-1. Signs of Dehydration Observed in an Infant.



See text for discussion. From: Miller-Keane Encyclopedia and Dictionary of Medicine, Nursing, and Allied Health, Seventh Edition. © 2003 by Saunders, an imprint of Elsevier, Inc.

Peripheral pulses and skin temperature are also useful guides when determining the intravascular volume of infants and children (**Fig. 1-1**). The examiner should use her/his *non-dominant* hand (left hand for right-handed persons and *visa versa*) to feel for pulses because the fingers of the non-dominant hand have fewer calluses and better sensation. Excessive pressure on the skin over a baby's artery can easily collapse the artery and lead to the false conclusion that the pulses are absent. Diminished or absent dorsalis pedis or radial artery pulses usually indicate a 5% reduction in intravascular volume. Diminished or absent popliteal and brachial artery pulses indicate a 10% reduction in intravascular volume, while diminished or absent axillary and femoral artery pulses are consistent with a 15% or more reduction in intravascular volume.

The heart rate of sick neonates and young children may be fixed at about 150 beats per minute (BPM) and not change with increases or decreases in blood pressure, indicating that the baroreceptor response is depressed or non functional. The heart rate of older children usually changes when the arterial blood pressure changes. If the blood pressure is more than two standard deviations below the normal mean blood pressure for age (**See Appendix 1**), the child is hypotensive.

In a warm environment (e.g., in an infant warmer or a warm climate), examining the infant's skin temperature is also a useful sign of whether the intravascular volume is adequate or not. The

examiner should place her/his hand on the patient's chest and move the hand outward along the skin of the chest, shoulder, and arm to the patient's fingers. Under normal circumstances, the patient's skin temperature will be warm all the way to the fingertips. A sudden decrease in temperature in the forearm is associated with an approximately 5% decrease in intravascular volume, while a sudden decreases in temperature in the upper arm or leg usually indicates a 10% reduction in intravascular volume. A decrease in temperature from the axilla or groin outward to the end of the extremity is indicative of a 15% or more reduction of volume.

The position of the liver relative to the right costal margin is another useful aid when determining intravascular volume. In infants and young children, the size of the liver is an index of how well the right ventricle is functioning and can be used as a surrogate for central venous pressure (CVP), which under normal circumstances is 2-8cmH_2O. An acute increase in central venous pressure or the presence of a pneumothorax decreases blood return to the heart and causes the liver to rapidly enlarge. Sudden liver enlargement is usually indicative of a failing right ventricle and inadequate ejection of the blood returning to the heart. Reduced ejection of blood from the right ventricle is often due to fluid overload, pulmonary hypertension, left ventricular failure, or disease of the cardiac valves. Failure of the right ventricle to adequately eject the returning blood increases right atrial and central venous pressures, which causes rapid enlargement of the liver. The liver edge of infants and young children can enlarge from its normal position two cm below the right costal margin to the pelvis in less than five minutes. Correcting the cause of right ventricle failure and liver enlargement leads to a rapid reduction in liver size and to return to its normal position in less than 5 min.

The position of the liver in the abdomen can be determined in several ways. The first is to gently run one's fingers back and forth across the right abdominal wall, without exerting excessive pressure, until the liver edge is felt. If excessive pressure is applied, the liver may be pushed up under the right costal margin and make the liver seem smaller than it really is. If the abdomen is distended or painful, a stethoscope can be gently placed on the abdomen and a fingernail can be gently scraped from the lower abdomen upwards towards the rib cage. The tone made by the fingernail scraping will suddenly become duller when the liver edge is encountered. Because the liver of hypovolemic children is smaller than normal, its edge may be up under the ribs. During abdominal surgery, the liver edge is usually visible to the surgical team and can be used by the anesthetist to both evaluate the intravascular volume of the child and to guide fluid administration. When adequate fluid has been administered and the heart is functioning normally, the liver edge will be approximately 2 cm below the right costal margin.

While any one of the above signs may not be indicative of the patient's intravascular volume status in all cases, taken together they give an accurate indication of the patient's intravascular volume. This examination takes only a couple of minutes to complete and should be part of *every* preoperative evaluation. With the exception of the liver edge and the fontanel, the above can be used to determine the volume status for patients of all ages.

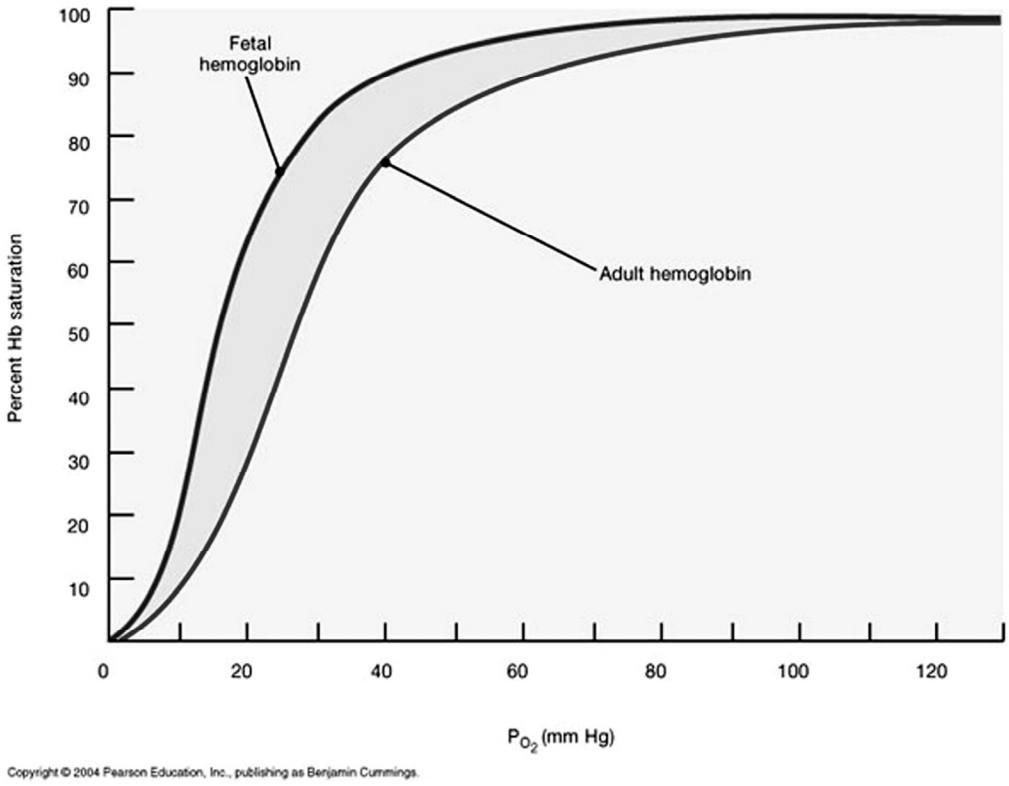
Respiratory System

The respiratory system begins its development very early in gestation but is not completely developed at birth. The larynx, trachea, bronchi, and lungs arise as outgrowths of the ventral wall of the foregut; failure of any of the steps required for normal organ development leads to airway anomalies, including laryngeal clefts, agenesis of the lung, and tracheal-esophageal fistulae. Surface-active material (SAM) prevents lungs from collapsing at the end of expiration and is usually secreted into the developing alveoli at 35-36 weeks gestation. Failure to secrete SAM, as often occurs in premature babies, results in hyaline membrane disease, a major cause of neonatal death worldwide.

Before birth the lungs are filled with about 30ml of fluid per kg of body weight that must be removed immediately after birth and replaced with approximately 30cc of gas per kg of body weight. Clearance of fluid from the lungs begins during labor and continues during both the birth process and breathing after birth.

Before birth the partial pressure of oxygen (PaO2) in blood perfusing the heart and brain is only 30-to-40mmHg. Despite this low PaO2, there is sufficient oxygen available for fetal growth because the concentration of hemoglobin is high (approximately 18g/dl) and the oxygen dissociation curve is shifted to the left (**Figure 1-2**).

Figure 1-2. Fetal and Adult Oxygen Dissociation Curves for Hemoglobin.



Note that the fetal curve is shifted to the left, which allows hemoglobin to take up oxygen at lower oxygen concentrations than adult hemoglobin. The PaO_2 associated with 50% hemoglobin oxygen saturation is 27mmHg in adults and 18mmHg in fetuses and babies less than about 3 months of age. ***From: Lissauer and Graham, 2002. With permission***

This left shifted oxygen dissociation curve allows fetal hemoglobin to carry more oxygen at lower PaO_2s. Due to the left-shifted oxygen dissociation curve and a higher hemoglobin concentration, fetal blood contains the same amount of oxygen as that of normal children and adults (20cc O_2/100ml of blood). If there is a decrease in umbilical cord blood flow, severe hypoxemia occurs very rapidly because there is no oxygen reserve in the lungs, as there is in older children. All of the fetus' oxygen is in the blood and tissues. Immediately after birth the baby breaths room air (21% oxygen), but his oxygen saturation (SaO_2) does not immediately increase. It takes approximately 10 minutes for the SaO_2 of normal babies to rise from about 70% at birth to 90%. The rapid respiratory rate of infants (30-60 breaths per minute) helps them maintain their functional residual capacity (FRC) because at these respiratory rates there is not enough time for more than their normal breath (tidal volume) to escape from the lungs during exhalation. Slowing the respiratory rate with narcotics or mechanical ventilation increases the time for exhalation and often causes atelectasis and hypoxemia, especially in premature babies.

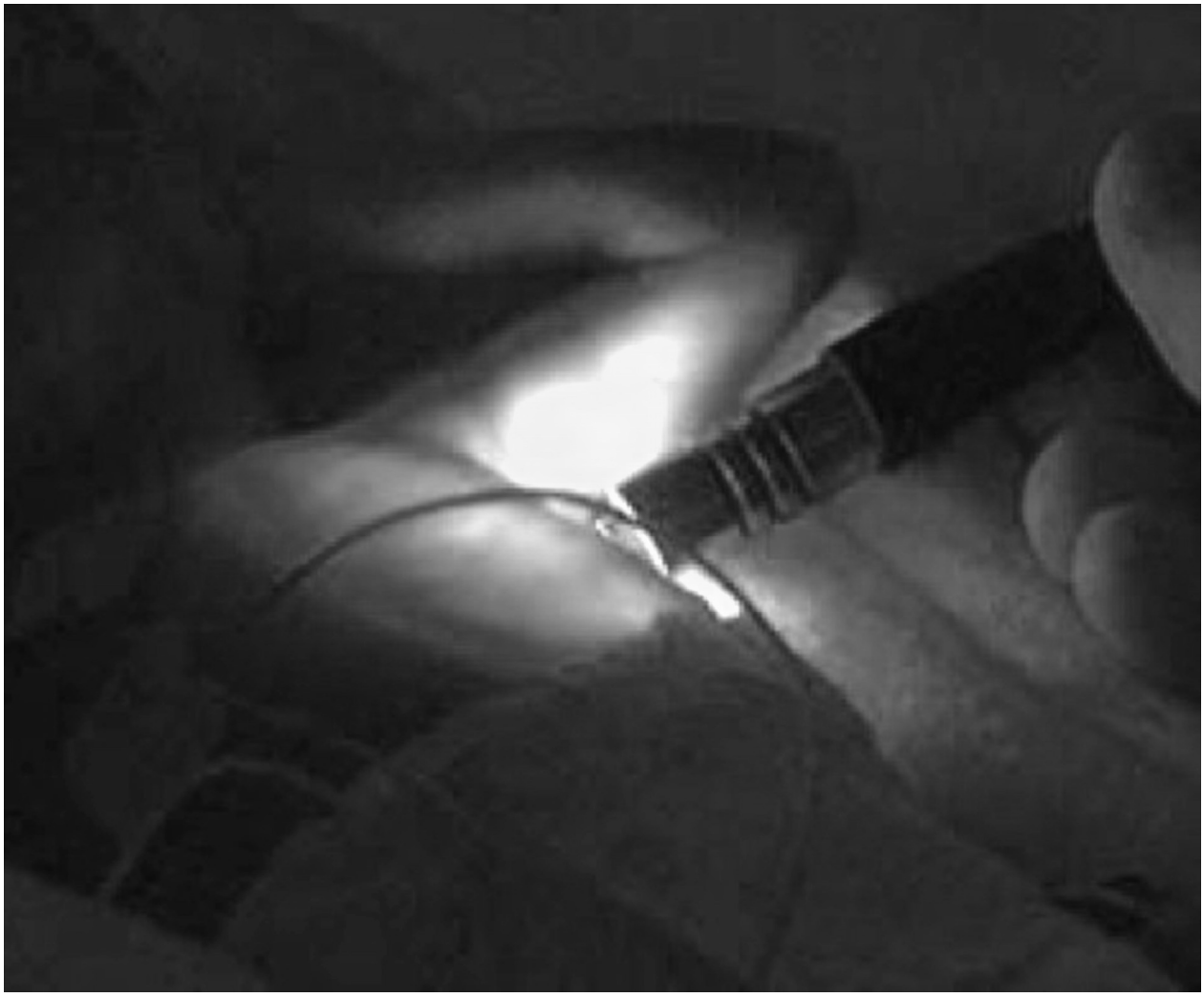
Over the first three months of life, the hemoglobin concentration decreases from about 18g/dl at birth to about 9-10g/dl, due to destruction of fetal red blood cells. New red blood cells produced contain adult hemoglobin; the hemoglobin concentration in blood rises to adult levels by about one year of age.

Visual examination of the child's chest and listening to her/his breath sounds must be part of every preoperative evaluation; these examinations provide important information about how well the lungs and the chest are functioning. A rapid respiratory rate (>60 breaths/min in babies and 20 breaths/min in young children) can be caused by lung injury or lung anomalies, hypercarbia, or hypoxemia. Intubation of the trachea frequently reduces FRC and causes atelectasis, which worsens oxygenation and acidosis, unless a positive pressure of 4-5cmH_2O is applied to the tracheal tube during expiration. Inward movement of the suprasternal notch, the spaces between the ribs, and the sternum during inspiration (retractions) are signs of increased respiratory work and are often due to bronchospasm, atelectasis, pulmonary edema or secretions, pneumonia, or congenital anomalies of the lungs. Grunting respiration (breathing out through partially closed vocal cords) is a sign of reduced FRC and is an attempt by the child to increase her/his FRC.

Listening to the chest provides useful information. Normally, the breath sounds are clear and equal bilaterally. "Dry rales" often indicate the presence of atelectasis. Wet rales, on the other hand, indicate fluid in the lungs, either within the alveoli or within the interstitium of the lung.

About one percent of babies have a pneumothorax immediately after birth, but most of these pneumotharacies are clinically insignificant. If the pneumothorax is clinically significant (i.e., difficulty breathing, decreased oxygen saturation, cyanosis, hypotension), the pneumothorax can often be detected by standing at the end of the bed and observing how the chest moves during breathing. If a pneumothorax is present, the affected side of a young child's chest will be elevated and move little or not at all with inspiration. The "normal" side of the chest will move more normally with inspiration. If a *cold* light, such as a surgeon's headlight or a small bright flashlight is available, it can be used to diagnose a pneumothorax in babies and young children. (This does not work in fat children.) When the light is placed on the non-affected "normal" side of the chest in a darkened room, a small amount of light is seen around the edges of the light source. If a pneumothorax is present, the involved hemi thorax will glow brightly when the light is applied (**(Figure 1-3)**.

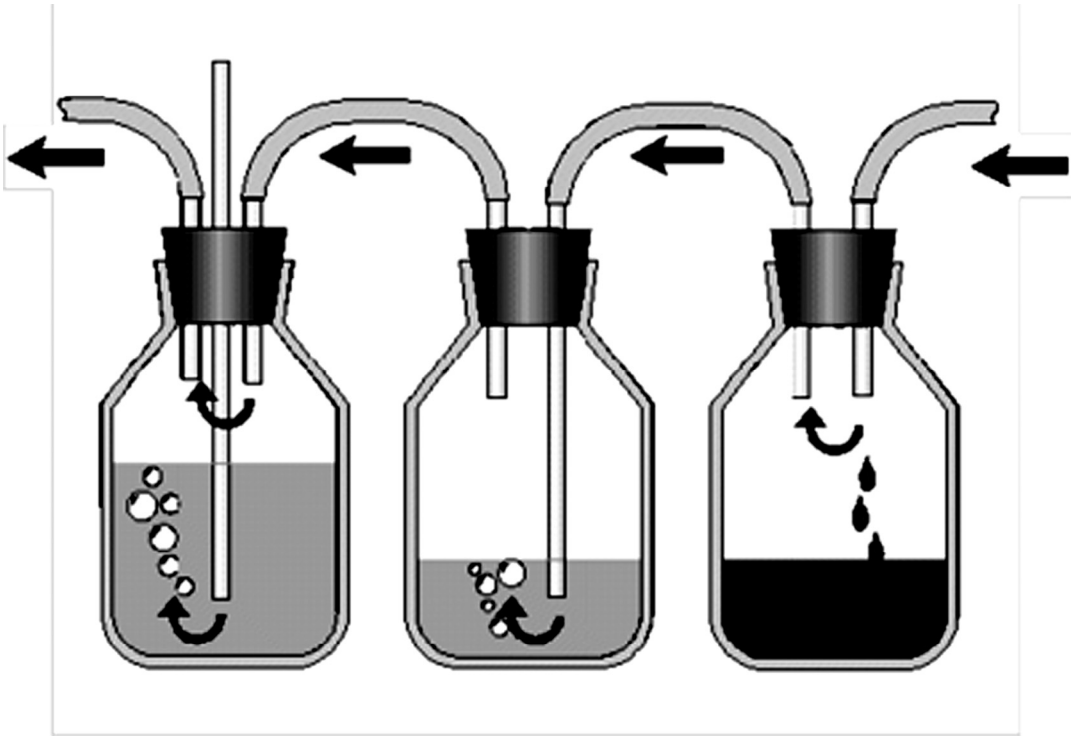
Figure 1-3: Use of a Cold Light to Diagnose a Pneumothorax in a Baby



A light applied to the right chest of the infant causes the entire chest "light up", demonstrating the presence of a pneumothorax. In the absence of a pneumothorax, only a small arc of light would show around the light source. ***With permission from http://www.NICU-pedia.com***

A pneumothorax that is causing distress must be treated immediately by inserting a needle or chest tube into the affected chest. If the air leak is continuous, a chest tube should be inserted and connected to three-bottle suction. **(Figure 1-4)**

Figure 1-4. The Three-Bottle System Used for Removing Air and/or Fluid From the Pleura.



The bottle on the right is connected to the patient's chest tube and is used to collect blood, fluid, and air. The bottle in the middle is an underwater seal that prevents air from being drawn back into the lung during spontaneous breathing. The difference in liquid height between the bottle on the left and the bottle in the middle determines the amount of suction applied to the pleura. In addition to tubes entering and exiting the bottle on the left, this bottle also has a third tube that open to air. How far this tube is located below the fluid surface determines the amount of suction applied to the lung. ***With permission from: The Trauma Professional's Blog - http://www.thetra-umapro.com***

Cardiovascular System

Both ventricular contraction and heart rate contribute to cardiac output and tissue perfusion. The heart rates of babies are normally between 120 and 160 beats per minute (bpm) and 80-to-100bpm in young children. Before six months of age, increases in cardiac output are primarily due to increases in heart rate, not to increases in ventricular contraction. This is due to the fact that the ventricles of young infants are "stiff" because their myocytes have fewer contractile elements. This may cause difficulties for sick children who are less than six months of age because their heart rates are often "fixed" at about 150 beats/min. As their condition improves, their heart rate varies. Consequently, a heart rate of 150 beats/min is not always a sign of a healthy infant.

Knowledge of how the patient's cardiovascular system is functioning and of their intravascular volume are important for preoperative planning and for reducing the incidence of hypotension

and hypoperfusion with the induction of anesthesia. While there is no good way to measure cardiac output and tissue perfusion clinically, the presence of normal arterial blood pressures, normal peripheral pulses, and a normal capillary refill times are suggestive of adequate tissue perfusion. **Appendix 1** shows the normal arterial blood pressures and heart rates verses age.

Cardiac murmurs are usually caused by turbulent blood flow and are common in infants and young children. Some murmurs are functional (innocent) or due to anemia; they are usually clinically benign. Functional murmurs are usually the result of physiologic conditions outside the heart and are *not* caused by defects in cardiac structure. However, functional murmurs must be differentiated from those produced by abnormal myocardial structures; the latter are often associated with significant cardiovascular problems before and during surgery, especially if the anesthetics and other drugs administered during surgery reduce cardiac function further (**See Chapter 12**). The murmur associated with a ventricular septal defect (VSD) is a systolic ejection murmur that is best heard along the patient's left sternal boarder. If the pressures in the right and left ventricles are equal, no VSD murmur will be heard, even though the child has a large VSD. This is because the right and left ventricular pressures are similar, which prevents blood from flowing across the defect; this is an ominous sign if the elevation in right ventricular pressure is caused by pulmonary hypertension. During anesthesia and surgery, hyperventilation-induced alkalosis may decrease the pulmonary vascular resistance and allow significant *left-to-right* shunting of blood through the VSD. This can cause a murmur and congestive heart failure. Positioning a stethoscope over the heart throughout surgery allows the anesthetist to detect a new or worsening murmur. If this occurs, ventilation is reduced to correct the alkalosis.

Abdomen

Abnormalities of the gastrointestinal tract are common reasons for surgery in infants and young children **(See Chapter 13**). Most young children have a protuberant abdomen because their liver and other intra-abdominal organs are relatively larger than those of older patients. If a baby's abdomen is flat or sunken, there is usually a less than normal amount of bowel within the abdominal cavity, due to a diaphragmatic hernia, bowel atresia, gastroschesis, or omphalocoele. Enlarged peri-umbilical veins suggest abnormalities or infections of the liver. Increased intra-abdominal pressure from intra-abdominal fluid, gas, or infection often turns the normally pink, well-perfused neonate's abdomen blue or grey. The abdomen of patients born with an imperforate anus is often distended. Therefor, patency of the anus should be determined at birth.

Infants and young children normally have a soft, non-tender abdomen on palpation. If palpation causes pain, its source should be determined. If the pain worsens on release of the abdominal pressure (rebound tenderness), the patient may have peritonitis. Localization of the pain to a certain area of the abdomen suggests an abscess. If the examiner's fingerprints are visible on the abdominal wall after he/she applies modest pressure, this too is indicative of peritonitis.

"Fingerprinting" only occurs if the baby's abdominal wall is edematous, even though the edema is otherwise difficult to detect. The young child's liver edge is normally 1-2cm below the right costal margin. Cirrhosis, hepatitis, amyloidosis, hemochromatosis, glycogen storage disease, liver cysts, cancerous and non-cancerous tumors, and an inadequate number of bile ducts (biliary atresia) can enlarge the liver. Right heart failure is also a common reason for liver enlargement in infants and small children. Enlargement of the spleen is usually not due to primary splenic disease but is a sign of hepatic failure, cirrhosis of the liver, beta thalassemia, biliary atresia, or cystic fibrosis, among others. Applying undue pressure to an enlarged spleen may rupture it.

The kidneys of young children can be palpated by placing one hand behind the upper abdomen and the other on the anterior abdomen. By pressing up with the posterior hand and down with the anterior hand, and gently moving the fingers of the upper hand, it is possible to feel the kidney as a globular mass. Urethral obstruction or a kidney tumor can enlarge the kidney. The presence of renal abnormalities and renal injury must be detected before surgery because perioperative mortality and poor clinical outcomes are more common when abnormal renal function is present preoperatively. The baby should make at least 0.75ml of urine per kg/hr. Well-hydrated infants and children usually make 2-4ml/kg/hr. of urine.

Auscultation of the abdomen also provides useful information. Absence of bowel sounds is associated with torsion or distention of the intestine, peritonitis, trauma, bowel obstruction, and hypokalemia. All of these conditions cause ileus, which is a condition in which the bowel smooth muscle fails to normally contract and move food and liquid forward. When ileus is present, the infant is more likely to vomit, regurgitate, and aspirate gastric contents during the induction of anesthesia. Measures should be taken to prevent aspiration (oral gastric tube, cricoid pressure). Children who are sleeping and those who have had abdominal surgery, anesthesia (general or spinal), opiate administration, anticholinergic drugs, and phenothiazines often have *hypoactive* bowel sounds. *Hyperactive* bowel sounds, on the other hand, occur with diarrhea, gastrointestinal bleeding, infectious enteritis, and bowel obstructions. If "rushes" (bowel sounds that increase in intensity over a few seconds) are heard, one should suspect the presence of a bowel obstruction.

Extremities

Trauma or congenital anomalies are common reasons for abnormalities of the extremities of infants and young children. If abnormalities are found, the reason(s) for them should be sought. If trauma is the cause, the anesthetist should search for other injuries, especially for intra-abdominal injuries (liver or spleen) because hemorrhage, severe anemia, and reduced intravascular volume often accompany these injuries. Young children seldom have extremity edema unless their serum protein concentrations are low. Lack of visible veins in the extremities is common, especially in young children who are chronically ill or are dehydrated and intravascular volume depleted. Finding a vein large enough to permit rapid transfusion of blood or large volumes of fluid can be a problem. The

saphenous or external jugular veins are usually adequate for this purpose. Placing the child in a slight head down (Trendelenberg) position often distends the external jugular vein, making insertion of an I.V. catheter easier. If intra-abdominal bleeding is suspected, the I.V. should be placed in an upper extremity or the neck when possible because administering blood and fluid through a lower extremity I.V. often causes the fluids to be lost into the abdomen and never reach the central circulation.

The skin of young children is looser and less well fixed to the subcutaneous tissue than it is in older patients. To make inserting an I.V. easier, the skin should be pulled moderately taught to prevent it and the vein from moving while the I.V. is being inserted. If skin tension is not maintained, the vein will often be pushed away from the needle tip, and the needle will not enter the vein. If the tension on the skin is released before the needle or catheter is securely positioned in the vein, the skin and vein may retract off the end of the catheter or needle. Care must be taken to prevent air from entering the intravenous line or I.V. connector because it only takes 0.1-0.2cc of air to cause a cardiac arrest and death if the bubble of air passes right-to-left through a foramen ovale and lodges in a coronary artery. Once the I.V. is connected to the fluid source and is taped securely in place, the arm or leg should be secured on a board to reduce the possibility of accidentally dislodging the I.V. In some instances, it may be necessary for a surgeon to insert a central line to measure central venous pressure, for blood and fluid administration, and to permit blood withdrawal for laboratory tests during and after surgery (**See Chapter 2**).

As part of the preoperative evaluation, the patient's skin should be carefully examined because skin infections are common and reduce the number of available sites for I.V. insertion. Skin infections may also make it unwise to use a spinal, caudal, or an epidural anesthetic because inserting a needle or catheter through an infected area may lead to meningitis or a spinal abscesses. The skin of normal patients is usually pink and warm. If it is grey, blue or cold, the cause(s) should be sought before anesthesia is induced.

Laboratory

All necessary laboratory results must be reviewed and understood before anesthesia is induced. **Appendix 2** lists normal values for hemoglobin, hematocrit, clotting factors, and electrolytes in young children. While these are the generally accepted values for young patients, anesthetists must know what the normal values for her/his population of patients and for their hospital's laboratory. Knowledge of the patient's preoperative hemoglobin concentration is very important, especially for infants and young children, because they consume at least two times as much oxygen per minute as adults. (Anesthesia reduces the infant's and young child's oxygen consumption by about 50% and that of adults by about 15%.) Since approximately 98% of oxygen in blood is in red blood cells (RBCs), the hemoglobin concentration of blood must be high enough to provide sufficient oxygen to meet the patient's tissue oxygen requirements. Understanding

two important concepts (*oxygen content* and *oxygen delivery*) are necessary when trying to decide if the hemoglobin concentration is adequate. **Oxygen content = (1.39 X [Hb] X SaO_2) + 0.003 X PaO_2**. SaO_2 is the saturation of oxygen on hemoglobin; 1.39 is the cc of O_2/g of hemoglobin; [Hb] is the hemoglobin concentration; and 0.003 is the cc of oxygen dissolved in plasma (i.e., <1% of oxygen in blood). Multiplying 0.003 X PaO_2 gives the cc of oxygen dissolved in plasma. Because most oxygen is in hemoglobin, it is far more effective to increase the hemoglobin concentration than to increase the inspired concentration of oxygen (FiO_2) when attempting to raise the oxygen content of blood. For example, if the hemoglobin concentration is 4g/dl, the oxygen content of hemoglobin is 5.4cc of oxygen/100 ml of blood. If the PaO_2 is 100mmHg, the amount of oxygen dissolved in plasma is 0.3 cc of oxygen/100 ml of blood. Thus, there is 162 times more oxygen in hemoglobin than the plasma/100ml of blood. If the baby is transfused to a hemoglobin concentration of 10g/dl, the oxygen content of hemoglobin will increase to 13.4cc/100 ml of blood, but the amount of oxygen dissolved in plasma will remain at 0.3cc/100 ml if the PaO_2 remains at 100mmHg. An oxygen content of 5.4g/dl is very close to the lower limit for survival and leaves little room to compensate for oxygen desaturation or blood loss during surgery. Preoperative transfusion of blood will increase the patient's margin of safety. If little or no blood loss is expected (cleft lip repair, closed reduction of a fracture), a hemoglobin concentration of 7-8g/dl is sufficient.

Oxygen delivery is the second important concept. **Oxygen delivery = the oxygen content of blood x the cardiac output.** While it is not possible to measure cardiac output clinically, it is probable that cardiac output and tissue perfusion are adequate if the arterial blood pressure and capillary refill times are normal. When the hemoglobin concentration of a 3kg infant is 4g/dl and the cardiac output is normal (300ml/kg/min), oxygen delivery is only 5.4cc/dl x 0.9l/ min or about 4.9cc of oxygen delivered per deciliter of blood delivered to the tissues each minute. However, the oxygen consumption of normal babies is about 6cc/kg/min. Survival of a patient with this hemoglobin concentration depends on his ability to increase and maintain greater cardiac output. However, increasing the cardiac output further increases oxygen consumption, which further reduces the margin of safety. If hypoxemia occurs or the anemia worsens during surgery, the patient may suffer a cardiac arrest. If at all possible, the hemoglobin concentration of such a patient should be raised by blood transfusion before the induction of anesthesia. At times this may not be possible, but hopefully these times would be rare.

Appendix 3 gives the normal serum electrolyte concentrations for infants and children. Abnormal electrolyte concentrations are common in children who have had diarrhea, vomiting, losses of fluids and electrolytes into their tissues (bowel, peritoneum, pleura [third space losses]), or inadequate fluid intake. During the first week of extra uterine life, renal tubular acidosis (RTA) occurs; this is caused by loss of bicarbonate from the infant's immature kidneys. Potassium is also lost. RTA usually self corrects when intake of food and protein are adequate. World wide, vomiting and diarrhea are the most common causes of fluid and electrolyte abnormalities in

young children. Interestingly, rehydration of dehydrated patients may worsen their serum electrolyte abnormalities by unmasking the presence of low total body electrolyte stores. Commonly used diuretics also increase bicarbonate and potassium loss from the kidneys and increase serum chloride concentrations. Severe electrolyte abnormalities, (e.g., potassium and calcium) may give rise to dysrhythmias and myocardial dysfunction, especially during anesthesia. Therefore, electrolyte abnormalities should be corrected prior to the induction of anesthesia when possible.

Since glucose is the brain's main energy source, it is important to assure that the blood glucose concentration is normal for age before, during, and after surgery (**Appendix 3**). Under normal conditions, blood glucose concentrations are maintained by glucose intake (oral or I.V.), glycogenolysis, and gluconeogenesis. Glycogen is stored mostly in the liver and muscle and is deposited in these tissues, primarily during the last trimester of pregnancy. Infants born before the last trimester of pregnancy or those who had poor intrauterine nutrition or were stressed before or during the birth process may have low glycogen stores and difficulty maintaining normal serum glucose concentrations. If the glycogen stores are adequate, the stress of surgery may *increase* serum glucose concentrations, which may also be a problem. If a cardiac arrest occurs when a patient's blood glucose concentration is greater than 200mg/dl, it is less likely he/she will be resuscitated from the arrest and more likely that he/she will have central nervous system injury if resuscitated.

If the patient had X-rays done before surgery, they should be evaluated for the presence of lung abnormalities, infections, or a pneumothorax or pneumomediastinum before inducing anesthesia.

Transport of Patients

Transport of patients to and from the operating room can be dangerous. To reduce the danger, enough people must be available to push the patient's bed, monitor the patient's condition, administer drugs and fluids, and ventilate the patient's lungs if necessary. The minimum monitoring during transport should include SaO_2 and continuous auscultation of the breath and heart sounds with a stethoscope that is taped to the chest. Measuring arterial blood pressure during transport is also helpful. Covering the child with plastic bag and warm blankets helps maintain a normal body temperature during transport. If the patient required mechanical ventilation before surgery, her/his lungs should be ventilated during transport, usually with a bag connected to an endotracheal tube. The respiratory rate used during transport should be the same as the rate used before transport and one that is appropriate for the patient's age and lung condition. Very rapid respiratory rates can interfere with exhalation and result in gas being trapped within the lungs. This causes over expansion of the lungs, decreased lung compliance, decreased ventilation, hypoxemia, and on occasion, a pneumothorax. A respiratory rate that is too slow can result in atelectasis and hypoxemia. If before surgery the inspired oxygen

concentration (FiO_2) exceeded that of room air (21%), the same oxygen concentration should be used during transport to and from the operating room (if possible) to prevent hypoxemia or hyperoxia. The chest should expand normally with each inspiration. Excessively large tidal volumes will over expand the lungs and may cause a pneumothorax or pneumomediastinum.

Sick patients should go directly to the operating room from the ward, ICU, or emergency room and not be left outside the operating room while the room is being prepared for surgery. Nurses and doctors should be waiting for the patient; a sick patient should never wait for them. All equipment, drugs and a functioning suction should be readily available before the patient arrives in the operating room. If possible the operating room should be warmed before the patient enters the room to reduce the likelihood of her/him becoming cold. Hypothermia increases the pulmonary vascular resistance of neonates, which causes hypoxemia as poorly oxygenated venous blood is shunted right-to-left through a patent foramen ovale. Anesthesia induced under these circumstances is a formula for disaster.

Summary

Preoperative evaluation is very important because it makes existing problems known and allows the anesthetist and surgeon to correct the problems pre-operatively, which reduce the risks of anesthesia and surgery. It is important to have an adequate number of people who understand the patient's problems available; this helps prevent complications of anesthesia and surgery. Transport of sick patients can be dangerous for patients and requires the same level care provided pre- and intra-operatively.

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Chapter 2

VASCULAR ACCESS AND MONITORING

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Introduction

Obtaining vascular access and monitoring vital signs are basic skills utilized by anesthetists each day. In one form or the other, every case requires monitoring. Vascular access is also essential for all cases, except those requiring very brief periods of anesthesia. However, if the anesthetist does not plan on inserting an IV before inducing anesthesia, he/she must first determine where an IV can be quickly inserted if an emergency arises. The first part of this chapter presents techniques and sites for obtaining peripheral venous access and goes on to discuss obtaining arterial and central venous access. The second part discusses monitoring techniques and interpretation of data obtained and describes the physiology producing changes in the data.

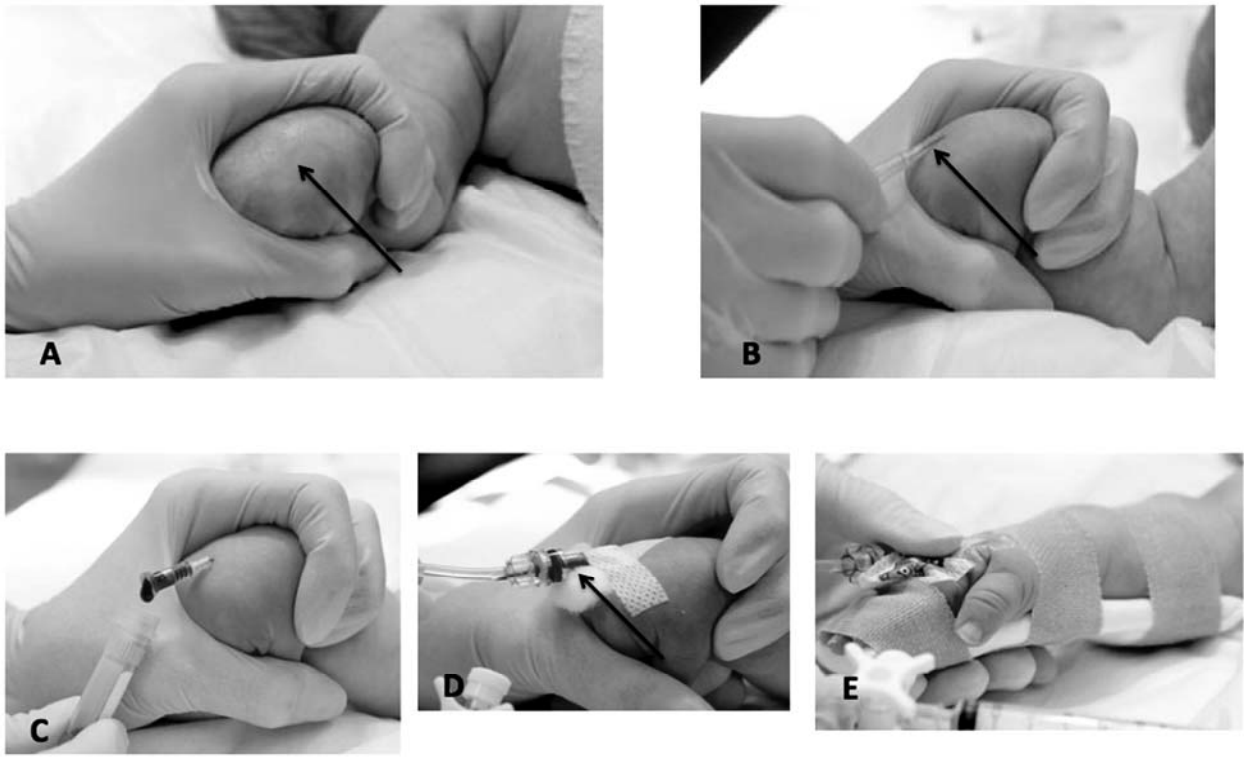
Vascular Access

Peripheral Venous Access

Peripheral venous access is an essential part of almost all anesthetics. However, access is often very difficult to obtain in some children, especially those who are small or obese, in shock, or have had multiple previous peripheral intravenous (IV) catheters. All visible veins can be used for peripheral IV access, but in infants and children, veins on the dorsum of the hand, wrist, antecubital fossa, and the saphenous vein at the ankle are particularly useful. In small infants, another useful vein is the vein that is frequently visible on the lateral side of the foot and runs parallel to the 5^{th} metatarsal bone. Sometimes it is necessary to place a small IV in a small visible vein at the wrist or side of the foot and then place a larger more secure IV after induction of anesthesia, which is possible because anesthesia dilates veins. In healthy patients undergoing simple surgery, a small IV (24g or 22g IV in infants; a 22g IV in larger children) is acceptable. If the anesthetist anticipates major blood loss or the need to administer large amounts of fluid during surgery, a larger IV is required; at least 22g for infants and young children up to age 3 years, 20g for 3-10 years, and 18g for children >10 years of age.

Technique: **Figure 2-1** shows placement of an elastic tourniquet on an extremity proximal to the puncture site. The tourniquet should be sufficiently tight to impede venous return from the limb but not so tight as to occlude arterial blood flow into the limb. This allows the veins to enlarge and makes it easier to start an IV. The skin is cleaned with 70% isopropyl alcohol to sterilize the puncture site. The alcohol is allowed to dry. Then an IV catheter or needle is inserted either slightly distal to the vein or directly on top of it at a 10°-to-30° angle. The needle or catheter is advanced parallel to the vein. If an IV catheter is used, the catheter and its inner stylet are slowly advanced until blood appears in the hub of the catheter. Then, both the needle and catheter are advanced together as one unit for several millimeters to ensure the catheter is inside the vein. Next, using either one or both hands, the catheter is very carefully advanced off of the stylet into the vein while the catheter is gently twisted back and forth. Minimal resistance should be felt as the catheter is advanced to its hub. Care is taken not to advance the inner needle (stylet) itself any further into the vein. The needle is removed from the catheter. Ideally, blood should drain through the hub of the catheter once the needle is removed. The tourniquet is only removed after the anesthetist has checked for blood return. Next, a saline filled T-piece, preferably with a Luer-lock end, is attached, and the catheter is *gently* aspirated with a 3-5ml syringe to remove any air bubbles and check for blood return. Next, saline is flushed through the catheter to ensure its proper placement in the vein. This last step is very important, as it prevents using a catheter for fluid infusion that is not properly situated in the vein. If the catheter is not in the vein, saline injected through the catheter will extravasate and the tissues will swell. If there is any question of fluid extravasation or infiltration, the catheter is removed. The catheter hub is then padded with a small cut gauze square and secured with clear adhesive dressing or tape. Placing a small padded board across the joint nearest the IV, i.e., the wrist or lateral aspect of the ankle, often prevents the IV from being dislodged. *Remember, only 0.1ml of air is required to cause a cardiac arrest and death in infants if the air passes through a patent foramen ovale and lodges in a coronary artery*.

Figure 2-1: Peripheral Intravenous Access Procedure



This figure shows placement of a 24g IV in the dorsum of an infant's hand. ***A:*** *The vein is visible after placing a tourniquet and flexing the wrist (Arrow);* ***B:*** *The catheter is inserted through the skin and advanced at a shallow angle of 10°-15°. Blood return is seen in the catheter (arrow).* ***C:*** *The inner needle has been removed after threading the catheter into the vein; at this point, blood can sometime be obtained for laboratory studies if needed.* ***D:*** *The T-piece is connected. Gentle aspiration with a syringe produces blood return in the hub of the T-piece (Arrow). A small piece of cotton padding is placed under the catheter hub.* ***E:*** *The hand and forearm are gently retrained on an arm board. Source: Royal Children's Hospital, Melbourne, Australia, Clinical Practice Guidelines: Peripheral Intravenous Access. http://www.rch.org.au/clinicalguide/guideline_index/Intravenous_access_Peripheral/*

If it is difficult to obtain IV access in a child, any small visible vein on the volar surface of the wrist, lateral and top surface of the foot, or volar or antecubital areas of the arms is cannulated with a 24g catheter.

External Jugular Vein

When IV access is difficult, the external jugular vein can be used as an IV site. Choose the patient's larger external jugular vein, place a small rolled towel under her/his shoulders, and place her/him in 30° Trendelenberg position (head down), prepare the site antiseptically, and have an assistant gently compress the vein by applying pressure just above the clavicle. This will distend the vein. Rotate the head 45-90° away from the side being cannulated and slightly extend the neck. Apply traction to the skin over the vein with one hand to tether it into a straighter course and facilitate successful cannulation of the vein. Puncture the vein high in its visible course in the neck using an angiocatheter attached to a heparinized saline filled syringe. With the catheter needle bent upwards 10°-20°, it is inserted into the vein, usually without puncturing the vein's back wall. With constant, gentle aspiration of the syringe, the vein is entered, blood obtained, and catheter advanced into the vein. Short catheters of the sizes recommended above should be used. To ensure that the catheter is properly positioned in the vein, blood is aspirated from the catheter and fluid is infused with a 5-10 ml syringe. Both should be easy. External jugular catheters infiltrate easily and must be checked frequently to determine they are still in the vein. It is more difficult to secure these catheters, due to mobile neck skin. Consequently, neck catheters should be sutured in place. One advantage of using an external jugular vein is its easy accessibility during surgery, which allows the anesthetist to monitor for fluid extravasation or kinking of the catheter.

Intraosseous Access

During a crisis intraosseous (IO) access can be used to administer fluid, blood, and drugs if no other venous access site is available. IO access is a last resort method for obtaining vascular access and is only used until secure peripheral or central IV access can be obtained. IO access can be used in anyone but is normally only used in small children. To obtain IO access, an IO needle is inserted into the flat surface of the proximal tibia (tibial plateau). Commercially available 14 or 16 gauge IO needles, or 16 gauge bone marrow aspiration needles may be used for this purpose. Regular hollow IV needles should not be used for this purpose because they often obstruct with a core of bone. The insertion site is aseptically prepared, the skin punctured, and the outer bony cortex of the proximal tibia (tibial plateau) contacted with the needle tip. The needle is advanced thought the outer cortex of bone into the marrow space with a twisting motion. Entrance into the marrow space is documented by a sudden loss of resistance. Because the infant's long bones have active marrow production, liquid bone marrow appears in the needle hub when the IO needle is aspirated. Rapid, free infusion of 10ml of normal saline without evidence of extravasation confirms proper placement of the needle. Emergency drugs and fluids can now be administered. Fluid and drugs given by the IO route reach the central circulation via bone marrow sinusoids, emissary veins of the bony cortex, and larger veins draining into the central circulation. IO needles should be replaced with conventional peripheral or central venous access as soon as

possible because infections are common problems with IO needles. **Figure 2-2** depicts insertion of an IO needle.

Figure 2-2: Intraosseous Access

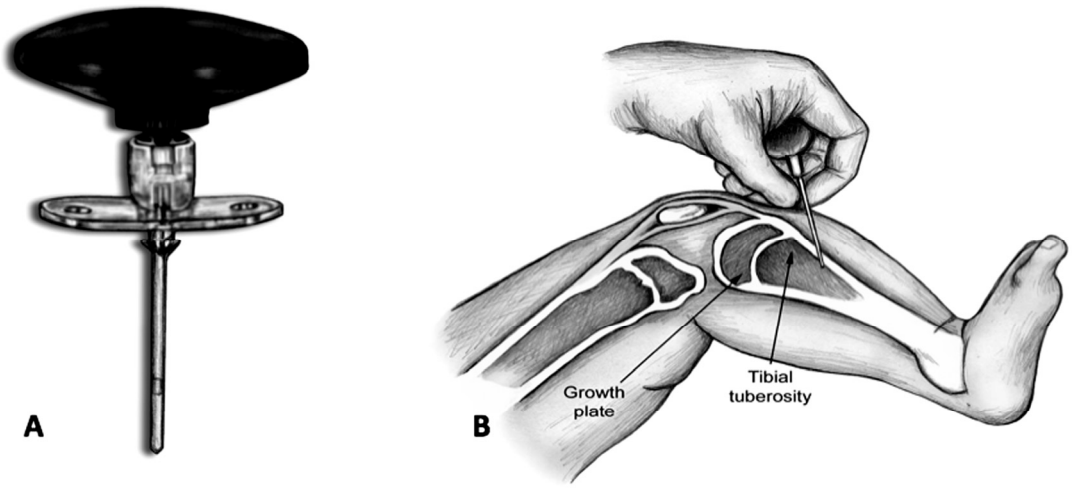


*Figure **A** on the left shows an intraosseous needle. It is either a 16 or 18 gauge hollow needle with a blunt tip, a stylet, and a knob or handle for a firm grip. **B:** Insertion technique. The flat area of the proximal tibia (tibial tuberosity) is the best site for inserting IO needles in infants. The needle is inserted with a pushing/twisting motion, taking care to avoid hitting and injuring the bony growth plate, until a sudden loss of resistance occurs. After the stylet is removed, bone marrow is aspirated to confirm correct placement of the needle. Then, 5-10ml of saline is rapidly injected through the needle to further confirm correct needle placement. Source: Medscape. Pediatric Intraosseous Access. Author: P. Eslami. http://emedicine.medscape.com/article/940993-overview - a30*

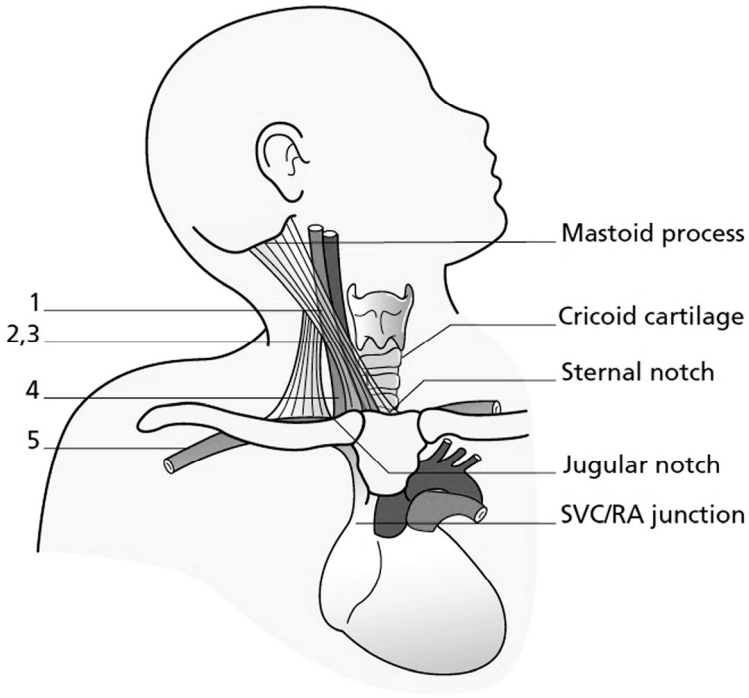
Central Venous Access

There are several indications for placing central venous catheters in infants and children: 1) for major surgeries where large blood loss or fluid shifts are expected. In this instance the catheter is used to: 1) monitor central venous pressure (CVP—see below) and to administer large amounts of fluid and blood when needed; 2) To provide peripheral IV access when it cannot be obtained or when prolonged IV access is needed, i.e., for a course of IV antibiotics lasting more than 7 days; 3) To administer inotropic and other vasoactive drugs for cardiac surgery or when major hemodynamic instability is expected. Because infection is a major complication of CVP catheter use, strict aseptic technique must be used, including: 1) Thorough skin preparation with chlorhexidine or an iodine-containing solution; 2) Thorough hand washing and the use of a surgical cap, mask, and sterile gown and gloves when inserting the catheter; 3) Draping the field with a large sterile drape and taking great care not to contaminate needles, catheters, or guide-wires; 5) Covering the insertion site with a clear sterile adhesive dressing; 6) Cleaning and redressing the entry insertion site every 72 hours; 7) Inspecting the insertion site carefully for signs of redness or discharge that suggest the presence of infection. Remove the catheter as soon as possible if signs of infection appear or when the catheter is no longer needed.

The right internal jugular (RIJ) vein is a reliable site for CVP placement because the incidence of malpositioning the tip of the catheter is very low. The approach strongly recommended for inserting a RIJ catheter is the high approach, which uses a puncture site midway between the mastoid process and the sternal notch (**Figure 2-3**), because it is less likely to cause a pneumothorax. The right side is used whenever possible because it is the least likely to injure the superior vena cava (SVC) or the innominate vein. The patient is placed in a 30-45° Trendelenberg position, a roll is placed under the shoulders, and the head is turned no more than 45° to the left. After the patient is sterilely prepared and draped, the anesthetist palpates the mastoid process and the sternal notch and visualizes a straight line between them. The puncture site is exactly midway along that line. After identifying the midway point, the anesthetist palpates the carotid artery impulse. The puncture site is 0.5 cm lateral to the carotid impulse, and the needle is directed along a line that points to the ipsilateral nipple. The anesthetist's left first and second fingers remain on the carotid pulse at all times to prevent inadvertently puncturing the carotid artery. A 3ml saline filled syringe is attached to the needle, and the needle is advanced slowly with continuous gentle aspiration on the syringe until dark blood appears in the syringe. If no blood return is seen while advancing the needle, remove the needle very slowly because blood return is often seen during needle withdrawal. Then, the anesthetist firmly grasps the hub of the needle with her/his left hand, and the syringe is removed without moving the needle. Blood should continue to flow through the hub. The Trendelenberg position, plus positive pressure ventilation, is maintained to avoid aspirating air through the needle and causing an air embolus. With the right hand, a J-tipped guide-wire is slowly advanced through the catheter. There should be no resistance to passage of the wire. If resistance is encountered, the wire must be withdrawn. If there is resistance to withdrawing the wire, the entire wire and needle are simultaneously removed, and another attempt is made to insert a catheter. If the wire passes easily, it is advanced while carefully observing the ECG. Development *of premature atrial contractions* [(PAC), a rhythm that has early appearance of a P wave and a narrow QRS complex] indicates that the guide-wire is in the atrium. If no PAC is seen, the wire has either not been advanced far enough or it is in the inferior vena cava. Another attempt should be made to insert the wire into the correct position. If a *premature ventricular contraction* (PVC) is the first ECG abnormality observed, the wire may in the carotid artery and has entered the left ventricle. If this occurs, the wire and catheter are removed immediately and pressure is held on the carotid artery. Once it is confirmed that the guide-wire is in the right IJ and not in the carotid artery, the carotid artery is again palpated to detect pulsations; the impulse should be medial to the wire. Then, a 1-2mm skin incision is made and a dilator that is slightly larger than the CVP catheter is inserted with a twisting motion. The dilator is removed, and a gauze pad is held against the insertion site with the heel of the left hand to limit bleeding. The correct size catheter is chosen according to the patient's weight and the insertion site (**Table 2-1**). Then, the catheter, which has been flushed with heparinized saline, is passed over the wire for the desired distance; if possible the catheter should have two lumens (**Table 3-2**). Great care is taken to ensure that the proximal end

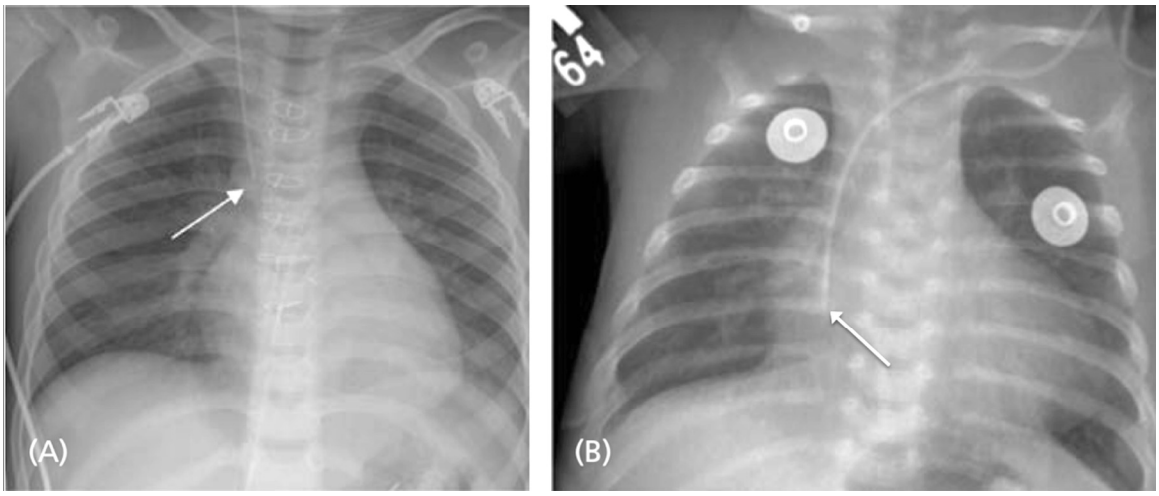
of the wire is beyond the end off the catheter. Both catheter lumens are then aspirated and flushed with heparinized saline, and capped. The catheter is sutured in place and the monitoring and IV tubing attached. Care is taken at all times to prevent air bubbles from being injected through a CVP line. A chest x-ray (CXR) is obtained as soon as possible to document correct placement of the CVP catheter and to determine presence or absence of a pneumothorax or hemothorax (**Figure 2-4**).

Figure 2-3: Sites for Central Venous Cannulation of the Superior Vena Cava.



The right internal jugular vein is in blue; the right subclavian vein is underneath the right clavicle in the thorax. ***1****: high approach, midway between mastoid process and sternal notch (best approach for safety);* ***2,3****: middle approach using apex of muscular triangle or cricoid cartilage;* ***4****: low approach using jugular notch;* ***5****: lateral approach for subclavian venipuncture.*

Figure 2-4: Chest Radiographs of Central Venous Catheter Placements



***A**: Correct placement of a right internal jugular vein catheter. The catheter tip is in the superior vena cava above the heart (Arrow). There is no pneumothorax. **B**: The catheter tip is in the right atrium (arrow), which is too deep. The risks of perforating the heart with the catheter and of arrhythmias from the catheter contacting the atrial wall is increased when the catheter tip in this position.*

The *subclavian* vein is often used for central venous access. However, there is a significant chance the catheter will not enter the SVC. The serious complication rate, especially pneumothorax, is also somewhat higher. On the positive side, subclavian vein catheters are easily secured to the chest wall and are more comfortable for the patient. The right subclavian vein is usually used because the complication rate is lower than with left subclavian vein cannulation. To insert a subclavian venin catheter, the patient is placed in the Trendelenberg position; a small towel is placed vertically under the patient's back between the shoulders. The head is turned to the right (to compress the right IJ and prevent the guide wire and catheter from passing upward), and the upper chest is steriley prepared and draped. The needle is bent upward at a 10°-15° angle and inserted about 1-2 cm lateral to the midpoint of the clavicle (**Figure 2-3**). Once the needle is under the clavicle, the needle is advanced toward the sternal notch during expiration. Stopping ventilation and deflating the lungs as much as possible reduces the chances of causing a pneumothorax while the needle is being advanced. When blood is observed in the syringe, the needle is firmly fixed in place with one hand and a guide-wire passed into the vein, all the while looking for PACs. A small skin incision is made, and a dilator is passed over the wire to make a track; this is sometimes difficult because the space between the clavicle and first rib is tight. Then, a catheter is passed over the wire into the vein. The same distance formula used for the right IJ (**Table 2-2**) is used here.

The right or left *femoral vein* can also be used for CVP access. The advantage of using this site is that these veins are easier to cannulate in small infants, and the incidence of complications from catheter placement is lower. However, because of its position in the groin, the insertion site can

easily become soiled with urine and feces. To insert femoral vein catheters, a small towel is placed under the patient's hips and the legs are placed in neutral position. Reverse Trendelenberg position (head up) of 15-20°is used to increase venous pressure in the femoral veins and makes it easier for the anesthetist to pierce the vessel. After sterile skin preparation and draping, a needle is inserted 1-2cm inferior to the inguinal ligament and 0.5-1cm medial to the femoral artery pulse. The needle is inserted at 15-30° angle and directed towards the umbilicus, all the while applying gentle, continuous negative pressure until blood appears in the syringe. A guide-wire is then passed through the needle into the vein. It is a good idea to pass the wire sufficiently far up the inferior vena cava for the wire to enter the right atrium and cause PACs. After making a 1-2mm skin incision, the anesthetist inserts a dilator over the wire. Finally, the catheter is inserted over the wire until its hub is at skin level. The catheter is aspirated to remove air, flushed with saline, and capped. It is then secured in place.

In neonates, the umbilical vein is patent for the first 3-5 days of life and can be used to insert a central venous catheter. When inserted the catheter passes through umbilical vein, the ductus venosus (which is in the liver), and into the IVC. Ideally, the tip of an umbilical venous catheter will be at the junction of the IVC and right atrium. Unfortunately, these catheters sometimes go into undesirable positions, including the portal vein. If caustic solutions (e.g., sodium bicarbonate) are injected into the liver, the drug will cause liver necrosis. Thus, before drugs and fluids are given through a UV catheter, it is important to obtain an X ray to know the catheter tip's position. If the catheter tip is in the liver, it should be withdrawn until it is in the umbilical vein or about 3-5cm from the catheter entry site before it is used like a peripheral IV.

Inserting the correct size catheter is very important for prevention of complications. **Table 2-2** provides recommendations for both the correct size and length of these catheters. **Table 2-1** has recommended depth of insertion for right IJ and right subclavian vein catheters, based on the patient's weight. If the patient's height is known, a very accurate formula is:

For patients <100 cm in height:

Depth of insertion (cm) = (height in cm ÷ 10) – 1

For patients >100 cm in height:

Depth of insertion (cm) = (height in cm ÷ 10) – 2

For example, the proper depth for insertion in an 85 cm patient is (85 ÷ 10) – 1 = 7.5 cm

Using these formulas allows correct positioning of the tip of the catheter in the mid SVC >95% of the time, which helps avoid perforating the heart and causing cardiac tamponade, two major complication of CVP catheter placement in infants.

Table 2-1: Recommended Central Venous Catheter Size and Length

Patient Weight	IJ/Subclavian Vein	Femoral Vein
< 10 kg	4 Fr, 2 lumen, 8 cm	4 Fr, 2 lumen, 12 cm
10-30 kg	4 Fr, 2 lumen, 12 cm	4 Fr, 2 lumen, 12-15 cm
30-50 kg	5 Fr, 2 lumen, 12-15 cm	5 Fr, 2 lumen, 15 cm
50-70 kg	7 Fr, 2 lumen, 15 cm	7 Fr, 2 lumen, 20 cm
>70 kg	8 Fr, 2 lumen, 16 cm	8 Fr, 2 lumen 20 cm

IJ, internal jugular vein; Fr, French size.

Table 2-2: Recommended Depth of Insertion of Right Internal Jugular or Subclavian Catheters According to Patient Weight

Patient weight (kg)	Length of CVC insertion (cm)
2–2.9	4
3–4.9	5
5–6.9	6
7–9.9	7
10–12.9	8
13–19.9	9
20–29.9	10
30–39.9	11
40–49.9	12
50–59.9	13
60–69.9	14
70–79.9	15
80 and above	16

CVC = central venous catheter

Complications of CVP catheter placement can be very serious and must be recognized and treated immediately when they occur. One complication is placing the needle in an artery rather than in a vein. This is detected by noting bright red blood from the needle and pulsatile blood return. When this occurs the needle is removed, the site is elevated, and pressure is applied over the puncture site for a full 10 minutes. A different site is chosen for subsequent CVP access. If there is question of whether the vessel is an artery or a vein and a guide-wire is in the vessel, a small short angiocatheter can be placed over the wire and the pressure can be transduced. It will

become immediately obvious if an artery has been cannulated. The situation is much more serious if a dilator and large catheter have been placed into the artery because they make a large hole in the artery, which tends to bleed. When this occurs, the dilator and catheter are removed and pressure is applied over the puncture site and vessel for at least 15 minutes. The anesthetist and surgeon should discuss what happened and decide whether to proceeded with the surgery or not. At times it is better to postpone the surgery and observe the patient for hematoma formation. Arterial puncture is best avoided during right IJ catheter placement by keeping the carotid impulse under the first and second fingers of the left hand and ensuring that the angle of the needle is lateral to the artery and directed toward the ipsilateral nipple. For subclavian CVP placement, ensuring that the needle is aimed directly at the sternal notch and not more cephalad, helps avoid arterial puncture.

A second potential major complication of IJV CVP catheter placement is pneumothorax, which develops when the needle is advanced too far toward the clavicle. This complication is more common with right-sided placement of catheters because the right pleura is slightly higher than left. Pneumothorax can also occur during subclavian CVP placement. To avoid producing a pneumothorax with subclavian placement, advance the needle only during expiration and maintain a very shallow course of needle placement, i.e., just underneath the clavicle. When a pneumothorax develops, breath sounds on the affected side will diminish. A tension pneumothorax occurs when a large amount of gas and pressure accumulate in the pleural space. Positive pressure ventilation usually worsens a tension pneumothorax. The elevated pleural pressure leads to respiratory distress and hypotension (from decreased return of venous blood to the heart). If the pneumothorax is small, treatment consists of avoiding excessive positive pressure ventilation and administering 100% oxygen to facilitate absorption of the pleural air. Larger or tension pneumothoracies are treated first by needle thoracostomy, that is, inserting a needle through the second anterior intercostal space into the pleura and removing some of the gas. A chest tube may be required for definitive treatment (**See Chapters 1 and 11**). Another possible complication is perforation of the heart by catheters, wires, or dilators, which is more likely to occur in small infants. This complication can be avoided by strictly adhering to the depth of insertion guidelines in **Table 2-2** and to the height formula above. Tachycardia, hypotension, distended neck veins, high CVP, muffled heart tones, and poor skin perfusion are signs of *cardiac tamponade*, another possible complication of CVP catheter placement. Cardiac tamponade is potentially life threatening and must be treated immediately by inserting a needle into the pericardial space by the subxyphoid approach and removing some of the blood. The patient may require pericardiotomy. Cardiac arrhythmias can also occur. Thus, the ECG must be carefully observed as the wire and catheter are advanced and immediately withdrawn a short distance when arrhythmias develop. Doing so usually terminates the arrhythmia. Catheter can also be malpositioned, i.e., not in the desired position. This is most easily detected by a CXR. If the catheter tip is in the internal jugular vein rather than the vena cava during insertion of a

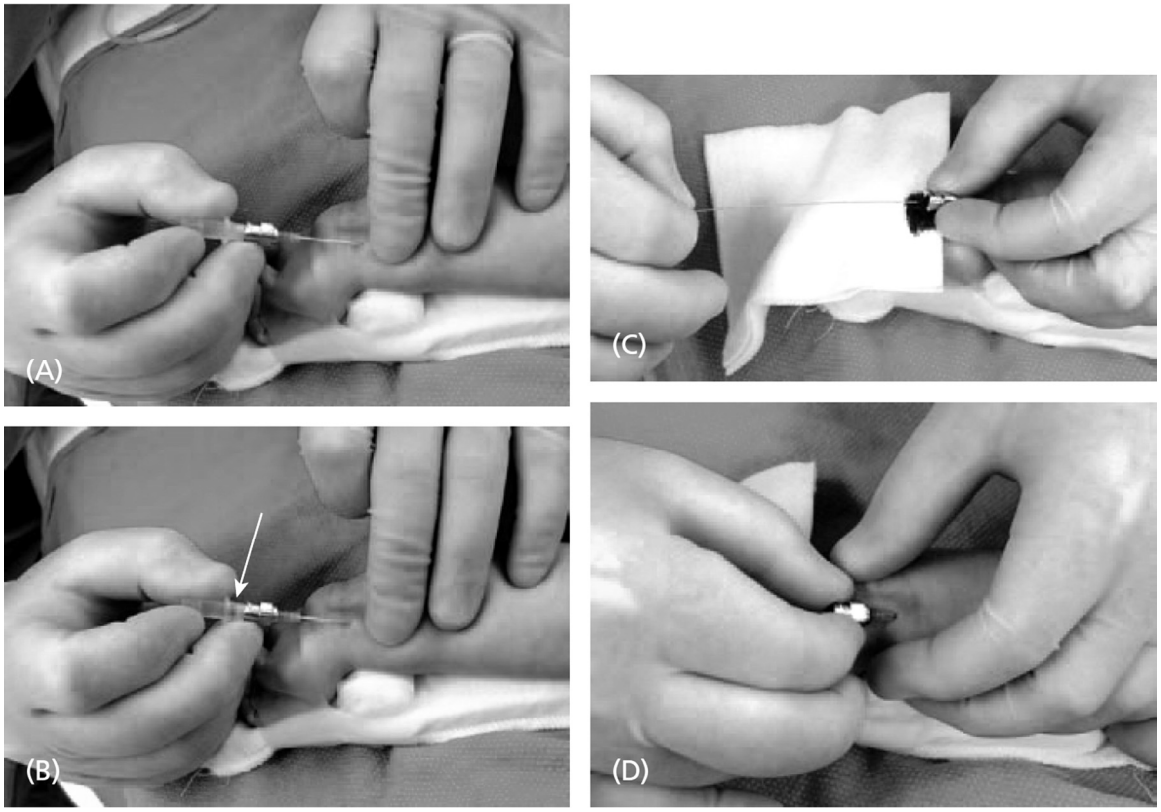
subclavian CVP, the catheter tip should be withdrawn and repositioned in the superior vena cava. Using the right IJV for catheter insertion usually prevents catheter malpositioning. Finally, infection is a constant concern with CVP catheters. Sterile technique must be used during catheter insertion (See above). Whenever drugs are given through the catheter, its hub is cleaned with alcohol and allowed to dry before anything is injected. The site should be cleaned and the sterile dressing changed at least every 72 hours. The best way to prevent CVP catheter-induced infections is to immediately remove the catheter when no longer needed.

Arterial Catheter Placement

Arterial catheters are indicated for: 1) Beat-to-beat monitoring of blood pressure during major surgery, e.g., cardiac surgery; major blood loss surgery, such as major trauma; or surgery in which there is anticipated hemodynamic instability; 2) Frequent blood sampling for arterial blood gases, hematocrit measurement, and electrolytes, glucose, and coagulation studies during surgery. Besides cardiac surgery, the most common indication for arterial monitoring is a neonate undergoing major surgery.

The best and most commonly used site for arterial catheterization is the radial artery. While ischemia is uncommon following radial artery catheter placement, it is best to place the catheter in the non-dominant hand (the one not used for writing, eating). The wrist is taped to a small arm board and enough rolled gauze placed underneath the wrist to gently extend it. The thumb is gently taped out to improve access to the artery. After the wrist is sterilely prepared, the radial artery is palpated at the lateral wrist. An angiocatheter flushed with saline is used to increase the speed of blood flow into the hub of the needle when the artery is accessed (**Figure 2-4**). At the point of maximal arterial impulse, the skin at the proximal wrist crease is punctured at a 15-20° angle to the skin. Puncture of the artery is recognized by a brisk return of blood into the liquid filled needle. The needle and catheter are then advanced together 1-2 millimeters into the artery, and the catheter is threaded over the needle into the artery until the hub of the catheter is at skin level. There should be little or no resistance to threading the catheter. Blood should easily flow into the needle hub. If the catheter cannot be threaded, the needle is carefully replaced in the angiocatheter and the needle and catheter passed together through the back wall of artery. The needle is removed and the catheter is slowly withdrawn until free flow of blood occurs. A small (0.015") guide wire, with a flexible tip, is inserted through the catheter into the artery and the catheter advanced over the wire into the artery. If unsuccessful, further attempts may be made at the same site, or at slightly more proximal sites to avoid repeated attempts at inserting a catheter into an area of arterial spasm, thrombosis, or dissection. The circulation distal to the catheter is assessed to determine if the color and capillary refill time of fingertips and nail beds, and the quality of the pulse oximeter signal are normal. The catheter is secured with a clear adhesive dressing and transparent tape so that the insertion site and catheter hub are visible at all times.

Figure 2-4: Insertion of a Radial Arterial Catheter in an Infant.



A: *The radial artery is approached with a saline-filled catheter that is inserted at the proximal wrist crease after the wrist was slightly extended on a sponge and taped to an arm board. A shallow angle of insertion is taken.* ***B:*** *Rapid flashback of arterial blood (arrow) is noted with arterial puncture using the "liquid stylet" technique. The catheter and needle are advanced together into the artery for 1-2mm. Then the catheter is gently threaded into the artery with a twisting motion.* ***C:*** *If the catheter does not thread easily, it can be withdrawn slowly until blood return is seen; then a 0.015" guide wire can be inserted into the artery through the catheter.* ***D:*** *Next the angiocatheter is threaded over the guide wire and into the artery.*

Other sites for arterial catheter placement include: 1) Dorsalis pedis (DP) artery on the top of the foot; 2) Posterior tibialis (PT) artery just below to the medial malleolus bone of the ankle. Catheters can be inserted into these arteries using the technique described above for radial artery cannulation. The foot is restrained by taping it to a footboard. Brachial and the ulnar arteries can be used for arterial access but are not recommended for this purpose because the risk for ischemic complications with them is high. Finally, the femoral artery can be used, using a similar technique to that described above for femoral vein access, except the femoral pulse is the target for needle placement. Recommended catheter sizes are listed in **Table 2-3**.

The umbilical artery can be catheterized in neonates for the first 3-5 days of life. To avoid complications, the catheter tip should either be low: at the level of the third to fourth lumbar vertebra by x-ray; or high, at the level of the 8^{th}-to-10^{th} thoracic vertebra. Problems with UACs include ischemia of the intestines and occlusion of a renal artery with subsequent hypertension.

These catheters should be removed as soon as they are no longer needed.

Complications of arterial catheter placement are mainly those of ischemia. Arterial catheters usually occlude blood flow in the artery beyond the catheter, especially in small infants. Because the intima of the arterial wall is often damaged, a thrombus can form, organize over time, and occlude the artery for months or permanently. Because infants and young children have excellent collateral circulation through the ulnar artery and through arteries in their foot, ischemia after radial, DP, or PT arterial catheter placement is rare. However, circulation of the hand and fingers and foot and toes distal to an arterial catheter must be evaluated periodically. The fingers and toes should be warm and pink and capillary refill should be rapid. If the fingers or toes are pale, cold, or have slow capillary refill, the catheter should be removed immediately, as these are signs of ischemia. Usually removing the catheter restores circulation. If not, heparin administration may be necessary. The risk of ischemia with *brachial artery or femoral artery catheterization* is high in small infants. Circulation to the arm and leg must be assessed frequently. Placing a pulse oximeter probe on a fingers or toe of the catheterized extremity allows continuous assessment of perfusion and oxygenation. Bleeding may occur when arterial catheters are removed. Applying firm pressure over the artery for 10-15 minutes usually stops the bleeding. Following the guidelines in **Table 2-3** and, in most instances, placing the smallest catheter possible in the artery prevents bleeding. Infection of arterial catheters is very rare.

Table 2-3: Recommended Arterial Catheter Sizes According to Weight and Arterial Site Used

Weight	Radial/DP/PT/ulnar arteries	Brachial Artery	Femoral Artery
<2kg	24 g	Not recommended	Not recommended
2-5 kg	22 g	24 g	22-24 g
5-30 kg	22 g	22 g	22 g
>30 kg	20 g	22 g	20 g

g = gauge; DP = dorsalis pedis; PT = posterior tibial. 24 g is equivalent to 2 French (Fr) size; 22 g is equivalent to 2.5 Fr size; 20 g is equivalent to 3 Fr size.

Monitoring

Adequate physiological monitoring of anesthetized pediatric patients is a core skill for anesthetists and is essential for the best possible patient outcomes. The level of monitoring varies greatly depending on the setting and institution, availability of equipment, and invasiveness of the surgical procedure. This section starts with a presentation of the basic monitoring methods available to all pediatric anesthetists, and then presents monitoring methods that rely more on technology and are only used for more invasive procedures. Physiologic changes that explain the information obtained from the monitor will be emphasized during the discussion.

Physical Examination

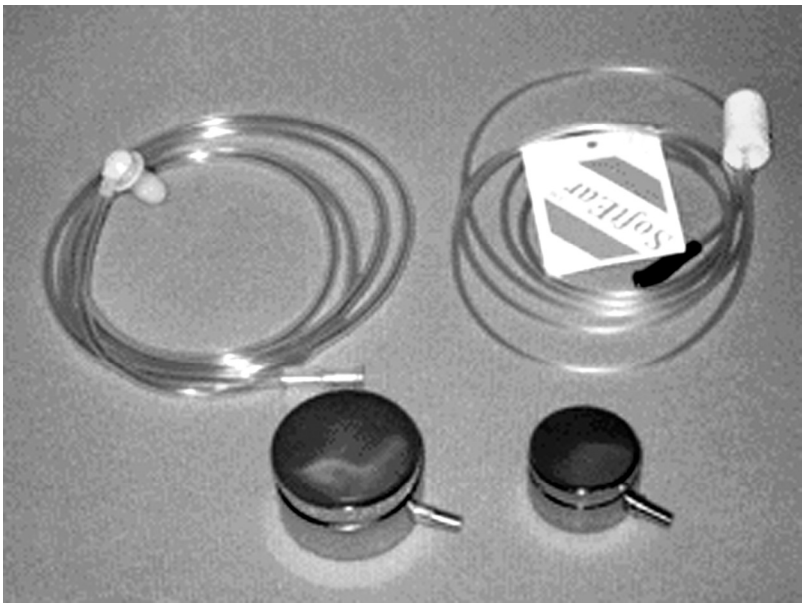
Elements of the physical examination are the most important monitors available to pediatric anesthetists during anesthesia. The examination starts with *inspection*, i.e., visual examination of the patient and the anesthetic and surgical areas. Start by evaluating areas that can be seen under the surgical drapes, the patient's head, scalp, fontanelle (if present), and face. A full or bulging fontanelle indicates increased intracranial pressure from brain edema, intracranial bleeding, or pneumothorax. A cyanotic or congested scalp indicates obstruction of the superior vena cava (SVC). Similarly, periorbital edema or plethoric appearance of the face and eyes can be signs of fluid overload or SVC obstruction. The skin is inspected for hives or erythema, as occurs during allergic reactions to drugs. Is the tracheal tube secure, and is the anesthetic circuit attached to the tracheal tube and anesthesia machine? Is the anesthesia circuit unobstructed? Is respiration spontaneous, what is the respiratory rate? Are there retractions or other signs of upper airway obstruction? If positive pressure ventilation is being used, is the chest rise adequate and appropriate for the ventilator settings? Is the color of the trunk and extremities pink and well perfused? Inspect the anesthetic machine and monitors: what values are displayed on the electronic monitor (if present)? Are they the expected values? Are the flowmeters appropriate for the desired fresh gas flows? Is there adequate volatile anesthetic liquid in the vaporizer? What is the vaporizer setting? If the child is breathing spontaneously, is the reservoir bag emptying and filling as expected? If the child is being mechanically ventilated, is the ventilator bellows filling and emptying as expected? Are the inspiratory/expiratory valves moving at appropriate times? Are IV fluids infusing as planned, and is the IV tubing free of air bubbles? Are IV, CVP, and arterial line connections secure and without bleeding? Next, attention is turned to the surgical field: what stage of the operation is occurring? Is there significant bleeding? What is the estimated blood loss based on the amount of blood in the sponges and suction bottle(s)? Is there concealed blood loss under the drapes? Is the surgeon affecting respiration, cardiac function, or venous return by her/his surgical manipulation, packing, or retractors? It is excellent practice to inspect or scan the anesthesia monitors, anesthesia machine, and IVs, the patient, and the surgical field at regular intervals during the anesthetic. Adopting this habit of scanning will detect many problems very early, sometimes before changes occur on the other monitors, e.g.

pulse oximetry or capnography.

Palpation is another important physical examination skill during surgery. Feel the patient's fontanelle (if present): is it flat (normal), depressed (volume depleted), or bulging (increased intracranial pressure)? Palpate the arterial pulses: the temporal artery is almost always accessible to the anesthetist; otherwise palpate the radial, brachial, carotid, or femoral pulses if they are accessible. What is the pulse rate? Is the pulse strong? Is the pulse pressure of a neonate very prominent and wide, as occurs with patent ductus arteriosus? Is the pulse weak and thready, as occurs with hypovolemia from bleeding or third-space fluid loss from major intestinal surgery? Is the pulse weak from cardiac dysfunction? Are the central pulses (i.e., carotid artery) strong and the radial artery pulse weak, as in low cardiac output? Palpate the extremities: are the hands, feet, and forearms warm and well perfused, and is capillary refill time less than two seconds? Or, are the distal extremities cool with delayed capillary refill, as with hypothermia, hypovolemia, or low cardiac output? Are the distal extremities hot, with bounding pulses and very fast capillary refill <1 second, as occurs in early sepsis? Palpate the precordial impulse: is the heart pounding, as it does with light anesthesia or early sepsis? Is the precordial impulse weak, as occurs with shock?

Auscultation can be performed with a standard stethoscope or with a precordial or esophageal stethoscope. A precordial stethoscope can be connected to a standard dual-earpiece stethoscope or to a special single earpiece, which allows the anesthetist to listen to heart tones and breathing and to the monitors, surgeon, etc. (**Figure 2-5**). Are the breath sounds clear and equal? Are there diminished breath sounds unilaterally, as with right mainstem bronchus intubation? Are the breath sounds absent or greatly diminished on one side, as occurs with tension pneumothorax, a large pleural effusion, or a hemothorax? Are there muffled, bronchial breath sounds on one side as occurs with pneumonia or lung consolidation? Is there wheezing, as with asthma, cardiac pulmonary edema, or an allergic reaction? Are there rales, as is found with pneumonia or pulmonary edema? Is there inspiratory or expiratory stridor, as occurs with upper airway obstruction, or airway foreign body? Are the heart tones strong, full, regular; indicating good cardiac output? Is there a systolic murmur; indicating a PDA, anemia, high cardiac output? Is there a harsh systolic murmur indicating heart disease, e.g., ventricular septal defect, or aortic or pulmonic stenosis? Is there a diastolic murmur, indicating poor cardiac function, or mitral stenosis? Are the heart tones muffled, as with low cardiac output, poor cardiac function, or the presence of a pericardial effusion? Are the heart tones irregular, as with a cardiac arrhythmia? Listen to sounds emitted from the upper airway: is there a leak around the tracheal tube? At what inspiratory pressure does this occurr? Is there stridor that is audible with the ear or stethoscope?

Figure 2-5: Precordial Stethoscopes, Tubing, and Earpieces

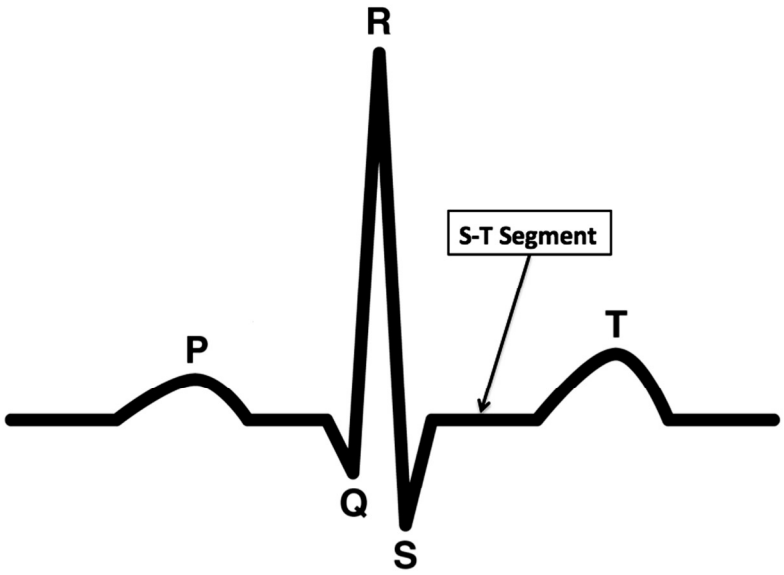


Large and small precordial stethoscopes are displayed. These can ether be taped or attached with double-sided adhesive discs or tape to the chest. The tubing and two types of earpieces are shown. The pink earpiece (left) is not moldable to the anesthetist's ear; the yellow foam earpiece (right) is moldable to the shape of the anesthetist's ear canal.

Electrocardiogram

The electrocardiogram (ECG) is an essential basic cardiac monitor during all pediatric anesthetics. The standard 3-lead configuration positions leads on the left and right shoulder or upper chest; the third lead is placed on the left thorax near the point of maximal cardiac impulse. This configuration is adequate for most anesthetics. This allows the ECG is continuously displayed on an electronic monitor screen. Lead II is the most useful lead for diagnosing arrhythmias. The 3-lead system allows leads I and III to be monitored also. It is often useful to switch between lead II and one of the other two leads if there is a question about the cardiac rhythm; leads I and III may show P or QRS waves better than lead II. The P wave signifies atrial contraction, the QRS complex signifies ventricular contraction, and the T wave signifies repolarization of the ventricular myocardium. The ST segment is the area between the QRS complex and the T wave. T waves give information about myocardial ischemia and strain (**Figure 2-6**). Normal size and configuration of the P wave, QRS complex, and T wave, and a flat, isoelectric ST segments are signs of normal, healthy myocardial function and are observed in the vast majority of pediatric patients during anesthesia. Normal sinus rhythm, (indicating a normal progression of the electrical activity of the heart), and a normal heart rate for age are also important measures of cardiac wellbeing. Normal P-R intervals, QRS width, and Q-T intervals are reassuring signs of normal cardiac physiology and function for age.

Figure 2-6: Normal ECG Waveform: Lead II

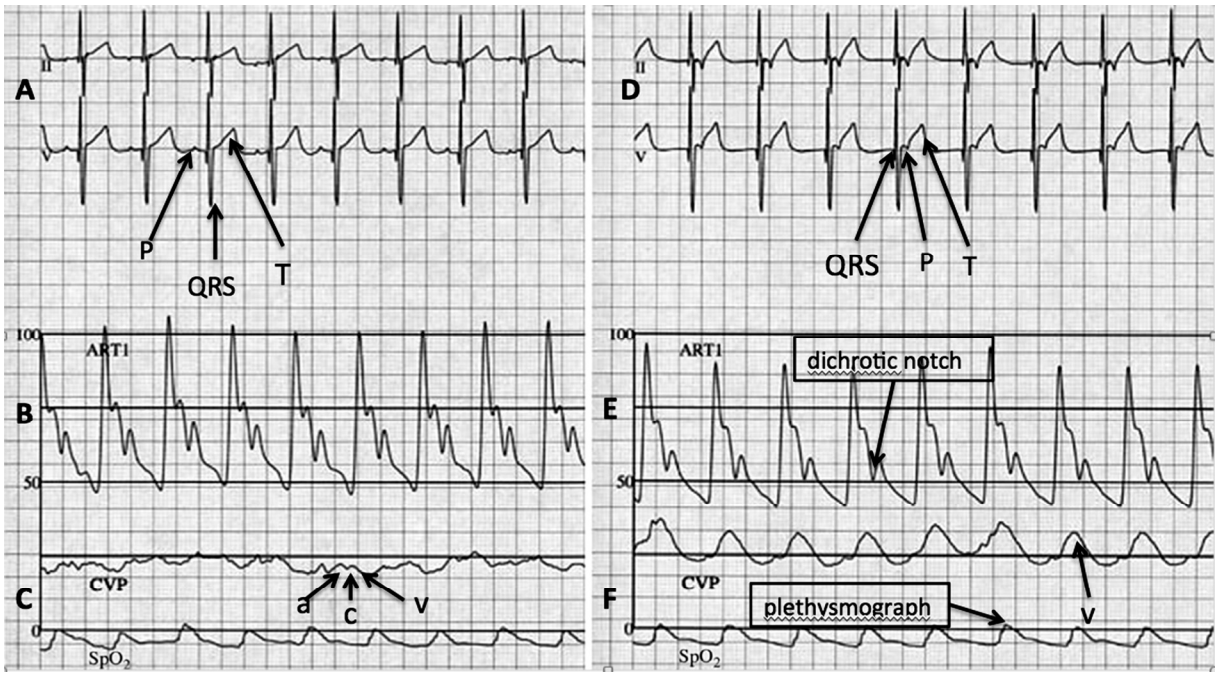


This figure shows a normal lead II ECG waveform, which is the result of electrical signal generated by the heart. The electrical activity is detected with electrodes on the surface of the body. The P wave corresponds to atrial contraction; the QRS waves together make up the QRS complex, which corresponds to ventricular contraction. The T wave corresponds to the electrical repolarization (return to normal intracellular electrical charge) of the heart. The S-T segment is a measure of normal electrical status of the ventricle; an elevated S-T segment usually indicates myocardial ischemia.

Slow heart rates for age (bradycardia) may indicate excessive anesthetic depth, the effects of high opioid doses, or the effects of vagotonic agents (e.g., neostigmine), or the use of beta-blocking agents, such as propranolol. Hypoxemia is a much more worrisome cause of a very slow ECG rates. If the SaO_2 is normal, the patient is not hypoxic, and the bradycardia is due to something else. When heart block is seen on ECG, each P wave is not immediately followed by a QRS wave. Rapid heart rates can indicate light anesthesia, which increases catecholamine release and heart rate; or it can indicate hypovolemia and is an attempt by the child to compensate for low stroke volume by increasing heart rate to maintain cardiac output. Atropine, ketamine, epinephrine, and some non-depolarizing muscle relaxants can also produce tachycardia, as can an elevated $PaCO_2$. Tachycardia is also seen with fever, which increases heart rate by increasing sinus node electrical discharge. Fast heart rates on ECG can be associated with atrial or supraventricular arrhythmias. P waves may not be visible on ECG, but QRS complexes are. A *junctional rhythm* has no P wave, only QRS complexes (**Figure 2-7**). *Atrial fibrillation or flutter* is recognized by absence of a P wave. There is instead a wavy line before the QRS, which is usually fast and irregular (**Figure 2-8**). More ominous ECG changes include *ventricular tachycardia*, which has a fast, wide complex QRS wave that is accompanied by low arterial blood pressure or cardiac arrest. Ventricular fibrillation has no recognizable P, QRS, or T waves, only a fast wavy oscillation around the baseline (**Figure 2-9**). Any abnormal ECG pattern must be investigated immediately by asking several questions: 1) What

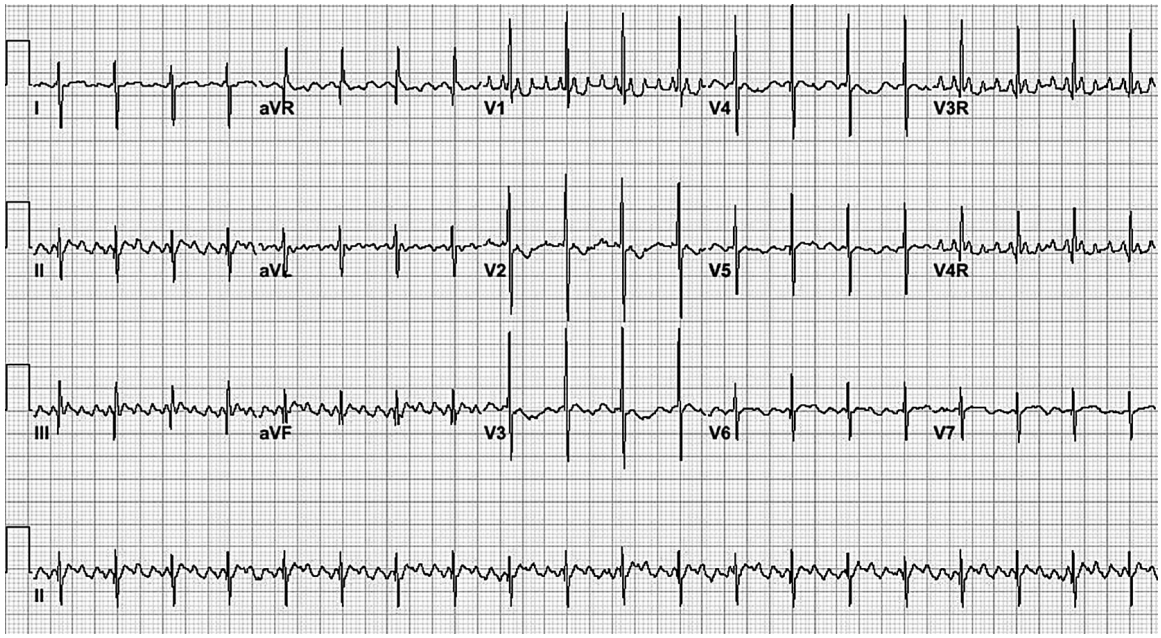
is the arterial blood pressure? 2) Are the pulses and heart tones normal or abnormal? If any one of these is low or absent, immediate action must be taken to restore normal circulation. Many times an abnormal ECG is accompanied by relatively normal blood pressure, pulses, and heart tones, in which case, there is more time to determine the cause of the abnormal ECG and decide if treatment is required or not.

Figure 2-7: Normal Sinus Rhythm (Left) and Junctional Rhythm (Right)



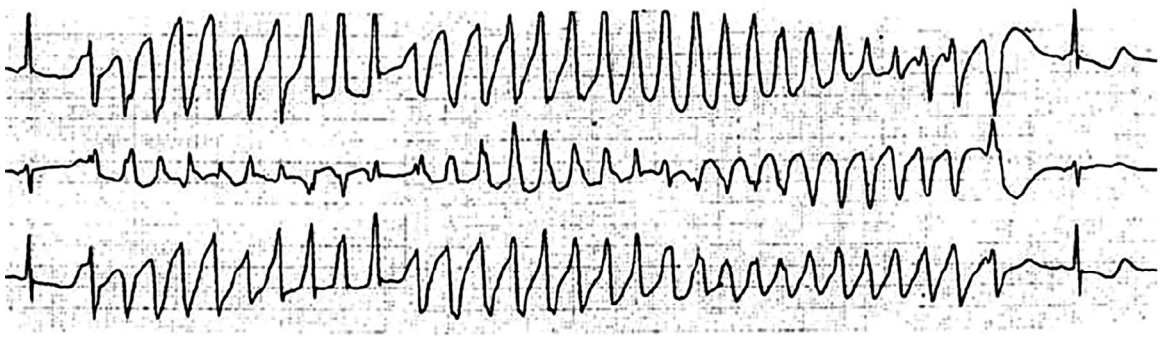
Normal sinus rhythm with ECG. ***(A);*** *arterial waveform* ***(B),*** *and CVP waveform, and pulse oximeter (SpO_2) plethysmograph* ***(C).*** ***A****: the P, QRS, and T waves are in normal sequence.* ***B:*** *normal arterial waveform. The upslope is very steep and brisk, indicating good cardiac function. The dichrotic notch is low and prominent, indicating low systemic vascular resistance.* ***C:*** *normal CVP tracing with "a", "c", and "v" waves, all of normal size.* ***D, E, and F*** *junctional rhythm.* ***D:*** *note that the QRS complex is followed by the P wave, and then the T wave. This means that the ventricle is contracting first and the atrium next.* ***E:*** *arterial waveform, note the systolic blood pressure, which was 100-105mmHg, is now 85-90mmHg.* ***F:*** *the CVP waveform shows only large "v" waves with no "a" and "c" waves.* ***F:*** *The plethysmograph on the pulse oximeter looks like the arterial waveform; in this case it is normal. A dampened plethysmograph tracing indicates poor peripheral perfusion. See text for full explanation.*

Figure 2-8: Atrial Flutter



Atrial flutter is shown on a standard ECG. Leads I, II, III, avR, avL, avF, v1-v7, and V3R and V4R are the leads shown on a full ECG that would be done as part of a preoperative evaluation. In the operating room, only leads I, II, and, III are usually available. Note the wavy ECG tracing before each QRS complex. This "sawtooth" form is characteristic of atrial flutter; the atrium beats much faster than the ventricle, and only every 3rd or 4th atrial beat is conducted to the ventricle.

Figure 2-9: Ventricular Tachycardia



This figure demonstrates ventricular tachycardia. Note the wide complex QRS pattern with no P or T waves. The changing amplitude of the QRS complex in this tracing is called "Torsade de Points." This is a very dangerous cardiac rhythm that must be immediately treated with drugs such as lidocaine, or with defibrillation if the patient has a very low arterial blood pressure.

Arterial Blood Pressure by Cuff

An important part of anesthesia care is determination and recording arterial blood pressure every 3-5 minutes in stable patients and more often in unstable patients. Very unstable patients require continuous pressure measurements (see below). Many methods are available for for determining arterial blood pressure, including listening for Korotkoff sounds and palpating the pulse, which are universally available and commonly used. Blood pressures are usually measured in the upper arm or the lower leg. For these pressures to be accurate, the width of the cuff should be 125-150% of the upper arm or lower leg diameter or 40-50% of the circumference. When the cuff is is too large, pressures will be falsely low; when it is too small, pressures will be falsely high.

To determine blood pressure by the *Korotkoff method*, a precordial or standard stethoscope is taped over the brachial artery beneath the blood pressure cuff. The cuff is inflated above systolic pressure and slowly deflated while listening for sounds made by each pulse; the first sound heard is the systolic BP. The loudest pulse sounds heard are near the mean arterial pressure, and the pressure at which the pulse tones disappear is the diastolic pressure. For ease of use during anesthesia, the anesthetist can connect a three-way stopcock to an earpiece (**Figure 2-5**). The stethoscope over the brachial artery is connected to one side of stopcock and the precordial stethoscope to the other side. This allows her/him to listen to heart tones and breath sounds continuously until he/she wants to determine the blood pressure. When this occurs, the stopcock is turned to the stethoscope, and the arterial pressure is measured.

There are three other simple methods to determine systolic blood pressure with a sphygmomanometer and cuff. With the *palpation method*, the blood pressure cuff is inflated above systolic BP and slowly deflated while feeling for a pulse distal to the cuff. The pressure at which the pulse is first felt is the systolic BP. With the *flush method*, the cuff is inflated above systolic pressure, which blanches the hand (becomes pale). The cuff pressure is slowly reduced while observing the hand. The pressure at which the hand flushes and becomes pink is the systolic pressure. With the *bounce method*, the cuff is inflated above systolic BP and slowly deflated while carefully observing the sphygmomanometer needle. The pressure at which the needle first starts “bouncing” (oscillating) is the systolic blood pressure. Many operating rooms use automated blood pressure monitoring, which is a variation of the oscillometric method. These devices have two air hoses connected to the BP cuff and the monitor, one for inflation and one for controlled cuff deflation of the cuff. At specified intervals, a small computer inflates the bladder inside the cuff above systolic BP. The pressure in the cuff is then slowly decreased. When the computer senses the first pulse in the bladder, it records it as the systolic BP. The pressure at maximum oscillations is the mean arterial BP, and the pressure when pulse oscillations disappear is the diastolic BP. The computer “remembers” the last systolic pressure and inflates the cuff to just above that pressure for the next measurement.

Hypotension (**Table 2-4**) in an anesthetized infant or child must be investigated and immediately addressed. Common causes of this problem are excessive anesthesia and hypovolemia from prolonged fasting, blood loss, or third space fluid loss. It is important to detect hypotension early because it can be a late sign of deep anesthesia. If it occurs, the depth of anesthesia must be reduced immediately. Halothane is more likely to cause hypotension than the other inhaled anesthetics. Furthermore, it frequently causes bradycardia, which further reduces the infant's cardiac output. When hypotension occurs, the inspired concentration of volatile anesthetic is immediately reduced (or discontinued) and the child is given a bolus (10-20ml/kg) of intravenous fluid. If the heart rate is slow, atropine or glycopyrrolate is given. At times it may be necessary to give a catecholamine, such as epinephrine or ephedrine, to increase persistently low systolic BPs and HRs. Other anesthetics and analgesics, such as propofol, thiopental, methohexital, and midazolam, also lower BP. Abnormal cardiac function is an additional important cause of hypotension. With congenital heart disease, hypotension is an indication of poor cardiac output. Some abnormal cardiac rhythms also reduce the BP. Sepsis with shock is another cause of hypotension. Surgical manipulation of the heart and great vessels often interferes with return of venous blood to the heart and causes hypotension. Finally, in a premature infant, a large PDA will reduce BP. When BP is low, assess the ECG, auscultate heart tones, palpate the pulses, and assess the anesthetic depth, blood loss, and surgical manipulation. Are they normal or not? If not, take appropriate actions. If the BP is very low, cardiac resuscitation with epinephrine and possibly with chest compressions may be needed (**See Chapter 5**). Turn off the anesthetic agent, determine if blood loss or something else is causing the hypotension, and treat the cause. With very low blood pressures, the oscillometric method only provides a mean BP because it cannot "read" the systolic and diastolic pressures.

Hypertension is usually due to light anesthesia and is treated initially by increasing the depth of anesthesia. However, medications, such as ketamine or topical vasoconstrictors (epinephrine or phenylephrine containing eye drops or nose drops), can also increase BP. A medication error, i.e., inadvertently giving the wrong medication (e.g., epinephrine) or dose of medication is a common cause of hypertension and tachycardia. Severe hypertension accompanied by bradycardia may indicate intracranial hypertension from intracranial hemorrhage or cerebral edema. Pressure or manipulation of the brainstem can cause either hyper or hypotension.

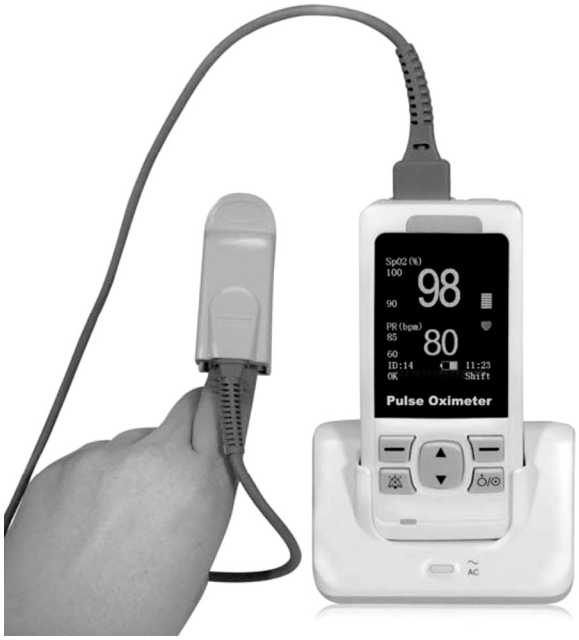
Table 2-4: Normal Heart Rate and Systolic Blood Pressure Ranges by Age During General Anesthesia

Age	Range of normal heart rates (beats per minute)	Range of normal systolic blood pressures, (mmHg)
Neonate (<30 days)	120–160	60–75
1–6 months	110–140	65–85
6–12 months	100–140	70–90
1–2 years	90–130	75–95
3–5 years	80–120	80–100
6–8 years	75–115	85–105
9–12 years	70–110	90–115
13–16 years	60–110	95–120
>16 years	60–100	100–125

Pulse Oximetry

Pulse oximetry uses the light absorption characteristics of oxygenated and deoxygenated hemoglobin to estimate the oxygen saturation (SpO_2) of arterial blood. Standard pulse oximeters use two wavelengths (660 and 930nm) that are transmitted through tissue to a detector whose algorithm only measures the pulsating arterial portion of oxyhemoglobin and filters out light absorption by non-pulsating capillaries, veins, bones and soft tissues. Since the mid 1980's, the widespread availability of pulse oximetry has changed anesthetic and critical care practice and improved patient safety more than any other monitor, which has made it a standard monitor for all pediatric anesthetics. Its availability all over the world has made anesthesia safer (**Figure 2-10**). Pulse oximeters should have a variable pitch tone that changes with SpO_2, accuracy to ± 2% at SpO2 range of 70-100%, a data-refreshing rate of <15 seconds, audible alarms, accurate pulse rate readout, and the ability to be used for both pediatric and adult patients. The oximeter pulse wave strength (plethymograph) must be indicated by either a waveform or by light bars that changes with changing pulse strength. A pulse oximeter that meets these standards is available from the LifeBox organization. To people in resource poor countries without charge (http://www.lifebox.org)

Figure 2-10: Life Box Pulse Oximeter.



The oximeter from Lifebox is lightweight, portable, and battery powered. Courtesy of Lifebox.org.

There are many manufacturers of pulse oximeters. The instructions for using each device should be followed carefully, particularly those related to proper use of disposable or reusable probes for each size patient. In infants weighing less than 3kg, it is often desirable to wrap a disposable probe around the patient's hand or foot, as this allows adequate light transmission, is more secure, and works better than trying to attach the probe to their tiny digits (**See Chapter 23**). The probe should be shielded from bright ambient light by covering it with a cloth otherwise the readings may be inaccurate (usually high). Normal arterial oxygen saturation exceeds 90%. The oximeter is accurate to ± 2%, but there is some potential loss of accuracy at SpO_2s below 90%. At SaO_2s below 70%, oximeter accuracy is greatly affected.

Cyanotic congenital heart disease (CHD) is a common problem encountered by pediatric anesthetists. Several studies have compared SpO_2 to co-oximeter determined arterial oxyhemoglobin saturations in these patients. Although some newer devices are more accurate at lower SpO_2 ranges, in general, the lower the oxygen saturation the less accurate the SpO_2. Thus, although the pulse oximeter is an excellent trend monitor for patients with cyanotic congenital heart disease, it consistently overestimates the true arterial saturation, especially when the SpO_2 is below 70-80%. When the SpO_2 is rapidly decreasing, it lags behind true SaO_2, because signal averaging occurs over 15 seconds. This means that the patient's SaO_2 is often lower that that displayed on the monitor.

Poor peripheral perfusion states are common during pediatric anesthesia, due to hypothermia, hypovolemia, cardiogenic shock, and many other causes. Since pulse oximetry relies on adequate digit perfusion to detect oxyhemoglobin saturation in pulsating tissue, significant vasoconstriction prevents the monitor from detecting minimal levels of arterial pulsation and determining SpO_2. The strongest predictor of inaccurate SpO_2 readings is a skin temperature below 30°C. Cold skin temperatures occur with hypovolemia, poor cardiac output, or simply cold core temperatures. **Figure 2-7** shows a normal plethysmographic waveform at the bottom of the figure. It looks very similar to a normal arterial BP waveform. When the waveform is dampened, perfusion and arterial pulsations under the probe are reduced, often by low cardiac output, hypovolemia, cold tissue temperature, or an arterial catheter-caused obstruction of blood flow.

Intravascular dyes that absorb light at the same wavelengths as hemoglobin have effects on SpO_2. Among the commonly used dyes, methylene blue is known to produce a significant, short lived, apparent oxygen desaturation, but this is due to interference with the signal, not true oxygen desaturation. Indocyanine green produces a less profound "desaturation" effect, and indigo carmine's effect is even less profound. Although bilirubin's light absorption spectrum has some overlap with that of that of hemoglobin, hyperbilirubinemia has little effect on pulse oximeter accuracy. Fetal hemoglobin also has little effect on SaO_2. Carbon monoxide (CO) falsely elevates SpO_2 because CO is tightly bound to hemoglobin and has a light absorption spectrum similar to that of oxyhemoglobin. Even with severe CO-induced oxyhemoglobin desaturation (CO levels >50%), SpO_2 still reads >90%. A blood gas is required to determine oxy- and carboxyhemoglobin saturations, or a special pulse oximeter is required to measure CO saturation.

When the usual sites for pulse oximetry (extremities) are unavailable, due to burns, trauma, surgery, or congenital malformations; conventional pulse oximeter probes can be placed on the earlobe, bridge of nose, buccal mucosa, tongue, and penis. More central locations (buccal mucosa, tongue, nose) experience earlier changes in desaturation and resaturation than the hand or foot. In cases where major vessels may be occluded during surgery or where intraoperative access to the extremities is limited (e.g., cardiac surgery), one oximeter is placed on both an upper and a lower extremity, in case one of them fails to function during surgery. If only one oximeter is available, the anesthetist must be able to access the site on which the pulse oximeter is located so it can be adjusted or moved to another location if need be.

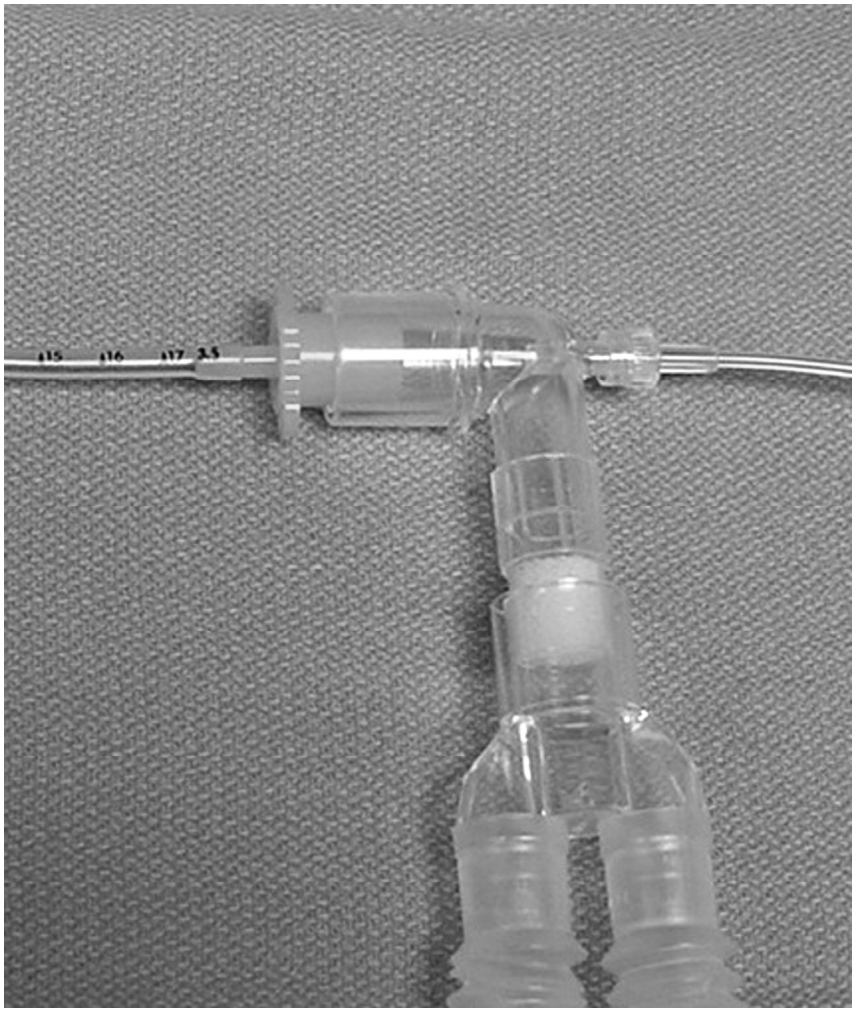
The normal SpO_2 of patients without CHD or significant lung disease exceeds 94%. However, it must be understood that a SpO_2 of 95-96% is associated with an arterial PaO_2 (oxygen tension in the blood) of 60-80mmHg when breathing room air (FiO_2 0.21), which is very marginal. If the patient is receiving supplemental oxygen and the SpO2 is only 95%, the anesthetist should assess the adequacy of ventilation and oxygenation and determine if the tracheal tube is in correct position and not obstructed. Does the patient have lung pathology, such as asthma, pneumonia, pneumothorax, or a pleural effusion? Is there loss of lung volume (functional residual capacity)

because surgical retractors are pushing the abdominal contents and the diaphragm cephalad, making it difficult for the diaphragm and lung to move during inspiration? If the SpO_2 is 99%-100%, the PaO_2 can range from 80 – to - >600mmHg. In premature infants, the SpO_2 should be kept between 90% and 94% to reduce the likelihood of causing pulmonary oxygen toxicity and retinopathy of prematurity. Neonates have high levels of fetal hemoglobin (>95%), which has greater affinity for oxygen. At a given pulse oximeter saturation, neonates with fetal hemoglobin will have lower PaO_2s than children six months of age or older. For example, at a SpO_2 of 95%, neonates have PaO_2s of about 50mmHg, whereas older children have PaO_2 of about 70mmHg.

Capnography

Monitoring end-tidal carbon dioxide (CO_2) is useful during all general anesthetics, both to confirm the initial correct placement of tracheal tubes and other airway devices and to continuously monitor adequacy of ventilation. Most capnographs utilize infrared light to quantify the amount of CO_2 in the exhaled gases. For pediatric patients, these devices work best when the exhaled CO_2 is measured as close to the tracheal tube (ETT) as possible. It is also important to minimize the space between the tracheal tube and CO_2 measurement line or device and to remove bulky airway connectors, adaptors, or Y-pieces and replace them with low volume pediatric models to reduce this “dead space” (**Figure 2-11**).

Figure 2-11: Minimal Dead Space Configuration for Capnography



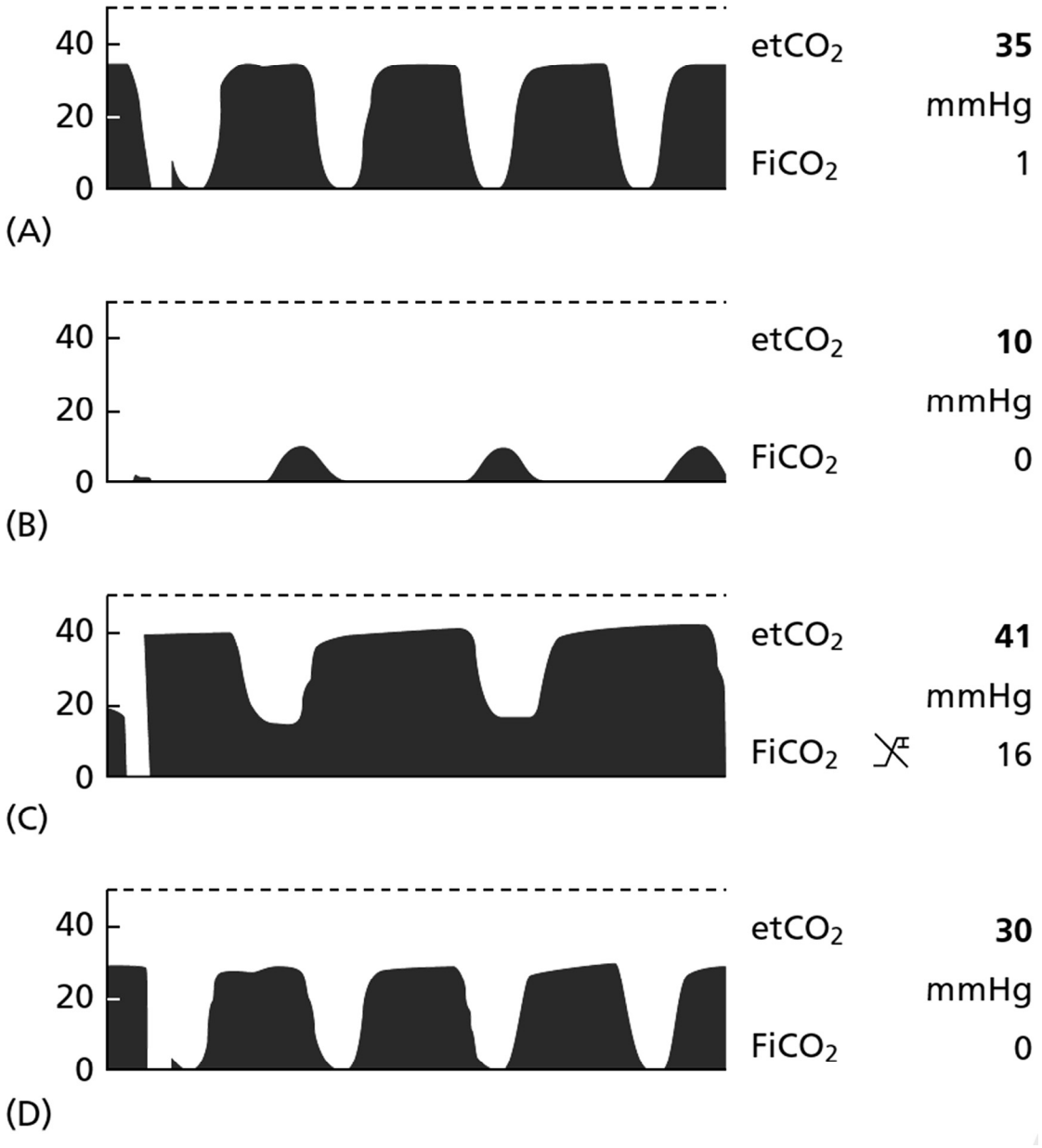
Configuration of the anesthesia circuit and tracheal tube connector to minimize dead space and make CO_2 sampling more accurate: pediatric circle system; small infant condenser-humidifier proximal to sampling line; minimum dead-space (0.5 ml) connector. The CO_2 sampling line is attached to the connector that attaches to the tracheal tube.

After placement of a tracheal tube, capnography is used to confirm correct tube placement. However this is not foolproof. A CO_2 waveform is often present with esophageal intubation or from CO_2 introduced into the stomach during mask ventilation. However, in both instances the concentration of CO_2 is low, and no CO_2 will be detected within 5-6 breaths. CO_2 can be detected when the tracheal tube is just at or just above the larynx; however, a tracheal tube situated in this position can easily be dislodged. Even when the tracheal tube is correctly placed, the amount of CO_2 detected during cardiac arrest or very low cardiac output is extremely low or absent because there is insufficient pulmonary blood flow to deliver CO_2 to the lungs. Low end-tidal CO_2s may also be seen when severe bronchospasm prevents or markedly reduces ventilation.

Normal end-tidal CO_2 tracings have a rapid upslope, a long flat plateau with minimal upslope, a rapid return to a baseline of zero, and an immediate transition to the next inspiration (**Figure 2-12A**). Other findings include separation between end expiration and the next inspiration when significant amounts of expired gas leaks from around a tracheal tube (**Figure 2-12B**). If the exhaled CO_2 does not return to baseline (zero), rebreathing of expired gases is occurring, which may be caused by a faulty expiratory valve or by increased dead-space in the breathing system, e.g., a condenser humidifier that is too large for the patient's tidal volumes (**Figure 2-12C**). A steep upslope during the expiratory phase often signifies expiratory obstruction**,** most often from bronchospasm or a blocked tracheal tube. Oscillations of $ETCO_2$ values during the plateau phase of expiration are due to cardiac stroke volume.

Besides monitoring the adequacy of ventilation, capnography provides an estimate of the patient's arterial CO_2 tension. This aids the anesthetist in preventing hypercapnea and its undesirable effects on pulmonary artery and intracranial pressures. It also helps prevent hypocapnea and its undesirable effects (decreases) on cerebral blood flow. Patients with normal hearts and lungs have a 3-5mmHg gradient (difference) between end-tidal and arterial CO_2, which, for several reasons, increases during anesthesia. Dead-space in the breathing circuit, an extension of the patient's anatomic dead space, dilutes the exhaled CO_2 and causes false "hypocapnia", especially in small patients. The dead space volume of tracheal tubes, tracheal tube connectors, condenser-humidifiers, Y pieces and elbows, and mainstream capnographs are other reasons for significant underestimation of arterial CO_2. In general, the effect is greater in smaller patients. Premature infants weighing <1.5kg are especially affected. Using small volume tracheal tube connectors, placing condenser humidifiers proximal to the CO_2 sampling line, and using special tracheal tubes that have a CO_2 sampling lumen that extends to the tip of the tracheal tube improves accuracy of capnography in small patients (**Figure 2-11**). Cyanotic congenital heart disease is another common cause of apparent "hypocapnea" in pediatric patients. With right-to-left intracardiac shunting, blood bypasses the lungs, reduces pulmonary blood flow, and decreases the amount of CO_2 in the exhaled gases. The end tidal-to-arterial CO_2 gap may be 15-20mmHg or more in patients with significant cyanosis (**Figure 2-12D**). The relationship varies with each patient, but in general the more cyanotic the patient the greater the reduction in pulmonary blood flow, and the greater the CO_2 gap. Improving pulmonary blood flow (e.g., placing a systemic-to-pulmonary artery shunt) decreases the end-tidal-to-arterial CO_2 gap. Patients with significant pulmonary hypertension, with or without an intracardiac shunt, frequently have large gaps. A decreasing end-tidal-to-arterial CO_2 gap usually signifies increased pulmonary blood flow, reduced pulmonary hypertension, and/or increased cardiac output. Finally, intrapulmonary shunting, such as that caused by lobar consolidation (pneumonia, atelectasis), produces variable increases in end-tidal-to-arterial CO_2 difference, depending on the degree of associated hypoxic pulmonary vasoconstriction.

Figure 2-12: Capnography Waveforms



Common capnography variants: ***A:*** *Normal: note rapid upslope and flat plateau of the waveform and minimal inspired CO_2;* ***B:*** *Large leak around the tracheal tube causing low measured CO_2 concentrations (the $PaCO_2$ is much higher): causes for this include a large leak around the tracheal tube. It can also be caused by partial disconnection of the sampling line and entrainment of room air into the capnography line;* ***C:*** *Rebreathing CO_2: the CO_2 does not return to the zero baseline at the end of exhalation. Causes include increased anatomic dead-space in the patient or circuit, exhausted CO_2 absorber, addition of inspired CO_2.* ***D:*** *Large $ETCO_2$-to-$PaCO_2$ gap: $PaCO_2$ was 40 mm Hg in this patient with cyanotic heart disease, but the$ETCO_2$ is only 30 mm Hg. In this patient, blood is bypassing the lungs without CO_2 being removed, which accounts for the difference between the blood CO_2 and exhaled CO_2. $FiCO_2$ = fraction of inspired CO_2; $etCO_2$ = end-tidal CO_2.*

Capnography is also useful for monitoring ventilation in non-intubated patients. Although facemasks have a significant amount of dead-space, capnography effectively monitors the adequacy of minute ventilation during spontaneous or assisted mask ventilation. Similarly, the dead-space is slightly (and at times greatly) increased, especially in babies, by the large bore tubing connecting the laryngeal mask airway (LMA) to the anesthesia circuit. Thus, monitoring $ETCO_2$ is essential when a LMA is used. Finally, a divided CO_2 sampling nasal cannula is available for monitor ventilation during spontaneous breathing during sedation and monitored anesthesia cases. This is especially useful when the anesthetist cannot be in direct proximity to the patient, i.e., during sedation cases.

As with any monitor that uses a mechanical-electrical interface, spurious capnograms and end-tidal CO_2 values may occur during equipment malfunctions or failure. A partially disconnected CO_2 sampling line, or cracked connector can entrain room air and artificially lower end-tidal CO_2 values. An occluded CO_2 sampling line (moisture or secretions) will detect little or no end-tidal CO_2. Automatic machine calibration at inopportune times, e.g., immediately after tracheal intubation, prevents CO_2 detection for about a minute. These potential malfunctions make it necessary for the anesthetist to have and use backup monitoring, e.g., a precordial or standard stethoscope. For further information on interpreting $ETCO_2$ waveforms, go to http://www.capnography.com.

Anesthetic Agent, Ventilation, and FiO_2 Monitoring

Whenever possible, the concentration of volatile anesthetic in exhaled gases should be measured. This can be done with portable monitors that also measure both end-tidal CO_2 and FiO_2. Monitoring respiratory gases, including the anesthetic agent, CO_2, and FiO_2, improves safety during general anesthesia, ensures that equipment is working, and allows the anesthetist to detect any problems with ventilation and oxygenation early. Many anesthesia machines also monitor ventilation pressures and volumes. The pressures measured are accurate, but the tidal volume not only includes the gas entering and exiting the lungs but also includes gases compressed in the anesthesia circuit (*compression volume*) and CO_2 absorber. The combination of the tidal volume plus the compressed gases increases the apparent tidal volume. Thus, tidal volume measured by anesthesia machines is not a true reflection of the patient's tidal volume. It is larger than the true volume. These measured tidal volumes are often very inaccurate with small infants. The best monitor of adequacy of tidal volume is still watching the chest rise a normal amount with each inspiration or the feel of the anesthesia reservoir bag during inspiration. A significantly obstructed upper airway results in diaphragmatic contractions that move the abdominal contents outward, while the thorax moves inward (paradoxical respiration) because of the negative intrathoracic pressure generated against the obstruction. The reservoir bag stops filling and emptying, and breath sounds are greatly diminished or cease. In anesthetized and paralyzed patients, a significant decrease or complete cessation of chest rise

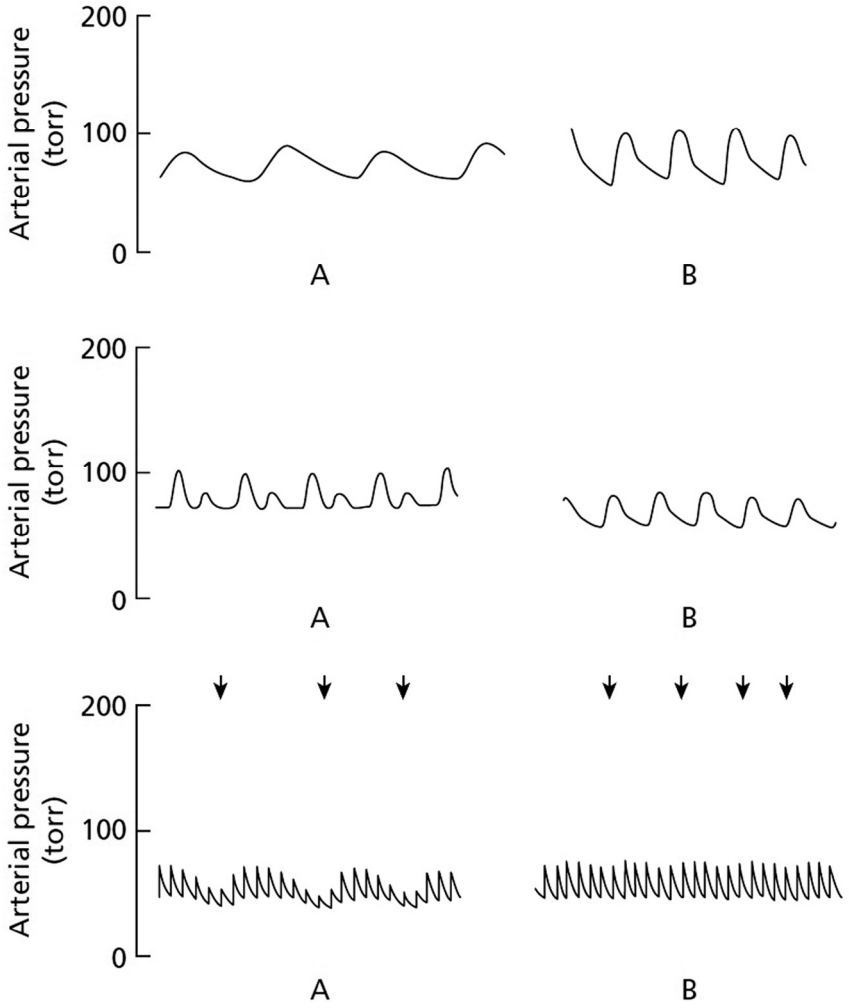
and fall can indicate dislodgment of the tracheal tube, disconnection of the ventilator, plugging of the tube with secretions, or severe bronchospasm.

Arterial Blood Pressure Waveform

The arterial blood pressure waveform provides very important information beyond systolic, diastolic, and mean arterial blood pressures. Beat-to-beat BP monitoring allows the anesthetist to detect decreasing or increasing BP early and to begin evaluation and treatment of the problem before significant difficulty arises. The arterial pressure waveform also provides information about cardiac function, systemic vascular resistance, and intravascular volume (**Figure 3-13**). Patients with normal cardiac function have rapid up strokes (initial part) on their arterial pressure waveform. Those with depressed myocardial contractility have a blunted waveform, and the angle of the upslope of the arterial pressure waveform is not steep. This change in upslope occurs because the reduced strength of ventricular contraction delays opening of the aortic valve and prolongs the time to peak (systolic) pressure (**Figure 2-13A and B**). Low systemic vascular resistance (low arterial BP, left-to-right shunting of blood through a large patent ductus arteriosus) produces a very prominent dichrotic notch, which is the "bump" on the downslope of the arterial pressure wave that corresponds with closure of the aortic valve at the end of systole. With low systemic vascular resistance, the aortic valve remains open longer, causing the dichrotic notch to occur lower on the downslope of the arterial waveform (**Figure 2-13C and D**). In hypovolemic patients, positive pressure ventilation reduces venous return to the heart. This reduces right ventricular stroke volume. Within 1 or 2 heartbeats, left ventricle stroke volume decreases and arterial BP declines. When positive pressure is released (exhalation), intrathoracic pressure rapidly diminishes, venous return increases, and right and left ventricular stroke volumes and arterial BP increase. The arterial BP varies with each ventilator breath (**Figure 2-13E and F**). If the patient's intravascular volume is normal, the variation in arterial BP is about 5mmHg during positive pressure ventilation.

Artifacts appear on the arterial BP tracing that give erroneous information that may cause the patient harm if the anesthetist does not recognize the data as spurious and acts on it. Accurate "zeroing" of the pressure transducer is very important. The stopcock on top of the transducer is placed at the level of the heart (mid-axillary line), and opened to air by turning it off to the patient and open to the transducer. The "zeroing" function of the monitor is activated. This process is repeated if the OR bed is moved up or down, or after several hours has passed (because the zero may "drift"). The arterial pressure tubing should be small bore, stiff, as short as possible, and have no air bubbles or loose connections. If an arterial BP reading unexpectedly changes, the anesthetist must quickly assess the patient and determine if the pulses, heart tones, and cuff blood pressure have changed before taking action that may be unnecessary.

Figure 2-13: Arterial Waveform and Hemodynamic Status



***Top panel**—the arterial pressure tracing with depressed (**A**), and normal (**B**) myocardial contractility. With depressed heart function, the angle of the upslope of the pressure wave is low, indicating a long systolic time, and the peak of the arterial waveform is rounded. **Middle panel**—Low (**A**) and normal (**B**) systemic vascular resistance (SVR). With lower SVR, the position of the dichrotic notch is low on the down slope of the pressure wave, indicating a long systolic ejection time (rapid runoff of blood into peripheral vessels) before the aortic valve closes. **Lower panel**—hypovolemia (**A**), and normovolemia (**B**)—arrows represent positive-pressure ventilations. With hypovolemia, positivie pressure ventilation significantly reduces venous return, resulting in variation in stroke volume, and arterial pressure. Source: Gregory GA. Monitoring During Surgery. In: Gregory GA, Pediatric Anesthesia. New York: Churchill-Livingstone, 2002:249-65.*

Central Venous Pressure Waveform

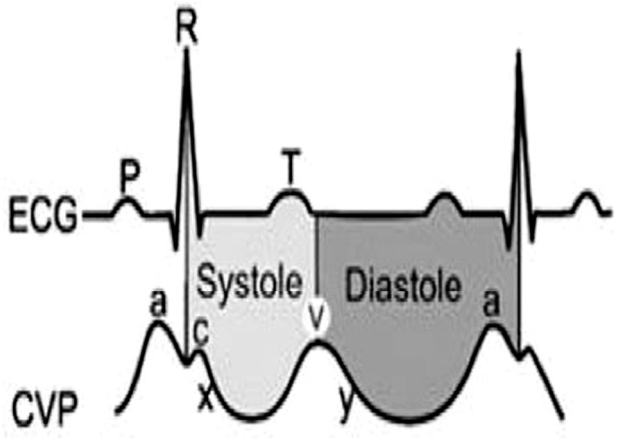
Central venous pressure (CVP) provides important information during cardiac or thoracic surgery, major trauma surgery, or other surgery in which significant blood loss or fluid shifts occur. Normal CVP in a supine, spontaneously breathing infant or child is 4-8$_{mmHg}$. CVP is proportional to and is a good estimate of right ventricular end-diastolic pressure and volume. The latter correlates with stroke volume, a major component of cardiac output. Many factors affect

whether CVP accurately reflects stroke volume, such as cardiac function, intrathoracic pressure, tricuspid valve regurgitation or stenosis, and position of the patient during surgery. But, under most conditions the relationship of CVP to stroke volume holds true. CVP should be measured in the superior vena cava (SVC) near the heart. The tip of a CVP catheter should not, however, be located in the right atrium, because serious complications may occur if it does (See above). CVP catheters whose tips are in the inferior vena cava (IVC) provides accurate CVP readings at end-expiration if the intra-abdominal pressure is not elevated or the IVC is not obstructed.

CVP waveforms consist of "a", "c", and "v" waves (**Figure 2-14**). The "a" wave corresponds to right atrial contraction during diastole (tricuspid valve open. The "c" wave follows the "a" wave and is a due to a small increase in right atrial pressure that occurs after the tricuspid valve closes. The "v" wave is a second large wave produced by right ventricular contraction. A normally shaped CVP waveform with normal "a", "c", and "v" waves means that atrial contraction, tricuspid valve closure, and ventricular contraction are occurring in normal sequence. A change in the CVP waveform is often a clue that the ECG rhythm has changed and is now abnormal. For example, if the "a" and "c" waves disappear and the "v" wave is very large (**Figure 2-7**), this usually indicates a change in ECG rhythm from the normal P-QRS-T sequence to a junctional rhythm, atrial fibrillation, or flutter. The loss of coordinated atrial contraction reduces right ventricular filling and decreases stroke volume by 20-30%, which reduces systolic blood pressure by 10-15% (**Figure 2-7**).

Changes in CVP during surgery provide a very important trend monitor for intravascular volume status. Best practice encourages getting a baseline CVP after the induction of anesthesia and before beginning surgery when possible. Other pressures are obtained following initiation of positive pressure ventilation and following patient positioning for surgery. It is important to compare changes in CVP during anesthesia to their "control" or baseline values. The CVP at end-expiration is normally 5-10mmHg during anesthesia and positive pressure ventilation. A CVP below 5mmHg often suggests hypovolemia. Observation of how fluid infusion affects CVP and arterial BP is important. CVPs >10mmHg occur with hypervolemia, right-sided cardiac dysfunction or failure, or with a pneumothorax. A CVP >15-20mmHg suggests right heart failure, pleural or pericardial effusions, or ascites. Usually, positive pressure ventilation with mean airway pressures of 15-20cmH_2O and positive end-expiratory pressures (PEEP) of <10cmH_2O have minimal effect on CVP. However, higher ventilating pressures can increase intra-thoracic pressures if some of the pressure is transmitted to the SVC and heart; the CVP will be falsely elevated. The patient's true CVP can be determined by discontinuing mechanical ventilation for a few seconds. During spontaneous respiration, a CVP below 3mmHg can decrease to -5-to--10mmHg during inspiration. This is a very dangerous situation because air can easily be entrained and produce an air embolus, hypotension, cardiac arrest, and death if a CVP stopcock is left open to air. CVP catheters should never be left open to air during insertion or any other time.

Figure 3-14: Normal Central Venous Waveform with Electrocardiogram



Central venous pressure (CVP) waveform matched with a normal ECG showing the P, R, and T waves. Systole and diastole are indicated. The "a" wave corresponds to the slight pressure increase occurring with atrial contraction. The "c" wave occurs with closure of the tricuspid valve (just before ventricular systole begins). The "v" wave occurs with ventricular contraction. Source:
http://www.uichildrens.org/uploadedImages/UIChildrens/Health_Professionals/PICU/CentralVenousPressureMonitoring.jpg

Temperature

Maintaining a patient's normal body temperature is an important task for pediatric anesthetists. Because the infant's body surface area to weight ratio is large, they are more likely lose heat by radiation, convection, evaporation, and conduction. This is especially true of small infants whose heads are large relative to the rest of their body, which further increases heat loss. Low body temperature (*hypothermia*) is associated with delayed emergence from anesthesia, shivering in older patients, and brown fat metabolism in neonates, which significantly increases oxygen consumption, apnea, slows metabolism of anesthetic drugs, delays blood clotting, and slows wound healing. Hypothermia-associated peripheral vasoconstriction increases systemic vascular resistance and cardiac work. Hypothermia and shivering are extremely uncomfortable and unpleasant for patients.

Hyperthermia is a relatively late sign of malignant hyperthermia, but is nonetheless extremely important to recognize and treat when detected. Infection, sepsis, and excessive environmental heat raise patient temperatures, increase oxygen consumption, increase metabolism of anesthetic drugs, and trigger tachycardia. Hyperthermia worsens the detrimental effects of ischemia. For example, it greatly increases neuronal loss during cerebral ischemia, and this increases the risk of long-term neurological damage. For all of these reasons, pediatric anesthetists should maintain the core temperature of children between 36.0°-37.0°C when possible.

Every pediatric patient should have her/his temperature measured during anesthesia. With short cases (e.g., myringotomy and tubes), skin or axillary temperatures are acceptable. For longer or more involved surgeries, core temperature should be measured with rectal or esophageal temperature probes. These sites provide equivalent temperatures to those of vital organs. For cardiac surgery, it is best to place a temperature probe through the nares to the level of the tragus of the ear, which puts the temperature probe just beneath the cribriform plate. This provides an accurate measure of brain temperature, an organ for which protection is essential during pediatric cardiac surgery. The bladder and tympanic membrane are other sites used to measure temperature, but they provide less accurate measures of true vital organ temperature. Forehead skin temperature can be determined with adhesive temperature strips that change color, depending on the temperature, but this is a poor way to accurately determine core temperature. If possible, skin temperature should not be used as the primary method of determining temperature.

Temperature should be monitored continuously. When found to be abnormal, measures should be taken to restore it to normal, i.e., warm the OR and apply forced air warming if available; cool the room and uncover the patient in cases of hyperthermia.

Urine Output

Inserting a catheter into the bladder and monitoring urine output is useful during blood loss surgery, fluid shifts, or hemodynamic changes. Although influenced by many factors, it is generally thought that a urine output of at least 1ml/kg/hr is indicative of adequate intravascular volume and kidney perfusion. Low or absent urine output may be the result of mechanical obstruction of the catheter, hypovolemia, antidiuretic hormone (ADH) secretion. The commonest cause, however, is hypovolemia. It is also important to remember that excessive urine output is abnormal and can be caused by hypervolemia or hyperosmolarity, hyperglycemia being the most common cause. Children and adults spill glucose in their urine when their serum glucose reaches approximately 180 mg/dl. Term infants spill glucose at about 150mg/dl, and premature infants spill it at 125mg/dl. Osmotic agents (mannitol) or diuretics (furosemide) also increase urine output. Urine color provides important clinical information. Bloody urine indicates frank bleeding or hemolysis (cardiopulmonary bypass, transfusion reaction); tea-colored urine suggests myoglobinuria (malignant hyperthermia or muscle crush). Cloudy urine occurs with oxalate crystals in the urine calcium, proteinuria and concentrated urine, or a urinary tract infection.

Blood Gases: Arterial and Venous

Arterial blood gas (ABG) monitoring is important for cardiac, thoracic, major neonatal, trauma, and for any surgery where pulmonary or cardiovascular instability or major blood loss or fluid shifts are expected. Many portable, compact, lightweight, accurate devices are now available that allow point-of-care testing in the operating room. The results of these tests are usually available in 1-2 minutes. After 3-5ml of blood is withdrawn (which is re-infused into a vein), 0.5-

1.0 ml of blood is drawn into a heparinized syringe for blood gas determinations. The heparin can be either in a dry form (pre-packaged ABG syringe), or a small amount (0.1ml) of regular heparin (100unit/ml), which is drawn into a 1 or 3ml syringe and flushed out with air to leave a residual of heparin on the syringe walls and barrel. This amount of heparin is sufficient to prevent the blood sample from clotting in the syringe.

Blood gas machines determine pH, $PaCO_2$, and PaO_2. pH is a measure of the patient's acid-base status and ranges from 7.35-7.45 normally. Keeping the pH within this narrow range optimizes body systems that depend on enzymes for functioning (cardiac function, drug metabolism, optimal cellular function, and function of essentially every organ and tissue in the body). $PaCO_2$ measures the partial pressure of carbon dioxide in arterial blood (plasma) and is primarily a measure of respiratory function. The $PaCO_2$ is normally 35-45mmHg. PaO_2, on the other hand, is the partial pressure of oxygen in blood (plasma) and is primarily a measure of the lung's ability to oxygenate pulmonary blood. In many disease states, oxygen consumption will affect PaO_2. Normal PaO_2 values while breathing room air vary with age. During the first hours and days of life, premature and full term neonates have normal PaO_2s of 50-70mmHg; by the end of the first month of life, it increases to 80-100mmHg; and after 1-2 months postnatal age, normal PaO_2 should be 90-110mmHg during room air breathing.

Acidosis is defined as a pH <7.35 and is classified as either respiratory, metabolic, or mixed respiratory/metabolic acidosis. In the body, $PaCO_2$ is converted to hydrogen ion (H+), which decreases pH. If the only cause of a low pH is CO_2, the patient has respiratory acidosis. *Respiratory acidosis* is due to respiratory insufficiency, which can be caused by a myriad of lung diseases (asthma, pneumonia, cardiac-induced pulmonary edema, pleural effusion, pulmonary hemorrhage or contusion, respiratory distress syndrome of the premature, adult respiratory distress syndrome, etc). Diseases that depress the central nervous system, such as brain trauma, intracranial hemorrhage, or drug intoxication, can depress the respiratory drive and elevate $PaCO_2$. During anesthesia and spontaneous ventilation, volatile anesthetics or opioids often depress respiration and elevate $PaCO_2$. With mechanical ventilation, inadequate tidal volumes, respiratory rates, or both, can lead to hypercarbia. Surgical retraction that interferes with ventilation is also a common cause of respiratory acidosis.

Metabolic acidosis occurs when fixed acids (lactate, pyruvate, etc.) are present. The most common cause of this form of acidosis is inadequate blood flow and oxygen delivery to organs or tissues. Reduced flow results in anaerobic metabolism, lactic acid production, and metabolic acidosis. If fixed acids are produced faster than the liver can metabolize them, metabolic acidosis develops. Thus, metabolic acidosis is an important warning sign of insufficient tissue oxygen delivery. Severe anemia, poor cardiac function, and blood or fluid loss are the major causes of lactic acidosis. Other causes include giving large volumes of IV fluids or parenteral nutrition solutions that are not pH balanced, e.g., normal saline.

A *mixed metabolic/respiratory acidosis* occurs when $PaCO_2$ is elevated and pH is lower than predicted from the respiratory acidosis alone. Most blood gas machines calculate a base deficit or base excess, which are measures of the metabolic component of acidosis. Base deficit is normally -2 to +2mmol/l and reflects the amount of bicarbonate needed per liter to correct the pH to 7.40. The base deficit is a reflection of how efficient pH balancing systems in the blood and tissues are in maintaining normal pH. Metabolic acidosis is signified by a pH <7.35, and base deficit greater (more negative) than -2mmol/L.

Respiratory alkalosis is defined as a $PaCO_2$ <35mmHg with a pH of >7.45. During spontaneous ventilation, excessive minute ventilation (anxiety, fear, light anesthesia, or the early stages of asthma) causes this condition. Respiratory alkalosis is very common during anesthesia and mechanical ventilation, due to excessive ventilation rates, tidal volumes, or both. Respiratory alkalosis constricts cerebral arterioles and reduces cerebral blood flow (**See Chapter 10**), which under most circumstances is undesirable. If the $PaCO_2$ is <25mmHg, there is significant risk of cerebral ischemia, especially when the patient is also hypotensive.

Metabolic alkalosis occurs with a pH >7.45 and normal $PaCO_2$. Metabolic alkalosis occurs commonly with diseases such as pyloric stenosis and other gastric outlet or high intestinal obstructions. In these conditions, large amounts of acid containing stomach fluids (H^+) and chloride (Cl^-) are vomited, which increases blood pH. With metabolic alkalosis, base excess is greater than +2. Other causes of metabolic alkalosis include long-term administration of diuretics (e.g., furosemide) that cause the loss of huge amounts of Cl^- ions from the kidney. Chloride is replaced with bicarbonate ($HCO3^-$) ions to maintain a neutral electrochemical environment in the plasma. This elevates pH.

Finally, some patients have both *respiratory acidosis* and a *metabolic alkalosis* at the same time, which is signified by a $PaCO_2$ >45mmHg and a base excess greater than +2. This combination often occurs as compensation for respiratory acidosis. The pH is usually normal. Respiratory alkalosis with simultaneous metabolic acidosis also occurs and is indicated by a $PaCO_2$ <35mmHg and base deficit in excess of -2mmol/L. This usually occurs as compensation for metabolic acidosis. Normal values for pH, $PaCO_2$, and PaO_2, along with conditions resulting in metabolic and respiratory acidosis and alkalosis are listed in **Table 2-5**.

Table 2-5: Blood Gas Values and Base Deficit in Normal and Pathologic States

Condition	pH	$PaCO_2$ mmHg	PaO_2 mmHg	Base deficit/excess mmol/l	Examples
Normal	7.35-7.45	35-45	50-80 (neonate); 90-110 (older infant/child)	-2 to +2	Healthy infant under anesthesia
Respiratory acidosis	<7.35	>45	Variable	-2 to +2	Asthma, surgical retraction, inadequate ventilator rate/tidal volume
Metabolic acidosis	<7.35	35-45	Variable	>-2	Anemia, hypovolemia, low cardiac output
Respiratory alkalosis	>7.45	<35	Variable	-2 to +2	Excessive tidal volume/ventilator rate under anesthesia
Metabolic alkalosis	>7.45	35-45	Variable	> +2	Long term diuretic use; pyloric stenosis
Mixed respiratory/metabolic acidosis	<7.30	>45	Variable	>-2	Inadequate tidal volume/ventilator rate with large blood loss
Mixed respiratory/ metabolic alkalosis	>7.50	<35	Variable	> +2	Excessive ventilator rate/volume with long term furosemide

Blood gases can also be determined from blood obtained from a central venous catheter, which gives the best measure of lung function, oxygen delivery, and acid base status for the whole body. Because central venous blood has not yet passed through the lungs to remove CO_2 and add oxygen, the normal $PvCO_2$ is 45-55mmHg, and the PvO_2 35-45mmHg; the pH is 7.25-7.35. The normal base deficit is still -2 to +2mmol/L. If arterial blood is unavailable, central venous blood gases can be used to evaluate the patient's respiratory and metabolic status. The examples in **Table 2-5** are still valid, but the differences in normal values for venous blood must be taken into consideration.

Some blood gas machines also measure hematocrit (hemoglobin), glucose, lactate, and electrolytes, including ionized calcium. Some also measure oxyhemoglobin saturation; if this is available, it should be compared to the SaO_2.

Conclusion

To provide the best possible care of pediatric patients, anesthetists must master monitoring and vascular access skills. Although we rely heavily on electronic monitoring for pulse oximetry, oscillometric blood pressure measurement, and arterial and central venous pressure monitoring, physical examination skills are still the most important way to monitor our patients. These skills are always immediately available for every patient, even when high technology monitoring is unavailable, or fails to work properly during use.

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Chapter 2: VASCULAR ACCESS AND MONITORING

Chapter 3
FLUIDS AND ELECTROLYTES

Claire Brett, MD

Managing perioperative fluids and electrolytes requires an in-depth understanding of a wide range of topics, including renal, hepatic, cardiorespiratory, endocrine, and central nervous system physiology. Electrolyte derangements both evolve from and lead to multi-organ dysfunction. For example, electrolyte and acid base abnormalities accompany renal, neurologic, hematologic, and/or hepatic disorders and often accompany myocardial or pulmonary failure. On the other hand, fluid and electrolyte imbalance often leads to electrophysiological abnormalities that affect multiple organ systems simultaneously. Age-related differences in function (e.g., cardio-respiratory, renal, hepatic) and pharmacologic responses, coupled with congenital anomalies and genetic abnormalities add to the complexity of fluid and electrolyte management in infants and children.

In this chapter, the newborn is discussed separately because managing perioperative fluids and electrolytes in this age group requires understanding of the unique aspects of distribution of total body water as well as specific renal, hepatic, cardiorespiratory, and central nervous system physiology. In all age groups, meticulously assessing and stabilizing the pre-operative status of the patient and promptly responding to intra-operative events sets the stage for a smooth transition to stable post-operative fluid and electrolyte balance.

General Considerations

Intravenous fluids replace water and electrolyte losses when oral intake is inadequate or impossible. Administration of intravenous fluid has at least four components: 1) replacing deficits secondary to NPO status or excessive losses (i.e., treating dehydration/hypovolemia); 2) calculating basal requirements, also called “maintenance” water and electrolytes (i.e., primarily from insensible and urinary losses and normal metabolism); 3) estimating ongoing fluid losses (i.e., diarrhea/vomiting, bleeding, intra-operative events); and 4) identifying additional derangements [e.g., hypo- or hyperglycemia, acid-base abnormalities, pathophysiologic states such as birth asphyxia, excessive secretion of arginine vasopressin (AVP)]. Although this framework for analyzing fluid delivery applies to all ages from newborn to the elderly, the unique developmental physiology of the newborn and young infant alters aspects of each component. For example, estimating “maintenance” fluid depends on gestational and postnatal age, and normal requirements for glucose change with postnatal age. Finally, responses to a similar insult may vary depending on developmental status.

At all ages, sodium is the primary positively charged ion (cation) and chloride the major negatively charged ion (anion) in the plasma. Potassium, calcium, and magnesium comprise the remaining cations in the extracellular space; bicarbonate, chloride, and proteins are notable anions. The intracellular fluid cations primarily include potassium and magnesium and proteins and organic/inorganic phosphates are the primary anions (**Table 3-1**). The tight regulation of the extracellular fluid compartment ultimately determines the volume of the intracellular space. That is, if the osmolality of the extracellular compartment changes, water moves into or out of the cell; water readily equilibrates across cell membranes in response to changes in solute concentrations. Movement of a solute, on the other hand, depends on the permeability of the solute in a specific membrane and on hydrostatic and osmotic gradients.

Table 3-1: Composition of the Extracellular and Intracellular Fluid Compartments

	Extracellular Fluid	Intracellular Fluid
Osmolality (mOsm)	290-310	190-310
CATIONS (meq/L)	155	155
Sodium (Na^+)	138-142	10
Potassium (K^+)	4.0-4.5	110
Calcium (Ca^{++})	4.5-5.0	
Magnesium (Mg^{++})	3	40
ANIONS (meq/L)	155	155
Chloride (Cl^-)	103	
Bicarbonate (HCO_3^-)	27	
Hydrogen Phosphate (HPO_4^{-2})		10
Phosphate (PO_4^{-2})	3	
Organic Acids	6	
Protein	16	40

Regulation of the extracellular compartment is directly related to cardiovascular function. For example, blood pressure and intravascular volume, as well as serum sodium concentration, are sensed by baroreceptors and osmoreceptors. The responses of the heart, the peripheral vasculature, the kidneys, and the brain to changes in pressure and osmolarity are tightly regulated by a complex interaction of hormones and mediators [e.g., renin-angiotensin-aldosterone system (RAS), arginine vasopressin (AVP) (also known as antidiuretic hormone (ADH), atrial natriuretic peptide, and catecholamines]. Other mediators fine-tune these regulatory hormones. Thus, the extracellular compartment volume and composition are regulated by a variety of hormones regulate, primarily by effects on renal sodium and water balance and on cardiac and peripheral vasculature.

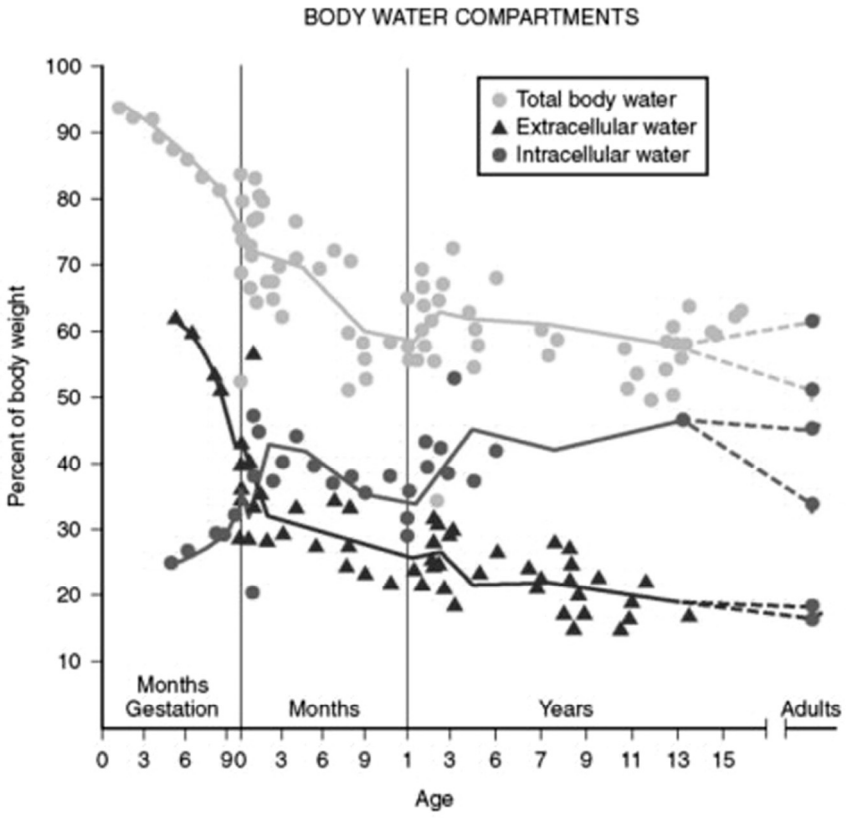
The Newborn: Developmental Aspects of Fluids and Electrolytes

Developmental Changes in Fluid Compartments and Distribution of Water

Water accounts for 78 ± 5% of a newborn's body weight, decreasing to 60% (mostly from the extracellular fraction) by six months of age, and to 57% later in childhood. Total body water is composed of intracellular (ICW) and extracellular (ECW) fluid compartments (**Figure 3-1**). In the fetus the extracellular compartment predominates, contributing approximately 62% of body weight during the first trimester of pregnancy but only about 43% at term. At the same time, ICW increases from 25%-to-32% of body weight.[1,2] The change in total body water distribution, as a function of weight, continues postnatally so that by three months of life, the intracellular compartment is larger than the extracellular compartment. The adult ratio of ECW to ICW is achieved by about one year of age. ECW is divided into plasma and interstitial compartments. The plasma compartment remains constant throughout life, comprising approximately 5% of body weight. Although two additional body compartments [slowly exchangeable areas (e.g., bone and cartilage) and transcellular] are actually extracellular, neither contributes to clinically important sites for water exchange under normal circumstances.

With their larger surface area, premature infants have a higher percentage of their body weight as total body water and a larger ECW compartment than infants born at term. Blood volume gradually decreases (as a function of weight) with growth and development.[3,4] That of a premature infant is approximately 100ml/kg, that of a full term infant approximately 90ml/kg, that of the two year old approximately 80ml/kg, and that of the older child and adolescent approximately 75-80ml/kg.

Figure 3-1: Body Compartments: Fetus to Adult



Total body water, extracellular water, and intracellular water as a percent of body weight. From: Friis-Hansen B. (1961) Body water compartments in children: changes during growth and related changes in body composition. Pediatrics 28:169-181.

Insensible Water loss.

Insensible water loss occurs primarily by evaporation from the skin (approximately 70%) and from the respiratory tract (approximately 30%). The total volume and source of this loss varies with ambient temperature and relative humidity[5] and is directly proportional to the amount of exposed surface area as well as metabolic rate. Because the ratio of skin surface area to body weight increases as gestational age decreases, premature infants lose the most water transcutaneously. Other factors, including increased permeability of and blood flow to the skin, as well as lower amount of subcutaneous fat, increase transcutaneous water loss.[6] The higher minute ventilation of the newborn magnifies the insensible water loss from the lungs. Tachypnea caused by acidosis or cardiorespiratory disease further exaggerates transpulmonary water loss.

Neutral Thermal Environment

In a neutral thermal environment (skin temperature 36-36.5˚C in an environment that has a temperature between 32˚-to-34˚C),[7] newborns expend the least amount of energy to maintain a normal body temperature. Minimizing basal expenditure of energy improves growth.[8]

Surgery in the intensive care nursery or the operating room often necessitates moving the patient from a protected and/or humidified environment to an open bed. Overhead warming and enclosed humidifying devices can seldom be used during surgery (they limit access to the patient) and during transport of infants to an operating room (inoperable without electrical power). To minimize heat and fluid loss during transport and surgery, newborns should be covered (other than the surgical site) with plastic shields, polyethylene blankets, or semi-occlusive skin barriers (including Seran Wrap™), and a hat. In addition, portable warmers that rest under the infant will help maintain the child's temperature during transport to and from the operating area. A variety of electrical warming devices are utilized intraoperatively on operating room beds.

Developmental Physiology

Various aspects of age-related characteristics of renal, cardiac, hepatic, hematologic, and neurologic function directly impact fluid and electrolyte balance in the newborn. Each of these factors will be discussed briefly.

Renal Physiology: Glomerular Filtration Rate (GFR), Tubular Function

Maintenance of normal extracellular fluid volume, electrolyte concentrations, and water balance are interrelated and undergo significant postnatal changes that are highly dependent on renal function. With nephrogenesis completed by 34-36 weeks gestation (60% occurring during the last trimester of pregnancy), at birth the number of nephrons approximates that of the adult.[9] Thereafter the size of both the glomeruli and tubules increase, but the number of nephrons does not increase more. The renal vasculature matures in parallel with the nephrons. In general, fetal and neonatal renal function is characterized by low renal blood flow (high vascular resistance), low GFR, low excretion of solids, and limited ability to concentrate the urine.

Glomerular filtration is present as early as the tenth week of gestation, increases steadily throughout fetal and postnatal life (10-13ml/min/1.73 m^2 at 25-28 weeks gestation and 20-25ml/min/1.73 m^2 after 34 weeks gestation,[10] reaching adult levels between 1-2 years of life (approximately 125ml/min/1.73 m^2, although values vary between men and women). Urine production also increases steadily throughout gestation (2-5ml/hr. at 20 weeks gestation, 10-12ml/hr. at 35 weeks gestation, and 35-50 ml/hr. at 40 weeks gestation).[11]

At the same time that GFR and renal blood flow are increasing dramatically during fetal and postnatal life, the renal tubules are simultaneously maturing. The plasma ultrafiltrate formed as a

result of glomerular filtration is presented to the proximal tubules, which play a major role in regulating the serum concentrations of a wide variety of molecules (e.g., sodium, chloride, potassium, phosphate, acid, glucose, amino acids) and excreting water. Neonates (especially premature infants) neither excrete nor reabsorb sodium as well as more mature infants.

Tubular immaturity accounts for other acid base and metabolic features of the newborn. For example, immaturity of the renal tubules impairs re-absorption of glucose; glucosuria in preterm infants may occur at serum glucose concentrations as low as 100mg/dl. Similarly, bicarbonate loss caused by incomplete tubular reabsorption leads to decreased "normal" serum bicarbonate concentrations in both premature and term newborns (12-16mEq/l, <26-28 weeks gestation; 18-20mEq/l, 30-35 week gestation; 20-22mEq/, term infant; 25-28mEq/l, adult).[12] As usual, mild hyperkalemia co-exists with metabolic acidosis (i.e., intracellular potassium moves extracellularly in exchange for protons moving intracellularly in response to acidosis). Neonatal renal function changes with both gestational and postnatal age. Due to rapid maturation after birth, the three-week old, ex-27 week gestation infant usually demonstrates more mature renal function than a one-day old term infant.

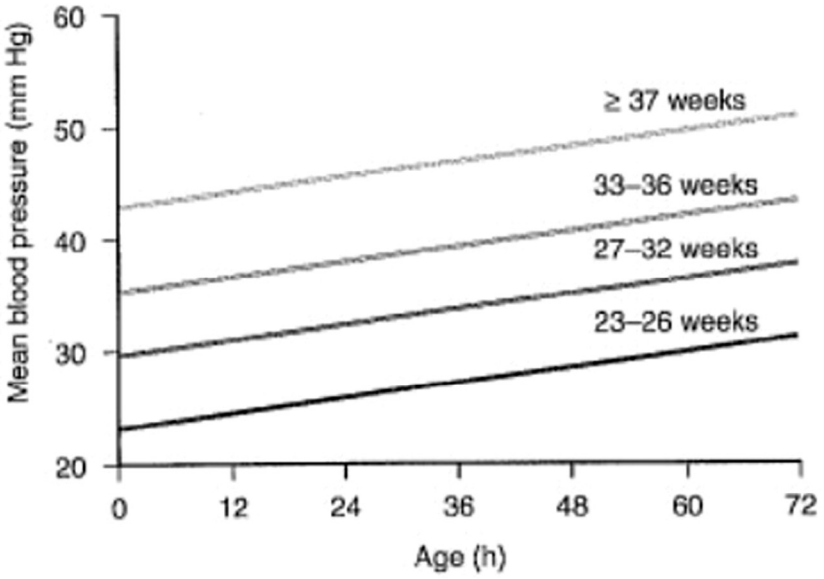
Cardiovascular Physiology: Cardiac Output, Heart Rate, Flux of Calcium

At birth, elimination of the placental circulation and initiation of breathing decreases pulmonary vascular resistance and increases pulmonary blood flow. At the same time, systemic vascular resistance increases. Left atrial pressure rises, functionally closing the foramen ovale. These physiologic events plus the closure of the ductus arteriosus and ductus venosus are the most critical aspects of the cardiovascular transition from intra- to extrauterine life. Cardiac output per kilogram body weight is higher in the newborn than at any other age. At the same time, volume loading of the immature ventricle increases cardiac output, but to a far lesser degree than occurs at older ages.[13] Finally, since the resting heart rate of the newborn is high, increasing the heart rate above normal has less effect on cardiac output. Decreasing the heart rate drastically reduces cardiac output.

Differences in myocardial ultrastructure (e.g., receptors, channels, transporters, pumps, contractile proteins) and the immaturity of various intracellular structures (e.g., myofibrils, sarcoplasmic reticulum, microtubules) influence clinical management of fluids and electrolytes, especially in the often-unstable perioperative period. For example, the volume of sarcoplasmic reticulum, as well as its ability to pump calcium, increases in utero and postnatally. In addition, the various sub-types of sarcoplasmic reticulum are less differentiated functionally in the immature heart.[14] These differences, compared to the adult, are probably the reason immature hearts are more sensitive to calcium channel antagonists,[15] and why maximal contractility is more dependent on extracellular calcium.[16]

The relationship between blood pressure, cardiac output (CO), and systemic vascular resistance (SVR) remains the same throughout life: BP = CO X SVR. That is, pressure and blood flow are not equal but are related to each other by resistance. Thus, flow to an organ may increase, decrease, or remain constant over a wide range of blood pressure depending on changes in vascular resistance. In fact, defining hypotension in the newborn is not simple.[17,18] Although the normal ranges for blood pressure correlate with gestational age,[19,20] the definition of hypotension remains elusive (**Figure 3-2**). Nonetheless, the variability in "normal" for blood pressure and heart rate among infants of the same gestational and/or postnatal age creates a dilemma in the setting of clinical care, especially in the tumultuous perioperative period.

Figure 3-2: Predicted Lower Limit of Mean Blood Pressure in Newborns (initial 72 hours of life).



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Thus, until bedside monitoring allows easy monitoring of cardiac output and/or direct measurement of blood flow to specific organs, acid base status and intravascular volume must serve as indirect guides for appropriate fluid administration. That is, rational intravascular fluid therapy requires integrating readily available cardiovascular parameters (e.g., blood pressure, heart rate) with renal, hepatic, respiratory, and central nervous system function. For example, electrolyte concentrations, pH, pCO_2, and urine output must be interpreted in the context of an infant's overall clinical condition (e.g., sepsis, bleeding, neurologic status, urine output) as well specific trends in arterial blood pressure, heart rate, and peripheral perfusion over 1-6 hours or longer. Changes over time provide more and better information than an isolated measurement.

Central Nervous System Physiology: Autoregulation of Cerebral Blood Flow

Although cerebrovascular autoregulation is present in preterm and term neonates, the range of arterial blood pressures over which cerebral blood flow is regulated is narrower for preterm infants and seems to be easily disrupted.[21,22] Of importance, autoregulation seems to be disturbed or disrupted by hypoxia, acidosis, seizures, and by the low diastolic blood pressures of patients with a patent ductus arteriosus.[23,24] Rapid increases in arterial blood pressure (e.g., from overly-aggressive administration of fluid boluses) can abruptly increase flow to the fragile vessels of the immature brain, while hypotension (e.g., inadequate intravascular volume) and low cerebral perfusion pressures may cause cerebral ischemia. More than at any other developmental stage, the intravascular volume status of the neonate, especially the premature infant, can have rapid and significant effects on the brain.

Hepatic Physiology: Glucose and Coagulation

To discuss hepatic function from the perspective of neonatal fluids and electrolytes, two topics are most relevant: glucose homeostasis and coagulation (**See Chapter 4**).

Glucose Homeostasis

The transplacental supply of glucose to the fetus abruptly stops at birth, requiring neonates to convert the glycogen stored in their livers to glucose by glycolysis or to produce glucose by gluconeogenesis. Compared to the adult, the liver of term infants is larger and has a greater store of glycogen[25] than that of adults. Thus, after the first day of life, most term infants are able to maintain a normal serum glucose concentration during a 10-12 hour fast. Since glycogen storage and the capacity for its degradation mostly occur during the last trimester of pregnancy, infants born in the second or early third trimester of pregnancy often develop hypoglycemia if they do not receive oral or parenteral glucose (i.e., fasting), especially in the first 24-48 hours of life.

Fetal glucose concentrations remain constant (approximately 50-55mg/dl) during the third trimester of pregnancy if maternal carbohydrate metabolism is normal. In experimental animals, glucose utilization at mid-gestation is about 9.5mg/kg/min and decreases to approximately 5mg/kg/min at term.[26] These data provide estimates of normal serum glucose requirements for premature infants of similar gestational ages. A surge in the concentration of catecholamines, glucagon, and cortisol (e.g., during birth or stress) increases glycogenolysis and gluconeogenesis, often resulting in hyperglycemia. In high-risk newborns (e.g., large or small for gestational age infants, premature infants, asphyxiated infants), glucose concentrations must be monitored frequently and infusion rates for glucose adjusted to maintain normoglycemia (approximately 60-90mg/dl).

Initially, glucose should be infused at rates of 5-6mg/kg/min in NPO newborns (e.g., 10% dextrose, 4ml/kg/hr. = 6.6mg/kg/min of glucose) and the infusion rates adjusted to maintain normal serum glucose concentrations of 60-90mg/dl. For infants who are stable on a well-established regimen, the infusion rate of glucose in the operating room should mimic that used in the intensive care nursery and should only be altered if hypo- or hypergylcemia develop. In many cases, especially in small infants, it may be difficult to obtain a blood sample without an arterial or central venous catheter in place, but currently available devices for measuring glucose only require a drop of blood, which can be obtained from a finger, toe, or ear lobe.

Hypoglycemia can have devastating effects on the central nervous system, primarily mediated by its effects on cerebral blood flow. MRIs of the brains of newborns with documented hypoglycemia demonstrate white matter abnormalities, and clinical evaluation at 18 months reveals persistent central nervous system impairment.[27] Older pediatric patients who also have abnormal glucose metabolism also have increased morbidity and mortality.[28]

Coagulation

Coagulation factors do not efficiently cross the placenta. (**See Chapter 4**) Although these factors are produced by the fetus, plasma concentrations and the laboratory tests used to monitor function [prothrombin time (PT); activated partial thromboplastin time (APTT)] differ markedly in both normal term and preterm infants compared to adults (**Table 3-2**). While the concentrations of vitamin K dependent factors (II, VII, IX, X), factors XI and XII, prekallikrein, kininogen, and thrombi are approximately 50% less than those of adults, the levels of fibrinogen and factors V and VIII are similar to those found in adults.[29,30] Coagulation disturbances associated with liver dysfunction are linked to decreased synthesis of clotting and fibrinolytic factors and with abnormal platelet function. The PT correlates with availability of factor VII. APTT primarily reflects the amount of thrombin generated.

Table 3-2: Normal Values of Coagulation Function in Newborns Compared with Adults.

	Prothrombin Time (sec)	Partial Thromboplastin Time (sec)	Thrombin Time (sec)
Adults	13.0 (11.2-14.5)	44.0 (36.8-50.0)	10 (9.0-11.1)
Term Infants	13.6 (12.8-14.4)	65.2 (50.0-84.0)	12.5 (10.0-15.0)
ELBW Infants	15.4 14.6-16.9)	108 (80.0-168)	14.9 (11.0-17.8)

Barnard DR, Simmons MA, Hathaway WE. Coagulation studies in extremely premature infants Pediat Res 1979; 13:1330-1335.

In spite of the differences in liver function between newborns and adults, clinically significant bleeding is uncommon in normal neonates whose have adequate vitamin K levels. On the other hand, sepsis and/or asphyxia-induced disseminated intravascular coagulation (DIC) increase the risk for bleeding. Both asphyxia and sepsis deplete coagulation factors (e.g., fibrinogen, factors V and VIII) and platelets and produce fibrin degradation products. To compensate for abnormal levels of coagulation factors, especially if accompanied by cardiovascular instability and ongoing blood loss, administration of specific blood components not only improves clotting function but can also replete intravascular volume.

Hematologic Physiology

In otherwise normal appropriate for gestational age (AGA) fetuses, hemoglobin concentrations vary with gestational age (14gm/dl at 25 weeks gestation, 16gm/dl at 30 weeks, 17gm/dl at 35 weeks, 18gm/dl at term).[31] The normal postnatal decrease in hemoglobin is more rapid in premature infants.[32]

Indications for transfusion of red blood cells or other blood components pre-, intra-, or post-operatively must be determined in the context of the patient's cardiorespiratory status, ongoing blood and fluid losses, and response to delivery of crystalloid and colloid.

Neonatal Perioperative Fluids: General Concepts

For the first 2-3 hours of life, the neonate's electrolyte concentrations reflect those of the mother and of perinatal events (e.g., asphyxia, placental or umbilical cord hemorrhage). Afterwards, the electrolyte concentrations reflect a balance among normal metabolism, cardiovascular, renal and hepatic function, and ongoing metabolic derangements (e.g., sepsis, congenital metabolic diseases, complex cyanotic congenital heart disease, etc.). Until adequate tissue perfusion is

established and washout of accumulated acid and other metabolic byproducts of anaerobic metabolism from tissues have occurred, metabolic acidosis may persist in depressed newborns, especially those who were severely asphyxiated and required resuscitation.

Sodium is seldom added to intravenous fluids of term and late preterm infants until day two of extra-uterine life. Then 2-6meq/kg/day of sodium is added. In the ELBW infant, sodium-containing fluid is often required as early as 12-24 hours of life to maintain adequate intravascular volume, especially with excessive transcutaneous fluid losses (**See section, Insensible Water Loss**). After the first few days of life, adequate sodium intake is essential for infants of all gestational ages to maintain normal growth and appropriate weight gain. Poor skeletal and tissue growth, as well as adverse neurodevelopment, are associated with chronic sodium deficiency.[33]

Ensuring normal fluid and electrolyte balance in the newborn in the presence of the high metabolic rate is associated with growth, marked insensible water loss, and limited ability to salvage and excrete water and solutes requires accurate monitoring of the newborn's fluid loss (i.e., urine, gastrointestinal, cerebrospinal fluid, blood sampling) and electrolyte concentrations to guide the quantity and composition of replacement fluid. Based on the complex interactions of illness and immaturity, the anesthetist must consider the following when determining appropriate fluid and electrolyte management in the newborn:

1. The normal postnatal diuresis contracts the extracellular space in all newborns but does so to a greater degree in preterm infants. During the first few days after birth, negative fluid and electrolyte balance occurs normally in healthy term infants, but prematurity and/or hemodynamic instability may demand aggressive intravascular fluid administration in these patients.

2. Transepidermal fluid loss is inversely related to gestational age and can be as much as 60-100ml/kg/day in ELBW infants. During the first few postnatal days, naked preterm infants lose 15 times more water by evaporation than naked term infants.[34]

3. Providing a warm, humidified environment and inspired gases and/or the use of plastic shields reduce transepidermal fluid loss, especially in the ELBW infant. Shielding devices are difficult or impossible to use during surgery but should be used during transport of the patient to and from the operating room and in the operating room before and after surgery.

4. The hypothesis that over-hydration or hyper- or hyponatremia increase the incidence of patent ductus arteriosus, necrotizing enterocolitis, and chronic lung disease should be considered, but the data documenting a strong correlation are inconsistent.

5. An intravenous infusion of calcium is usually required in preterm, asphyxiated, or LGA or SGA infants until they establish adequate enteral nutrition. Ideally, calcium is infused into

a central venous catheter, but most often only peripheral intravenous access is justified in an uncomplicated SGA or LGA newborn. The site of peripheral infusion of calcium must be meticulously monitored, since subcutaneous infiltration of calcium can cause extensive tissue necrosis. As stated above, calcium should be administered through a central line if possible.

Preoperative Evaluation of the Neonate

Understanding how fluids and electrolytes have been managed before surgery will help avoid dangerous and unnecessary alterations in therapy (e.g., rate of glucose delivery). Of course, intra-operative events, such as general anesthesia, blood loss, increased insensible losses (e.g., open abdomen with exposed intestine), often require changes in both the volume and type of fluid delivered. Developing an effective relationship with the intensive care nursery staff facilitates intraoperative consultation with the primary medical team.

The following summarizes the most significant aspects of pre-operative evaluation of the newborn:

1. **Review of labor and delivery:**
    a. Documentation of appropriate fetal growth (AGA, LGA, or SGA)

2. **Review of systems:**
    a. Trend in weight (daily or more frequent)
    b. Trend in intravenous and oral intake and urine output/urine specific gravity
    c. Trend in other output (gastrointestinal, cerebrospinal fluid, etc.)
    d. Composition of intravenous fluid (glucose, sodium, calcium) correlated with corresponding trends in plasma concentrations of these electrolytes
    e. Hemodynamic instability: trends in heart rate, arterial blood pressure, peripheral perfusion; presence and effect of patent ductus arteriosus or other cardiovascular dysfunction (e.g., tricuspid regurgitation after asphyxia)
    f. Current and recent trend in hemoglobin concentration correlated with hemodynamic function; determine if the patient "requires" a minimum level of hemoglobin (e.g., increased heart rate or acidosis if the hematocrit is <40%)
    g. Central nervous system insult (presence of intraventricular hemorrhage)
    h. Current coagulation status, history of bleeding, and trends in requirements for blood components; availability of blood products
3. **Adequacy of intravenous access and monitors** (functioning of arterial, venous, or central venous lines)

4. **Estimate "allowable blood loss"** as part of the pre-operative evaluation. For example, blood volume in a 3-day old, 1 kg infant is 100 ml/kg, or 100ml. A loss of 20 ml of blood is 20% of the blood volume. Anticipating the need for transfusion depends on current hemoglobin concentration and history of tolerance for decreasing blood volume and/or a decrease in hemoglobin.

Intra-Operative Fluid Management

During transport from the intensive care nursery to the operating room, intravenous infusions, cardiorespiratory support, and body temperature must be maintained, and hemodynamic status and oxygenation monitored. At a minimum, heart rate and oxygen saturation should be continuously monitored, and more invasive hemodynamic monitoring (e.g., arterial blood pressure) may be required, depending on the clinical status of the patient. The infant's head and body should be covered with semi-occlusive material and/or blankets/hat. Portable warming devices (e.g., a heating pad set on low) are often placed under the infant. Intravenous infusions for convenient and effective use in the operating room should be set up before the patient leaves the intensive care nursery. For example, the extensions on the intravenous and intra-arterial tubing should be long enough to allow easy access to the lines when surgical drapes are in place.

Initially, glucose is infused in the operating room at the same rate that provided normoglycemia in the intensive care nursery. Often, simply continuing the infusion of "maintenance" fluid or total parenteral nutrition is appropriate. In surgical procedures lasting longer than one hour, monitoring glucose concentrations should be considered to ensure normoglycemia. In most cases, additional non-glucose-containing fluid must be delivered to compensate for intra-operative events and blood and fluid losses. To avoid hyperglycemia and its side effects (e.g., osmotic diuresis, central nervous system injury), *glucose-containing solutions should **never** be used to replace intraoperative intravascular losses* (i.e., increased insensible losses during abdominal surgery, blood loss, etc). *In some cases (e.g., newborns, patients totally dependent on intravenous alimentation, some diabetics) glucose is delivered intraoperatively as part of maintenance fluid. All other losses should be replaced with non-glucose containing fluids.*

As always, during induction of anesthesia, anesthetic agents should be titrated to maintain an appropriate hemodynamic status. Often, dramatic hemodynamic responses occur with the administration of anesthetics (e.g., hypotension, tachycardia) and require prompt rapid delivery of a bolus of a normal saline or plasmalyte (5-10ml/kg or more).

As surgery proceeds, blood loss and coagulation abnormalities may occur and require the administration of various components of blood to maintain intravascular volume and hemodynamic stability and to restore normal levels of hemoglobin and/or coagulation factors. Monitoring blood loss requires meticulous observation of the surgical field, since loss of small volumes of blood is difficult to determine and is easily hidden in drapes and body cavities. Since

the blood volume of a 1kg infant is only 100ml, a 10-20ml blood loss can induce significant hemodynamic instability. Theoretically, urine output should be monitored, but in the smallest of infants, it may be difficult or impossible to measure small volumes of urine accurately. For example, after placing a tube into the bladder and attaching it to a drainage system, at least 5ml or more of urine must be produced before the anesthetist can detect and measure any urine in the drainage device. In addition, the small urinary catheter and drainage system are frequently soft and easily compressed, kinked, or occluded during positioning of the patient.

Inspired gases should be warmed and humidified to near body temperature whenever possible to reduce insensible fluid losses and help maintain body temperature. Overhead, servo-controlled warmers should be used during pre-operative preparation (e.g., inserting or adjusting intravenous lines and ventilation devices, preparation of the surgical site). The surgical bed should include a warming device (varies among institutions), and the room temperature should be adjusted to minimize development of hypothermia. Once the child is draped for surgery, the room temperature can usually be decreased, depending on the response of the infant's body temperature to the surgery and to decreases in the environmental temperature. Intravenous fluids and blood products should be warmed to 37°C when possible, especially if large volumes of either are required. However, some blood warming devices can overheat blood and cause hemolysis.

The following summarizes the most significant aspects of intraoperative fluid management in the newborn (**See case study, 3-3**):

1. Ensure that the intravascular volume is adequate before induction of anesthesia.

2. Estimate the need for blood products in the operating room before starting anesthesia/surgery. (**See Chapter 4**) This process should begin in the intensive care nursery to ensure availability of specific blood products when they are needed.

3. In spite of normovolemia, the newborn may respond to judicious doses of either inhaled or intravenous anesthetic agents with dramatic changes in heart rate, arterial blood pressure, and/or peripheral perfusion. To maintain hemodynamic stability, fluid and/or colloid boluses or infusions may be required.

4. The infusion of glucose should be continued at the same rate that was associated with normoglycemia in the intensive care nursery. This can be considered the "maintenance" component of fluid administration. The infusion of this solution should be adjusted, based on intraoperative measurements of glucose.

5. In most cases, delivering crystalloid compensates adequately for insensible losses, but the required rate of delivery depends on the type of surgery. For example, during a laparotomy to treat necrotizing enterocolitis, 10-50ml/kg/hr. (or more) of *non-glucose*

containing, isotonic crystalloid (normal saline or plamalyte) may be required to compensate for ongoing insensible losses from exposed bowel and peritoneum. During less invasive procedures (e.g., inguinal hernia repair), less crystalloid is required (0-5ml/kg/hr.), since ongoing losses are minimal to none. At some point after delivering large volumes of crystalloid (30-40ml/kg/hr.) for several hours (sometimes longer, sometimes shorter, depending on cardiovascular and clotting status), colloid may be a more appropriate intravenous therapy (**See #s 6, 7 below**).

6. Critically-ill newborns, especially premature newborns, are often more hemodynamically stable (e.g., less apnea and bradycardia) when their hematocrit is >40%. In those cases, frequent packed red blood cell transfusions (and/or erythropoietin) are required in the intensive care nursery. In contrast, after days to weeks in the intensive care nursery a stable infant who undergoes elective surgery (e.g., inguinal hernia repair) often tolerates lower concentrations of hemoglobin. For example, an ex-26 week gestation infant undergoing an inguinal hernia repair at 10 weeks of age (36 weeks gestation) may have no cardiorespiratory problems (i.e., no apnea/bradycardia, tachycardia, or hypotension) despite a hematocrit of 30%. In contrast, perfusion and metabolic status of a 5-day old, 26-week gestation infant with a patent ductus arteriosus and necrotizing enterocolitis, who is receiving mechanical ventilatory support, usually improves when the hematocrit is >40%. The hematocrit associated with a stable preoperative cardiorespiratory status provides the initial guideline for the appropriate perioperative level.

7. Many newborns, especially critically ill newborns, respond more promptly to the administration of colloid than to crystalloid after losing between 10-20% of their blood volume. Colloid may include packed red blood cells, fresh frozen plasma, or fibrinogen, depending on the coagulation status and the patient's hemoglobin concentration.

8. Monitoring of urine output, arterial blood pressure and/or central venous pressure helps guide intravenous fluid therapy.

9. Heat loss and insensible fluid loss should be minimized during transport of the infant to and from the operating room.

Table 3-3. Intraoperative Fluids: Newborn

12 h old, 3.2 kg, 39-week gestation infant, noted to have increasing abdominal distension. Abdominal imaging consistent with bowel obstruction, but free air also present. Nasogastric tube in place, with bilious drainage. She is breathing spontaneously (50 breaths per minute). BP= 58/34, Heart rate=160 bpm, Urine output has decreased from 2ml/kg/h to 0.5ml/kg/h over prior 6h. Two peripheral IV's are in place. D10 in NS infusing, 15 ml/h

- *Arterial blood gas: pH=7.30, pO_2=62, pCO_2=47 (blow-by supplemental oxygen).*
- *Electrolytes: Na^+, 135; K^+, 4.6; Cl^-, 106; HCO_3^-=16; Ca^{++}, 1.0; Glucose, 75 mg/dl*
- *Renal Function: BUN 32mg/dl/creatinine 0.7mg/dl*
- *Hemoglobin, 15g; Platelets, 125,000; PT, 17 seconds; PTT 48 seconds; Fibrinogen 120mg/dl*
- *Before transport to the operating room to treat bowel obstruction/perforation, 3 additional boluses of 10ml/kg administered: BP 68/40, HR 150*
- *Before transport, packed RBC's available.*

Deficit	Maintenance	Ongoing Losses
• 0 ml • Allowable blood loss: about 30-40ml	• 15ml/hr D10 infusion	• Blood products available in operating room: RBC's, fresh frozen plasma available • Infuse crystalloid, 10-50ml/kg/hr or more; boluses to maintain hemodynamic instability • RBC's for blood loss (1ml for each 2-3ml of blood loss). Fresh frozen plasma infusion/boluses in response to bleeding/laboratory values • Monitor Hg, PT/PTT, platelets

The same patient, now 6 m old, presents for elective take down of ileostomy. She tolerates a combination of breast milk and formula and now weighs 6.1 kg. Normal physical examination, with ileostomy in place. *Hemoglobin, 10g/dl; glucose, 90mg/dl; Normal electrolytes. Parents report that she has been NPO 8 hours.*		
Deficit	Maintenance	Ongoing Losses
4 X 6.1 kg X 8 h = 195 ml	• 4 X 6.1 kg = 25ml/h, isotonic fluid during surgery	• Open abdomen, but without acute injury (e.g., no ischemia) • Consider 20-30ml/kg of isotonic fluid to infuse over initial 2 hours • Adjust rate in response to hemodynamic status • Continue isotonic fluids in PACU and on ward; measure Na^+ after 24h • Transfusion unnecessary intra- or postoperatively unless unexpected blood loss/anemia

The Older Infant and Child

Unlike the newborn, older infants and children often present for elective surgery with a clearly defined medical and/or surgical history. That is, congenital anomalies and metabolic/genetic abnormalities have been identified and a specific, stable treatment plan has been established. Outside of trauma, accidents, or an acute/new illness (e.g., malignancy, gastrointestinal events, systemic or localized infection, etc.), emergent/urgent surgery is usually required to treat complications of a recognized anomaly or because diagnostic studies are needed to evaluate an underlying illness.

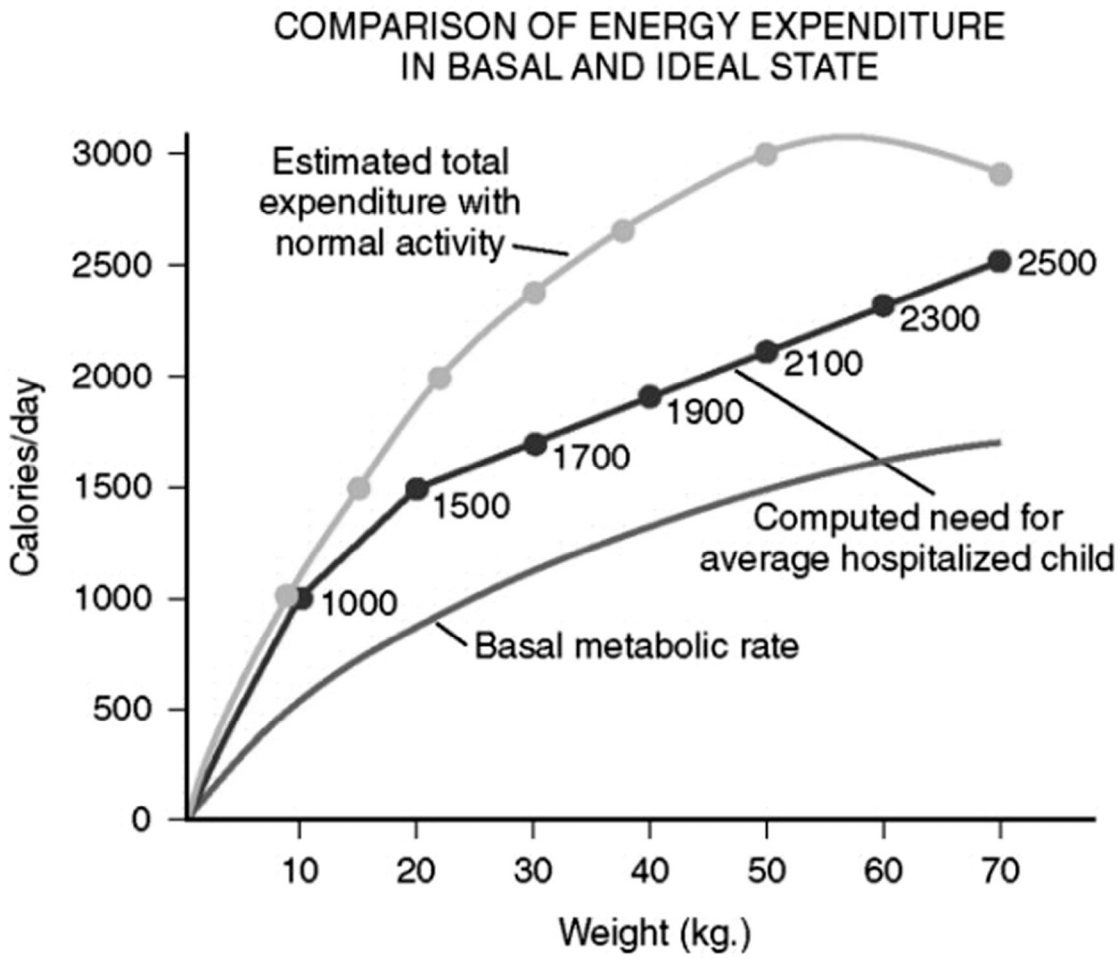
As in all age groups, pre-operative evaluation of the fluid and electrolyte status of older infants/children requires assessing intravascular volume by integrating findings from the physical examination, review of cardiorespiratory/renal/hepatic/central nervous system status, and relevant laboratory studies. And, similar to the newborn infant, when devising a rational plan for intra-operative fluid management of older children, the clinician must consider maintenance fluid requirements, replacement for ongoing losses, and the need to correct specific abnormalities (e.g., glucose, calcium, coagulation). Fine-tuning fluid and electrolyte abnormalities must be balanced against the urgency of the surgery. In some cases, monitoring laboratory values during

surgery demands ensuring easy access to central venous, arterial, or a large peripheral intravenous catheter during surgery.

Maintenance Fluid: Classic Concepts

Maintenance fluids should contain appropriate amounts of water and electrolytes to replace urine, gastrointestinal, sweat, breathing, and skin evaporation losses. Oral, intravenous, and other routes (e.g., intraosseous) can be used to administer maintenance fluid.

Figure 3-3: Comparison of Energy Expenditure in Basal and Ideal State



Based on Holiday and Segar's data, energy expended (calories) is the approximately the same as the requirement for fluid (milliliters). Although caloric expenditure varies (basal rate vs. normal activity) three linear weight categories are easily identified (0-10 kg, 10-20 kg, >20 kg). Energy expended by hospitalized patients was estimated to be approximately between that for basal and normal activity. From the curve for the hospitalized patient, a 10kg infant expends 1,000 calories (requires 1,000ml/day, 100ml/kg) and a 20kg child, 1,500 calories (requires 1,500 ml/day, 100ml/kg for the first 10kg + 50 ml/kg for each kilogram between 10 and 20kg. if the child weighs >20kg, he/she requires 1500ml + 1ml/kg he/she is above 20kg). From: Holliday MA, Segar WE. The maintenance need for water in parenteral fluid therapy. Pediatrics. 1957; 19:823-832

Any discussion of "maintenance" fluids and electrolytes inevitably notices Holliday and Segar's classic paper, *"The maintenance need for water in parenteral fluid therapy"*.[35] These authors simplified the calculation of fluid requirements by correlating insensible loses with the metabolic rate of healthy children at rest and during activity (**Figure 3-3**). They noted that the requirements for water paralleled those for energy and that the metabolic rate correlated with weight: ≤10kg, 100kcal/kg; 10-20kg, 1000+5kcal/kg; >20 kg, 1500+2kcal/kg. For a 10kg child, they calculated a net loss of approximately 34ml/kg/day of insensible loss (i.e., evaporative loss of water from the skin and respiratory tract) and approximately 66ml/kg/day of urine, for a total of 100ml/100kcal/day (approximately 4cc/kg/hour) of maintenance fluid for the ≤10kg patient. Based on curves generated from plots of weight versus estimated calories expended/day, the authors estimated that required maintenance fluid is 100ml/kg/day (approximately 4ml/kg/hour) up to 10kg, an additional 50ml/kg/day (approximately 2ml/kg/hour) for each kilogram between 10-20 kilograms, and 20ml/kg/day (approximately 1ml/kg/hour) for each kilogram beyond 20 kilograms. This is the basis for the "4-2-1" formula widely used to calculate rates for hourly maintenance intravenous fluid (**Table 3-4**).

Table 3-4. Calculating Maintenance Fluid: The "4-2-1 Rule".

Weight (kg)	Hourly Rate	Daily Volume
<10	4ml/kg	100ml/kg
10-20	40ml + 2ml/kg (for each kg between 10-20)	1000ml + 50ml/kg (for each kg between 10-20)
≥20	60 ml + 1 ml/kg (for each kg >20)	1500ml + 20ml/kg (for each kg > 20)

To characterize the composition of maintenance intravenous fluid, Holliday and Segar assumed that electrolytes in intravenous fluid should mimic those in breast milk. They estimated the amount of sodium and potassium in 100mls of human milk and extrapolated those data to determine the concentration of electrolytes needed in maintenance fluid (i.e., approximately 3-5meq/kg/day for sodium and approximately 2-3meq/kg/day for potassium). This led to the practice of adding 0.2-0.45% saline to 5% dextrose in water for routine intravenous fluid. Holliday and Segar's estimated intravenous fluids for patients was based on data derived from normal children.

However, over the last two decades, severe neurologic injury and death have been reported in both pediatric and adult patients who develop hyponatremia while receiving hypotonic intravenous solution, suggesting that Holliday and Segar's data are not easily extrapolated to the acutely ill infant/child,[36] especially after surgery.[37] Moritz's recent review emphasizes the enormous impact of hyponatremia (serum sodium <135mEq/l) since he found that approximately 25% of hospitalized children were hyponatremic.[38] Severe adverse outcomes (death or neurologic injury) have been reported in 30% of severely hyponatremic children.[39]

Thus, although Holliday and Segar's principles for calculating routine intravenous fluid have been incorporated into clinical care for over 60 years, the high incidence of hyponatremia occurring with administering intravenous hypotonic fluids has prompted many experts to strongly recommend 0.9% normal saline for the vast majority of hospitalized children, especially peri-operatively. In addition, some authors have suggested that in hospitalized patients, predicting fluid excretion/reabsorption) are not prompt or are incomplete.

Arginine vasopressin (AVP or ADH) modulates normal osmolality by increasing free water reabsorption by inducing water channel [vasopressin-sensitive water channel (aquaporin 2)] insertion into the distal tubule and collecting ducts.[33,42] In most cases, hyponatremia is linked to an excess of water rather than sodium imbalance. That is, hyponatremia evolves with or without a deficit of sodium, and with either hypovolemia or euvolemia, but in the presence of AVP.

Hypovolemia (i.e., inadequate intravascular volume) appropriately elicits a stimulus to decrease free water excretion (i.e., release of AVP is normal). Regardless of the etiology (e.g., salt wasting states, sepsis, blood/fluid loss), hypovolemia initially is usually treated with boluses of an isotonic crystalloid (e.g., 0.9% normal saline), and, in some cases, followed by colloid or blood products. Treatment is guided by various parameters, such as the hemodynamic response, acid-base status, and urine output. In most cases, after the initial repletion of the intravascular volume (with normal underlying renal, hepatic, and cardiorespiratory function), isotonic fluid should be continued at maintenance rates during the transition to oral hydration/feeds. At that point, if intravenous supplement is required, intravenous hypotonic fluid might be considered.

In the absence of renal pathology, congestive heart failure, or hepatic disease (cirrhosis), there is euvolemic non-osmotic, un-physiologic (excessive) release of requirements (i.e., fluid losses) ased on energy expenditure overestimates the volume needed for maintenance fluid[40] (**See section, Intravenous Fluids and Hyponatremia: Current Controversy**).

In all cases, intravenous fluid should be considered an "invasive therapy" or a "medication" rather than an exact replacement for breast milk, formula, or other

oral fluids. Patients who require intravenous fluid often have a significant illness that is associated with hormonal imbalance or physiologic derangements (e.g., renal or hepatic disease,

chronic lung disease) that complicate maintaining adequate intravascular volume and electrolyte balance. Holliday reminds us, "when fluid therapy extends beyond the first day, monitoring is needed to adapt orders to special cases, avoiding the consequences of 'one plan fits all".[41]

Physiology of Hyponatremia: Clinically Relevant Concepts

More than any other molecule, sodium establishes osmolality (normal, 280-290 mOsm/l) in the extracellular space (Plasma Osmolality = 2 x [Na^+] + [glucose]/18 + [BUN]/2.8), and consequently contributes significantly to maintaining extracellular fluid volume, blood volume, and perfusion pressure. Sodium cannot freely move across cell membranes. On the other hand, water does freely pass through cell membranes in response to osmolar gradients. Arginine vasopressin (AVP) is secreted by the pituitary gland in response to low circulating blood volume and results in the retention of free water by increasing water reabsorption in the collecting ducts of the kidney nephron.

The release of AVP is often a response to central nervous system abnormalities (brain tumors, meningitis, trauma), pulmonary disease (asthma, pneumonia), various non-CNS malignancies (lung, leukemia, lymphoma, thymoma), or other non-specific pathology (e.g., nausea, stress due to pain, post-operative state). In these cases, the etiology of hyponatremia is attributed to abnormal release of and response to AVP, despite the absence of both osmotic and baroreceptor mediated stimuli.[26] Consequently, the high incidence of hyponatremia is not surprising in perioperative patients who commonly encounters disorders associated with both physiologic and non-physiologic excessive levels of AVP.

SUMMARY: Perioperative Intravenous Fluids: Current Controversy

Reports of hyponatremia-induced morbidity and mortality (e.g., death, permanent brain injury) have elicited heated debate about whether hypotonic or isotonic crystalloid should be the routine maintenance fluid, especially in the perioperative period (**SeeGeneral Considerations**). Thus, isotonic solutions do not change cell volume, but intravenous hypotonic solutions may increase (water moves intracellularly) and hypertonic solutions decrease (water moves extracellularly) cell volume. Infants and children who require surgery have an especially high risk for hyponatremia from a combination of events:

1. Co-existing significant medical illnesses that predispose to AVP levels (e.g., leukemia/chemotherapy, congenital heart disease/diuretic therapy, chronic lung disease/ex-premature infant).

2. Non-osmotic stimuli for AVP secretion are commonly encountered perioperatively (e.g., pain, nausea and vomiting, narcotic administration, inhaled anesthetic agents, positive pressure ventilation), and increase the risk for hyponatremia.

3. When the concentration of AVP is high, hypotonic solutions greatly predispose to hyponatremia.[42,43]

4. Specific surgical patients are at high risk for hyponatremia (those with central nervous system injury[44] or scoliosis repair).[45,46]

Administering hypotonic intravenous fluid in the presence of AVP has been considered to be the primary etiology of postoperative hyponatremia. Burrows[45] noted that serum sodium concentrations decreased postoperatively in pediatric patients who had received either hypotonic or isotonic fluids, but more so when hypotonic solutions were administered. Similarly, Eulmesekian[47] reported hyponatremia (Na≤135mmol/L) in 21% (by 12 hours) and 31% (by 24 hours) of patients admitted to a PICU after orthopedic surgery.

Several prospective randomized studies,[37,48-52] as well as smaller observational reports[53] and recent reviews,[38,54] document that administering isotonic intravenous fluid minimizes perioperative hyponatremia but does not induce hypernatremia. The rate of fluid delivery is apparently less important than the concentration of sodium delivered.[49,50] Large or even normal volumes of hypotonic intravenous fluid are an obvious source of excess water.

Minimizing the risk for hyponatremia requires meticulous attention throughout the perioperative period (**See case study, 3- 5**):

1. **Preoperative:**
   - Ensure adequate intravascular volume
   - Measure serum sodium if intravenous therapy is required for >12-24 hours, especially during intravenous therapy with hypotonic intravenous fluid
   - Maintain a low threshold for symptoms of hyponatremia, especially with high-risk physiology/disease states.

2. **Intraoperative**

   - Since NPO guidelines now allow *clear liquids* orally up to two hours before the induction of anesthesia, children are ideally fasted for no more than 3-4 hours.[55] Thus, pre-operative dehydration is more likely linked to an underlying disease state. If the pre-operative fast was appropriate and without evidence for intravascular deficit or hemodynamic response to induction of general anesthesia, intravenous fluid administration beyond those required for maintenance rates may not be necessary during non-invasive surgery or may be deliberately avoided (e.g., stable patient undergoing neurosurgery). The clinician must assess the physical status of the patient and the nature of the surgery rather than simply delivering intravenous fluid to compensate for the hours of NPO.

- On the other hand, families may not follow NPO instructions and/or the operating room schedule may be delayed. If the fasting period has been prolonged or there is a high-risk disease state, the anesthetist should consider giving a bolus of 20-40ml/kg of isotonic fluid over the initial 1-2 hours of surgery and then adjust the rate of administration based on maintenance plus ongoing fluid losses. This may inhibit excessive AVP release and avoid hypovolemia. After initial vigorous fluid delivery, "maintenance fluids" might be decreased to half the classic rate recommended by Holiday and Segar ("2-1-0.5" ml/kg/h).[56]

3. **Postoperative**

   Isotonic solutions are appropriate for postoperative maintenance fluids and for replace ongoing losses. Frequent monitoring of the patient's clinical status and serum electrolytes should guide both the rate and electrolyte composition of the intravenous fluid.

As Bailey notes, "no single IV fluid can be used safely in all situations".[54] Excessive delivery of any solution to patients with cardiovascular or renal failure could be disastrous. Finally, hypotonic intravenous solutions may be required when attempting to compensate for excessive free water loss (e.g. diabetes insipidus).

Summary: Perioperative Fluids and Electrolytes

During pre-, intra-, and post-operative periods, the clinician must differentiate between delivery of intravenous fluid to treat hypovolemia (e.g., to compensate for NPO status) and delivery of "maintenance" fluids. Boluses of isotonic fluid are appropriate to correct hypovolemia (i.e., hemodynamic instability, poor perfusion). In the perioperative period, one indication for hypotonic fluids is excessive loss of free water, which must be diagnosed and monitored by simultaneously measuring serum and urine electrolyte concentrations. At all times, meticulous monitoring of the patient's clinical status and serum electrolytes is essential to avoid perpetuating or inducing electrolyte abnormalities.

Intra-Operative Glucose Delivery in the Older Infant/Child (See Case Study, Table 3-5)

In the era of prolonged preoperative fasting, hypoglycemia was common.[57,58] Now, with more liberal NPO regimens, the incidence of hypoglycemia ranges between 0-2.5% and occurs almost exclusively in patients who have had an inappropriately long fast (8-19 hours).[59] Nonetheless, in spite of its rare occurrence, when hypo- and hyperglycemia do occur, the consequences can be catastrophic. For example, administering glucose-containing fluid at high rates during surgery may cause hyperglycemia, glucosuria, and an osmotic diuresis. Of greater significance, hyperglycemia has been associated with increased mortality and infection in both adult and pediatric patients in intensive care units.[60,61] In the absence of an underlying metabolic requirement (e.g., diabetes, methylmalonic acidemia) intraoperative fluids do not routinely include glucose.

Table 3-5. Intraoperative Fluids: Beyond infancy

<table>
<tr><td colspan="3">Three year old previously healthy child is scheduled for excision of a Wilm's Tumor. NPO X 3h; Wt = 15kg; hemoglobin, 1 g/dl; open laparotomy</td></tr>
<tr><td>Deficit</td><td>Maintenance</td><td>Ongoing Losses</td></tr>
<tr><td>150 ml</td><td>50 ml/hr</td><td rowspan="2">• 20-30ml/kg over initial 1-2h of surgery to ensure adequate intravascular volume, suppress AVP

• After initial 1-2h, 10-20ml/h for insensible losses with "open abdomen"

• Until transfusion, replace EBL with crystalloid (volume = 2-3 times the EBL)

• Consider RBC's if Hgb <8g/dl &/or hemodynamic instability/or massive ongoing losses</td></tr>
<tr><td colspan="2"></td></tr>
<tr><td colspan="3">A 6 yo presents to the ER after being struck by automobile. He is conscious, but has X-Ray documented right femur fracture. Abdominal examination is normal.

Parents report no significant past medical history. Patient ate full meal two hours prior to trauma. Weight 22kg.

On admission, BP= 75/40mmHg, Heart rate=130bpm

Two peripheral IVs have been placed. 0.9 NS infusing, about100ml/h.

• Arterial blood gas: pH=7.42, pO_2=98, pCO_2=33 (blow-by supplemental oxygen).
• Electrolytes: Na^+, 135mmol; K^+, 4.6mmol; Cl^-, 106mmol; HCO_3^-=20 mmol/l; Ca^{++}, 1.0mmol/l; Glucose, 110 mg/dl
• Renal Function: BUN 12mg/dl/creatinine 0.7mg/dl
• Hemoglobin, 9g/dl; Platelets, 125,000; PT, 12 seconds; PTT 40 seconds;
• Before transport to the operating room, three boluses of 10ml/kg 0.9 NS administered: BP 85/50mmHg, HR 110bpm
• Before transport, packed RBC's available
• Four doses of 1mg morphine titrated in ER, minimal hemodynamic effect</td></tr>
</table>

Table 3-5. (Continued)

Deficit	Maintenance	Ongoing Losses
• 10ml/kg (200ml, 0.9 NS) infused rapidly prior to rapid sequence induction	• 60ml/h, isotonic fluid (no glucose)	• BP decreased from 90/50-to-65/40, heart rate 110bpm, after bolus of 20mg propofol, 25mg rocuronium • 20ml/kg of isotonic fluid to infused rapidly, BP increased to 80/50, HR stable at 100-110bpm • Sevoflurane/fentanyl titrated while delivering additional 0.9 NS; no glucose • Decrease isotonic fluid delivery as hemodynamic status improves • Measure hemoglobin and ongoing blood loss to assess need for PRBC • Continue isotonic fluids in PACU and on ward; measure Na^+ after about 24h, if NPO • transfusion unnecessary intra- or postoperatively if blood loss controlled and hemodynamic status stable

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Chapter 4

HEMOSTASIS AND BLOOD TRANSFUSION

Tony Chang, MD, Elizabeth Donegan, MD

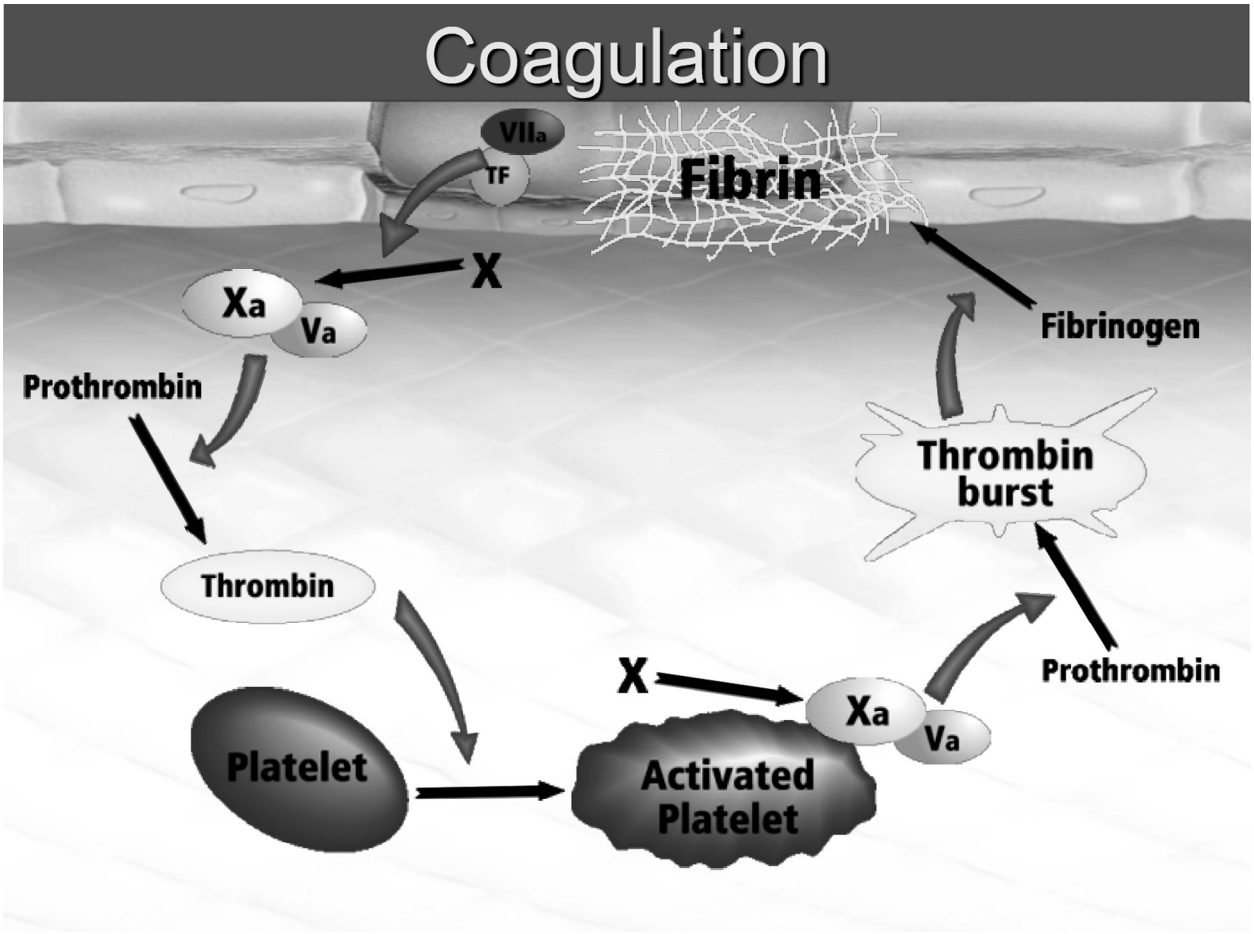
The infusion of blood and blood products from one individual to another to stop or prevent bleeding, provide adequate oxygen delivery, and to prevent death from hemorrhage saves millions of lives. Nevertheless, the practice of Transfusion Medicine remains in its infancy. In the 20th century, blood transfusion became a relatively safe and common practice, despite the relative absence of transfusion guidelines based on randomized, prospective controlled trials. Most transfusion practices are based on convention and convenience. The understanding of hemostasis, safe transfusion practices, appropriate testing of blood and blood products, as well as the consequences of transfusion continues to unfold. An understanding of these principles is very important for anesthetists who transfuse about half of the blood transfused in hospitals. Without this knowledge, transfusion practices often result in poor outcomes.

This chapter will discuss the current understanding of hemostasis, laboratory tests that assist in the decision of which components or products to transfuse and when to transfuse. Current pediatric transfusion practices, and the laboratory collection, preparation, and testing of blood and blood components are included.

HEMOSTASIS

Hemostasis is now understood to be a complex system of checks and balances designed to prevent abnormal clotting and uncontrolled bleeding. Coagulation is a cell-based event initiated on the surface of endothelial cells, in the subendothelium, and on platelets (**Figure 4-1**).

Figure 4-1: Coagulation



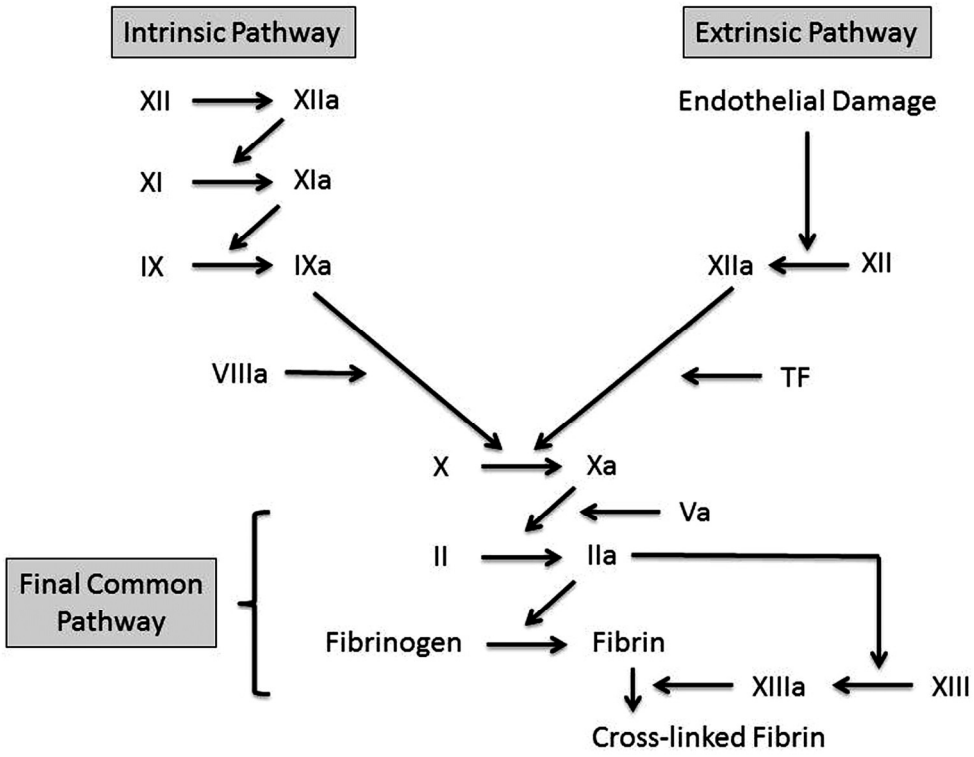
Overview of coagulation: Coagulation is activated when endothelium is disrupted and blood contacts tissue factor (TF). TF activates factor VII in turn activators factor X, which in combination with factor V, forms the prothombinase complex. Further amplification occurs with platelet surface interaction resulting in a thrombin burst, which catalyzes formation of fibrin.

Following endovascular injury, control and termination of vascular bleeding consists of two sequential responses: primary hemostasis and secondary hemostasis. Primary hemostasis has four overlapping phases 1) vasoconstriction at the site of endovascular injury 2) von Willibrand factor (vWF) mediated platelet adhesion to exposed underlying tissue factor 3) platelet activation 4) platelet aggregation. Secondary hemostasis is the activation of serine proteases and their cofactors on endothelial and platelet surface phospholipids, which culminates in the formation of cross-linked fibrin and stabilizes the platelet plug.

Coagulation Cascade

In the 1960s, coagulation appeared to be the result of a serial stepwise activation (cascade) of either an intrinsic (intravascular) or extrinsic (extravascular) system of coagulation factors (**Table 4-1**) leading to a common pathway ending in the formation of fibrin (**Figure 4-2**). The prothrombin time (PT) assessed adequacy of the extrinsic system, partial prothrombin time (PTT) assessed the intrinsic system and the thrombin time (TT) the common coagulation pathway. A stable clot formed with the contribution of platelets activated and trapped at a bleeding site. These laboratory tests, in addition to the hemoglobin/hematocrit and platelet count continue to be useful in determining the need for transfusion of blood and blood products.

Figure 4-2: Intrinsic and Extrinsic Pathways



Classic coagulation cascade depicting the intrinsic (left), extrinsic (right), and final common pathway: Refer to Table 4-1 for the names of coagulation factors.

Table 4-1: Coagulation Factors

Coagulation Factors	
Number	Name
I	Fibrinogen
II	Prothrombin
III	Tissue Factor
IV	Calcium
V	Labile factor/proaccelerin
VII	Stable factor/proconvertin
VIII	Antihemophilic factor
IX	Christmas factor
X	Stuart Factor
XI	Plasma thromboplastin antecedent
XII	Hageman factor
XIII	Fibrin stabilizing factor

Coagulation Tests

Prothrombin time evaluates the extrinsic pathway of the coagulation cascade as well as the common pathway (factors I, II, V, VII, X) (**Figure 4-2**). Due to differing reagents used in laboratories to measure PT causing varying normal reference ranges, the *international normalized ratio (INR)* was developed to standardize results. It is a ratio of the patient's PT to a normal control sample raised to the international sensitivity index, a value assigned to tissue factor used to activate clotting by the manufacturer. Most often, PT is used to monitor therapeutic levels of warfarin that are being given to prevent clotting. PT may also be prolonged in patients who have liver disease, vitamin K deficiency, disseminated intravascular coagulation (DIC), or factor I, II, V, or X deficiency.

Partial Thromboplastin Time, or *activated partial thromboplastin time (aPTT),* assesses the intrinsic pathway and the common pathway (factors I, II, V, X, VIII, IX, XI, XII) (**Figure 4-2).** PTT is often used to monitor therapeutic levels of unfractionated heparin. Low molecular weight heparin (LMWH) must be monitored by anti-Xa levels. Mild factor deficiency may result in a normal PTT. Prolonged PTT may not be observed until factor levels are 40% of normal. Von Willebrand disease, liver disease, DIC, factor I, II, V, VIII (Hemophilia A), IX (Hemophilia B), X, XI deficiencies may prolong PTT. Lupus anticoagulant (LA), a non-specific inhibitor, may prolong PTT as well. The LA-sensitive PTT or dilute Russell viper venom test should be done if LA is suspected. Mixing studies may also be used to differentiate between factor deficiency and non-specific inhibitors by mixing normal plasma with the patient's prolonged PTT sample. If mixing results in a normalized PTT, factor deficiency may be diagnosed. If the results remain prolonged, a non-specific inhibitor may be suspected, and further studies may be indicated.

Thrombin time or thrombin clotting time (TCT), is used to evaluate the coagulation cascade at the level of fibrinogen by measuring the time to clot after thrombin is added to plasma. The test is sensitive to heparin, fibrinolytics, and antifibrinolytics. Prolonged TCT times may be due to the effects of heparin, fibrinogen dysfunction or deficiency, fibrin degradation products, or factor XIII deficiency. Methylene blue, which neutralizes heparin, can be added to the assay to determine whether heparin contamination of the specimen is the cause of a prolonged TT. A functional fibrinogen assay has largely replaced this test. Malnutrition, liver disease, DIC, and fibrinolysis may contribute to low fibrinogen values. Fibrinogen is an acute phase reactant and elevated levels are non-specific.

Blood to be tested for PT, PTT, and TT is collected in a tube containing buffered sodium citrate to allow chelation of calcium and to maintain proper plasma pH. The tube is then centrifuged to remove platelet phospholipids. Standard amounts of calcium and phosopholipid are added back to the plasma for testing at 37^{o} C. Under-filled anticoagulated tubes will result in excess anticoagulant, prolonging the results of these tests. The idea ratio of blood to anticoagulant is 9:1.

Activated clotting time (ACT), or *activated coagulation time*, is commonly used to monitor the effects of anticoagulants during procedures that require anticoagulation, such as cardiopulmonary bypass, extracorporal membrane oxygenation, or other vascular surgeries. Most frequently, the anticoagulant that is monitored is unfractionated heparin, but it may also be used to monitor other anticoagulants, such as direct thrombin inhibitors. Fresh whole blood is added to a tube containing surface activators. The activator may be celite, glass, or kaolin. These activators initiate coagulation through the intrinsic pathway to form clot after the sample is warmed. Results are measured in seconds. ACT is a fully automated point of care test that results in obtaining more rapid results than PTT. PTT is insufficient for monitoring the effects of heparin because the PTT is overly sensitive to high doses of heparin that cause coagulation to remain incomplete. Factors that may affect the ACT results include temperature, thrombocytopenia or other qualitative platelet abnormalities, platelet inhibitors, anticoagulants, factor deficiency, hemodilution, and antithrombin deficiency. A baseline ACT is commonly obtained prior to anticoagulation. After heparinization, another ACT is obtained to ensure adequate anticoagulation for the procedure. A target ACT of 480 seconds is commonly used prior to initiation of cardiopulmonary bypass. Subsequent ACTs are obtained to determine the need for additional heparin to maintain the desired ACT level, depending on the procedure. ACT does not correlate with heparin levels. These measurements are less precise than PTT and large variability exists between patients and their response to heparin.

Platelet count is included as part of complete blood counts. Thrombocytopenia is defined as a platelet count $<150x10^{3}/\mu L$. Patients with platelet counts $>50x10^{3}/\mu L$ are generally asymptomatic, unless undergoing surgical procedures that have hemorrhage risk or if there is

significant trauma. At counts of <$10x10^3/\mu L$, the risk of spontaneous bleeding is increased; petechiae and spontaneous bruising occur. Platelet count <$5x10^3/\mu L$ is considered a hematological emergency, given the risk of severe spontaneous hemorrhage (intracranial, mucosal, gastrointestinal). The etiology of thrombocytopenia can be divided into increased platelet consumption, decreased platelet production, and platelet sequestration in the spleen. Increased platelet consumption may be immune mediated (idiopathic, autoimmune, drug induced), platelet activation (DIC, hemolytic uremic syndrome, thrombotic thrombocytopenic purpura, necrotizing enterocolitis), or mechanical destruction. Decreased platelet production may result from infection, cyanotic congenital heart disease, bone marrow failure, malnutrition, or impaired thrombopoiesis. Platelet sequestration may occur in liver disease, von Willebrand disease (Type 2B, platelet-type), and hypersplenism related to malaria or other diseases.

Bleeding time (BT) is a classic test used to determine functional platelet deficiency and disorders in primary hemostasis. Currently, this test is rarely used because more reliable, comprehensive, and less invasive tests are available. The principles of bleeding time involve a controlled cutaneous puncture on the forearm and observation of the time required for bleeding to stop, measured in 30 second intervals. In the Ivy method, a blood pressure cuff is placed on the upper arm and inflated to 40mmHg. A standard incision of 1mm deep and 1cm long is made on the hairless ventral side of the arm. Blood is blotted away every 30 seconds. Normal bleeding time is less than 9 minutes. Von Willebrand disease, Glanzmann's thromboasthenia, Bernard-Soulier disease, connective tissue disorders, thrombocytopenia, medications affecting platelet function, liver failure, uremia, and hypofibrinogenemia may prolonged bleeding time. Bleeding time is not performed when the platelet count is less than $100x10^3/\mu L$ and not recommended in children under the age of three years or children unable to cooperate. This test is primarily used as a screening test and is not sufficient for diagnosing specific conditions.

Platelet aggregation studies are used to determine the presence of intrinsic platelet abnormalities by examining the platelet clumping response to platelet agonists. Whole blood is collected in a citrated blood tube and centrifuged to obtained platelet rich plasma, which is then placed in a cuvette. Whole blood may also be used in a variant method. Continuous light transmission aggregometry is performed by placing the cuvette between a light source and photocell where transmission is detected. After a platelet aggregate is added, platelets begin to clump and more light is passed through the sample, which is sensed by the photocell. Most agents that induce platelet aggregation (collagen, epinephrine, and thrombin) act through effects on the adenosine diphosphate (ADP) normally released by platelets upon activation.

Decreased collagen and epinephrine aggregation occur in samples from patients taking aspirin and anti-inflammatory agents. Abnormal thrombin aggregation occurs in patients with some intrinsic platelet defects (Bernard-Soulier disease, storage pool defects). The addition of exogenous ADP causes direct platelet aggregation. Patients with Glanzmann's thrombasthenia

fail to aggregate their platelets when ADP is added. Patients with von Willibrand's disease have normal platelets and consequently show aggregation with the addition of ristocetin. This test is sensitive to platelet count, fat content of blood, temperature, pH, fibrinogen content, and anticoagulants. Patients with uremia or severe liver disease may develop complex bleeding disorders, which can include platelet dysfunction.

Platelet function assay (PFA) is a screening test that measures platelet adhesion and aggregation in primary hemostasis. It depends on platelet function, number, von Willebrand factor, and hematocrit. Anticoagulated whole blood is passed through a membrane that contains collagen and epinephrine or ADP at a rate that simulates small capillaries. The platelets adhere to the membrane and gradually occlude the membrane. The time to complete occlusion is referred to as the closure time. This test has a high *negative* predictive value of platelet dysfunction if the collagen/epinephrine membrane closure time is normal. If this value is prolonged, a collagen/ADP test is automatically performed. If this closure time is normal, an aspirin-induced platelet dysfunction may be inferred. If both values are prolonged, anemia, thrombocytopenia, or other platelet function defects are present, such as von Willebrand disease, renal failure, release defect, Bernard-Soulier disease, or Glanzmann thrombathenia; further work up should be pursued.

Thromboelastography (TEG) is a method of evaluating the viscoelastic properties of blood coagulation. Whole blood is placed in a cup. A metal pin oscillates in the cup as clot formation is initiated. The instrument rotates the cup and as fibrin and platelet aggregates form, it begins to adhere with the cup. The torsion wire connected to the pin measures clot strength over time. Lysis of the clot causes a decrease in clot strength. *Rotational thromboelastometry (ROTEM)* is another version in which there is a rotating sensor shaft and oscillating pin with an optical sensor.

Figure 4-3a: Tissue-Based Coagulation

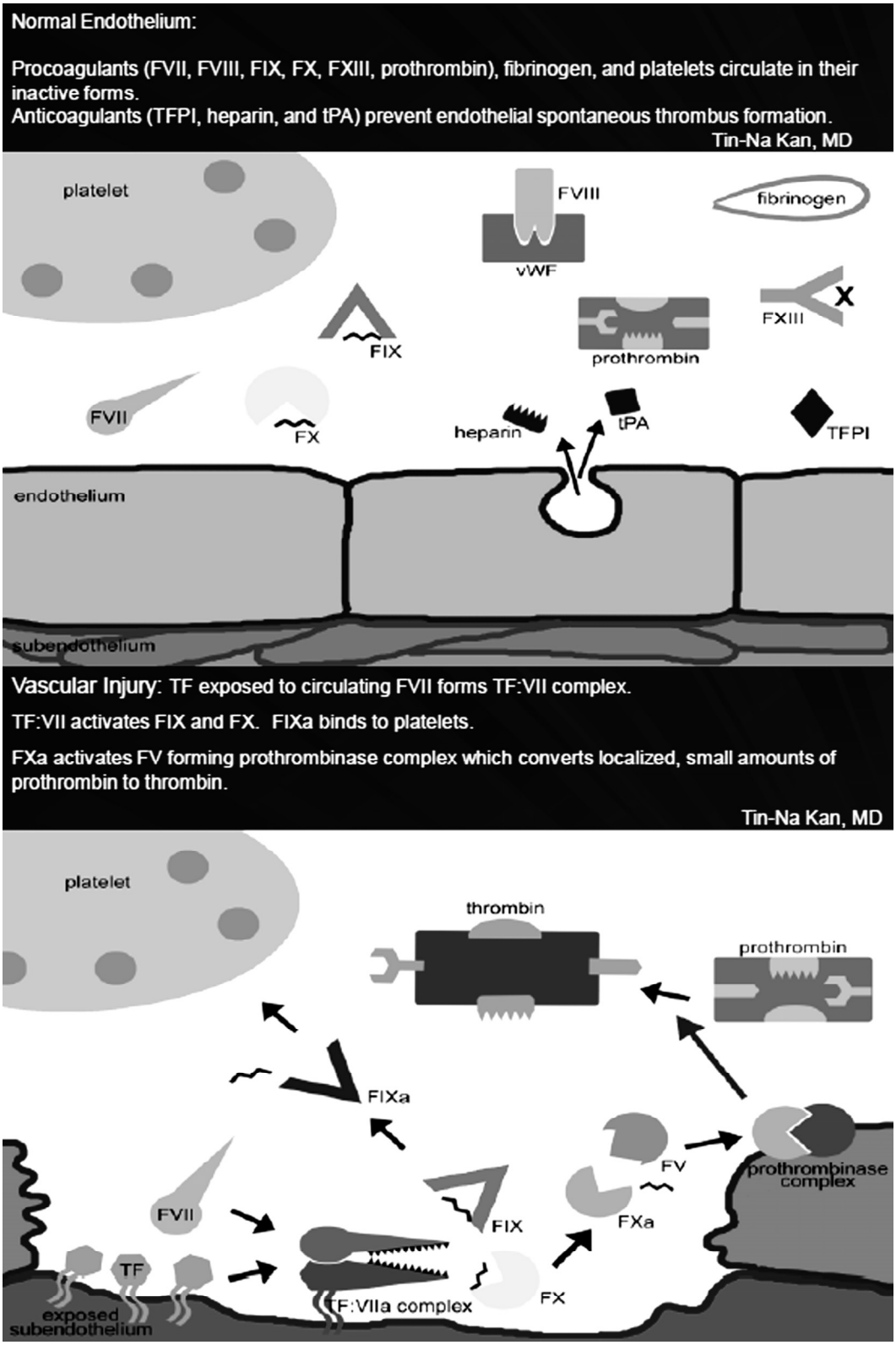
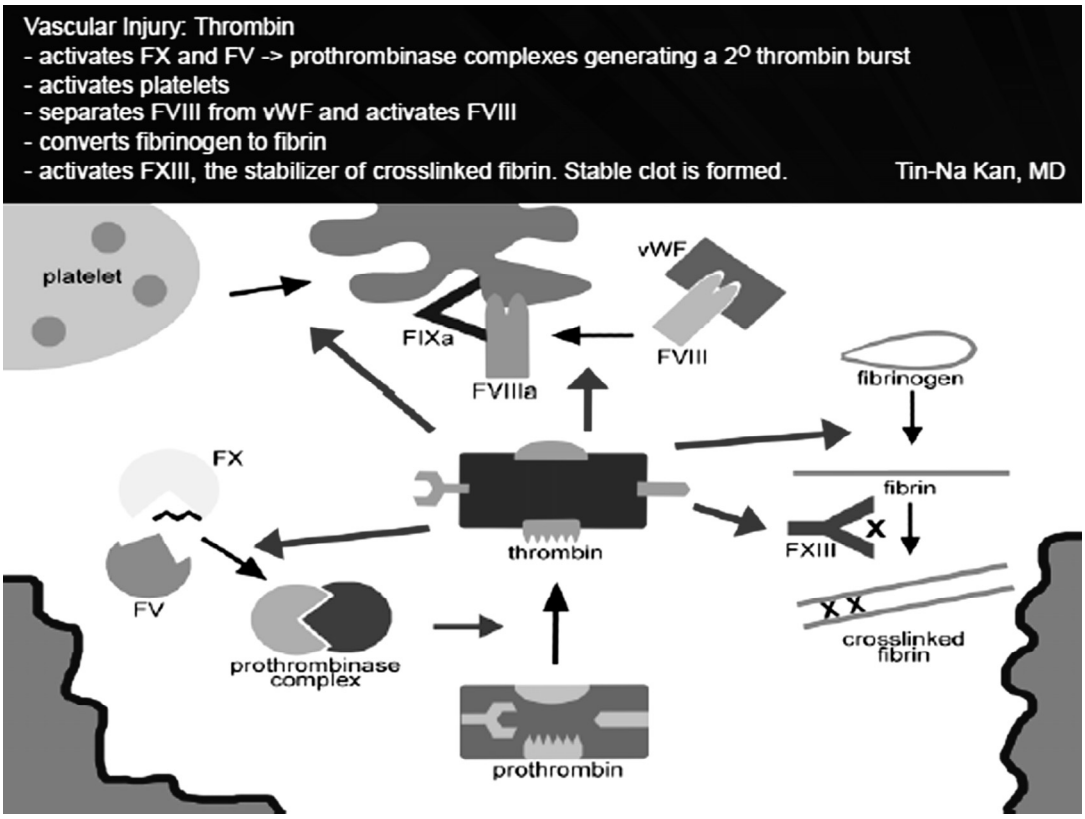


Figure 4-3b: Tissue-Based Coagulation



See text above figures for details. vWF, von Willebrand factor; TFPI, tissue factor pathway inhibitor; tPA, tissue plasminogen activator.

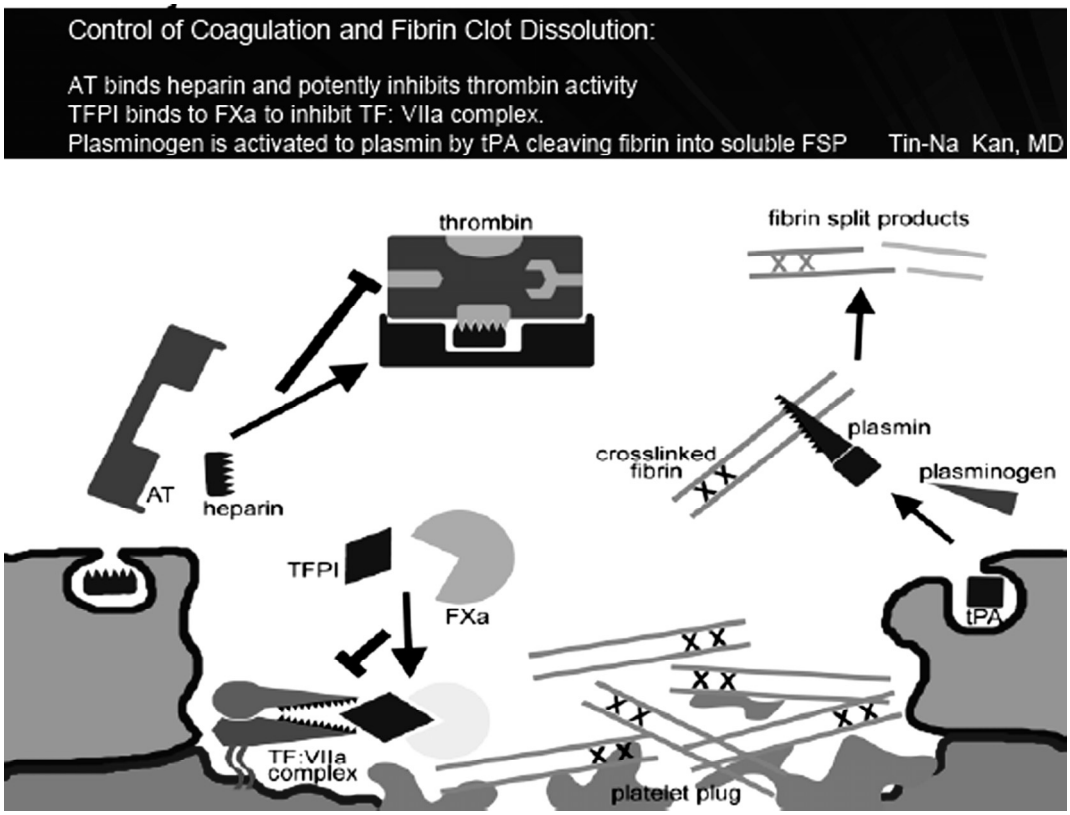
The physiologic response to bleeding is initiated with endothelial damage and intravascular exposure of tissue factor. Vessel wall-platelet interactions cause vasoconstriction and exposure of *tissue factor*, a transmembrane protein, to the intravascular environment. Platelets aggregate at the site of injury and are activated, releasing cytoplasmic granules with the formation of an unstable primary plug. Coagulation then proceeds with three overlapping stages resulting in the formation of fibrin plug: tissue factor initiated activation of coagulation factors, amplification of activated coagulation factors, and thrombin propagation on activated platelet surfaces.

Intravascular surface exposure of tissue factor activates small amounts of local factor VII (FVIIa) to which it attaches (**Figure 4-3a**). The TF-FVIIa complex activates factor X (FXa) and factor IX (FIXa). FXa activates and complexes with factor V (FVa). Prothrombinase complex (FXa-FVa) catalyzes the conversion of small amounts of prothrombin to thrombin (initiation phase). This thrombin activates local platelets that change membrane configuration and release cytoplasmic granules (ADP and serotonin, vWF and factor V), causing changes in platelet shape and membrane expression of surface receptors. Thrombin, through a positive feedback mechanism, activates additional FV, FVIII, FXI and platelets (amplification phase). The propagation phase proceeds with FIXa binding to FVIIIa (the tenase complex). Tenase activates additional FX (FXa) which again

complexes with FVa forming large amounts of prothrombinase complex and consequently large amounts of thrombin (*thrombin burst*) (**Figure 4-3b**). Secondary hemostasis proceeds on the platelet surfaces with binding of fibrinogen to platelet surface receptors, leading to thrombin cleavage of fibrinogen to fibrin. Large numbers of platelets are entrapped and activated forming an unstable fibrin monomer plug. Activated factor XIII cross-links and stabilizes fibrin monomers, which are irreversibly polymerized. Thrombin also activates FXIII and thrombin-activatable inhibitor, which participate in stable clot formation. Thrombin participates in its own down-regulation by complexing with thrombomodulin, which activates protein C and its cofactor protein S, inactivating FV and FVIII. Circulating thrombin is inactivated by antithrombin.

Fibrinolysis

Figure 4-4: Fibrinolysis



Refer to text above figure for details. TFPI, tissue factor pathway inhibitor; tPA, tissue plasminogen activator.

Normally, plasmin circulates as *plasminogen* (the inactive form). Endothelial cells secrete plasminogen activator inhibitor type 1 to inhibit the activation of plasminogen. Endothelial injury initiates secretion of tissue plasminogen activator generating plasmin by stimulating the cleavage of plasminogen (**Figure 4-4**). Plasmin degrades cross-linked fibrin to soluble D-dimers and fibrin degradation products, both of which inhibit thrombin. Since tissue plasminogen activator also binds to fibrin, the generation of plasmin remains a localized event. Circulating plasmin, by comparison, is regulated by a more powerful (100x) inhibitor, α2-antiplasmin. Fibrinolysis is also contained in surgery and trauma by release of acute phase reactants that limit fibrinolysis.

Developmental Hemostasis

Procoagulant and anticoagulant proteins form in utero. Most of these proteins reach normal adult levels by six months after birth. Some reach adult levels during adolescence. These differences can complicate the correct diagnosis of coagulation disorders in early childhood and can affect monitoring of anticoagulation treatment. Differences in levels of coagulation proteins, as compared with adult levels, may not be important in hemostasis. Some coagulant proteins, such as TF and thrombomodulin, are important in angiogenesis, inflammation, and wound healing.

Coagulation factors do not cross the placenta. The fetal liver begins production of coagulation factors at five weeks gestation, with measurable, albeit low, plasma levels at 20 weeks gestation. Fetal forms of protein C and fibrinogen change to the adult forms at birth. The only procoagulant proteins in the normal adult range at birth are fibrinogen, FV, and FVIII. Vitamin K dependent factors (II, VII, IX, and X) and contact factors (XI, XII, prekallikrein) are at 50% of normal adult values. By six months of age, values overlap with the normal adult range values, but the average of most factor levels is 20% lower than average adult levels until adolescence. FVIII levels are normal at birth, and levels of vWF are elevated until three months of age.

Low levels of *vitamin K dependent factors* and *contact factors*, FV, FVIII and fibrinogen between 19 and 30 weeks gestation result in prolonged values for PT, PTT and TT.

The *anticoagulants* antithrombin (AT) and heparin cofactor II (HCII) are at 50% of normal adult values at birth, normalizing to adult levels by three months of age. Proteins C and S are less than 50% of normal adult values at birth and reach adult levels by six months of age. On the other hand, anticoagulant α-2 macrogloblin values are higher than adult levels at birth and twice the adult levels at six months of age, not normalizing to adult levels until the third decade.

Developmental Hematopoiesis

Blood cell formation (hematopoiesis) is an ongoing self-renewing process. The life span of mature blood cells is short. The average circulating life span for mature red cells is 110 – 120 days, for neutrophils 5 – 6 days, and for platelets 5 – 9 days. Some memory lymphocyte cells survive for

years, but the majority of circulating lymphocytes survive for weeks. Hematopoietic stem cells replenish precursor and progenitor blood cells over a person's lifetime.

Initially, hematopoietic stem cells appear in the embryonic yolk sac at four weeks gestation; progenitor cells then migrate to the fetal liver for expansion and maturation. Stem cells are later produced in the aorta-gonad-mesonephros, major blood vessels, and the placenta. Fetal liver blood progenitor cells migrate to the thymus, spleen, and then to the bone marrow, where they mature under the influence of multiple hematopoietic growth and transcription factors. Extra embryonic hematopoiesis stops by 10 – 12 weeks gestation. From 20 – 24 weeks gestation, hematopoiesis occurs, primarily in the fetal liver. Bone marrow hematopoiesis gradually increases in the second trimester of pregnancy as liver hematopoiesis diminishes.

Red cell hemoglobin production evolves through a variety of forms, starting with the embryonic forms (Gower-1, Gower-2, Hb Portland) to fetal hemoglobin, and finally progressing to the adult forms (Hbg A and Hbg A2) during infancy. The majority of hemoglobin, a tetrameric protein containing four iron-containing units and four globin (protein) chains, will ultimately mature in composition to two α and two β chains manufactured in the bone marrow. Embryonic and fetal hemoglobin have higher oxygen affinity than hemoglobin A. This higher affinity for hemoglobin facilitates transplacental oxygen delivery.

Thrombopoiesis

Megakaryocyte progenitors (burst forming units and the more mature colony forming units) ultimately give rise to bone marrow megakaryocyte under the influence of thrombopoietin. Megakaryocytes undergo duplication without cell division (endoreplication). As a megakaryocyte matures, cytoplasmic pseudopods are formed. These pseudopods initially have thick membranes, which thin with maturity. When mature the cytoplasmic pseudopod fragments are released as platelets. Generally, circulating large platelets are the result of early megakaryocyte platelet release in response to thrombocytopenia.

TRANSFUSION

Blood is transfused based on blood type. The distribution of blood types varies worldwide. Red blood cells have surface antigens to which antibody may form. There are more than 400 known red cell antigens and 30 of these antigens are of major importance. Transfusion of donor red cell antigens to a recipient who does not have the antigen may elicit an antibody response. Each antigen is controlled by one gene. One gene is inherited from each parent. Antigenic determinants of a red cell group are produced by alleles at a single gene locus or at a locus so closely linked that crossing over is extremely rare. For any antigen of a group, a single allele is present at a locus, excluding other antigens. Antigens on red cells are usually determined by phenotype after mixing red cells with antigen specific commercial antisera and observing

hemagglutination. The number of antigenic determinants per erythrocyte and the ability to elicit an immune response varies from antigen to antigen.

ABO and Rhesus (Rh) Red Cell Antigen Groups

The two most significant red cell antigen systems are ABO and the Rh blood group. These two antigenic groups are the most immunogenic and generally illicit the most frequent and strongest antibody response in antigen negative individuals exposed to antigen. Transfusion of incompatible blood into a sensitized individual (previously exposure to the antigen with the production of IgG antibody) may result in antibody formation and a subsequent hemolytic transfusion reaction.

The *ABO blood group* consists of blood types A, B, AB and O and all individuals belong to one of these blood groups. Groups A and B represent carbohydrate antigens bound to surface glycoprotein and to some extent to membrane lipid on red cell and endothelial surfaces controlled by transferase enzymes conjugating either N-acetylglucosamine (A) or galactose (B) to a terminal fructose on the stem carbohydrate added by the H gene coding for a fucosyl transferase. The absence of H gene (hh) is rare and phenotypically termed O_h or Bombay type which results in high titer anti-H and no addition of either A or B carbohydrate despite the presence of A or B genes. Group O indicates the absence of both A and B genes. Consequently, neither the A nor B carbohydrate is present on erythrocytes or endothelial cells. AB individuals have both antigens. There are subgroups of A and B antigens, most of which are rare, with differences between subgroups quantitative, i.e., in the number of surface antigenic sites. Weak variants of group A may be difficult to detect with routine antisera. Antibody to group A and B are “naturally” occurring, meaning that they are thought to be stimulated by normal intestinal flora bacteria and plant and food proteins rather than by transfusion. A and B antibodies develop in the absence of antigen during the first 3-6 months of life, peak at age 5 – 10 years, and decrease with age and some immunodeficiency states.

Second in importance is the *Rh blood group*. Anti-Rh antibody is the leading cause of hemolytic disease of the newborn and an important cause of hemolytic transfusion reactions. There are more than 40 antigens in this group. Antigens are inherited as a gene complex set with three products, one codominant set from each parent. There are two nomenclatures for the Rh system: Fisher-Race and Weiner. The Fisher-Race system is now in more common use. In that system, there are five commonly produced antigens: C, c, E, e, D (no d has been identified). The other antigens in this system are minor variants. Individuals who are Rh negative (Rh^-) are those who do not have D. Individuals who are Rh^+ have the D antigen. Rh^- individuals are uncommon in most of the world but are present in roughly 15% of Caucasians. People with weaker forms of D (fewer antigens per red cell membrane) are designated as D^u. Transfusion of Rh^- individuals with D^u positive erythrocytes can cause sensitization.

Other Important Red Cell Antigen Groups

Of the remaining red cell antigens and groups, the four red cell groups most often implicated in hemolytic transfusion reactions are Kell, Duffy, Kidd and MNS. These reactions are generally delayed hemolytic reactions, often detected by the absence of an expected increase in hemoglobin/hematocrit after transfusion. For a red cell antigen to stimulate antibody production following transfusion, the antigen must be both immunogenic and prevalent. If an antigen is highly immunogenic but is low incidence, it is unlikely to be transfused. Hemolytic antibodies are IgG antibodies and react at 37^0C. Hemolysis is rare with IgM (cold reacting) antibody.

Kell system antigens are frequently associated with delayed hemolytic transfusion reactions. Originally described as K and k (more frequent) allelic pairs, now there are two additional allelic pairs and several variants identified. Kell antigens are highly immunogenic. One-in-20 recipients transfused with K^+ blood develop antibody. Antibodies to Kell cause hemolytic transfusion reactions, hemolytic disease of the newborn, and at times hemolytic anemia. The McLeod phenotype lack Kx, a precursor to Kell antigen, results in depressed expression of k. Those with the McLeod phenotype have erythrocyte and neuromuscular abnormalities. This phenotype has been associated with chronic granulomatous disease. *Duffy system* antigens (Fy^a, Fy^b) are codominent with Fy^a more frequently implicated in delayed hemolytic reactions. The Fy^{a-b-} phenotype, more common in people of African origin, provides resistance to *Plasmodium vivax* (not *P falciparium*) infection as Duffy antigens facilitate erythrocyte entry receptors for the parasite. The *Kidd system* comprises four genetic phenotypes: JK^{a+b-}, JK^{a-b+}, JK^{a+b+}, and JK^{a-b-} (found in some Pacific Island populations).

Pre-transfusion Testing of Red Cells

"Type and Screen": testing for both blood type and preformed red cell antibody in the blood recipient

Blood Type

The ABO and Rh type of all potential blood recipients must be completed prior to non-emergent transfusion of blood. This is accomplished by mixing recipient red cells with commercial anti-A, anti-B and anti-D. Red cell clumping indicates the presence of antigen on the red cell surface. The blood type is confirmed by mixing commercial A, B or D red cells with recipient serum or plasma (back typing). The likelihood of a hemolytic transfusion reaction transfusion with type specific blood is approximately 1:1000 units administered.

Antibody Screen

The recipient's serum or plasma is screened for antibodies, other than A, B or D, by mixing recipient serum with a set of three commercial O red cells containing the most common red cell

antigens. Antigen-antibody complexes form following either hemolysis or agglutination. The screen can be completed with the indirect antiglobulin test (IDAT) looking for agglutination. Should one of the screening cells react, an expanded red cell antigen panel is used to identify the antibody, delaying the availability of cross-matched blood. In laboratories without three cell antigen panels and expanded antibody identification panels, an alternative strategy is to test both donor and recipient red cells using commercial anti-sera with a single common red cell antibody (more stable than red cells) such as Kell, Duffy, Kidd antibody to lower the possibility of antibody sensitization.

Crossmatch

Red cells from the unit to be transfused are mixed and incubated with the intended recipient's serum or plasma prior to transfusing a unit of packed red cells. The method for accomplishing the cross-match depends upon the resources available and recipient circumstance. In the least complicated form, the cross-match can be made at the bedside using a clean glass or ceramic plate. In environments with more abundant resources, red cells are washed and incubated with various reagents, which promote interaction between antigen and antibody and fixation of complement.

Blood intended for use in the crossmatch should be collected no longer than three days prior to the intended transfusion unless it is certain the patient has not been pregnant or transfused within the preceding three months. The crossmatch methodology varies with circumstance and available resources. In an emergency, donor type O negative packed red cells or donor packed cells known to be of the recipient's blood type ("type specific") can be transfused and the crossmatch performed retrospectively. The most simple, but least sensitive crossmatch, consists of mixing donor segment red cells with recipient serum/plasma on a clean non-reactive surface and observing agglutination. The routine tube crossmatch employs mixing donor cells in saline with recipient serum/plasma and recording hemolysis or agglutination at room temperature, after incubation at 37°C and after addition of antihuman globulin. Various reagents (albumin, low ionic strength saline, polyethylene glycol, etc.) can be added to enhance reactions.

Table 4-2: ABO Compatibility

Donor Red Cells	Recipient Blood Type
O (universal donor)	O, A, B, AB
A	A, AB (B and O have anti-A)
B	B, AB (A and O have anti-B)
AB	AB (A, B, O have anti-A or B or both)

Generally, donor red cells of the same type as the recipient are transfused. However, it is not necessary for donor red cells to be of the same blood type as the recipient provided that the blood types are compatible with one another. For example, blood type O which has neither A nor

B antigen can be transfused as packed red cells to any recipient. For this reason, O red cells are considered to be from *"universal" donors* as long as the crossmatch is non-reactive for other red cell antibodies. Recipients who are type A can receive either A or O red cells. Type B recipients can receive either B or O blood and AB recipients can receive either O or AB red cells that are crossmatch compatible (**Table 4-2**).

Compatibility testing for infants less than four months of age

Infant red cells must be tested for ABO and Rh type pretransfusion. Serum/plasma from either the infant or the mother may be used to detect unexpected antibodies and for crossmatch. Infant serum/plasma need not be tested for antibody when O cells are to be transfused. In the absence of an unexpected antibody, donor red cells need not be crossmatched. Repeat infant testing can be eliminated during any single hospital admission.

Check Specimens, Immediate-Spin, and Computer Generated Crossmatch

The vast majority of ABO compatible packed red cell units are crossmatch compatible, provided the patient has not been pregnant or transfused previously. The majority of hemolytic transfusion reactions are due to ABO incompatible transfusions resulting from clerical errors, such as recipient blood samples labeled with the incorrect patient name or misidentification of the recipient at the time of transfusion.

Many transfusion services require two separate ABO type tests prior to releasing blood or blood components from the Blood Bank. One sample collected at the time of the "Type and Screen" and a second consisting either of a record from previous ABO testing in the same Blood Bank or the testing of a second sample (i.e. the "check specimen"). This procedure markedly reduces the number of major hemolytic transfusion reactions.

Transfusion Services can employ an "*immediate-spin" crossmatch* for recipients who do not have previous or current clinically significant antibodies. Saline suspended washed donor segment red cells are mixed with recipient serum/plasma at room temperature and the tube centrifuged immediately. This method is employed to detect ABO incompatibility.

A *computer-generated crossmatch* is an electronically generated match between the recipient and the red cell unit and/or other blood component that matches the ABO and Rh of inventory donor blood and the confirmed recipient blood type. Recipients without clinically significant antibodies by testing or history and without recent transfusion are suitable for computer-generated crossmatch. However, a number of rigorous on site requirements for computer system validation are required. This practice is increasingly adopted by large blood services with sophisticated laboratory and transfusion service computer programs.

Table 4-3: Transfusion Reactions

Transfusion Reactions		
RBC	**WBC**	**Plasma Proteins**
Immediate (intravascular)	Febrile	Allergic
Delayed (extravasvcular)	TRALI (transfusion, other)	Anaphylactic (IgA, other)
	GVHD	
	Alloimmunization	

Although increasingly safe, reactions to blood transfusions continue to occur. Each transfusion must to be monitored during and following infusion. It is important that vital signs be obtained and recorded before transfusion, at 30-minute intervals during transfusion and 30 minutes after transfusion. Reactions to transfusion must to be reported to the transfusion service and reviewed by the hospital Transfusion Committee.

Transfusion reactions are either immune-mediated or non-immune mediated. Reactions can be due to recipient antibody to donor red cells, reactions to white cells, platelets or plasma. Preventable reactions are sepsis due to blood component bacterial contamination (most frequently platelets) and fluid overload (transfusion associated cardiac overload or TACO). Sepsis and TACO are the most common adverse outcomes of transfusion.

Red Cell Reactions

Hemolytic reactions following transfusion of red cells can be immediate or delayed. Immediate reactions are intravascular and are due to IgG antibody to donor red cells. These reactions are fatal in 10% of ABO incompatible transfusions and generally occur following large volume infusion of incompatible blood as a result of clerical error. Clerical error is the consequence of mislabeling the blood specimen used for crossmatch with the wrong patient name or not matching the name on the unit of cells or blood to be transfused with the recipient at the time the transfusion is initiated. Immediate hemolytic transfusion reactions occur in less than 0.02% of red cell transfusions. Fever, chills, and burning at the infusion site herald a reaction. Hypotension, dyspnea, sometimes back or joint pain, and then hematuria follow. These reactions can proceed to cardiovascular collapse, disseminated intravascular coagulation and renal failure. The transfusion should be stopped at the first sign of a hemolytic transfusion. The remainder of the red cell unit and a blood and urine sample should be sent to the laboratory for testing. The unit will be examined for signs of bacterial contamination, a repeat crossmatch, and a *direct Coombs test* will be performed. If a hemolytic transfusion reaction has occurred, recipient antibody will coat transfused donor cells. Anti-Ig is added to recipient red cells. Following incubation, red cell agglutination occurs in a positive direct Coombs test. The recipient blood sample can be tested

for haptoglobin, which will be cleared from the blood with hemolysis, and the patient's urine will be positive for free hemoglobin.

Delayed hemolytic transfusion reactions are extravascular, occurring in the liver and spleen. These reactions occur 3-10 days after transfusion. A delayed hemolytic transfusion reaction is suspect in blood product recipients with or without fever in which the appropriate increase in hemoglobin is not observed following transfusion. This reaction is detected with a positive direct Coombs test and elevated bilirubin levels. They are the consequence of primary immunization to red cell antigens or are due to an anamnestic response in a previously sensitized recipient with undetectable antibody levels at the time of pre-transfusion testing. These reactions are thought to occur once in every 6,000 red cell transfusions but are often undetected.

Inadvertently overheating or freezing donor blood or the shear stress caused by rapid transfusion of donor red cells through small-bore needles can artificially produce red cell destruction. This has lead to hyperkalemia, lack of predicted hematocrit elevation, and hematuria.

Leukocyte Reactions

There are several types of leukocyte reactions and reactions to leukocyte produced cytokines within the donor unit after collection. The frequency of adverse leukocyte reactions and the transmission of leukocyte associated viruses, such as cytomegalovirus, declines markedly when blood is leukoreduced (leukocytes are removed) at the time of collection or at the time of transfusion. The most common leukocyte reaction is febrile non-hemolytic transfusion reaction (FNHTR), occurring in 1-2% of non-leukoreduced red cells and platelet transfusions. These reactions are characterized by fever ($\geq 1^{o}C$) 30 – 60 minutes following transfusion and often accompanied by chills. The reactions are generally self-limiting with fevers of 38 - $39^{o}C$ and must be distinguished from the fever associated with a hemolytic transfusion reaction. Bacterially contaminated blood components commonly present with high fever ($\geq 40^{o}C$) and rigors. Only one in eight recipients will have a repeat febrile reaction during the following transfusion. These reactions most commonly occur in multiply transfused recipients and in multiparous women who were leukocyte sensitized during past pregnancies. Recipients with recurrent febrile reactions can benefit from pre-treatment with antipyretic drugs.

Transfusion associated acute lung injury (TRALI) is a rapid onset pulmonary edema of generally 24 – 48 hours duration. It is uncommon for TRALI to be fatal. High-titer anti-leukocyte antibody, usually in the donor plasma to recipient leukocytes, cause leukocyte aggregates that are filtered in the lungs and lodge in pulmonary capillaries. Complement activation results in endothelial damage, flow of intravascular fluid into alveoli, and pulmonary edema. Recipient leukocyte antibodies to donor leukocytes can also cause TRALI. All plasma containing blood components have been implicated in TRALI, but fresh frozen plasma is most frequently associated with it. For that reason, there has been a shift towards only using male plasma for production of fresh frozen

plasma, reducing the incidence of TRALI in both Europe and the United States. Female blood donors who have been pregnant can make white cell antibodies and HLA antibodies to white cells of their fetus. Females with a history of multiple pregnancies have been most frequently implicated in cases of TRALI.

Transfusion associated graft versus host disease (TA-GVHD) is a rapidly fatal form of GVHD following transfusion of HLA haplo-compatible lymphocytes between first-degree relatives. Designated donations between first-degree relatives are gamma irradiated (25 gray, Gy, to the central portion of the blood bag and no less than 15 Gy to the outer portions), inhibiting T lymphocyte blastogenesis and engraftment. Irradiation damages red cell membranes with consequent increases in plasma potassium over time and decreases post-transfusion cell viability. For this reason, red cell units expire at their original outdate or 28 days from the time of irradiation, whichever comes first. Blood is generally irradiated immediately prior to transfusion. Platelets expiration dates are not affected since platelets are relatively resistant to irradiation. TA-GVHD can also occur in some immunocompromised patients triggering pre-transfusion irradiation. Blood transfused during intrauterine transfusion or transfused to infants $\leq$1500 grams and to severely immunocompromised children (congenital cellular immunodeficiency disorders, children undergoing bone marrow or progenitor cell transplant, treatment for hematologic malignancies, Hodgkin's disease or cancer patients undergoing intense chemotherapy) is irradiated to prevent GVHD.

Cytomegalovirus (CMV) seronegative children at risk of severe CMV infection should be transfused with CMV minimal risk blood. Latent CMV infection of leukocytes follows primary infection with periods of reactivation. CMV infection in healthy individuals is of little consequence, but infection can be serious and even fatal in the imunocompromised. Transfusion associated CMV is most often transmitted by viremic acutely infected otherwise healthy, asymptomatic blood donors prior to specific CMV antibody formation. Although not perfect, leukoreduced blood ($<5 \times 10^6$ WBC) or CMV seronegative blood rarely transmits CMV. Previously frozen blood components do not transmit CMV. CMV reduced risk blood is indicated for recipients of intrauterine transfusions, premature infants <1500gms, and for seronegative children with congenital immunodeficiency disorders, AIDS, and for bone marrow, progenitor cell, and organ allograft recipients, as well as for cancer patients undergoing intense chemotherapy.

Allergic Reactions

Allergic reactions are the most frequent type of transfusion reaction, occurring in 1-3% of recipients of plasma containing components. Allergic reactions are more common with platelets (3.7%) than with red blood cells (0.15%). Most reactions consist of hives, itching, and local erythema. These reactions can, however, proceed to bronchospasm and rarely to anaphylaxis. When an allergic reaction is detected, the transfusion should be stopped until the severity of the

reaction is determined. Mild reactions respond to antihistamines. The transfusion can be continued when the hives abate. Reduced plasma or washed red cells/platelets can be transfused in patients with recurrent allergic reactions uncontrolled with antihistamines. While washed components are more effective in preventing reactions than reduced plasma volume components, the volume of transfused cells is also reduced and the quality of platelets may not be the same. There is no evidence that pretreatment with prophylactic antihistamine prevents allergic reactions.

Plasma proteins are often implicated as the cause of allergic reactions and rarely cause anaphylaxis. These reactions were first described when normal donor blood was transfused into IgA deficient patients with anti-IgA and later in haptoglobin deficient patients making haptoglobin specific antibody. It is now also understood to occur in patients with subclass and allotype variations who have been transfused and formed antibodies. There are racial differences in the incidence of IgA and haptoglobin deficiency. The incidence of haptoglobin deficiency is 1-in-1,000 – 1-in-4,000 in East and Southeast Asians but very uncommon in Africans, Europeans and western or eastern Asians. On the other hand, the incidence of IgA deficiency in Europeans is 1-in-2,500 and 1-in-30,000 in the Japanese. Also described are anaphylactic reactions to complement C4 in deficient individuals and to Factor IX in hemophiliacs with Factor IX inhibitors.

Chemically induced anaphylaxis is less common and is described as caused by methylene blue added to fresh frozen plasma for viral inactivation. Also reported is anaphylaxis precipitated by donor derived food allergens, such as peanuts, transferred to a sensitized individual. Subpathways to anaphylaxis, not evolving IgG or IgE, are described. *Allergen-independent mechanisms* caused by biological response modifiers (BRMS), such as inflammatory cytokines and chemokines accumulating in blood components during storage, can cause allergic reactions. Passive transfer of sensitization has been described from donor-derived allergy, in one case to cepholthin and in another to peanuts.

Two laboratory tests are used to determine occurrence of an allergic reaction. Elevated levels of serum or *plasma tryptase* indicate mast cells activation and degranulation in allergic and in the more severe form of anaphylactic reactions. Secondly, there is interest in applying the *basophil activation test (BAT)* to transfusion medicine to detect allergic reactions. In this test, patient's blood is incubated with the allergen in question, tested for upregulation of the basophil activation markers, CD63 and CD203c, using flow cytometry.

Alloimunization

Recipient antibody can be formed following transfusion of red cells, white cells, and platelets. Red cell antibodies complicate the ability to provide crossmatch compatible blood, and they add additional costs and delay the delivery of blood. White cell antibodies are a cause of TRALI. HLA and platelet specific antibodies in platelet recipients rapidly destroy transfused platelets leaving

some patients vulnerable to fatal hemorrhage. Factors that determine antibody formation are poorly understood. Antigenic differences between donors and recipient, number of transfusions, genetic predisposition, and an underlying inflammatory state may be of importance.

Antibodies to red cell antigens are not common in the general population and are estimated to occur in between 0.5 – 1.5% of individuals. These antibodies are formed during pregnancy or following transfusion of red cells. When sensitive methods for antibody detection are used, 8.4% of patients receiving a mean of three RBC units form alloantibodies. In contrast, individuals with diseases such as sickle cell disease (SCD) or some forms of thalassemia, who may require lifelong RBC transfusion, develop RBC alloantibody at a much higher rate (18 – 76%) and face difficulty and delay in finding compatible blood. Several strategies to limit the prevalence of RBC alloimmunization in these patients are in use. In addition to ABO and D matching, limited RBC phenotype matching and transfusion with blood from C, E and K similar donors result in 5 – 14.5% of patients forming alloantibody. Antibody to C, E and K comprise two thirds of the antibodies found in SCD. Extended RBC matching beyond C, E and K antigens can be even more successful with 7% of transfused patients forming alloantibody.

Antibodies to human leukocyte antigens (HLA) are responsible for a number of different transfusion reactions: FNHTRs, TRALI, and TA-GVHD (see above). Refractoriness to random platelet transfusions occurs in 30-50% of platelet dependent patients. Immune mediated platelet refractoriness is primarily due to HLA class I antibodies and occasionally due to anti-platelet specific antibodies or high titer ABO antibody. Non-immune factors may contribute to a lack of an adequate platelet increase following transfusion, such as poorly stored platelets, sepsis, disseminated intravascular coagulation, and certain drugs, such as amphotericin B and ciprofloxacin. Leukoreduction of donated blood, best done at the time of blood collection (but can also be done prior to transfusion) decreases alloimmunization and adverse leukocyte reactions.

Neonatal alloimmune thrombocytopenia (NAIT), although not a transfusion reaction, is the result to maternal alloimmunization against paternally inherited platelet antigen that is similar to red cell antibody seen in *hemolytic disease of the newborn (HDFN)*. Contrary to HDFN, NAIT can occur with the first pregnancy. IgG antibody is transplacentally transferred to the fetus with destruction of fetal platelets. Antibody is most often (80%) directed against platelet antigen-1a (HPA-1a). In Asian populations, HPA-1a is very rare and anti-HPA-4b is the most common cause of NAIT. About 20% of infants born to women with anti-HPA-1a will have severe thrombocytopenia and clinically manifest signs of bleeding, such as petechiae, purpura, or mucocutaneous bleeding. The risk of intracranial hemorrhage is 1 – 14% in severely affected infants. Maternal antibody clears from the infant in 1-3 weeks. NAIT can be treated in utero by giving IVIG and dexamethasone to the mother; it can be treated after birth with IVIG and/or platelet transfusion to the infant.

Immunomodulation by viable white cells from blood transfusion is poorly understood and the mechanism causing it is unknown. Blood transfusion prior to renal transplantation decreases allograft rejection and has a beneficial effect in women with recurrent abortion. Although controversial, blood transfusion is reported to decrease survival in patients with cancer and to increase infections in patients undergoing major abdominal, cardiac, and orthopedic surgery.

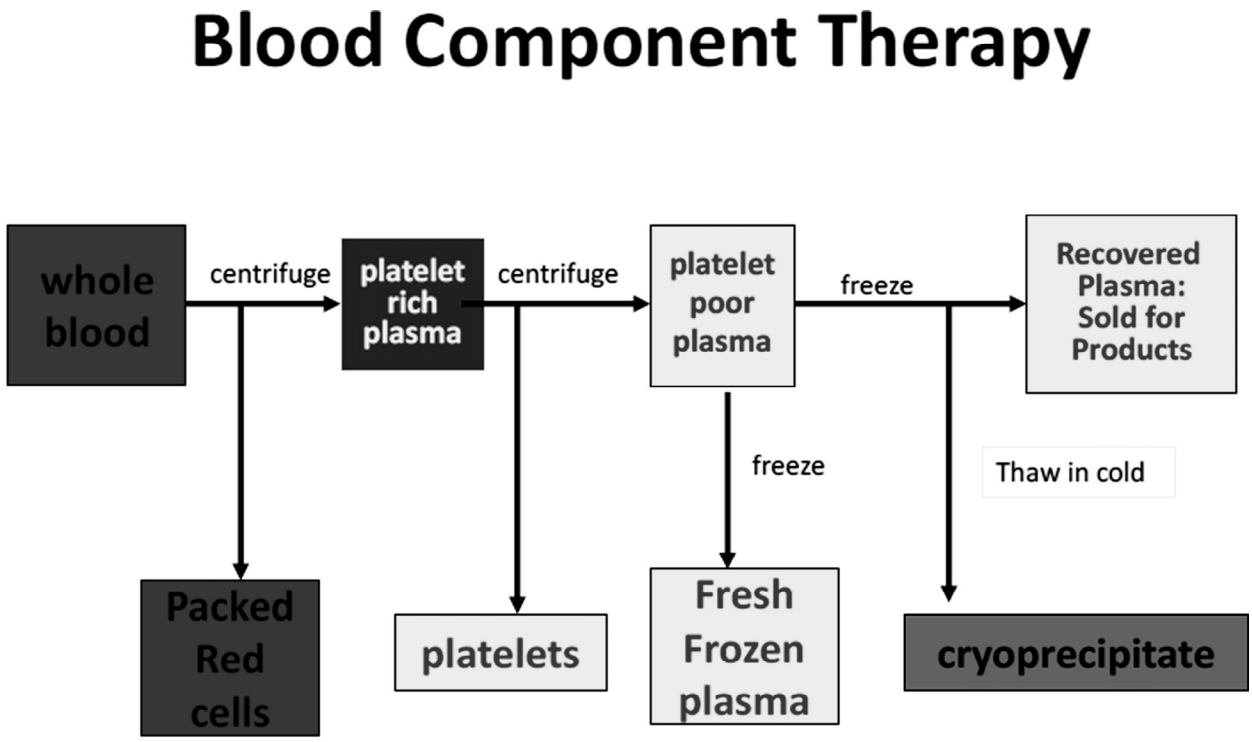
Transfusion Transmitted Infection

Great strides have been made in improving the safety of the blood transfusion. The risk of acquiring an infectious agent remains a constant threat to transfusion recipients and requires ongoing vigilance by practitioners. The risk of infection may vary by season for some organisms, such as West Nile virus, babesiosis and dengue, by the geographic prevalence for other microorganisms such as HIV, HTLV, hepatitis viruses and Chagas disease, or by methods of component collection, processing and storage, such as the majority of bacterial contamination. Progressive interventions have improved the safety of the blood supply beginning with testing for syphilis in late 1930, collection and storage of blood in integral system plastic containers, cold storage of components other than platelets, and improved donor screening and application of an expanding number tests to the blood collected. The delayed response to hepatitis and HIV transmission via blood and blood derivatives rightfully resulted in a hypersensitivity to disease transmission by blood transfusion. At this point, however, the cost of applying an increasing number of tests balanced with the decreasing benefits of those tests has been called into question.

Clearly, resources need to be balanced with risks. At a minimum, the World Health Organization (WHO) recommends all blood donations be screened for HIV-1 and HIV-2 antigen-antibody or antibody alone, hepatitis B surface antigen, hepatitis C antigen-antibody, or antibody alone and for syphilis (*T pallidum* antibody). Also, blood donations should be collected from volunteer, non-paid donors, preferably from repeat donors who are less likely to transmit infection. Other screening tests for infection, such as malaria, Chagas disease, or HTLV, should be based on local epidemiological evidence.

The United States has developed a number of different screening strategies, depending on the transmissible agent and the risk of transmission. A sample from each blood donation is pooled in batches and tested for HIV 1/2 and hepatitis B and hepatitis C viral nucleic acid. Individual nucleic acid testing for West Nile virus, a seasonal infection, is triggered based on the positivity of pool testing. Specific antibody testing is employed on each donation for HTLV-I/II, hepatitis B core, hepatitis C, HIV-1/2 and syphilis. Antigen testing is utilized for hepatitis B surface antigen. Geographic exposure, using donor history, triggers temporary donor deferral for malaria but may be indefinite as is the case for variant Creutzfeldt-Jacob disease. Once-in-a-lifetime donor testing is utilized for *T. cruzi* and sometimes for cytomegalovirus antibody.

Figure 4-5: Blood Component Therapy



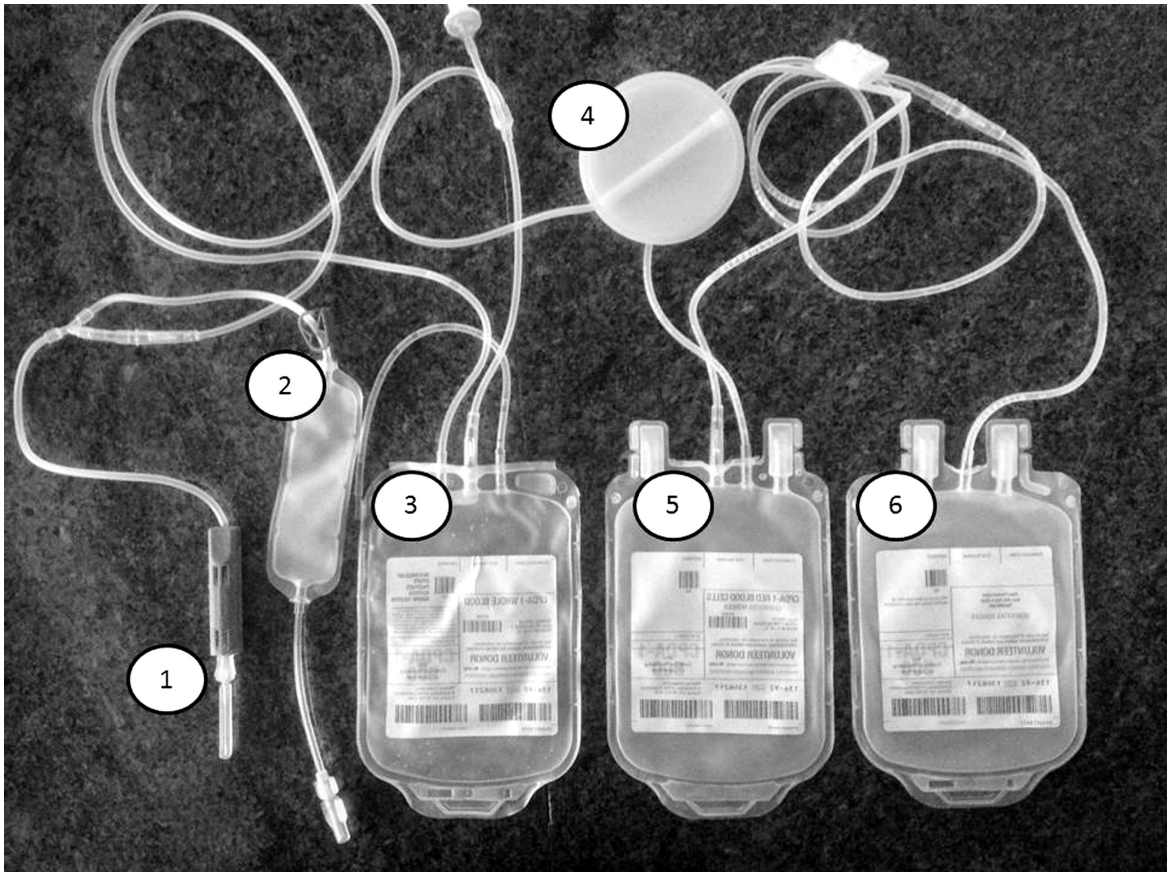
Overview of the blood fractionation process

Blood Collection and Transfusion

Using sterile technique, whole blood is collected in a closed system with a hollow bore needle leading to a sterile plastic collection bag that contains a fixed amount of preservative. Collection systems with a variable number of attached satellite bags are available, depending on the planned number and type of derivative blood components to be produced (**Picture 4-1a**). Generally, the main collection bag is a 500 ml capacity bag with a suitable amount of preservative, which necessitates a donor weigh of at least 110 lbs., since no more than 10% of the donor's blood volume may be collected at one time. Smaller capacity collection bag systems (such as 250 ml or 350 ml) with suitably reduced amounts of preservative are available for donors who weigh less than 110 lbs.

Each whole blood unit can be transfused as whole blood or fractionated. A single whole blood unit can be fractionated into packed red cells, a platelet concentrate and either a unit of fresh frozen plasma or cryoprecipitate. Using the proper collection system, cryoprecipitate is produced using a refrigerated slow thaw technique, centrifuging, and then discarding or selling the supernatant (recovered plasma).

Picture 4-1a: A Collection System for Red Cells, Fresh Frozen Plasma, or cPyoprecipitate



1. *Hollow bore 16 gauge collection needle*
2. *In-line 10 ml bag for infectious disease testing*
3. *Main collection bag with CPDA-1 (whole blood)*
4. *In-line white cell filter*
5. *Satellite bag for leukoreduced packed red cells*
6. *Satellite bag for leukoreduced fresh frozen plasma*

The use of whole blood transfusions are discouraged by blood services able to fractionate blood into components because refrigerated blood looses platelet activity within 24-48 hours and coagulation factors begin to decline. After one week, 50% of factor V and VIII is lost in refrigerated plasma. Packed red blood cells (pRBCs) are suspended in thawed fresh frozen plasma (FFP) when needed for exchange transfusions.

Red Blood Cells

After whole blood is centrifuged, the platelet rich plasma layer is expressed into a pilot bag, leaving pRBCs in the primary collection bag (**Figure 4-5**). The tubing is sealed and the pRBCs refrigerated. RBCs may also be obtained by apheresis. RBCs should be stored at 1^0C. Shelf life depends on the preservatives and additive solutions. Citrate-phosphate-dextrose (CPD) and citrate-phosphate-dextrose-dextrose (CP2D) used as anticoagulants allow blood storage for 21 days (70% viability in the recipient at 24 hours). Citrate-phosphate-dextrose-adenine (CPDA) can extend shelf life to 35 days. Adenine saline (AS) solutions may further increase shelf life to 42 days. Coolers used to transport RBCs should maintain a temperature of 1-10 ^{0}C. RBCs should only be transfused with 0.9% normal saline or plasmalyte. Lactated Ringers (LR) and hypotonic solutions, such as D5W and 0.45% normal saline (NS), should not be used as carrier fluids for transfusions. All red cell transfusions must be filtered with a minimum filter size of 170 – 200 microns.

Low volume red cell transfusions for intrauterine and repeated low volume neonatal transfusions present unique difficulties. The red cells to be transfused are usually O^-, CMV negative, and irradiated. Often only 10 -15 ml of red cells are required. Exposure to multiple donors is not desirable and sterility of the unit must be maintained for each transfusion. Several strategies are in use. Large institutions with multiple neonates requiring transfusion employ four to six aliquot bag collection units attached to the pRBC unit. If the satellite packed red cell unit is entered using a spike attached to the aliquot bags (**Picture 4-1b**), then the unit expires in 24 hours. If, however, the red cell unit is attached using a sterile docking device (**Picture 4-1c**), then the aliquots expire on the original red cell expiration date. More than one child is assigned to a single donor unit. Other hospitals sterilely draw blood from the pRBC unit, using a syringe. They filter the blood either at the time of withdrawal or at the time of transfusion in the neonatal intensive care unit. In the past, “walking donors” were used, but now the risk of infection using this technique is considered too high. However, in some low-income countries walking donor donations are still used as a source of blood, especially in an emergency. This blood has higher levels of coagulation factors and platelets.

Picture 4-1b and 4-1c: Aliquot Bags for Small Volume (Infant) Red Cell Transfusions

Picture 5-1b

1. *Packed red cell satellite bag*
2. *Spike attached to four aliquot bags*

Picture 5-1c

Sterile docking device

Red cells are transfused to increase oxygen-carrying capacity in patients with anemia due to infection (malaria, dengue fever) with or without severe malnutrition, congenital hemoglobin disorders (sickle cell disease, thalassemia, other hemoglobinopathies), iron deficiency, or acute blood loss. Red cell transfusion should be avoided in non-emergent situations when other appropriate therapies are available, such as oral iron for iron deficiency. The child's hemoglobin/hematocrit and signs/symptoms of anemia and the rapidity of blood loss guide the appropriateness of red cell transfusion. Generally, in otherwise healthy children, acute loss of more than a 15 - 20% of their blood volume is needed before transfusion is initiated. Usually a hemoglobin (Hbg) of 6-7g/dl is well tolerated. Pallor, hypotension, tachycardia, and mental status guide the need for transfusion in the absence of a reliable measure of hemoglobin. Children with cyanotic cardiovascular disease may require a higher hemoglobin concentration. Studies in pediatric intensive care units support a transfusion trigger of 7g/dl in stable critically ill children, including children with non-cyanotic cardiac malformations. This restrictive transfusion trigger has also been repeated in pediatric patients with sepsis and postoperative general and cardiac surgery settings with similar results. A higher threshold of 9g/dl for children with cyanotic heart malformations is indicated. Children with chronic anemia, such as the hemolytic anemia of malaria, can tolerate an Hbg of 4.5 – 5g/dl or lower before transfusion is necessary.

Hemoglobin triggers for transfusion in neonatal ICU depend on age and clinical status of the patient (need for respiratory support or mechanical ventilation, hemodynamic instability and vasopressor requirements, presence of active bleeding, cardiac abnormalities). Most published guidelines are based on expert opinion and not clinical studies. Risks and benefits should be weighed before transfusion.

Low potassium or washed PRBC units may be indicated in patients who require ECMO, cardiopulmonary bypass (CPB), or who have poor renal function, until their hyperkalemia resolves. Washing is a process using 0.9% normal saline to remove unwanted plasma proteins and electrolytes. Washed RBCs may also be considered in pediatric patients with an unexplained severe or anaphylactic response to prior transfusion. The shelf life for RBCs after washing is 24 hours. Washing damages the cells. As many as one third of the red cells are lost during the washing process.

Hemoglobin/hematocrit transfusion increments are difficult to determine and several methods are used. Three common methods are:

1) 10-15ml of red cells/kg body weight are thought to increase the hemoglobin by 3gm/dl
2) A formula for *maximum allowable blood loss (MABL)* can be used to guide transfusion.

   Volume of pRBC transfused = Total Blood Volume x (desired Hgb – actual Hgb)/ pRBC Hgb.

   Total Blood Volume (TBV): pre-term neonates: 90 – 100ml/kg; full-term neonates: 80 - 90ml/kg; 6mos – 2 years: 80ml/kg; >2 years: 70ml/kg

3) Volume of pRBCs to be transfused = 4.8 x weight (kg) x desired rise in Hgb (gm/dl)

Platelets

Whole blood derived platelet concentrates are prepared from expressed platelet rich plasma that is not cooled below 20^{o} C and is processed within eight hours of blood collection (**Figure 4-5**). The platelet rich plasma pilot bag is centrifuged and the surface platelets expressed into a second pilot bag. Platelets from multiple sources can be pooled at the blood center prior to transfusion. Apheresis platelets are obtained from one donor, limiting donor exposures, the possibility of alloimmunization, and infectious disease transmission, at an added expense. One pheresis unit is equivalent to six single donor platelet concentrates. Platelet pheresis is often divided in half (3x 10^{11} platelets) for pediatric platelet transfusion. Sterility while obtaining and preparing platelets is vital, given the high risk of bacterial contamination. Platelets may only be stored for five days at 20-24^{0}C, and they must be constantly agitation.

Prophylactic platelet transfusions can be considered for stable premature infants at a platelet count of <30,000 and at <50,000 for more unstable premature infants. Term infants <4 months old should be prophylactically transfused at <20,000 and at <10,000 if a term infants is older than four months. Patients who are scheduled for procedures having a risk for bleeding should be transfused when platelet counts are <50,000. Traditional dosages of platelets are approximately 10-15 ml/kg in the neonatal population with the expectation of a 6 x 10^{3} increase in platelet count. Cross match and ABO compatibility are normally not needed prior to platelet transfusion, but transfusing platelets for patients with the same ABO type may have advantages. Lack of response to platelet transfusion may indicate refractoriness. HLA-matched or cross-matched

platelets may be used in this situation. Residual RBCs may also remain in platelet units, especially when using whole blood derived platelets.

In actively bleeding patients, platelet transfusions may be indicated when counts are <50,000. Platelets can be transfused regardless of count in actively bleeding patients with a qualitative platelet abnormality. In more specialized situations, such as diffuse bleeding related to CPB or ECMO, platelet transfusion may be required if counts are <100,000.

Platelets are issued as the most compatible blood type available but are not crossmatched. The more compatible the blood type, the longer the platelets will circulate in the patient's blood. Platelets concentrates can be "volume reduced" to remove the majority of the plasma for patients in whom volume overload is problematic. Platelets can be "washed" to remove leukocytes and cytokines accumulated during storage as well as chemokines to decrease FNHTR where leukodepletion is not available. However, platelets are lost with washing, and the concentrate expires 24 hours after washing. Platelets must be filtered prior to transfusion.

Fresh Frozen Plasma

Fresh frozen plasma (FFP) can be obtained by apheresis or from whole blood after platelets are removed from platelet rich plasma (**Figure 4-5**). Following this process, the plasma is frozen at -18^{0}C within 8 hours and may be stored in this state for one year. Once needed for transfusion, FFP is warmed at 30-37^{0}C. This thawed plasma is stored at 1-6^{0}C for up to 24 hours. If not transfused within 24 hours, it may be refrozen and thawed at a later date for use (FFP-24).

FFP is transfused to provide coagulation factors in bleeding patients when the PT/PTT is 1.5 times greater than the mid-point of the normal range. This indicates that less than 30% of the normal amount of a coagulation factor is present, which is the point at which bleeding can occur in the face of a normal level of platelets. The International Normalized Ratio (INR), developed to standardize PT measurements within laboratories using tissue thromboplastin of variable strength, approximates this point within the usual clinical range for the PT. Importantly, the INR of FFP itself is approximately 1.2-1.5.

FFP transfusion may be considered when INR is greater than 1.5 to 2.0 in a non-bleeding patient scheduled for an invasive procedure or when INR >1.5 in diffusely bleeding patients. FFP is also indicated in patients who have a warfarin overdose and will undergo major surgery or an invasive procedure. Other indications include patients with TTP undergoing transfusion or plasma exchange, protein C, protein S, AT deficiencies with heparin resistance, C1 esterase inhibitor or other single factor deficiency (with the exception of Factor IX - where it is relatively ineffective) when no product is available and a patient is bleeding. FFP may also be used to treat bleeding secondary to vitamin K deficiency. FFP should not be used for volume expansion or for ITP. FFP must be filtered with a large pore filter (150-170 micron). The dosing is approximately 10-15ml/kg in pediatrics.

Cryoprecipitate

Cryoprecipitate is a source of factor VIII, fibrinogen, von Willebrand factor (vWF), factor XIII, and fibronectin. It is quality controlled to contain at least 80 IU (international units) of factor VIII and >150mg fibrinogen per concentrate. It is the milky white precipitate that forms when FFP is thawed at 4^{o}C and remains in small volume following centrifugation and removal of the surface plasma (now termed recovered plasma). It is commonly pooled from multiple units prior to storage. Cryoprecipitate may be stored for one year at -18^{o}C. Once thawed at 30-37^{0}C, it may be stored for six hours at 20-24^{0}C.

First developed as a source of factor VIII and used in pools for small children who could not tolerate the volume load of FFP, it is used in the operating room as a source of fibrinogen where fibrinogen concentrate is not available or affordable. The amount of fibrinogen in one concentrate is roughly equivalent to the amount of fibrinogen contained in the one unit of FFP from which it is made. Cryoprecipitate is indicated when fibrinogen levels are <100mg/dl and an invasive procedure is scheduled, or for patients with qualitative fibrinogen disorders experiencing diffuse bleeding or undergoing a scheduled procedure with the potential for blood loss. It may also be used in von Willebrand disease or factor VIII deficiency when there is active bleeding; it may also be used for scheduled procedures when the patient is unresponsive to DDAVP or factor concentrates. Cryoprecipitate may be transfused without compatibility testing, though testing may be the preferred method by some practitioners. Cryoprecipitate is dosed at one concentrate per 10 kg body weight in pediatrics.

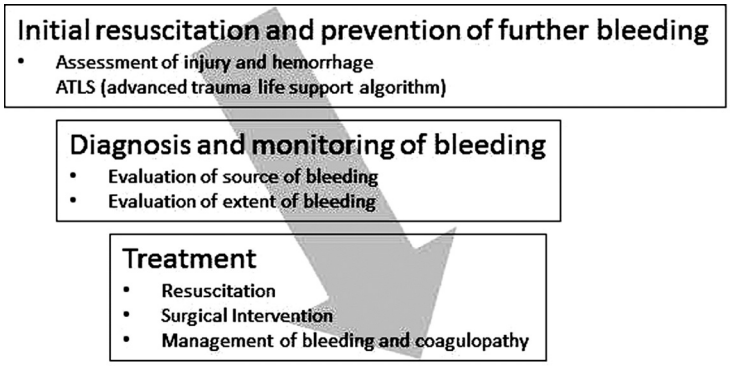
Resuscitation of Acute Surgical Bleeding and Trauma

Massive bleeding resulting from trauma or acute, active surgical bleeding may produce shock via hypovolemia and decreased cardiac output, with the end result being decreased oxygen delivery to end organs. Metabolic acidosis, inflammation, and signs of hypovolemia and hypoperfusion often accompany this presentation. Hypothermia is common during active resuscitation, especially in trauma situations, from environmental exposure, use of unwarmed intravenous (IV) fluid, and disruption of thermoregulatory mechanisms. Acute coagulopathy related to shock, hypoperfusion, and hemodilution follow. Combined, acidosis, hypothermia, and coagulopathy form what is known as the “lethal triad” or “triad of death,” and thus need to be corrected rapidly and aggressively. The primary treatment of hemorrhage is surgical hemostasis and fluid resuscitation.

With initial management of bleeding (Figure **4-6)**, large bore IV access is obtained and crystalloid is infused in acutely unstable patients while avoiding hemodilution, which may further decrease oxygen carrying capacity, oxygen delivery, and coagulation factors. Blood samples should be sent for type, screen, and crossmatch when time allows. If the patient is in emergent need of blood, O negative blood should be transfused without testing. If O negative blood is not available, type

specific blood may be transfused in emergent scenarios. Crossmatched blood should always be transfused first when available. Refer to the above *hemoglobin/hematocrit transfusion increments.*

Figure 4-6: General Algorithm for Management of Acute Bleeding Following Trauma



General algorithm for evaluation and treatment of trauma patient: Each step is part of a continuum, which may be performed simultaneously during resuscitation.

Massive Transfusion Protocols

In some institutions, massive transfusion protocols (MTP) exist and may be activated when faced with active and massive bleeding. Massive transfusion has been defined many ways in the literature, but a common definition in adults is transfusion of 10 units in 24 hours or replacement of one blood volume. In pediatrics, it has been defined as transfusion of greater than 40 ml/kg in the first 24 hours. The principles of MTP involve clinician activation. Type and screen and a second confirmatory specimen are usually required. Emergent uncrossmatched O negative blood is immediately released. MTP "packs" made up of crossmatched PRBCs, FFP, and platelets are then created. Cryoprecipitate is sometimes included as well. In some pediatric protocols, these packs are based on weight. The blood bank stays in close contact with the clinical team in order to determine the ongoing need to provide "packs" of blood products. The protocol is discontinued at the clinician's discretion.

Transfusion of PRBCs, FFP, and platelets in proportions similar to whole blood is used to prevent dilutional coagulopathy. The ideal ratio of PRBC to FFP is controversial. Commonly ratios of 1:1-to-1: 2.4 are used and have been shown to improve outcomes in retrospective studies on adults. Pediatric massive transfusion data is more limited and further studies need to be done for validation in this patient population. Calcium repletion is needed due to citrate in the storage bags. Ideally regular laboratory studies, including blood gasses, complete blood counts, potassium, calcium, PT/PTT, fibrinogen, and thromboelastography, should be obtained to guide transfusion therapy.

Leukoreduction

Most cellular components are leukoreduced in the United States prior to transfusion. Universal leukoreduction is the rule in Europe. Leukoreduction decreases the incidence of FNHTR, CMV transmission to patients at increased risk, and alloimmunization to leukocyte antigens. The concentration of leukocyte content in whole blood is on the order of 10^9 per adult collection. Approximately 90% of leukocytes are fractionated with RBCs. The recommended goal of leukoreduction is that each component contains $<5 \times 10^6$ residual WBCs per collection. The recommended residual WBC content for platelets is $<8.3 \times 10^5$ per unpooled unit. FFP does not require leukoreduction, as the residual WBC content is below the recommended limit. This is accomplished primarily through filtration but also by cell adsorption to the filter. Filtration should occur through a closed system that involves an in-line filter and sterile tubing. All blood products should be leukocyte reduced within five days of collection and prior to storage to ensure quality control and standardization of this process. It is possible to leukoreduce at the bedside if an appropriate filter is available, but this has been associated with the occurrence of hypotension. Filters for platelets are not interchangeable with filters intended for RBC use, due to differences in design. Modern filtration systems have effective pore sizes <10 microns. All blood products should be infused through at least, a large pore filter (170-260 micron).

Limiting exposures

It is important to limit donor exposures in the pediatric population, if possible. One strategy includes blood collections from a limited pool of known, screened donors that are available when transfusions are needed for a specific patient. Another strategy is to divide one adult component unit into smaller aliquots in a sterile fashion.

Irradiated Components

It is also important to note that leukocyte depleted products cannot be used to prevent graft versus host disease. Irradiated components are needed. Candidates for irradiated products include infants <6 months old, all pediatric patients with malignancies, neonatal exchange transfusions, recipients of components from first degree relatives, myelosuppressive therapy, candidates for marrow or peripheral blood stem cell transplants, and congenital immunodeficiency syndromes. Irradiation adds to component cost and alters red cell metabolism, causing accelerated potassium loss, increased free plasma hemoglobin, and shortened red cell survival.

IgA Deficiency

Special considerations for IgA-deficient patients are that platelet recipients with anti-IgA antibodies should receive platelets and FFP from other IgA deficient donors. These patients can receive washed RBCs, but frozen deglycerolized RBCs are preferred in this population.

CMV Negative Components

CMV is carried in a small minority of donor monocytes. CMV negative components should be used for CMV seronegative patients who are immunoincompetent including infants <6 months old, those with congenital or acquired immunodeficiencies, and bone marrow or organ transplant candidates or recipients of CMV negative grafts.

Special Transfusion Considerations: Hereditary Disorders of Hemostasis

A thorough medical history is the foundation of assessing the risk of bleeding/clotting. A disorder may be identified from the family history. Hemophilia A (factor VIII deficiency) and hemophilia B (factor IX deficiency) are sex-linked disorders occurring in the male children of mothers carrying the mutation. Autosomal dominant inheritance occurs with von Willebrand's disease. It is important to ascertain whether bleeding is spontaneous, longstanding, or follows injury. Seemingly spontaneous bleeding occurs in more severe bleeding disorders, such as the hemophilias or von Willebrand's disease. Milder bleeding disorders, such as mild factor deficiency, factor XI or XII deficiency, may present after surgery or following trauma. Bleeding from factor XIII deficiency typically occurs 24 – 48 hours after surgery. The physical location of bleeding can help identify the cause. Mucosal bleeding, bruising or petechiae typically occur with hereditary platelet disorders. Hemophilias present with spontaneous bleeding into joints, particularly elbows and knees.

Procoagulant Disorders: Congenital Platelet Disorders

Platelets can be abnormal in number (quantitative) or function (qualitative). Disorders can be inherited or acquired. Inherited platelet abnormalities are rare. von Willebrand's disease (VWF) and afibrinogenemia, which do influence platelet function, are more common but are factor abnormalities (see below) rather than platelet abnormalities.

Acquired quantitative abnormalities in which platelet numbers are low but function is normal are more common and are due to either decreased bone marrow production, as that caused by viral infection (HIV, EB virus, CMV, hepatitis B), aplastic anemia, paroxysmal nocturnal hemoglobinemia, or leukemia. More commonly, it can be caused by peripheral destruction of platelets, often due to antibodies against the platelet membrane, as occurs in autoimmune thrombocytopenia, or following transfusion, platelet consumption due to endothelial damage, as occurs with Rocky Mountain spotted fever, meningococcemia, HELLP syndrome in pregnant women, or in the renal microcirculation, as in hemolytic uremic syndrome. Platelets can also be sequestered in the spleen, which normally contains one third of circulating platelets.

Intrinsic platelet abnormalities include Bernard-Soulier syndrome, Glanzmann's thrombasthenia, and storage pool disorders. In Bernard-Soulier syndrome there is an absence of the platelet

surface receptor GP 1b to which von Willebrand's factor binds, inhibiting normal subendothelial binding. These patients have mild thrombocytopenia, giant platelets, and a prolonged bleeding time. Platelets fail to agglutinate on exposure to ristocetin. Glanzmann's thrombasthenia is an autosomal recessive disorder in which there is an absence of the platelet GP IIb-IIIa binding site for fibrinogen. Patients with thrombasthenia have severe mucous membrane and postoperative bleeding. Platelet transfusion may be required to stop bleeding. Bleeding time is very prolonged and platelets do not aggregate with exposure to physiologic stimuli (ADP, collagen, epinephrine, thrombin). Storage pool disorders are disorders of platelet granule secretion. Bleeding is mild and patients experience easy bruising, occasional excess mucous membrane bleeding, and postoperative bleeding. Platelets may be deficient in dense granules (decreased storage pool ADP), alpha granules (thrombospondin, factor V; "grey platelet syndrome"), absent thromboxane A2 synthetic material (cyclooxygenase or thromboxane synthetase), or thromboxane A2 functional deficiencies (defective surface membrane receptors for thromboxane A2). These patients have normal appearing platelets, moderately prolonged bleeding times and normal ristocetin-induced platelet agglutination but impaired or absent aggregation with exposure to collagen, epinephrine, and low concentration of ADP.

Table 4-4: Procoagulant Disorders: Congenital Factor Deficiency

	von Willebrand's Types
I	Low levels of vWF (80% of cases)
II a	Abnormal vWF Have low amounts of high LW multimers
II b	Abnormal vWF Avidity to platelet glycoprotein Ib Low platelets
III	Low vWF and low factor VIII
***Platelet type**	Increased avidity to platelets

Deficiency of von Willebrand's factor (VWF), a multimeric molecule that stabilizes circulating factor VIII and binds platelets to one another and to endothelium, is the most common bleeding disorder. There are three main forms of deficiency: decreased normal multimer (80% of cases), abnormal multimers, and absent multimers. VWF is synthesized in the endothelium, as is factor VIII. Bleeding in mild to moderate cases can be treated with desmopressin (DDAVP), which releases VWF from the endothelium. Endothelial VWF is released within 30 – 60 minutes. Synthesis of new endothelial VWF requires six hours. The platelet types of VWF (IIb and platelet type) are not treated with DDAVP because increased abnormal VWF lowers the platelet count. Type III VWF is not treated with DDAVP, since there is a complete absence of VWF. Other

treatments include intermediated purity factor VIII concentrates (Alphanate, Humate-P), fresh frozen plasma, and cryoprecipitate. Mucosal surfaces are rich in fibrinolytic agents. Consequently, antifibrinolytic agents, such as epsilon aminocaproic acid (EACA) and transexamic acid (10 times more potent than EACA), are useful adjuncts to other treatments for mucosal bleeding. Antifibrinolytic agents are contraindicated during episodes of hematuria, due to concerns with urinary tract obstruction.

The two most common coagulation factor disorders are *hemophilia A* (factor VIII deficiency) and *hemophilia B* (Christmas disease or factor IX deficiency). Both are X-linked congenital disorders occurring in 1 out of 5,000 males (80 – 85% hemophilia A and 10% hemophilia B). Hemophilia is graded as severe (<1% of normal FVIII or FIX levels), moderate (1 - 5% of normal levels) or mild (>5% of normal levels). Individuals with severe hemophilia bleed with little or no apparent trauma, those with moderate disease bleed following mild to moderate injury, while mild hemophiliacs bleed after severe trauma or surgery. Normal individuals have 100% or 100 units of factor/ml of plasma.

Patients with severe hemophilia are increasingly treated with scheduled prophylactic factor concentrate infusion to prevent spontaneous bleeding and its chronic sequelae. Mild to moderate trauma or bleeding is treated with sufficient factor to raise plasma factor levels to 40% of normal. For severe bleeding or major surgery, factor levels are raised to 100% of normal and maintained at a trough level of 80 – 100% for some time after the event. Some patients with mild hemophilia A will experience mild to moderate bleeding benefit from treatment with DDAVP. Episodes of severe bleeding are not controlled with DDAVP. DDAVP is not useful in the treatment of severe hemophilia A or in the treatment of any form of hemophilia B. Replacement factor concentrates are either high purity plasma concentrates or recombinant concentrates. Each unit of factor VIII concentrate increases the plasma factor VIII level by 2% with a half-life of 10 – 12 hours. The desired rise in plasma factor VIII level is calculated by: desired rise (U/dl) x kg body weight x 0.5 with repeat dosing every 12 hours. For factor IX concentrate, each unit increases plasma factor IX levels by 1% due to increased extravascular distribution; its half-life of 18 – 24 hours. Recombinant factor IX in children is generally dosed 1.2 – 1.4 times higher than adults due to the higher volume of distribution in children. The desired rise in plasma factor IX level is calculated by: desired rise (U/dl) x kg body weight x 1.2 with repeat dosing every 24 hours. Ideally, factor levels should be monitored during treatment whenever possible due to individual and product variable responses. Mucosal hemostasis can be augmented with EACA or tranexamic acid.

Some hemophiliacs (20-30% hemophilia A, 1-4% hemophilia B) form inhibitors i.e. neutralizing alloantibodies to factor concentrates. Subsets of hemophilia B patients with inhibitors develop anaphylaxis. Increasing the dose of factor concentrate can successfully treat patients with low levels of inhibitors. Bleeding episodes in hemophilia A patients with high levels of inhibitors are

treated with continuous infusion of factor concentrate or bypassing agents, such as recombinant factor VIIa or activated prothrombin complex concentrates (Factor VIII inhibitor bypassing agent, FEIBA). Bleeding hemophilia B patients who have inhibitors can be treated with recombinant factor VIIa or prothrombin complex concentrates (contraindicated in patients with anaphylaxis). Immune tolerance has been achieved in some patients using high dose factor concentrate, immune globulin infusion, immunosuppression and extracorporal immunoadsorption.

Deficiency of other coagulation factors is rare and may or may not be associated with bleeding. Congenital deficiency of fibrinogen, prothrombin, Factors V, V/VIII, VII, X, XI and XIII may be clinically significant and inherited as autosomal recessive traits. Each of these deficiencies may present as mucosal bleeding or as unexpected and sometimes life threatening bleeding during surgery. Quantitative specific factor assay may or may not predict the severity or frequency of clinical bleeding, particularly with factor VII and factor XI deficiency. Specific factor concentrates should be used, if available, during bleeding episodes or when the risk of bleeding is high (surgery) in order to minimize the risk of transfusion transmitted infection.

Deficiency in Factor XII, prekallikrein, or high molecular weight kininogen, has no effect on hemostasis but may protect from arterial thrombus formation.

Clotting Disorders: Congenital Hypercoagulable Disorders

Hereditary thrombophilias are a group of disorders in which individuals are more likely to develop either venous or arterial clotting. They comprise a group of mutations involving procoagulants (factor V Leiden, factor II, factor VII), natural anticoagulants (antithrombin, protein C, protein S), inhibitors of fibrinolysis (plasminogen-1 activator inhibitor) or other mutations (methylene tetrahydrofolate reductase deficiency- MMTHFR). All of these deficiencies have in common a propensity to abnormal clotting.

Antithrombin, a serine protease inhibitor, preferentially binds and neutralizes the active site of thrombin, Xa, IXa, XIa inhibiting coagulation. Antithrombin activity is markedly enhanced (100x) by heparin binding. Heparin is inactive in the absence of antithrombin (heparin resistance). Local release of platelet factor four from lysis of activated platelets also inhibits antithrombin. Deficiency of antithrombin leads to uninhibited thrombin conversion of fibrinogen to fibrin and consequent thromboembolic events. Homozygous AT deficiency is incompatible with life. Heterozygous AT deficiency, occurring in 1:2,000 individuals, is usually asymptomatic, but spontaneous thromboemboli and habitual abortion may occur.

Protein C and protein S work together and require vitamin K for anticoagulant effect. Both protein C and protein S are manufactured in the liver in inactive forms. When activated, protein C neutralizes Va and VIIIa. Protein C is also thought to participate in activation of fibrinolysis. Protein S accelerates the process. Homozygous protein C or protein S deficiency is thought to be

incompatible with life and heterozygous deficiency results in a propensity to thromboemboli. Factor V Leiden (8% of the white population) is resistant to activated protein C.

Perhaps the most common type of hereditary thrombophilia, particularly in individuals of Northern European ancestry, is resistance to activated protein C (APC resistance) caused by a single amino acid mutation on factor V that results in idiopathic recurrent thromboembolism, particularly when other factors are present, such as surgery, pregnancy, birth control pills, or prolonged inactivity.

Red Cell Disorders Requiring Repeated Red Cell Transfusion

In *Sickle Cell Disease (SCD),* the two β globin molecules of hemoglobin (two α and two β globins) each have a single amino acid substitution (valine for glutamic acid) in SCD. This substitution of a hydrophobic residue for a hydrophilic residue results in a predilection to hemoglobin polymerization on deoxygenation. Red cell dehydration and deoxygenation occur as red cells traverse the microcirculation. Hemoglobin then polymerizes trapping leukocytes and platelets, damaging the endothelium and occluding small vessels. Patients who have persistent high levels of fetal hemoglobin experience less severe disease. These changes give rise to the adverse consequences of SCD: acute anemia, stroke, acute chest syndrome and rarely hyperhemolysis syndrome.

Chronic transfusions are often used to reduce the incidence of these complications. However, complications of chronic transfusion therapy can include transfusion reactions, transfusion of transmitted infections, red cell alloimmunization, delayed hemolysis, and iron overload. The high rate of red cell alloimmunization (25% vs. 0.5 – 1.5% in the general population) is attributed to racial differences between donors and recipients, leading to discordance between donor and recipient red cell antigens, inflammation, immune responsiveness, number of transfusions, and age at first transfusion. Several strategies are in currently used to minimize antibody formation and delayed hemolysis. Some institutions phenotypically match the red cells to be transfused either before any transfusion or after the formation of the first red cell antibody. Limited antigen matching is done with C, E, and K. Extended red cell antigen matching usually entails the addition of Duffy, Kidd and MNS. Nevertheless, antibody may form to Rh variant alleles and/or uncommon red cell antigens. The distribution of red cells antigens is population dependent and influences alloantibody specificity. For example, in Asians the prevalence of K antigen is low and anti-K alloantibody uncommon. In comparison, the MNS antibodies anti-Mi^a /Mur are found in 31% of chronically transfused Chinese thalassemia patients with alloantibody.

Delayed transfusion reactions are estimated to occur in 5% of transfused SCD patients. These reactions generally occur two days to two weeks after transfusion and may or may not be accompanied by measureable antibody or a positive DAT. *Hyperhemolysis syndrome* is an uncommon but is a serious complication following transfusion; the post transfusion hematocrit

falls to a level lower than the pre-transfusion hematocrit. Although not well understood, hyperhemolysis syndrome is thought to be due to hemolysis of autologous red cells, suppression of erythropoiesis, or macrophage activation.

Individuals with sickle cell trait (one abnormal β globin) do not experience the complications of those with SCD. Individuals with HbSC and $HbSb^0$ (β thalassemia with no normal beta chain) may experience complications similar to those with SCD. Patients with $HbSb^+$, in which some normal beta chain is produced, do not experience the complications of SCD.

Chronic transfusion is often necessary for treatment of severe forms of ß thalassemia, ß thalassemia major and hemoglobin E-β thalassemia (HbE-βthal). HbE-βthal is relatively common in parts of India, Bangladesh, Myanmar and Southeast Asia.

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Chapter 5
PEDIATRIC RESUSCITATION

Lisa D. Heyden, MD

This chapter discusses the 2010 American Heart Association (AHA) guidelines for cardiopulmonary resuscitation and focuses on the resuscitation of pediatric patients from cardiac arrest. http://www.heart.org

The causes of arrest and the priorities and techniques for cardiopulmonary resuscitation differ in children and adults. Fortunately, cardiac arrest is uncommon in pediatric patients. When it does occur, it is usually preceded by a respiratory arrest or shock. The terminal cardiac rhythm in children is typically bradycardia followed by pulseless electrical activity (i.e., there are no pulses but there is ECG activity) or asystole. Few patients (6-8%) survive cardiopulmonary arrest when it occurs outside a hospital. Those who survive are frequently left with severe neurologic injury. If the cardiac arrest occurs in hospital, about 27% of children survive.

Congenital malformations, complications of prematurity, and sudden infant death syndrome (SIDS) are the leading causes of infant deaths in developed countries. The leading cause of death in children over one year of age is injury (trauma).

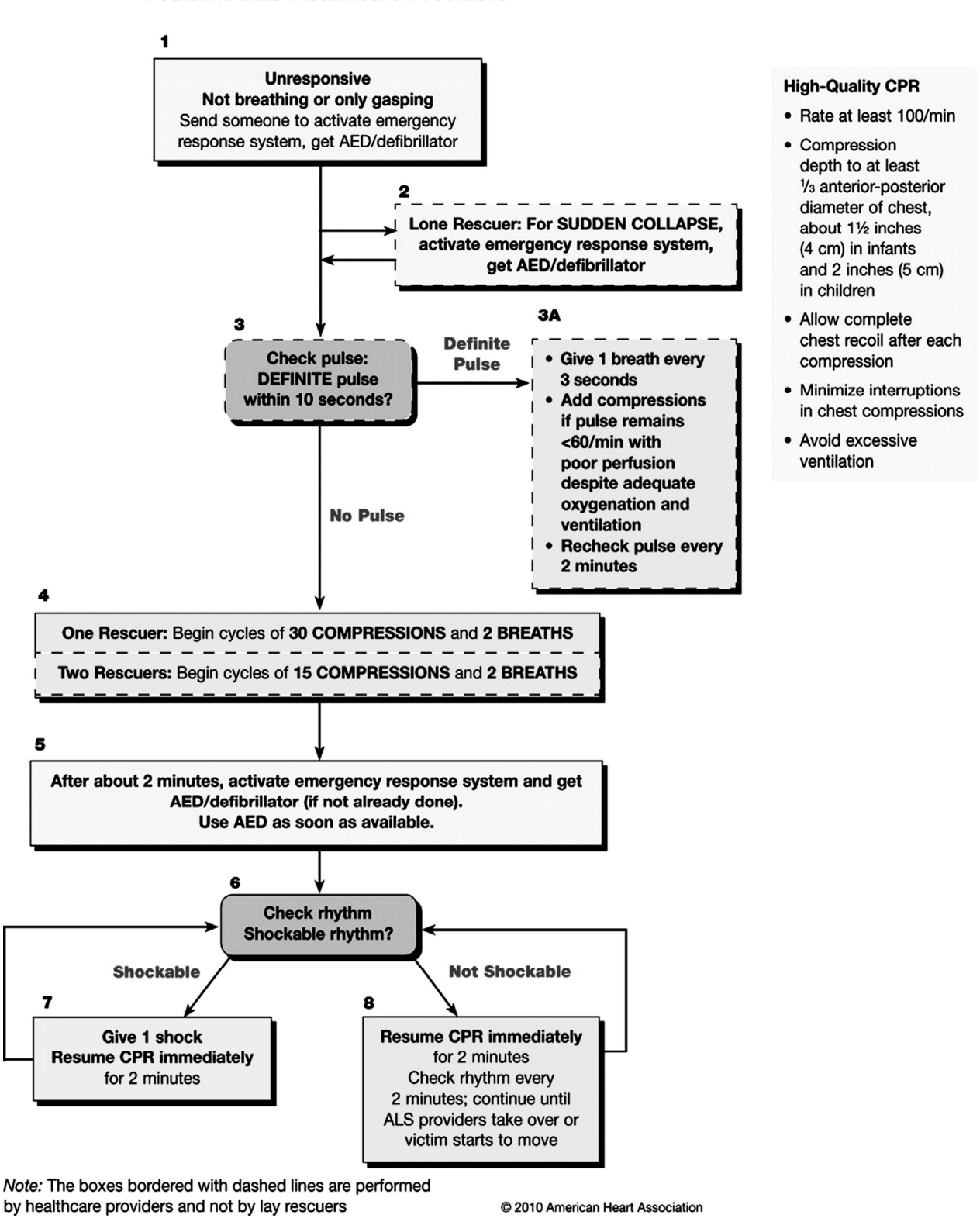
When caring for pediatric patients, it is important to address the unique and varied needs of patients of different ages (newborns, infants, children, and adolescents). Age and patient size are also important considerations when performing chest compressions, ventilating the lungs, giving drugs, and determining the cause(s) of the arrest. For the purposes of this chapter, infants are patients under one year of age. Children are one year of age to puberty (breast development in girls and axillary hair in males).

Prior to 2010, the AHA recommended a sequence for resuscitation of ABC (airway, breathing/ventilations, circulation/chest compressions) in this order. The 2010 the guidelines were changed to recommend a sequence of CAB (chest compressions, airway, breathing/ventilations). To simplify the guidelines and training of people doing resuscitation, the AHA made the resuscitation algorithm for adults and children the same; chest compressions are begun before ventilation. The majority of adults undergoing cardiac arrest have ventricular fibrillation (VF) when they arrest. More adults survive if chest compressions are started early and continue with minimal interruptions. Thus, it is important that all rescuers are able to perform effective chest compressions. Rescue breathing is harder to perform effectively, takes time to initiate, and may delay initiation of chest compressions. For rescue breathing to be effective,

pulmonary blood flow must be present. Thus, cardiac massage (chest compressions) must be initiated quickly.

In adults ventricular fibrillation (VF) is the most common cause of cardiac arrest. In infants and children asphyxia is the most common cause. This makes early initiation of artificial ventilation (rescue breathing) more important in children. However, resuscitation should start with giving 30 chest compressions before giving two breaths; giving these 30 chest compressions should not delay starting rescue breathing by more than about 18 seconds. Healthcare providers often tailor the sequence of rescue actions (ABC versus CAB) to the most likely cause of arrest. If the cardiac arrest is witnessed and occurs suddenly, or the child is at high risk for developing a life-threatening arrhythmia, VF cardiac arrest may be more likely than asphyxia induced cardiac arrest. In this case, an automatic external defibrillator (AED) (if available) and additional help with the resuscitation are extremely important.

Figure 5-1: Basic Life Support Algorithm for Health Care Providers



Chapter 5: PEDIATRIC RESUSCITATION

From Pediatrics 2006;117:e980-e1040, with permission pediatrics.aappublications.org

The following is an organized approach to the resuscitation of patients from cardiac arrest.

Assess the need for CPR – If the victim is unresponsive and is not breathing (or only gasping), send someone to both get help and to obtain equipment needed to resuscitate the patient.

Pulse check – If the child is unresponsive and not breathing, feel for a pulse (brachial pulse in an infant and carotid or femoral pulse in a child). If no pulse is felt within 10 seconds or you are not sure if there is a pulse, begin chest compressions (See below).

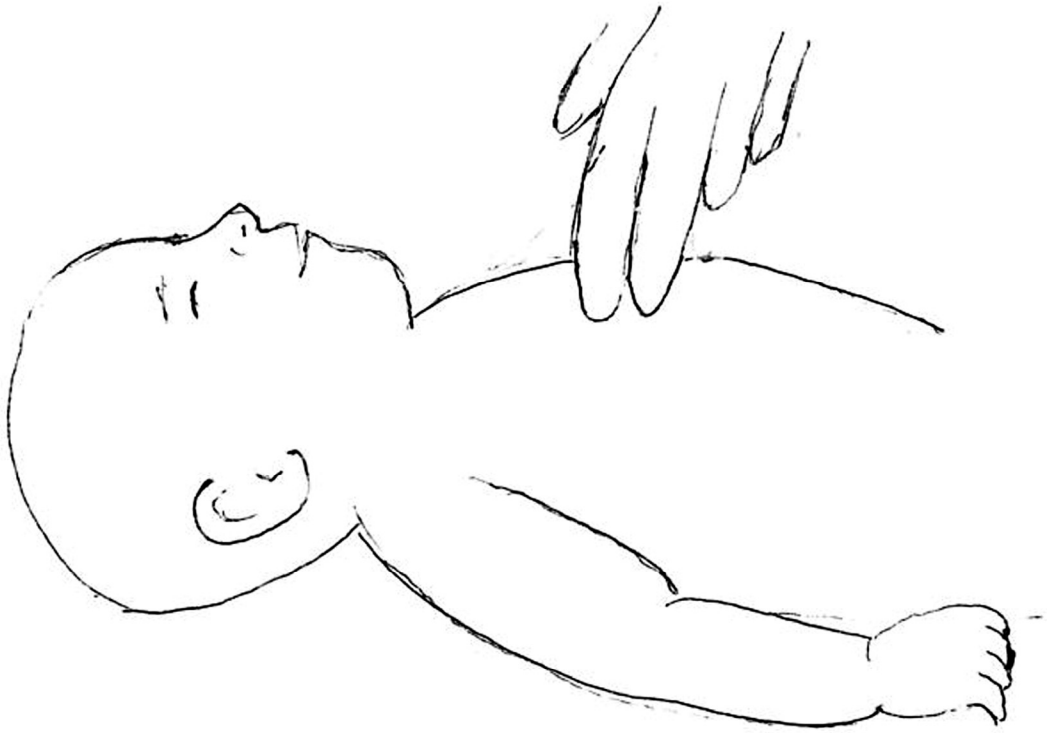
Inadequate breathing with a pulse - If the pulse is palpable and the pulse rate is greater than 60 per minute, but breathing is inadequate, give 10-to-20 breaths per minute. Reassess the pulses for no more than 10 seconds every two minutes and continue breathing for the patient. The heart rate should increase and be normal for that patient's age (**See Appendix 1**).

Bradycardia with poor perfusion - If the pulse rate is below 60 beats per minute and the patient has signs of poor perfusion (pallor, mottling, or cyanosis of the skin), begin chest compressions. *Cardiac output in infants and children depends largely on the heart rate.* When cardiopulmonary resuscitation (CRP) is started before a full cardiac arrest occurs, survival is improved.

Chest compressions - If the infant or child is unresponsive to voice and/or tactile stimulation, is not breathing, and has no palpable pulse, place the child on a hard surface (a table, a board, or the floor) and immediately start chest compressions; chest compressions done in a bed are ineffective. Deliver 30 chest compressions at a rate of *100 compressions per minute or more* and use sufficient force to depress the sternum at least *one-third of the anterior-posterior (AP) diameter* of the chest [1.5 inches (4 cm) in infants; 2 inches (5 cm) in children)]. Allow the chest to completely return to its normal resting position between compressions. Interruption of chest compressions must be minimized.

The method by which one does chest compression depends on both the patient's age and her/his size. In infants, lone rescuers place the second and third fingers of one hand on the lower half of the sternum, just below an imaginary line drawn between the patient's two nipples (**Figure 5-2**), and compress the sternum as described above. Do not compress over the xiphoid process or the ribs because this may injure the lungs, liver, spleen, and/or other intra-abdominal organs.

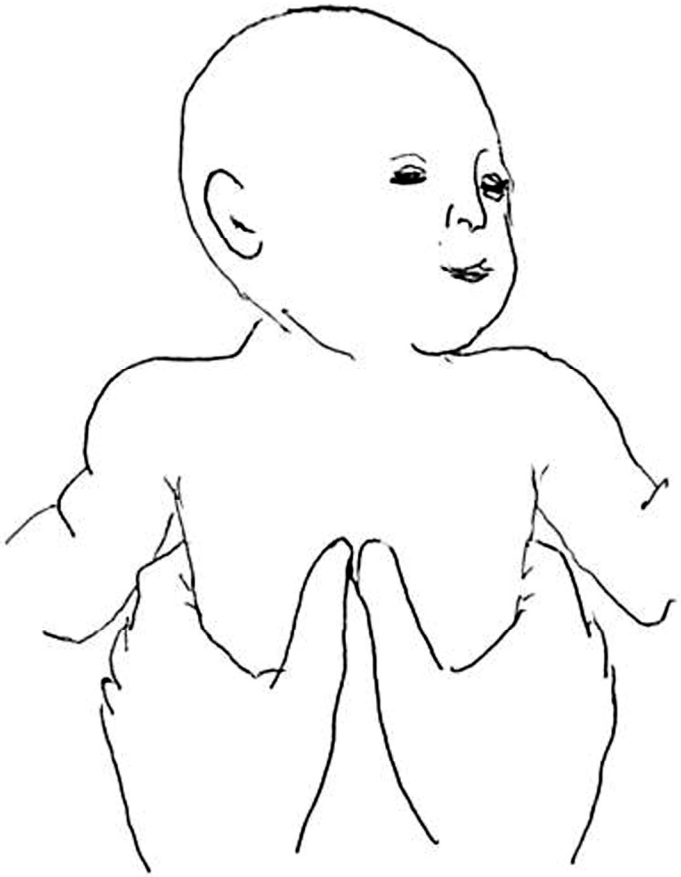
Figure 5-2: Two-Finger Method for Cardiac Massage



The image shows the two-finger method of doing cardiac compression in a baby. Note the fingers are in the midline just below an imaginary line drawn between the child's two nipples; the back is supported on a hard surface. Artificial ventilation should be done as soon as possible.

If two rescuers are available to perform CPR, the two thumb-encircling hands technique (**Figure 5-3**) is used in young children because it provides more effective chest compression and causes less rescuer fatigue. The fingers of both hands are placed around the thorax, and the thumbs are positioned in the midline over the lower third of the sternum while compressing the sternum. Avoid compressing over the xiphoid process and/or ribs. The fingers surrounding the chest support it during compressions. The second resuscitator can ventilate the patient's lungs if he/she is capable of doing this.

Figure 5-3: Two Thumb Method of Chest Compression in an Infant



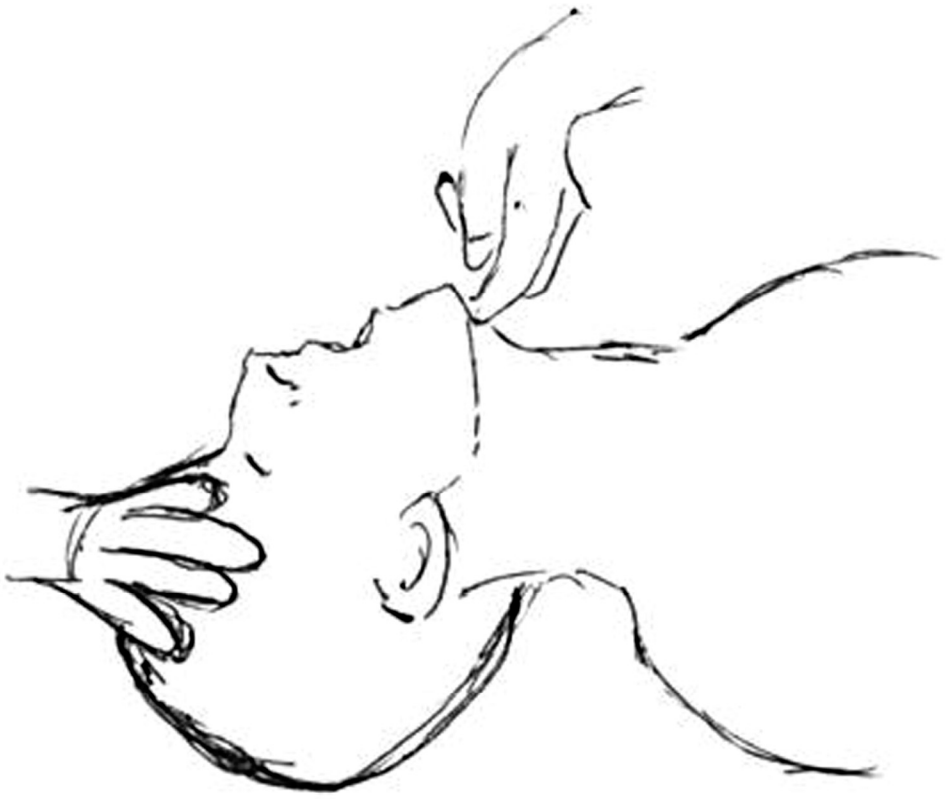
The figure shows cardiac compression using the two-thumb technique. The fingers of both hands are placed around the patient's chest and the thumbs compress the heart between the sternum and the spine. The fingers and thumbs are placed just below an imaginary line drawn between the baby's nipples and the sternum is compressed. Ventilation can be produced with a bag-and-mask.

Chest compressions can be done in children with either the heel of one hand (the back muscular part of the palm of the hand) while the other hand is situated on top of the first hand. The heel of one hand may also be used alone. In adolescents, chest compressions should be done using the two hands method, as in adults. To decrease resuscitator fatigue and to improve the quality of CPR, the person doing chest compressions should switch with the second rescuer every two minutes, if there are two rescuers. If only one rescuer is available to ventilate the patient's lungs (because of lack of training or for other reasons), chest compressions should be started and continue until help arrives.

Ventilation of the lungs - After 30 chest compressions for a single rescuer and 15 compressions for two rescuers (one person to compress the chest and the other to ventilate the lungs), breaths are given. Before starting artificial ventilation, the airway is opened by tilting the head back (head

tilt) and lifting the chin (**Figure 5-4**). Then two breaths are given. If one suspects that the patient has a cervical spine injury, open the airway by performing a jaw thrust *without* doing a head tilt. If this does not provide an airway, an airway adjunct is used (See below). If the airway still is not patent, a head tilt must be done to open the airway. Doing so may increase spinal cord injury if present, but will usually allow the lungs of the victim to be ventilated in this desperate situation. Airway adjunct can also be used in some patients to avoid having to extend the head (see below).

Figure 5-4: Head Tilt-Chin Lift Maneuver.



This figure shows the method for chin lift. The fingers of the hand lifting the chin should be on the bone of the mandible, not in the soft parts of the chin. Placing the fingers in the soft parts of the chin can push the tongue back into the throat, causing worse airway obstruction.

Figure 5-5 shows method for chin lift and application of a facemask at the same time.

Figure 5-5: Chin Lift While Holding a Facemask.

The fingers of the hand holding the mandible pull the mandible upward to move the tongue out of the pharynx. Since the tongue is attached to the posterior mandible, this maneuver moves the tongue away from the pharyngeal wall. T to avoid causing more airway obstruction, the resuscitator's fingers should be placed on bone, not in the soft tissues of the chin.

If the stomach becomes inflated with air during artificial ventilation, this may prevent the diaphragm from moving freely during inspiration and interfere with effective pulmonary ventilation. Distention of the stomach also increases the risk of regurgitation and aspiration of gastric contents. Thus, an oral gastric tube should be placed during resuscitation to remove gas that enters the stomach. Avoid applying excessive peak inspiratory pressures because they often force gas into the stomach. Ventilate the patient's lungs at a rate appropriate for the child's age, and use only enough pressure to raise the chest a normal amount with each breath.

Coordinate chest compressions and ventilation – Ventilation of the lungs should only interrupt chest compressions a minimal amount. If ventilation is being done through a tracheal tube, chest compressions should continue while breaths are given at rates of up to 60 breaths per minute at 3 months of age, 40/minute at 1 year of age, and 30 breaths per minute at 12 years of age.

Excessive ventilation rates and/or pressures are to be avoided. Most rescuers are frightened when resuscitating a patient from cardiac arrest and tend to ventilate very rapidly and give deep breaths. Both can injure the lungs and interfere with pulmonary blood flow.

When only one rescuer is available to resuscitate the patient, he/she should give 30 chest compressions and two breaths for two minutes or for five cycles of 30 and 2. If there is no one else to do it, leave the victim and call for help and obtain an Automated External Defibrillator (AED). The time away form the arrested patient should be very short. A lone rescuer should begin resuscitation as soon as possible, starting with chest compressions and continue the chest compressions until the second resuscitator or other help arrives or until the victim starts to breath spontaneously. When there are two rescuers, one should start CPR and the other should quickly call for help and obtain an AED, if available.

Defibrillation - Ventricular fibrillation and pulseless ventricular tachycardia are shockable rhythms' i.e., they often convert to a sinus rhythm or to one that perfuses organs when the heart is shocked by the electrical discharge from a defibrillator. A manual defibrillator (a device that monitors the heart rhythm and allow the user to manually set the energy to be delivered) is preferred for infants if a trained healthcare provider is available who can identify a shockable rhythm. The initial shock is 2 Joules/kg of body weight; 4 J/kg are used for subsequent shocks, if needed. If no one trained to use a manual defibrillator is available, an AED equipped with a pediatric attenuator can be used for patients who are less than nine years of age. An attenuator is a device that decreases the AED's power, allowing the AED to be used for young pediatric patients. This attenuator prevents delivery of adult doses of electricity to a pediatric heart, which can damage it. A standard adult AED is used for children who are nine years of age and older. If an AED with a pediatric dose attenuator is not available, an adult AED should be used. Stacked shocks (multiple shocks given one after the other) should be avoided in young pediatric patients. Minimize the time between chest compressions and shock delivery by coordinating the chest compressions and shock delivery.

Obtaining vascular access, administering drugs, or placing an advanced airway (tracheal tube, laryngeal mask airway), while very useful and usually necessary, should not significantly interrupt chest compressions or delay shocking the heart when needed.

Breathing Adjuncts

If starting artificial breathing will be delayed by waiting to obtain a barrier to place between the patient's and resuscitator's mouths, or to obtain ventilation equipment (i.e., bag and mask), mouth-to-mouth breathing should be initiated without waiting for these items if the resuscitator is willing and able to breath for the patient. If the resuscitator is unwilling or unable to provide mouth-to-mouth ventilation, chest compression alone should be provided without interruption until help arrives.

Bag and mask ventilation is the preferred method for ventilating a patient's lungs during cardiac arrest, but it takes training and practice to effectively ventilate a patient's lungs with these devices and requires training and practice to effectively ventilate a patient's lungs. Rescuers must be able to select the proper mask size for each patient, open the airway with a head tilt-chin lift, make a tight seal between the mask and the patient's face, deliver an effective volume of gas with each breath, and assess the effectiveness of the ventilation. A self-inflating bag, such as those produced by AMBU™, is very useful because it will function without added oxygen. *Avoid excessive ventilation rates and pressures.*

If the ventilation being used fails to produce adequate chest rise, readjust the head tilt-chin-lift and make sure the mask seal is adequate. If the chest rise is still inadequate, use an airway adjunct, such as an oropharyngeal airway (OPA) or nasopharyngeal (NPA) airway. A properly sized and placed OPA or NPA aids in opening the airway by displacing the tongue or soft palate away from the pharynx. In some children inserting a NPA will remove a portion of infected adenoids, which can cause significant bleeding once blood is again circulating. A laryngeal mask airway (LMA), if available, can also be useful. However, it is often difficult to generate more than 20cmH_2O airway pressure through this device. This amount of pressure may be inadequate in some patients to provide an adequate breath. The first priority (after chest compression) is to ventilate the patient's lungs using a bag and mask when available. Spending time trying to insert a tracheal tube (unless it is impossible to obtain an effective airway otherwise) wastes time and makes it more difficult to effectively resuscitate a patient from cardiac arrest. *Very few people die from lack of a tracheal tube. Almost everyone dies from a lack of oxygen*. Deliver basic ventilation with a bag and mask (if possible) until the patient's condition is stable. Then intubate the trachea if necessary.

If the caregiver is skilled in tracheal intubation and airway management and can place a tracheal tube into the trachea in less than 10 seconds, CPR may be paused briefly to insert the tube. Either cuffed or uncuffed tracheal tubes are used, but cuffed tracheal tubes are thought to decrease the risk of aspirating gastric contents or the need to change the tube because it is too small or too large.

Newborn Resuscitation

Since asphyxia is the primary cause of cardiac arrest in neonates, the prior recommendation of airway/breathing before starting chest compressions is still appropriate. If a patient is not breathing or is not adequately oxygenating (SaO_2), enough positive pressure ventilation is used to normally expand the chest; a respiratory rate of about 30 breaths per minute is used. *Avoid using excessive pressure* because it can injure the newborn lung.

If the patient has no pulse or her/his heart is rate below 60 beats/minute, start chest compressions as described earlier. The recommended chest compression-to-lung ventilation ratio

when asphyxia is presumed to be the cause of arrest is three compressions of the chest for each breath. If the arrest is known to be of cardiac origin, higher ratios (15:2) may be used.

The AHA guidelines recommend attaching a pulse oximeter probe to the right upper extremity of babies and basing the need for giving supplemental oxygen on the patient's oxygen saturation (SaO_2). For babies born at term (37-42 weeks gestation), these guidelines suggest starting resuscitation with room air rather than 100% oxygen. The amount of oxygen that is added to the inspired air should be based on the patient's SaO_2. However, if the SaO_2 is below 85% and does not increase rapidly with effective resuscitation, enough oxygen is added to the inspired gases to raise the SaO_2 to 90% or higher. Hyperoxia (SaO_2 above normal for age) may be toxic to newborns, particularly if they are preterm (<37 weeks gestation age). High concentrations of oxygen can cause chronic lung disease or blindness (retinopathy of prematurity). Thus, it is best to keep the SaO_2 of term babies between 94%-97% and that of premature babies between 88% and 94%. Airway suctioning after birth is reserved for babies who have obvious airway obstruction (intercostal, supra-sternal, infra-sternal, and sternal retractions), hypoxemia, or require positive pressure ventilation. There is little evidence that routine suctioning of the airway is beneficial, and there are risks associated with airway suctioning (hypoxemia, apnea, arrhythmias, damage to the airway).

Medications Used for Pediatric Resuscitation

The doses of medications used for resuscitation are usually calculated using the child's weight in kilograms, if known. A Broselow tape, a device used throughout the world to calculate drug doses to be given to pediatric patients during emergencies, is very useful for pediatric emergencies. The tape is laid beside the child and. based on her/his weight and height, predetermined drug doses are provided on the tape. In addition to drug doses, the tape also provides the appropriate size equipment (tracheal tubes, masks, etc.), and the joules needed to defibrillate the patient's heart.

Table 5-1 lists medications commonly used during resuscitation. Intravascular (IV) injection of drugs is the preferred method for drug administration, but if IV access is unavailable, lidocaine, epinephrine, atropine, and naloxone can be administered via a tracheal tube. However, drugs administered via the trachea may not be as effective as when the same drug given IV. An IV should be placed as soon as possible in an arrest.

Table 5-1: Medications for Pediatric Resuscitation

Medication	Dose	Remarks
Adenosine	0.1mg/kg (maximum 6 mg); Second dose: 0.2mg/kg (maximum 12 mg)	Monitor ECG. Give as a rapid IV/IO bolus with flush
Amiodarone	5mg/kg IV/IO; may repeat twice up to 15mg/kg; Maximum single dose 300 mg	Monitor ECG and Blood pressure; adjust administration rate to urgency (IV push during cardiac arrest, more slowly–over 20–60 min when the heart rhythm produces peripheral perfusion. Expert consultation strongly recommended prior to using this drug when a patient has a perfusing rhythm; Use caution when administering with other drugs that prolong the QT interval - (obtain expert consultation)
Atropine	0.02mg/kg IV/IO 0.04–0.06mg/kg/TT. Repeat once if needed. Minimum dose: 0.1mg. Maximum single dose: 0.5mg	Higher doses may be used with organophosphate poisoning
Calcium Chloride (10% solution)	20mg/kg IV/IO (0.2mL/kg) Maximum single dose 2 g	Administer slowly
Epinephrine	0.01mg/kg (0.1 mL/kg of a 1:10,000 solution) IV/IO; 0.1mL/kg of a 1:1000 solution per TT*. Maximum dose 1 mg IV/IO; 2.5mg/TT	May repeat every 3–5 minutes
Glucose	0.5–1g/kg IV/IO for hypoglycemia	Newborn: 5–10mL/kg $D_{10}W$ Infants and Children: 2–4mL/kg $D_{25}W$ Adolescents: 1–2mL/kg $D_{50}W$
Lidocaine	Bolus: 1mg/kg IV/IO; 20–50mcg/kg/minute by infusion	
Magnesium Sulfate	25–50mg/kg IV/IO over 10–20 minutes; give faster in torsades de pointes; Maximum dose 2g	
Naloxone	Full Reversal: <5 y or ≤20 kg: 0.1mg/kg IV/IO/ET*; ≥5y or >20 kg: 2mg IV/IO/ET*	Use lower doses to reverse respiratory depression associated with therapeutic opioid use (1–5 mcg/kg – *titrate the dose to effect*)

Table 5-1 (Continued)

Procainamide	15mg/kg IV/IO. Adult Dose: 20mg/min IV by infusion to a total maximum dose of 17 mg/kg	Monitor ECG and blood pressure; Give over 30–60 minutes. Use caution when administering with other drugs that prolong QT interval (obtain expert consultation)
Sodium bicarbonate	1mEq/kg per dose IV/IO slowly	After adequate spontaneous or mechanical ventilation is present

IV indicates intravenous; IO, intraosseous; and TT, via tracheal tube.

* Flush with 5 mL of normal saline and follow with 5 ventilations.
http://circ.ahajournals.org/content/122/18_suppl_3/S876.full.pdf+html

Adenosine

Adenosine briefly blocks conduction through the atrioventricular (AV) node and is used to treat supraventricular tachycardia (SVT), including super SVT associated with Wolff-Parkinson-White Syndrome.

Amiodrarone

Amiodarone should not be used without expert consultation (i.e., cardiologist).

Atropine

Atropine in an anticholinergic drug that increases the heart rate and is used to treat symptomatic bradycardia, organophosphate poisoning, and nerve gas exposure.

Calcium

Calcium administration is not recommended for pediatric cardiopulmonary arrest unless there is documented hypocalcemia, calcium channel blocker overdose, hypermagnesemia, or hyperkalemia.

Epinephrine

Epinephrine is indicated for anaphylaxis, asthma, symptomatic bradycardia, croup, pulseless cardiac arrest, hypotensive shock, and overdose of beta-adrenergic blockers or calcium channel blockers.

Glucose

Measure serum glucose levels during resuscitation if possible and treat hypoglycemia (<40 mg/dl in infants and <70 mg/dl in children.

Lidocaine

Lidocaine decreases automaticity (i.e., ability of the heart to respond *without* outside influences) and suppresses ventricular arrhythmias; it may be used in patients when ventricular fibrillation (VF) does not respond to electrical shocks and epinephrine. Avoid causing lidocaine toxicity by giving too much lidocaine (7 mg/kg or 500 mg total).

Magnesium

Magnesium is indicated for the treatment of documented hypomagnesemia or for torsades de pointes (polymorphic ventricular tachycardia). Magnesium is not used routinely during cardiac arrest.

Procainamide

Expert consultation is recommended.

Sodium Bicarbonate

Routine administration of sodium bicarbonate is not recommended in cardiac arrest because it produces large amounts of carbon dioxide (CO_2), 2,500 cc of CO_2/50 mEq of bicarbonate when fully reacted with hydrogen ions. The lungs cannot clear the CO_2 produced during an arrest, due to lack of pulmonary blood flow and ventilation. Bicarbonate is used to treat hyperkalemia and overdose of sodium channel blockers.

Vasopressin

There is insufficient evidence to make a recommendation for or against the routine use of vasopressin during cardiac arrest.

Summary

1. The AHA guidelines were changed in 2010 to emphasize chest compressions/circulation. Although asphyxia is the cause of most cardiac arrests in children, resuscitation begins with chest compression before the lungs are ventilated or the airway is manipulated. The exception to this rule is newborn resuscitation, where ventilation may be started before beginning chest compression.

2. It is possible to provide CPR with limited resources; the lungs can be ventilated with mouth-to-mouth breathing if no bag-mask equipment is available.

3. Chest compression should be "hard" and fast (100 times/minute) with the chest returning to its normal position following each compression of the chest.

4. Pauses to evaluate return of pulses, to give breaths if no tracheal tube is present, or to analyze the heart rhythm, and to shock the heart should be very brief (<10 seconds). Chest compression should be restarted immediately after a shock is delivered.

References

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3. http://circ.ahajournals.org/content/122/18_suppl_3/S876.full.pdf+html
4. http://pediatrics.aappublications.org/content/126/5/e1361.full
5. http://c.ymcdn.com/sites/www.masscrna.com/resource/resmgr/pals_2010_overview.pdf

Chapter 5: PEDIATRIC RESUSCITATION

Chapter 6
THE PEDIATRIC AIRWAY

E. Heidi Jerome, M.D.

Introduction

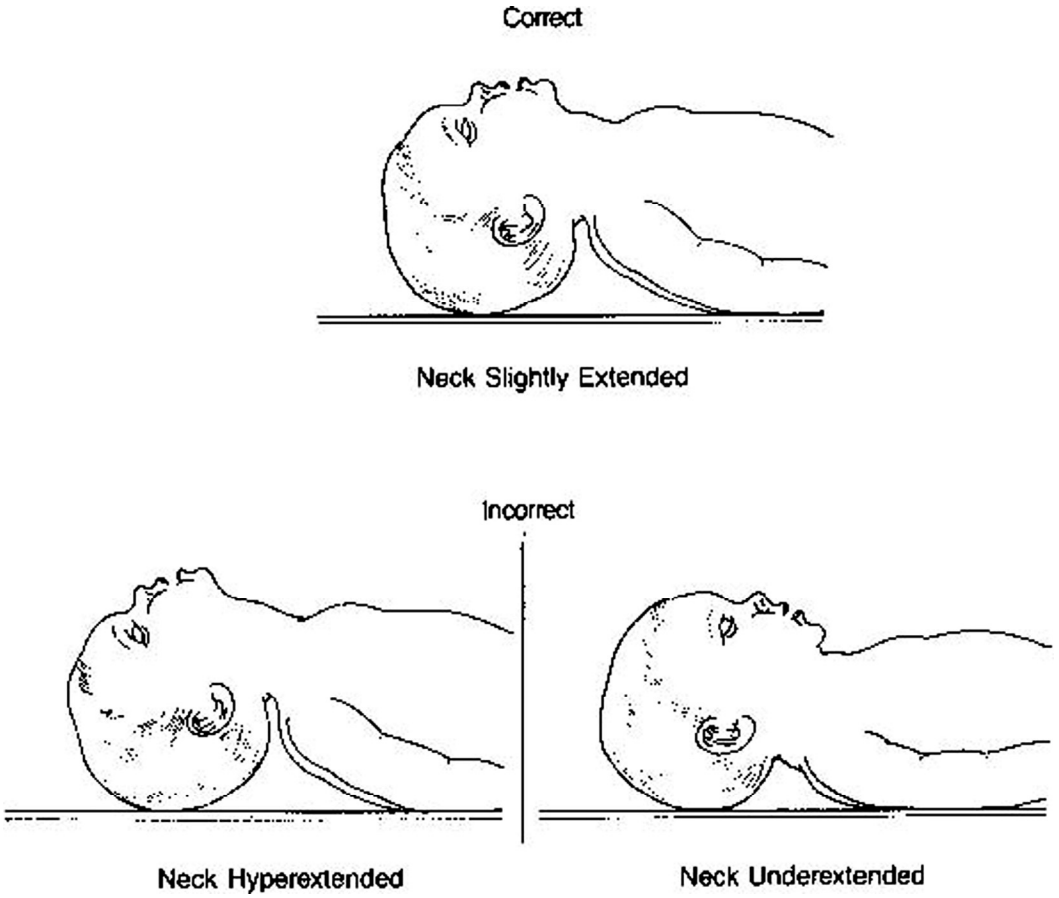
Because airway difficulties are the most common complications in pediatric anesthesia, knowledge and skills of pediatric airway management are crucial for providing safe conduct of anesthesia in children. By understanding normal developmental anatomy and physiology, the differences between pediatric and adult airways, and the specific issues of difficult pediatric airways, anesthetists will have the tools they need to care for patients with airway problems. This chapter first reviews pediatric airway anatomy and discusses appropriate use of airway devices. It then discusses management of the pediatric airway during pre-, intra- and post-operative periods. Finally, it discusses management of the difficult airway. Both established and newer methods of pediatric airway management are discussed even though some of these devices and techniques will not be available to all anesthetists. Knowledge of both is essential for those providing anesthesia for children.

Pediatric Airway Anatomy and Use of Airway Devices

From the head to the diaphragm, the pediatric airway differs from that of the adult in many ways. Consequently, the methods of pediatric airway management differ from those used for adults.

The heads, and particularly the occiputs, of infants are larger (relative to their body size) than those of older children or adults. The ideal “sniffing” position, i.e., the position that opens the airway and allows easier mask ventilation and tracheal intubation, is different in infants and toddlers, compared to adults. Infants and young children have the best airway position while lying flat on an operating bed because their large occiputs put them in the sniffing position (**Figure 6-1**).

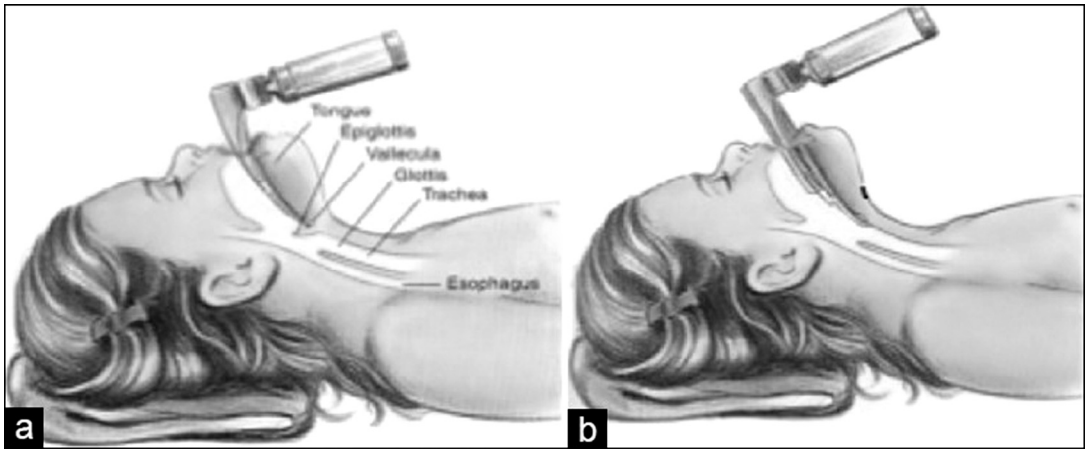
Figure 6-1: Best Position of the Head for Tracheal intubation and Bag-and-Mask Ventilation.



The infant in the top drawing is in a neutral position looking straight up (forward). Because of the infant's large occiput, this puts the airway in the best position for bag-and-mask ventilation and tracheal intubation. Both of the other two positions (hyperextended and under extended) make ventilation and tracheal intubation more difficult. Http://www.Glowm.com

For infants with very large occiputs, placing a small roll under their shoulders often improves their airway alignment. After five years of age, the child's occiput is less prominent. Placing a small pillow under the occiput of an older child helps align their airway (**Figure 6-2**). Keeping the head in a neutral position (neither extended or flexed) improves airway patency during both spontaneous and positive pressure ventilation.

Figure 6-2: Head on a Pillow to Align the Airway



a: shows placement of a curved (MAC) blade in the airway. Note the tip of the blade in the vallecuala. b: shows a straight (Miller type) blade in the airway of a child older than five years of age. In both instances, the laryngoscope blade is pulled up at a 45-degree angle to obtain the best view of the vocal cords. Http://www.ljcis.org

The nasopharynx of infants and children differs from that of adults. First, babies are obligate nose breathers for the first 3-5 months of life, which allows them to breast feed and breathe simultaneously. For this reason the nasopharynx must be kept patent during the induction of anesthesia and in the post-operative period. Second, in the first decade of life, many children have adenoidal hypertrophy that obstructs the nasopharynx and causes respiratory difficulties for both awake and anesthetized children. Caution is required when placing nasal airways or nasal tracheal tubes in these patients because doing so may injure the adenoids and cause bleeding. Consequently, oral airways are used more often than nasal airways in 4-10 year old children. A third anatomic consideration is sinus growth. Ethmoid and maxillary sinuses are present at birth and grow with the patient. Blocking these sinuses with long-term nasal intubation leads to sinusitis. Frontal sinuses develop at 5-6 years of age; the sphenoid sinuses develop during adolescence.

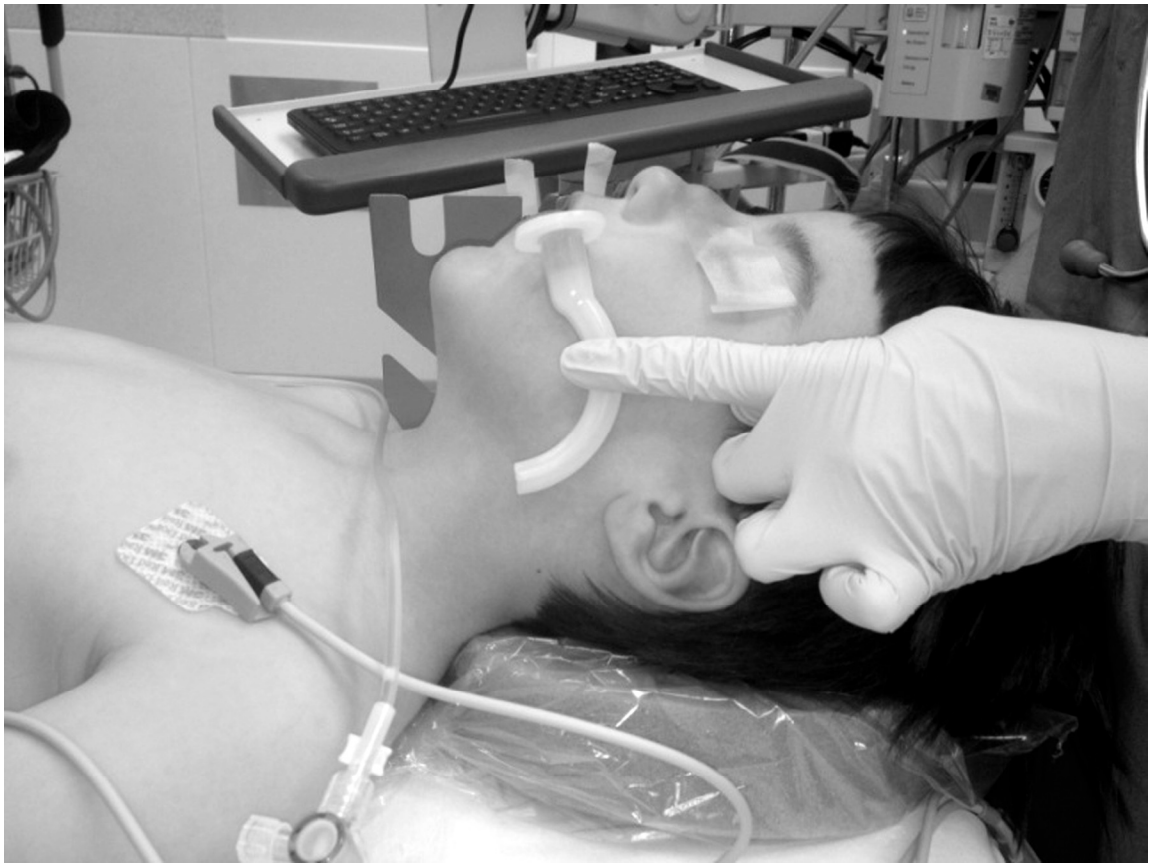
The mouth openings of children are smaller than those of adults. Thus, pediatric laryngoscope blades are narrower to allow these blades to fit between a child's gums or teeth. Teeth start appearing from the upper gums around one year of age and continue to emerge from both upper and lower gums through the first decade of life and the teen years. Deciduous tooth loss begins around 5-6 years of age, but when it begins is quite variable. Loose teeth should be identified during pre-operative discussions with the child and parents. Very loose teeth should be removed manually after the airway is secured to prevent inadvertent dislodgement of the tooth while the child is emerging from anesthesia and having the tooth "fall" into an unprotected trachea. Care must be taken during manipulation in the oral cavity not to injure or dislodge teeth.

The oropharynx contains the tongue, palate and tonsils. The tongues of pediatric patients are relatively larger than those of adults and can easily obstruct the airway during anesthesia. The tongue of a normal patient is easily moved away from the posterior oropharynx by pulling the mandible forward (anteriorly). However, a small mandible with a short ramus, as occurs in patients with Pierre-Robin syndrome (see below), may push the tongue posteriorly and obstruct the airway. When it does, manipulating the mandible often does not relieve the obstruction. It may be very difficult with standard laryngoscopy to visualize the glottis of patients who have short mandibles. The tongue position in utero may prevent the palate of patients with Pierre-Robin syndrome from forming normally and cause a posterior cleft palate.

Just as pediatric patients develop adenoidal hypertrophy, they also develop tonsillar hypertrophy and infection, which are well known causes of airway obstruction, even in non-anesthetized children. Long standing tonsillar and adenoidal hypertrophy that obstructs the airway can lead to obstructive sleep apnea (OSA), which puts patients at risk for post-anesthetic central and obstructive apnea. When OSA is suspected (based on physical examination and a history of apneic episodes lasting longer than 10 sec during sleep), the patient should be observed in the recovery area for at least three hours after surgery and monitored with pulse oximetry. He/she should not be discharged from hospital unless there is no evidence of apneic spells or the SaO_2 remains above 96%. Consideration should be given to observing the child overnight in an ICU or other monitored area.

During induction of or emergence from anesthesia, placing an oral or nasal airway may improve upper airway soft tissue obstruction and airway patency. The length of the oral or nasal airway is critical. If too short, oral airways push the tongue back into the posterior palate and worsen the obstruction. A nasal airway that is too short may not reach beyond the collapsed soft tissues. Oral and nasal airways that are too long may push the epiglottis over the glottic opening and worsen the obstruction. Checking the size of the oral or nasal airway, by holding it next to the patient's cheek, helps choose the correct size airway (**Figure 6-3**).

Figure 6-3: How to Determine the Correct Size of an Oral Airway



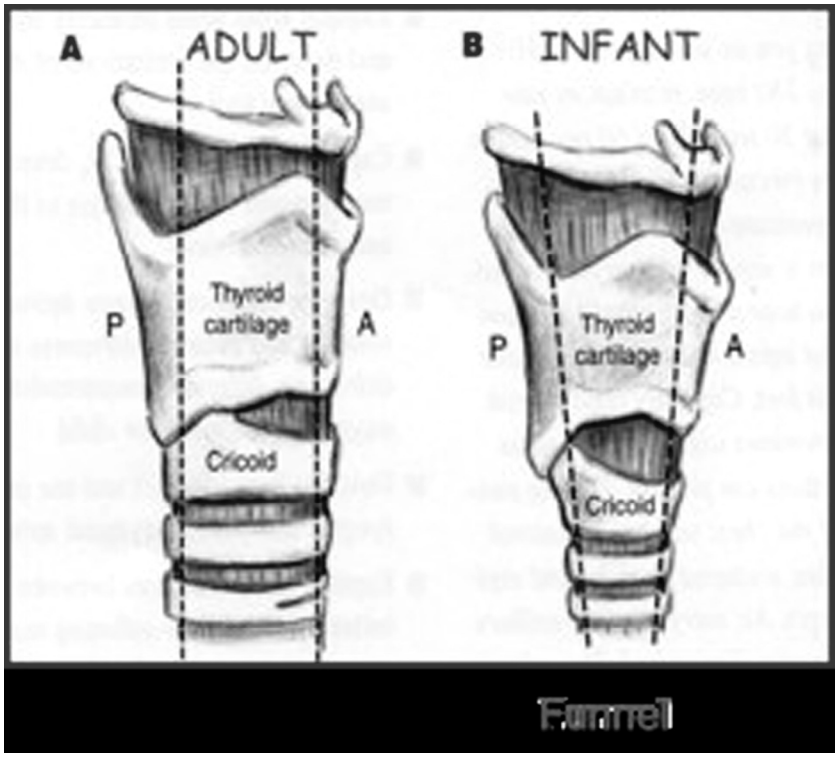
The anesthetist is holding an oral airway next to the face of a young child. The proximal end of the airway is even with the mouth and the distal end is at the angle of the jaw.
Basicsof pediatricanesthesia.files.wordpress.com

The distal tip of the airway should sit at the level of the angle of the jaw. To place an oral airway, a tongue depressor is positioned well back on the tongue to depress it. This makes it easier to pass the lubricated airway over the tongue. Inserting the airway upside down and rotating it into place may obstruct the airway, damage the palate, or injure loose or decayed teeth. (See video) Consequently, this should seldom be done.

The pediatric larynx differs in several important ways from that of the adult; it is located higher in the neck. The vocal cords are also different. They advance from a position opposite cervical vertebra 3-4 in neonates to a position opposite cervical vertebrae 5-6 by late adolescence. The infant's and child's vocal cords are angled, with the anterior attachment lower than the posterior attachment. A straight Miller blade usually provides a good view of infants' or toddlers' vocal cords. A curved Macintosh laryngoscope blade provides a good view of the deeper vocal cords of adolescent patients.

The larynx of pediatric patients is funnel-shaped and is narrowest at the cricoid cartilage, the deepest portion of the larynx (**Figure 6-4**). The adult larynx, by contrast, is more like a straight tube whose narrowest portion is at the vocal cords.

Figure 6-4: Shape of the Upper Airway of Infants and Adults



Notice that the larynx and upper trachea of an adult is straighter than that of the infant on the right. Thus the narrowest portion of the adult's airway is at the vocal cord and that of the infant is at the cricoid ring. http://o.quizlet.com

Transition from pediatric to adult laryngeal anatomy is completed between five years of age and puberty. The timing of these changes is quite variable among patients. The cricoid cartilage forms a complete cartilaginous ring through which cuffed or uncuffed tracheal tubes can be passed. In older children or adults, cuffed tracheal tubes are used to fill and seal the airway below the vocal cords. In patients less than six months of age, uncuffed tubes are usually chosen to maximize the internal diameter of the tube and decrease turbulent gas flow. When a cuffed TT is used in young patients, the tube should be 0.5mm smaller than an appropriate uncuffed tube to accommodate the cuff within the trachea. Newer "microcuff" tracheal tubes have lower profiles and shorter cuffs, which may allow passage of a slightly larger internal diameter TT without exerting excessive pressure on the tracheal mucosa. For example, a 3.0mm internal diameter microcuffed TT may fit in the trachea of a newborn only slightly more snuggly than a 3.0mm uncuffed tube. Microcuff tubes may also be less traumatic to the tracheal mucosa than older style cuffed tracheal tubes. Another advantage of cuffed tubes in toddlers and school age children is that a good tube fit can

be achieved after a single laryngoscopy and tracheal intubation by inflating the cuff with a small amount of air, rather than having to sometimes change uncuffed TTs more than once to achieve a proper fit. However, microcuff tubes are more expensive than uncuffed TT.

Whether a cuffed or uncuffed tube is chosen (**Table 6-1**), there is enough variation in cricoid cartilage diameter that it is necessary to test for a gas leak around the TT in all pre-pubertal patients to assure that excessive pressure is not being applied to the tracheal mucosa.

Table 6-1: Appropriate Tracheal Tube Sizes

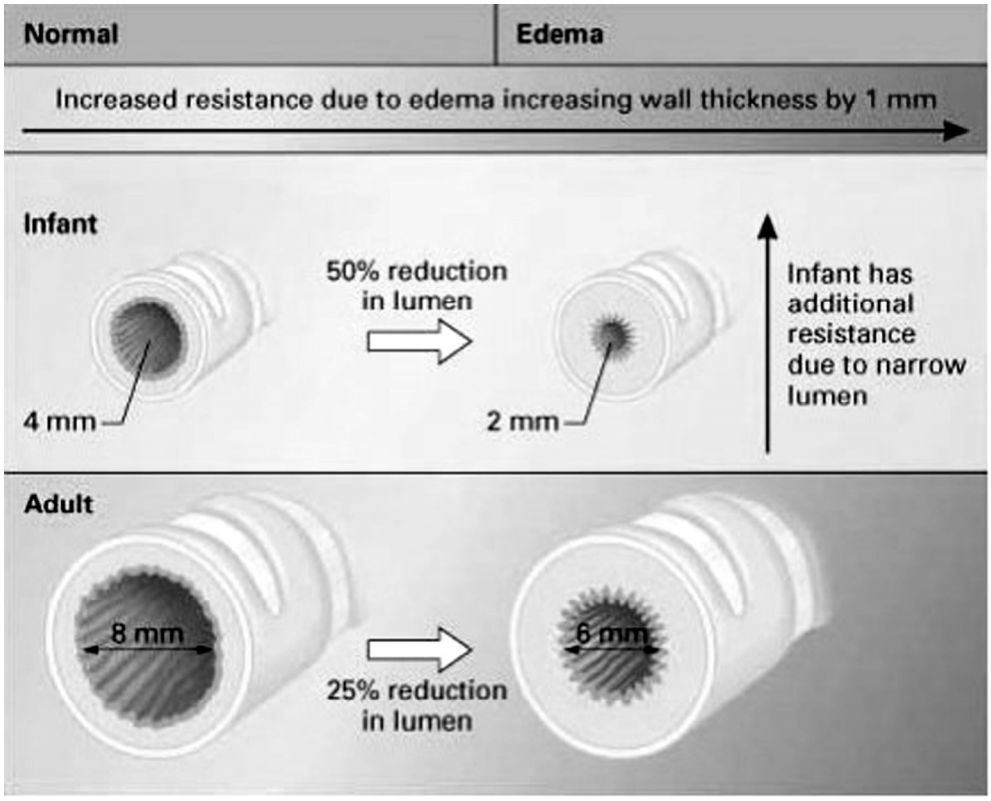
Age	Uncuffed ETT ID*	Cuffed ETT ID*	Initial ETT†	Central Line Size‡
Newborn	3.0-3.5	3.0	9-10	5-8 cm/4 Fr
1-5 months	3.5	3.0-3.5	10	5-8 cm/4 Fr
6-11 months	3.5-4.0	3.5	11	8-12 cm/4-5 Fr
1 year	4.0-4.5	4.0	12	8-12 cm/4-5 Fr
2-3 years	4.5-5.0	4.0-4.5	12-13	8-12 cm/4-5 Fr
4-5 years	5.0-5.5	4.5-5.0	13-15	8-12 cm/5.5-6.0 Fr
6-9 years	5.5-6.0	5.0-5.5	15	8-12 cm/5.5-6.0 Fr
10-12 years	6.5-7.0	6.0-6.5	17	12-15 cm/6.0+ Fr
13+ years	7.0-7.5	6.5-7.0	19	12-15 cm/6.0+ Fr

*Measured in mm.
†Depth measured at lips in cm.
‡Length is in cm, size is in French (Fr).

http://www.ebmedicine.net

This is done by slowly increasing the pressure in the breathing circuit while listening with a stethoscope at the thyroid notch for the sound of a gush of gas at airway pressures between 15-25cmH_2O. If a gas leak occurs below 25cmH_2O pressure, the anesthetist can be reasonably assured that the tracheal tube is not compromising capillary blood flow to the cricoid mucosa. Post-extubation croup that is due to mucosal ischemia and edema will be avoided. Younger patients with smaller tracheas are more likely to develop stridor if there is airway edema because 1mm of circumferential narrowing is more likely to occlude small airway lumens than 1mm of circumferential airway narrowing in a larger airway (**Figure 6-5**).

Figure 6-5: Changes in Airway Lumen Size With Edema

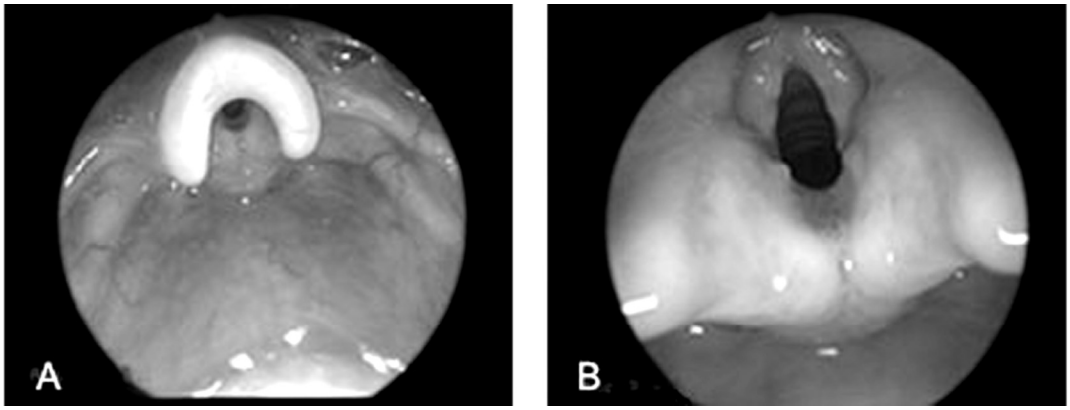


This figure shows the differences in the effects of 1mm of edema on lumen size of airways of infants and adults. *http://www.uwppc.org*

If croup does develop after tracheal extubation, aerosolized racemic epinephrine should be used to treat it. 0.25-0.5ml of 1:10,000 racemic epinephrine may be diluted in an aerosol chamber with 1.5ml saline and delivered to the patient via mask over 15 minutes. Repeated administration of racemic epinephrine may be necessary in severe cases of post-extubation croup.

Another difference between the pediatric and adult airway is the size and shape of the epiglottis. In infants it is stiffer and omega shaped (**Figure 6-6**).

Figure 6-6: Shape of the Epiglottis of Adults and Infants



In "A" the epiglottis is larger and rounder, making it easier to lift with the tip of a laryngoscope blade. In "B" the epiglottis is smaller and more "omega" shaped, making it more difficult to lift with a laryngoscope blade. In infants, it is often easier to place the tip of the laryngoscope blade in the vallecula (as is done with a curved blade in older children) and to pull up and out and not attempt to lift the epiglottis. http://chop.edu

The infant epiglottis may be difficult to manipulate with the tip of a laryngoscope blade compared to the adult epiglottis. The relatively longer Miller blade allows the anesthetist to pick up the epiglottis to have a good view of the vocal cords of younger patients. The larger, softer epiglottis of adults is usually easily moved with gentle laryngoscope blade pressure in the vallecula, which is the space anterior to the epiglottis. The relatively shorter MacIntosh blade is designed for this purpose (**Figure 6-7**).

Figure 6-7: The Curved Macintosh in Place in the Airway

The tip of the MacIntosh is in the vellecula and the blade is pulled up and out at a 45 degree angle. This lifts the tongue and mandible to provide a view of the glottis. http://comps.fotosearch.com

In toddlers and school age children, either a Miller 2 or MacIntosh 2 blade works well for intubating their tracheas, while a Miller 0 blade is appropriate for newborns and premature babies; a Miller 1 blade is appropriate for infants.

The trachea is shorter in children than it is in adults. Correct positioning of the tracheal tube places its tip in the mid-point of the trachea. Uncuffed TT's have centimeter markings on their sides to indicate the distance the tube tip has been advanced into the trachea with respect to the patient's gums, assuming the head is in the neutral position. For cuffed TT's, the entire cuff is placed just beyond the vocal cords under direct laryngoscopic vision. The depth of the tube relative to the gums or teeth should be noted immediately after removing the laryngoscope from the mouth so that the tube can be secured in this position with tape. During flexion or extension of the head, TT's may move several centimeters in the trachea (**Figure 6-8**) if the tip of the tube is

not in the mid-trachea, accidental tracheal extubation or mainstem bronchus intubation can occur (**Figure 6-9**).

Figure 6-8: Movement of a Tracheal Tube in the Trachea With Flexion and Extension of the Head

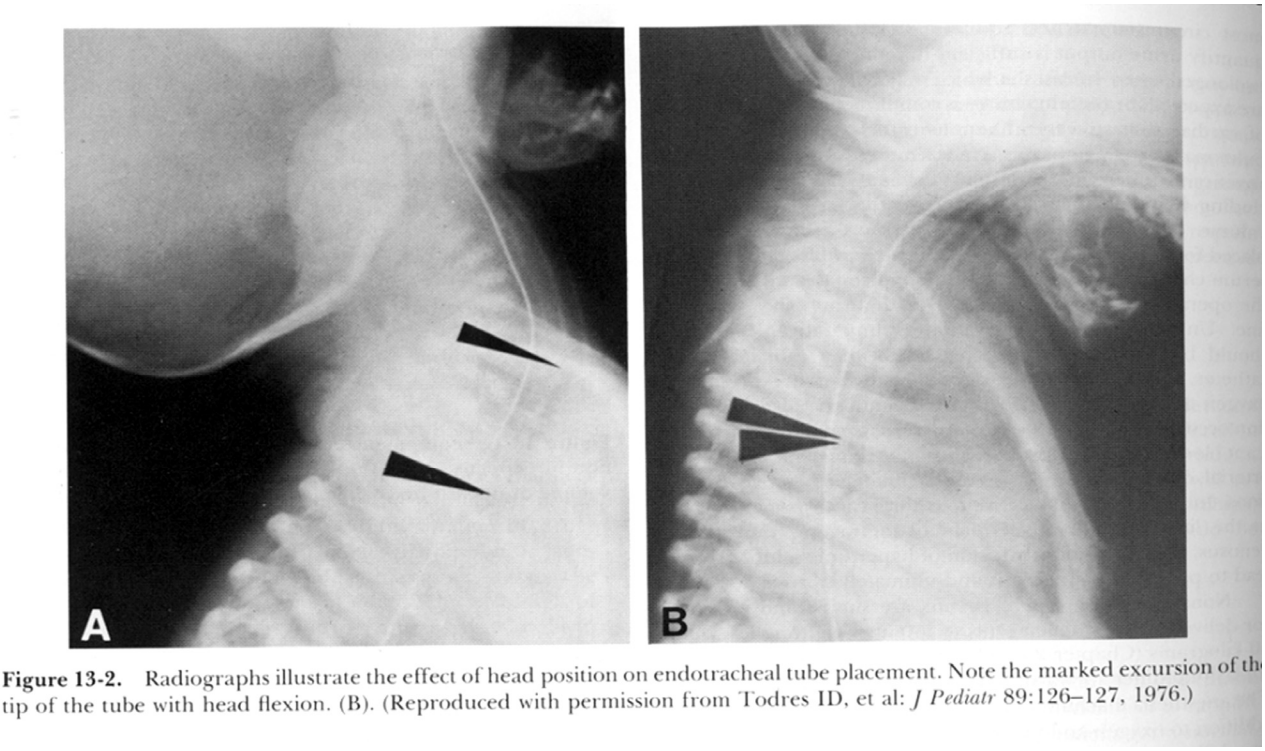
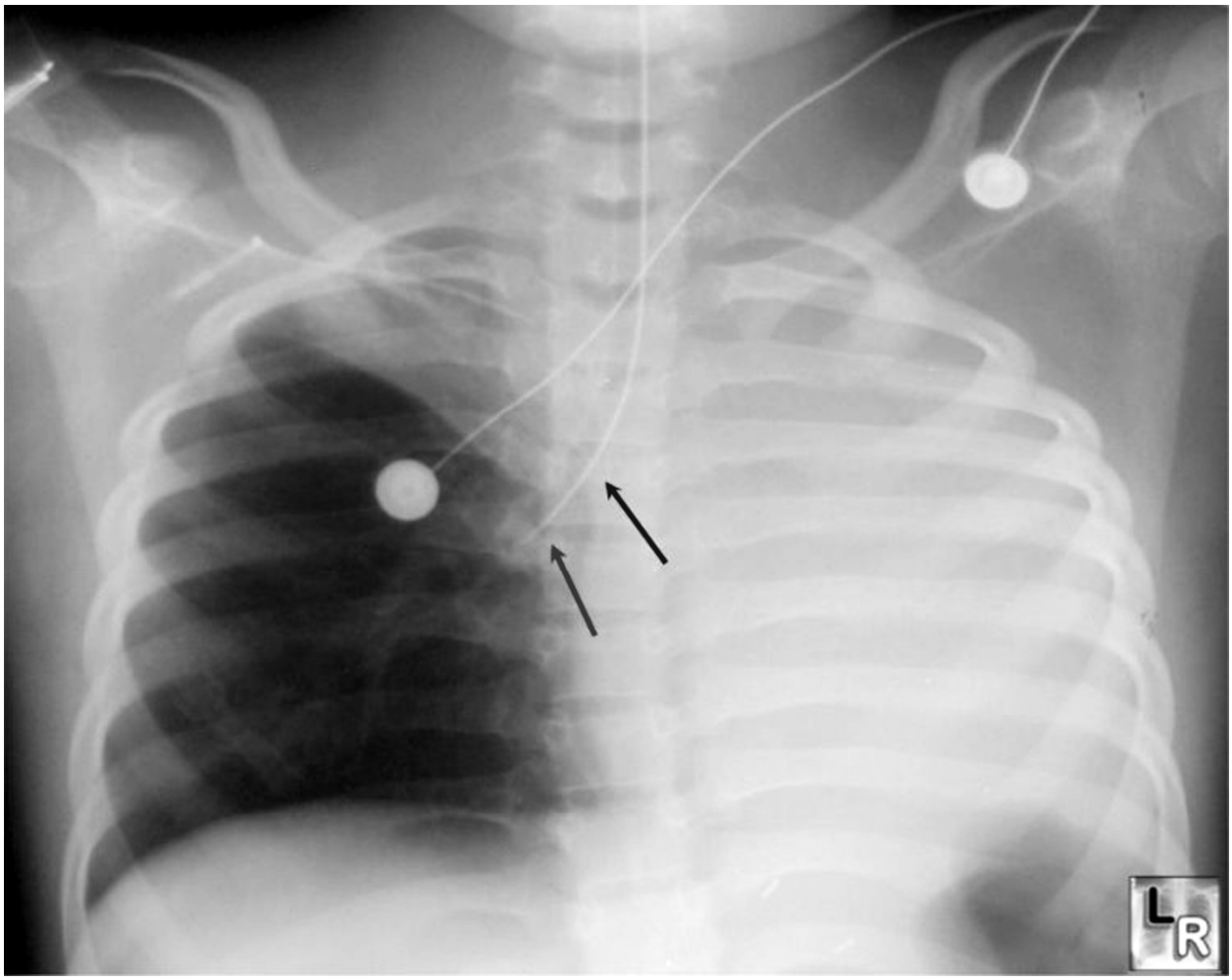


Figure 13-2. Radiographs illustrate the effect of head position on endotracheal tube placement. Note the marked excursion of th tip of the tube with head flexion. (B). (Reproduced with permission from Todres ID, et al: *J Pediatr* 89:126–127, 1976.)

These figures show movement of the tip of the tracheal tube with movement of the head. Reproduced with permission from Todres ID, J. Pediatr 1976;89:126-7

Figure 6-9: Tracheal Tube in Right Main Stem Bronchus



This photograph shows the tip (red arrow) of a tracheal tube in the right main stem bronchus, which has caused collapse of the left lung and the right upper upper lobe of the lung. See arrows. http://www.learningradio-logy.com

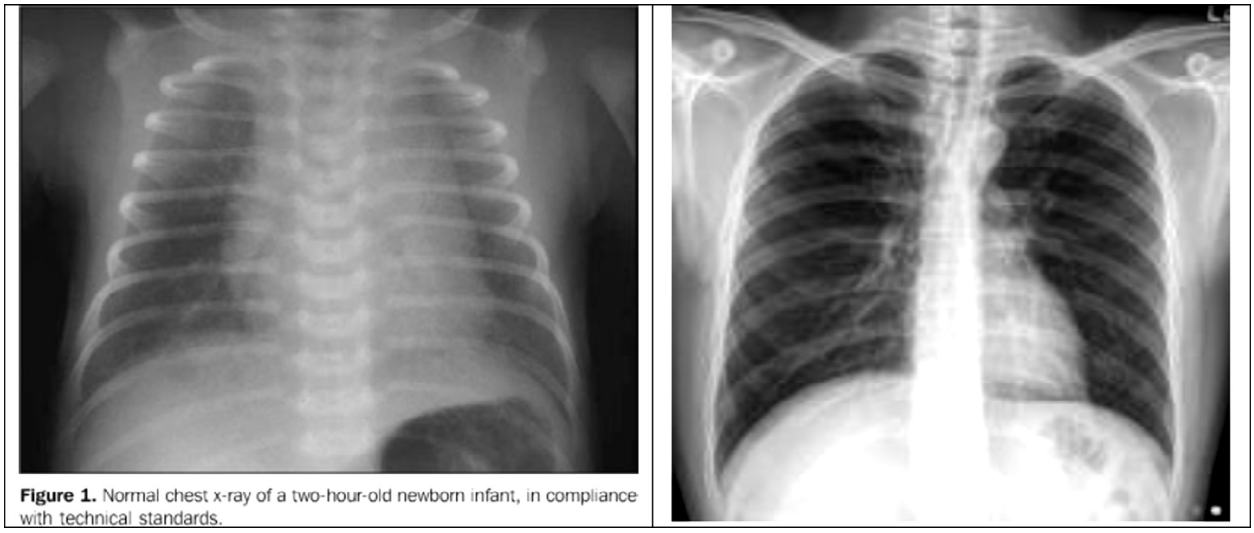
Pediatric patients' lungs have fewer and smaller alveoli than adults (**See Chapter 8)**. New alveoli continue developing until about eight years of age. After that they merely increase in size until the child reaches adulthood. Despite having fewer alveoli, oxygen requirements and carbon dioxide production are greater in neonates and infants because their metabolic rates are larger. While adults consume 2-3cc/kg/min of O_2, babies consume more (6-10cc/kg/min). To meet these needs, small patients require more minute ventilation per kilogram of body weight. The tidal volumes of adults and children are similar (6-10cc/kg) during positive pressure ventilation. Therefore, higher respiratory rates are needed to provide adequate minute ventilation in infants. Functional residual capacity (FRC) is the volume of gas in the lungs at end-expiration. FRC is important for two reasons: 1) it allows oxygenation to continue during the expiratory phase of breathing, and 2) less respiratory work is required to expand the non-collapsed lung during inspiration. An adequate FRC is the key to oxygenation. The FRC is similar in pediatric and adult

patients. During anesthesia the FRC of infants, children, and adults decreases, which compromises oxygenation. Physiologic dead space (non-gas exchanging portions of the respiratory tree, including the trachea, bronchi, mouth, and sinuses) is similar in children and adults, about 30% of tidal volume.

The tracheas and bronchi of pediatric patients are smaller in diameter and more compliant than those of adults. During spontaneous inspiration, the slightly negative intrathoracic pressure (about -5cmH_2O) dilates intrathoracic airways but slightly narrows the extrathoracic trachea. Patients who have extrathoracic airway obstruction (e.g., croup or epiglottitis) markedly narrow the extrathoracic trachea during vigorous crying or breathing because both conditions increase the amount of negative inspiratory pressure. This dynamic airway collapse results in stridor. The narrower and more compliant intrathoracic bronchi of pediatric patients are more likely to obstruct during forced expiration (bronchiolitis, asthma).

Pediatric chest walls are more compliant than those of adults because the ribs of neonates contain less calcium than those of adults. In neonates, infants and toddlers, the intercostal muscles and diaphragms are also weaker. During spontaneous respiration, the horizontal rib position of young children provides less mechanical advantage during inspiration than the more diagonal ribs of adults (**Figure 6-10**).

Figure 6-10: Rib Position of a Young Child (Left) and Adult (Right)



The X-ray on the left shows the flatter ribs of a neonate, which provide less advantage during inspiration. The film on the right shows the more curved (bucket handle) ribs of the older child and adult, which lift and move outward during inspiration http/:www.radiopaedia.org

There is less muscle and more cartilage in the chest walls of pediatric patients, making them more compliant. This rib configuration makes it harder for children with pulmonary edema or pulmonary disease to expand their chests during spontaneous breathing. In addition, the

relatively large abdominal organs of pediatric patients can push up against their diaphragms and prevent the diaphragm from descending appropriately. Taken together, all of these factors increase the respiratory work of young children.

Perioperative and Intraoperative Airway Management

Preoperative assessment includes recognition of airway abnormalities, such as asthma, pulmonary infections, or tobacco smoke exposure in the home. Active asthma or recent or current airway infection may be reasons to delay elective surgery, although many factors enter into such a decision (**See Chapter 18**).

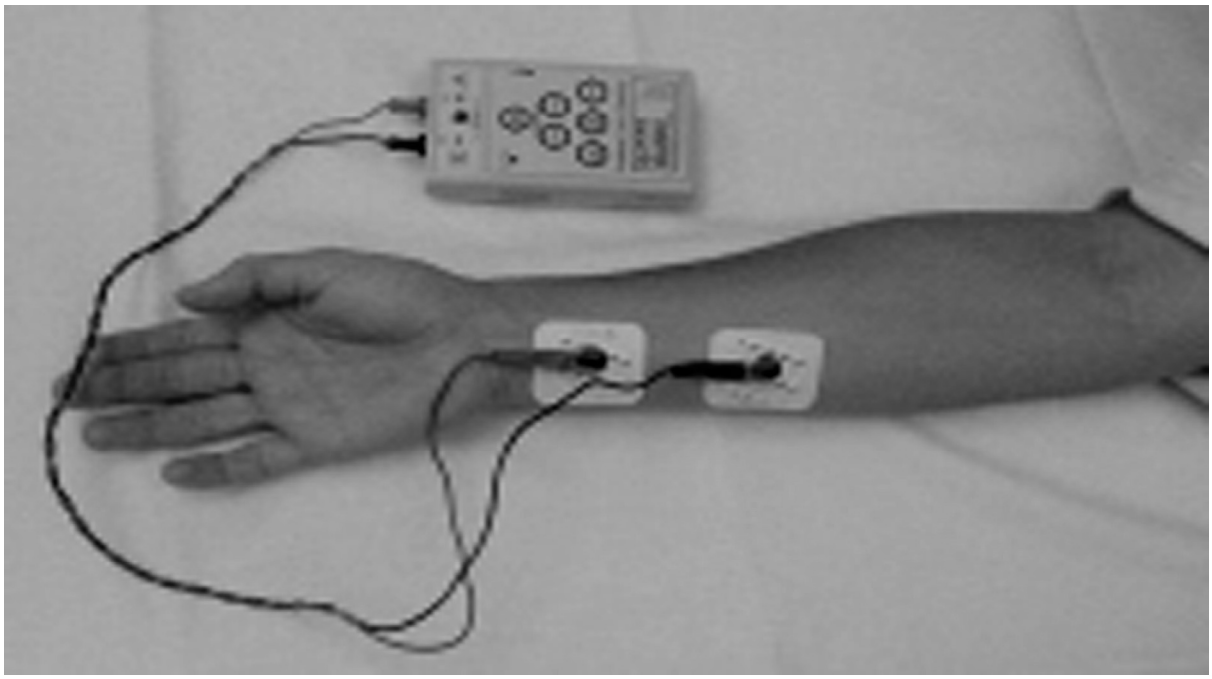
Particular attention should be paid to patients with syndromes that affect the airway, such as Down syndrome, Pierre-Robin syndrome, and Treacher-Collins syndrome. Patients with these syndromes may have difficult airways that require extra planning for anesthesia (see below).

Pediatric patients scheduled for elective surgery should have food and liquids withheld for appropriate amounts of time. For healthy children, this includes two hours for clear liquids at all ages, four hours for breast milk or formula for infants less than six months of age, six hours for food and non-clear liquids for those who are more than six months old. Scheduling younger patients for surgery early in the day and allowing them to drink clear liquids until two hours before anesthesia induction prevents dehydration, particularly in hot climates, while preventing aspiration during airway manipulations. These nul per os (NPO) times and the rationale for them should be made clear to parents, who may be reluctant to withhold food from their hungry, crying child. For emergency surgery, the anesthetist may have to accept the increased risks of a full stomach, but should plan to use a rapid sequence induction of anesthesia. Gastric emptying ceases in patients who are in pain, so the NPO time for a child with a traumatic fracture will be the time between the last meal and the accident, not the time between last meal and presentation in the operating theater.

*Monitoring***:** Pulse oximetry is a required monitor for patient safety during anesthesia (Life box). Having an audible tone on the pulse oximeter allows the anesthetist to “hear” the level of oxygen saturation while carrying out all other anesthesia tasks. As the oxygen saturation (SaO_2) decreases, the monitor tone decreases. When SaO_2 increases, the tone increases. Airway pressure measurement is built into modern anesthesia machines and is incorporated into many anesthesia monitors. It tells the anesthetist when the pressure in the system decreases, e.g., if the oxygen tank is empty. Measuring the oxygen concentration of inhaled gases and linking this measurement to an alarm tells anesthetists if the concentration of oxygen in the circuit is low. End-tidal CO_2 analysis, if available, is very helpful for confirming that the airway is patent and that the patient is breathing. Newer anesthesia machines and ventilators simultaneously measure inhaled and exhaled oxygen, CO_2, nitrogen (air), anesthetic gas concentrations, and tidal volumes. This information is very helpful when tailoring an anesthetic and should be used if available.

Standard cardiovascular monitoring with EKG and blood pressure measurements are also essential for safe airway management (**See Chapter 2**). If neuromuscular blocking agents are to be used, having a neuromuscular blocking monitor will be very helpful, particularly for determining if the effects of muscle relaxants have been adequately reversed prior to tracheal extubation (**Figure 6-11**).

Figure 6-11: A Neuromuscular Blockade Monitor



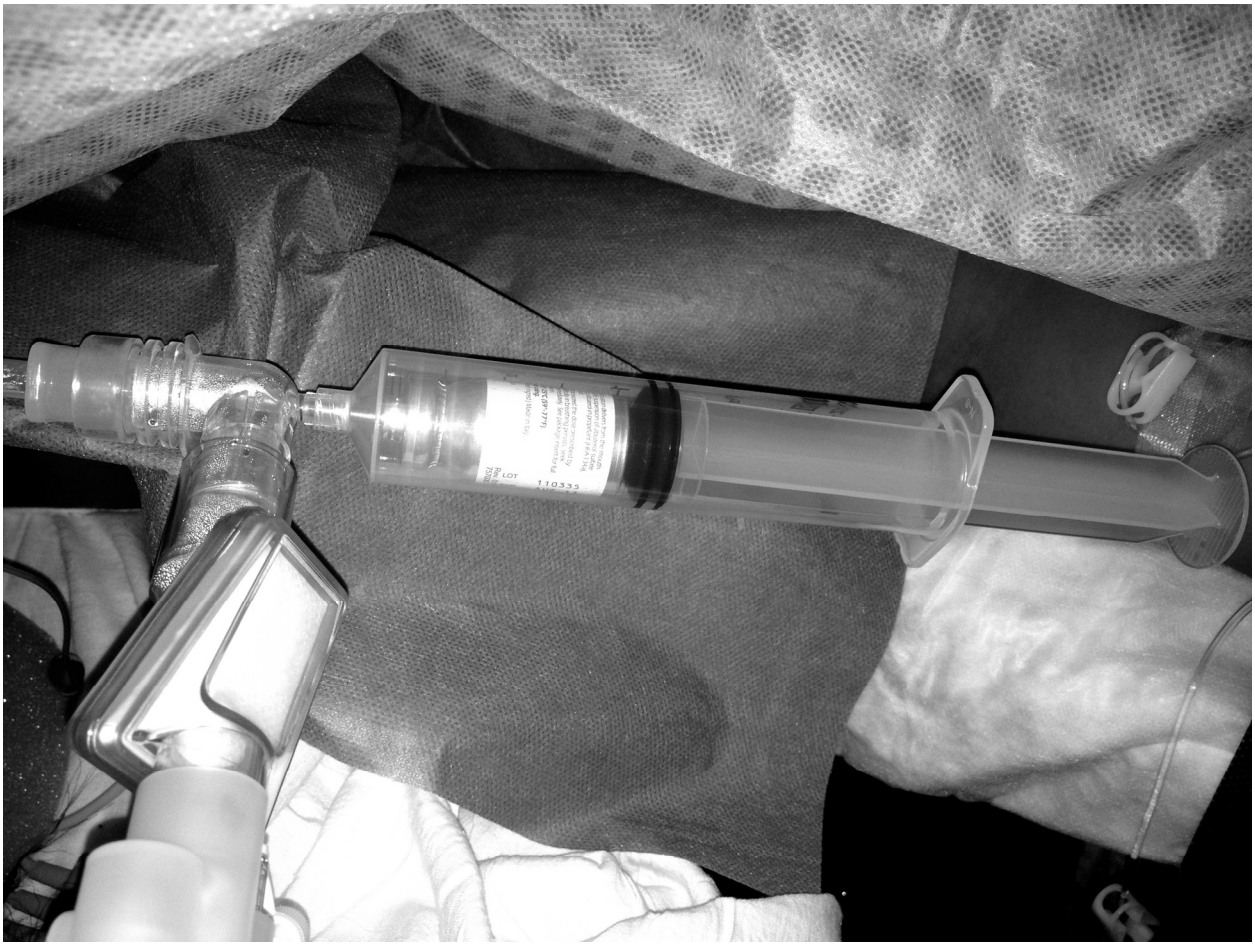
The system for determining the degree of neuromuscular blockade is shown above. Two electrodes are attached to a patient's forearm along the pathway of the ulnar nerve. The nerve is stimulated with short electrical pulses and movement or no movement of the thumb observed. Before a muscle relaxant is administered, the anesthetist determines the amount of movement (twitch) and bases observations after giving muscle relaxants or reversal drugs on an estimated percentage of the original twitch amplitude. If it is above 95%, most patients will sustain respiration on their own.

Equipment: Standard equipment for managing a pediatric patient's airway includes an anesthesia machine with oxygen, a breathing circuit and appropriate sized facemasks, a vaporizer with sevoflurane or halothane, a suction machine with rigid plastic yankauer suction tips and soft suction catheters, oral airways, a laryngoscope handle and blades, appropriate sized tracheal tubes, a stethoscope, and tape for securing tracheal tubes. Intravenous fluids and materials for placing intravenous lines are also essential for safe airway management (**See Chapter 2**). Laryngeal mask airways (LMAs), nasal airways and advanced airway devices provide the anesthetist with safety and flexibility when treating patients who have difficult airways. Scavenging anesthetic gases benefits all personnel in the operating theater.

Medications: For most pediatric patients, a simple anesthetic (including a plan for postoperative analgesia) is the safest choice. It is better to use a small number of drugs that the anesthetist understands than to have many drugs whose effects may be unknown or complicated. Consequently, an anesthetic that is based primarily on a reversible inhalation agent (halothane, sevoflurane) plus a short acting intravenous induction agent (pentothal, propofol), perhaps a small dose of narcotic, and perhaps a paralytic agent and drugs to reverse their effects (reversal agent) are all that are needed. The use of multiple sedatives, hypnotics, and vasopressor agents that might be appropriate for adults, may cause untoward reactions in pediatric patients. Because of the wide range of responses in children to hypnotic and narcotic drugs, use of these drugs may result in an obtunded patient at the end of anesthesia and the potential for airway compromise.

Emergency bronchodilator medications, such as albuterol and terbutaline, should always be immediately available in case a patient develops bronchospasm. An airway device for delivering the inhaled bronchodilator (**Figure 6-12**) must also be immediately available. Terbutaline can be given intramuscularly. Potent inhalational anesthetics and ketamine also have bronchodilator activity. Other emergency drugs required for safe airway management include neuromuscular blocking agents and atropine.

Figure 6-12: A Simple. Effective Method for Delivering Inhaled Bronchodilators



The 60ml syringe contains a canister of albuterol. During inspiration the plunger of the syringe is pushed two or three times and the inspired gas carries the bronchodilator released into the lungs. This maneuver is usually repeated with two or three separate breaths. The patient is then reevaluated and consideration given to administering more albuterol if needed. Basics of pediatricanesthesia.files.wordpress.com

Induction of anesthesia: It is important for the anesthetist to gain the trust of each pediatric patient and to keep a calm, reassuring environment around the child during induction of anesthesia. For toddlers, the presence of parents, if permitted by operating theater rules, may provide the most assurance. However, the parents must understand that they are to leave the operating theater as soon as the child is "asleep". For older children, playing games, such as blowing up the balloon (ventilation bag) or telling calming stories about the child's favorite activities, may distract her/his attention and prevent anxiety during induction of anesthesia.

Choice of whether to induce anesthesia with inhalation agents, intravenous agents or intramuscular injection of anesthetic drugs depends on many factors, including the age and maturity of the child, NPO status, comorbidities, surgical procedure, and body habitus. Infants and toddlers can be rapidly and painlessly anesthetized with sevoflurane or halothane because

these anesthetics have high minute ventilation and low muscle and body fat composition. The amount of time spent in the excitement phase during induction of anesthesia is fairly short, minimizing the risk of laryngospasm. It is important for the anesthetist to carefully observe the child pass from the first to second (excitement) stage of anesthesia where the eyes become disconjugate (looking in different directions), when tachycardia and tachypnea occur, and when body movements are common; they then pass to the third stage of anesthesia when the eyes are again conjugate, respirations become regular, heart rate slows, and movements cease. Positive pressure ventilation often replaces spontaneous ventilation at this point. An IV catheter can be inserted more easily once the child is anesthetized because commonly used anesthetics dilate the veins. Isoflurane and desflurane *should not* be used for inhalation induction of anesthesia because they irritate airway epithelium and precipitate laryngospasm or bronchospasm in 25-80 percent of children.

Obtaining intravenous access may be difficult in young, uncooperative patients. If it is deemed that induction of anesthesia with parenteral agents, such as ketamine or midazolam, will be safer for the patient than an inhalation induction, intramuscular injection of ketamine 5-8mg/kg is helpful. However, intramuscular injections are painful, due to the volume of fluid injected and drug pH. An alternative method for inducing anesthesia for more fragile pediatric patients is to administer oral midazolam 0.5-1mg/kg (maximum dose 15mg) to sedate her/him and then obtain vascular access.

Healthy school age children may prefer a painless inhalation induction of anesthesia or they may tolerate IV placement with or without oral premedication. Teenagers frequently have anesthesia induced by the intravenous route because their more mature body composition and lower alveolar ventilation prolong the time needed for inhalation induction, increasing risk of laryngospasm or other airway obstructions. Teens who are fearful of needle sticks frequently tolerate IV placement while breathing nitrous oxide, if it is available.

Pediatric patients with full stomachs should undergo rapid sequence IV induction and tracheal intubation with cricoid pressure held until the tracheal tube is confirmed to be in the correct position. Because younger patients have high oxygen consumptions, oxygen desaturation may occur if the patient remains apneic until the neuromuscular blocking agent takes effect. For this reason, the anesthetist may modify the rapid sequence induction/intubation by gently ventilating the patient's lungs with a bag-and-mask for 60-90 seconds while waiting for the onset of muscle paralysis. Neonates or weak infants may have their tracheas intubated awake if they are at risk for aspiration of their gastric contents, (e.g., a bowel obstruction). Additional oxygen may be provided during tracheal intubation by taping a plastic catheter for O_2 delivery to the blade of the laryngoscope and delivering one or two liters of oxygen per minute through the catheter (**Figure 6-13**).

Figure 6-13: A Method for Delivering Oxygen During Tracheal intubation.

This is a miller blade for neonates. A tube connected to oxygen has been taped to the top of the blade. It should be taped away from the light if possible. 0.5 liters of oxygen flows through the tube and provides increased inspired oxygen during tracheal intubation. If this is done in older children, the oxygen flow should be 1 liter per minute.

Patients with significant comorbidities, such as heart failure or severe respiratory disease, will be safer when an intravenous line is in place during induction of anesthesia. IV access allows use of a wider choice of anesthetic agents and a route for rapid delivery of fluids and resuscitation medications if needed.

Obese patients and those with abnormal airways are also more safely anesthetized when they have an IV in place for administration of anesthetic agents and rescue medications when needed. Sometimes it is difficult to see the veins of awake, obese children (**See Chapter 2**). In this case, an antecubital vein is often the easiest site for venous cannulation; inhalation of nitrous oxide may help with the process.

Following induction of anesthesia, the anesthetist secures the airway. For some minimally invasive procedures, an intravenous anesthetic may be chosen, and nasal cannula oxygen is supplied. For short procedures, oxygen can be delivered through a mask to spontaneously breathing patients. For longer and more invasive procedures, laryngeal mask airway (LMA) or tracheal intubation may be needed (**See Chapter 7**).

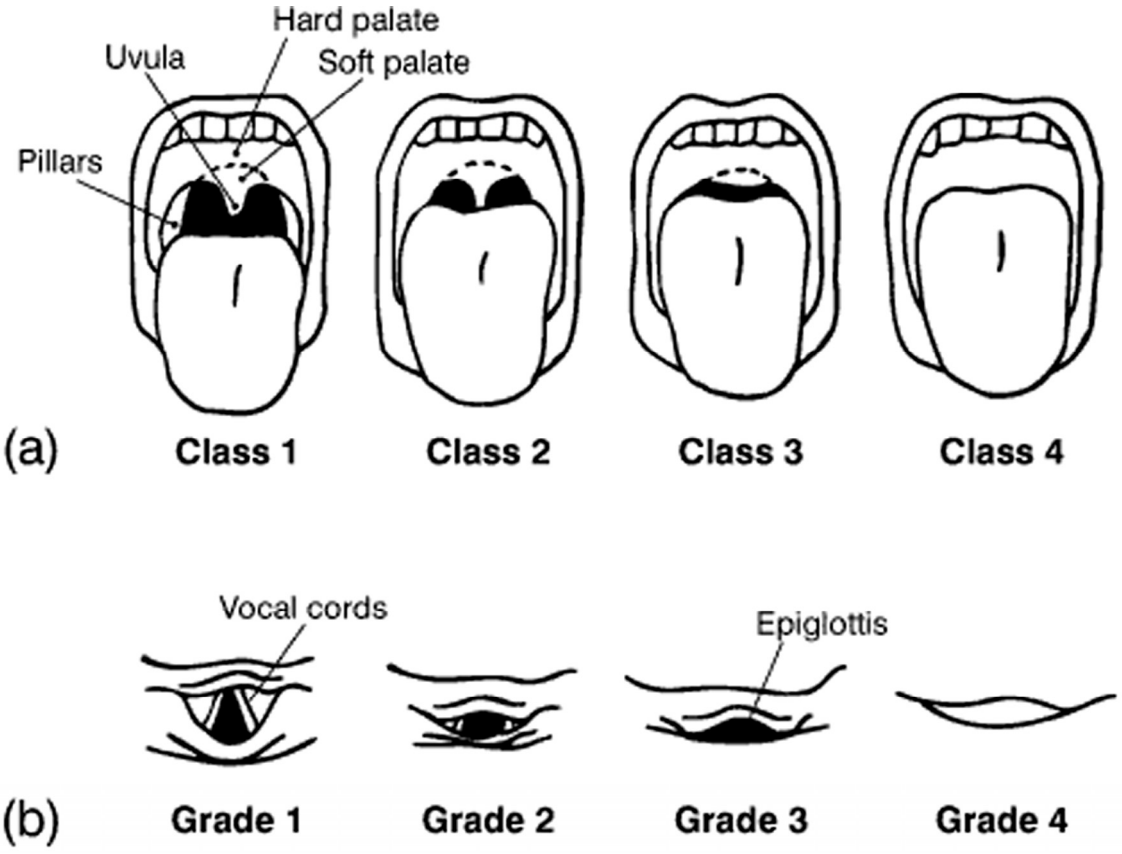
Once pediatric patients have passed through the excitement phase to the third stage of anesthesia and an IV is in place, administration of intravenous propofol, barbiturates, and/or rapid acting narcotics quickly deepens the anesthetic and facilitates tracheal intubation or placement of a LMA. Paralysis may make tracheal intubation easier in larger children but is often unnecessary in properly anesthetized smaller children. A non-depolarizing muscle relaxant is preferred to succinylcholine because of succinylcholine's risk of causing bradycardia and hyperkalemia in some children.

Airway Obstruction During Induction of Anesthesia: As their tongue and pharyngeal muscles relax, pediatric patients commonly develop airway obstruction during induction of anesthesia. Positive pressure ventilation or placement of an oral airway during the second stage of anesthesia is avoided because it can precipitate laryngospasm. A safer manoeuver for overcoming airway obstruction is to apply 5-10cmH_2O of continuous positive airway pressure while lifting the jaw forward. This pulls the tongue away from the posterior pharynx and allows gas to pass behind the tongue. If this manoeuver fails to relieve the obstruction, then an appropriately sized oral airway (**see above**) is inserted. If the oral airway does not relieve the obstruction or if the anesthetist believes that the patient has laryngospasm, he/she should apply a continuous high pressure 10-20cmH_2O via the mask. If this is unsuccessful, intramuscular (IM) or intravenous muscle relaxant must be administered quickly. IM succinylcholine 4-5mg/kg should be accompanied by IM atropine 0.01 mg/kg to prevent vagally mediated bradycardia. IM injections should be given into a deltoid muscle (not leg muscles) as it takes twice as long to absorb drugs from the legs and buttocks as it does from the deltoid muscles. If circulation is poor (due to hypoxia), the drugs should be injected into the muscle of the tongue (**See Chapter 7**). The vocal cords should begin to relax in <15 seconds.

Tracheal intubation: An appropriate sized laryngoscope blade and tracheal tube should be selected prior to induction of anesthesia. Various guidelines are used to choose the correct size TT. Use of a 3.0 TT in a newborn, a 4.0 tube in a one year old, and a 5.0 tube in a five year old is simple to remember. The formula of age divided by 4, plus 4 [(age/4) + 4] or age + 16 divided by 4 [(age+16)/4] quickly gives the approximate TT size for most older children. Matching TT size to the widest part of the fingernail of the patient's fifth digit is also useful. Each of these methods only gives the approximate size tube to use, so tubes of different sizes should be immediately available in case the tube chosen is too large or too small. Similarly, laryngoscope blades of different sizes in both Macintosh and Miller styles should be immediately available. If one style does not work, the other may. Intubation of the trachea of patients who have normal anatomy is most easily accomplished with the patient's head in neutral, "sniffing" position and by opening the mouth widely. Crossing the thumb and third fingers of the right hand and pushing the upper and lower gums or teeth apart accomplish the latter. The laryngoscope blade is then gently introduced into the mouth over the right side of the tongue and advanced until the tip of the blade lies near the posterior pharynx. This manoeuver moves the tongue leftward. The fingers of the right hand are removed and the laryngoscope handle is gently but firmly pulled upwards and outwards toward the patient's chin at a 45-degree angle. Levering the laryngoscope blade against the upper teeth or catching the lip between the teeth and laryngoscope blade must be avoided, as this causes unwanted injury. Now the epiglottis should be easily seen. If not, the blade can be gently advanced or withdrawn until the vocal cords and/or epiglottis come into view. The tip of the blade should be placed into the vallecula and gently tipped up to improve the view of the vocal cords (**video** endotracheal intubation). Alternatively, a Miller blade can be inserted in the same

way and advanced to pick up the epiglottis if it obscures the anesthetist's view of the vocal cords. Laryngoscopic views of the vocal cords may be assigned a grade to communicate information about the ease of intubation to future anesthetists (**Figure 6-14**).

Figure 6-14: Grade of View of the Airway and the View Seen at Laryngoscopy



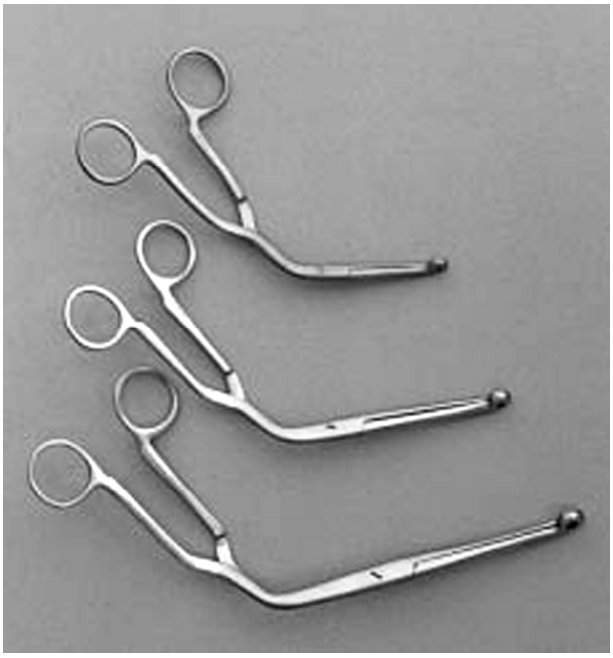
This figure shows the views of the airway using the Malampati classification (a). A Class 1 view shows the uvula, hard and soft palate, and the tonsillar pillars. In Class 4, only the hard palate is seen. Class 2 and 3 show progressive loss of these structures. (b) shows the view of the vocal cords with each Malampati grade from clear in Class 1 to not at all in Class 4. This scoring system is useful when attempting to determine if a child has a difficult airway. ***http:www.scielo.br/image/revistas.jped***

Once the anesthetist has a clear view of the glottis, a TT is inserted into the mouth to the right of the laryngoscope blade, not through the laryngoscope channel, which would block the anesthetist's view of the vocal cords. The tube is then passed through the vocal cords. Both lungs are auscultated near the axillae; the abdomen is auscultated over the stomach to assure the TT is in the trachea and not in the esophagus. The presence of continuous end-tidal CO_2 confirms correct positioning of the TT in the trachea. However, anesthetists must be aware that the sounds caused by insufflation of the stomach can be transmitted to the lungs, that some amount of CO_2 may appear in gas from the stomach for a few seconds (**See Chapter 2**), and that breath sounds may be transmitted from one lung to another if a main stem bronchial intubation occurs. If there

is *ANY* question in the anesthetist's mind about the position of the TT, laryngoscopy, and possibly tracheal re-intubation, should be done. To place the tip of the TT in the mid trachea, the tube can be temporarily advanced into a main stem bronchus and the difference in breath sounds between right and left lungs determined. The TT is then slowly withdrawn until the breath sounds are equal on both sides of the chest. The location of the carina has now been determined. The TT is then withdrawn an additional 1-2 cm, depending on the child's size, to place the tube's tip in the mid-trachea. TT's easily go into the right bronchus of children, but they easily go into either main stem bronchus in infants.

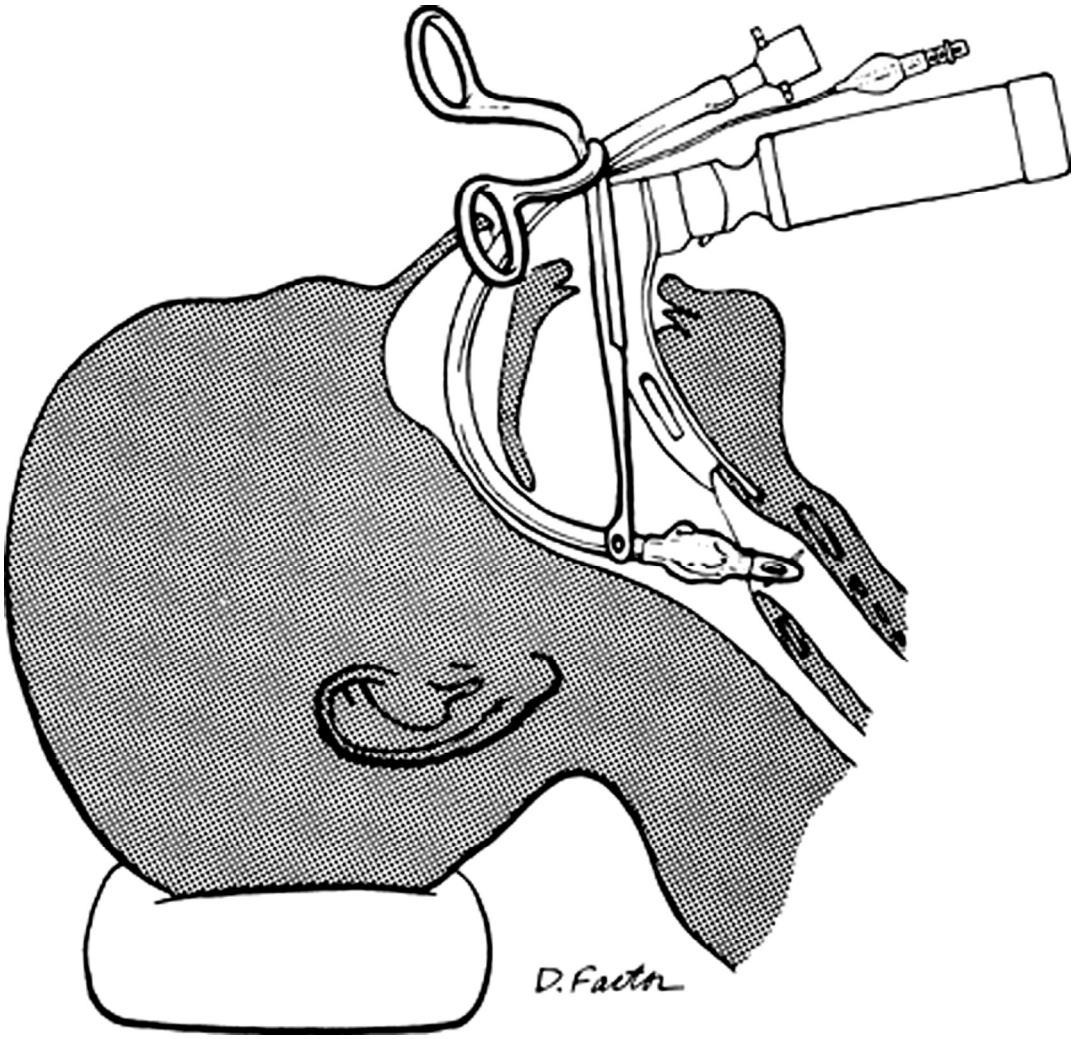
Placement of an oral TT is appropriate for most surgical procedures, but patients who are having facial or dental surgery may benefit from a nasal tracheal intubation. Softening the TT in warm water and lubricating it with gel reduces the amount of trauma caused to the nasal passage and adenoids. Vasoconstricting agents have been used to prevent bleeding produced by nasal intubation, but these drugs are best avoided when possible because their systemic absorption can cause hypertension and tachycardia. The TT is gently passed into the nostril and advanced until it reaches the posterior pharynx. Then under direct laryngoscopic vision, the tip of the TT is grasped with a Magill forceps or hemostat (**Figure 6-15**) and directed through the vocal cords into the trachea (**Figure 6-16**).

Figure 6-15: Magill Forceps



When available, the Magill forceps are ideal for grasping the tracheal tube in the pharynx and directing it through the vocal cords into the trachea. The three different sizes are designed for infants (top), children (middle), and adults (bottom). When Magill forceps are not available, a curved hemostat of appropriate length can be used.

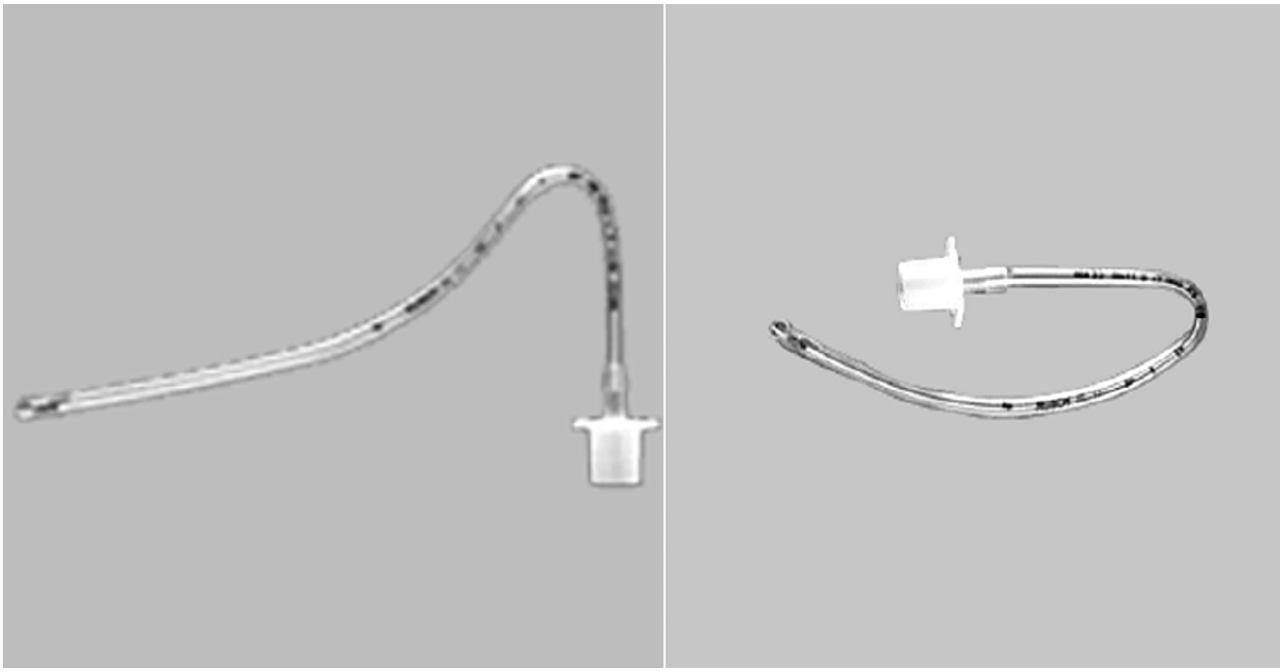
Figure 6-16: Tracheal Tube Being Directed Through Vocal Cords Into the Trachea



The figure shows a tracheal tube being grasped with a Magill forceps and being directed into the trachea. Once the tracheal tube is in the trachea, the forceps are removed. If a Magill forceps is not available, the tube can be directed into the trachea with a curved hemostat of appropriate length. http://www.ezio.com

Preformed nasal RAE tubes (**Table 6-17**) are appropriate for some facial surgeries. An oral RAE tube (**Figure 6-18**) is an excellent choice for cleft lip and cleft palate repairs and for adenoidectomy and tonsillectomy because these tubes provide the surgeon with better access to the surgical field and because there is less likelihood the surgeon will accidently dislodge the tube.

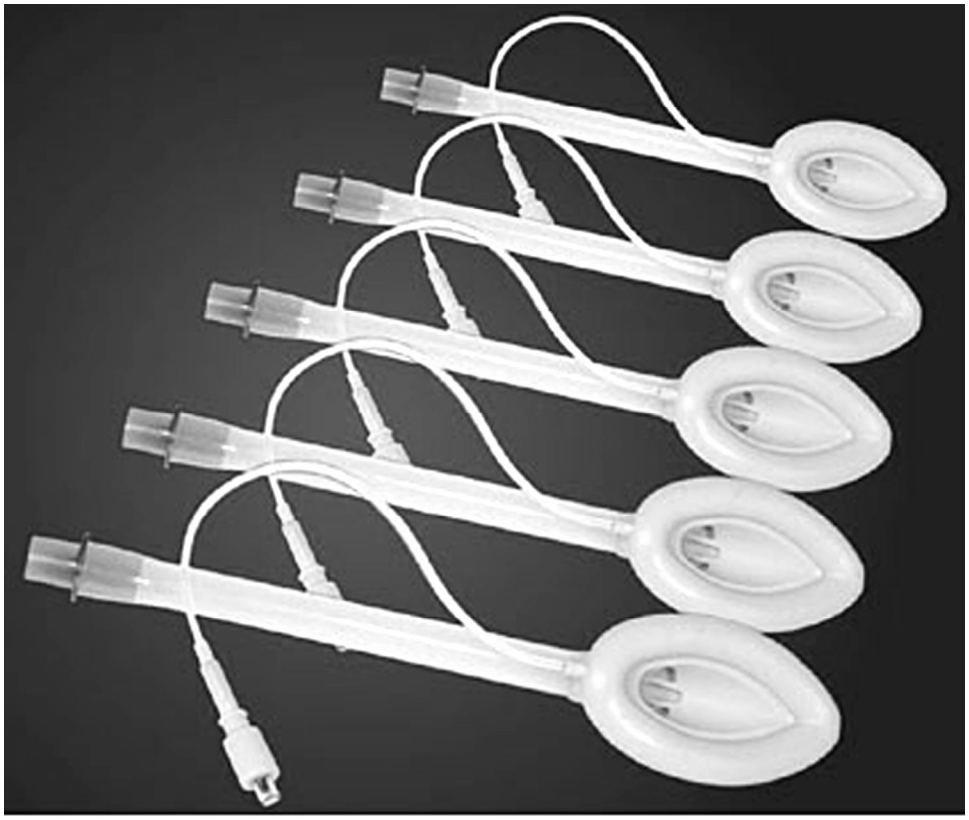
Figure 6-17: Nasal and Oral RAE Tracheal Tubes



The tracheal tube on the left is longer distal to the bend to account for the distance through the nose when the tube is placed through the nose. The distal portion of the tracheal tube on the right is shorter and is used for oral intubations. In both instances, the connector is positioned away from the surgeon, which improve her/his view into the mouth.

Anesthetists throughout the world are using *laryngeal mask airways (LMA)* more frequently. These devices come in several styles and sizes. A few LMA's are configured for the different anatomy of the pediatric pharynx, but smaller sized LMAs are simply scaled down versions of adult LMAs (**Figure 6-18**).

Figure 6-18: Laryngeal Mask Airways



Various size LMAs. Note that they all have the same shape and do not account for the structural airway differences in infants and young children. Consequently, it is often more difficult to get an appropriate seal between the LMA cuff and the glottis in infants. This may make it more difficult to generate sufficient pressure to ventilate the lungs of some young children.

Thus, the smaller the patient, the less likely a LMA will seal the glottis. Once the technique for insertion is learned, LMA's are quite easily placed. They relieve most upper airway obstructions and allow spontaneous or gentle positive pressure ventilation. It is important that LMA's fit properly and that gas leaks around the LMA cuff when less than 20cmH_2O pressure is applied. This prevents pain and pressure-induced ischemia of the pharyngeal mucosa. LMA's are also important tools for emergency management of the difficult airway (**see below**). LMA's are not appropriate if there is a risk of aspiration (full stomach, gastroesphageal reflux) and should be used cautiously for laparoscopic or open abdominal procedures. LMA's can be placed in anesthetized patients by gliding the lubricated, deflated, or semi-inflated mask into place with one or two fingers placed on top of the mask. However, for small pediatric mouths, it may be easier to hold the LMA near the end that connects to the anesthesia circuit and advance the LMA with steady, gentle pressure while the first and second fingers of the other hand grasp the lower teeth or gums and pull the mandible forward (see video). If the LMA does not easily pass through the posterior pharynx, the mask can be rotated 90 or 180 degrees in the pharynx, then de-rotated

into the correct position after it has passed the base of the tongue. As with tracheal intubation, the chest and upper abdomen are auscultated to assure that the LMA is providing gas to the lungs and not the stomach.

Maintenance of a patent airway is always the key to safe anesthesia. When the patient is moved, when surgical drapes push on the TT or LMA, or when abdominal retractors are used, it is possible to displace or kink the tube. The only protection against this is to be constantly vigilant of vital signs, tidal volume, airway pressures, and the CO_2 tracing. Early recognition of problems allows time to safely correct them before a disaster occurs. Auscultation in the right and left axillae should be performed whenever there is concern about TT position or there are changes in airway pressures. Bronchospasm is readily appreciated by auscultation.

Intraoperative ventilation: The anesthetist must decide whether controlled ventilation or spontaneous ventilation is best for each pediatric patient being anesthetized. For abdominal or thoracic surgery, positive pressure ventilation via a tracheal tube is almost always appropriate. For limb, head, neck, and genitourinary procedures, spontaneous ventilation may suffice if muscle paralysis is not required. If a laryngeal mask airway is used, spontaneous ventilation is usually adequate but will be associated with some elevation of end-tidal CO_2 and CO_2 rebreathing because these devices add additional dead space, and anesthetic drugs depress respiration. Positive pressure ventilation via a tracheal tube requires a well maintained ventilator that reliably provides the small tidal volumes and more rapid rates required by pediatric patients. End-tidal CO_2 monitoring makes mechanical ventilation safer, but must not be entirely relied on, especially in small babies in whom the amount of CO_2 in a small exhaled breath may be easily diluted by fresh gas flow from the anesthesia machine and cause falsely low end-tidal CO_2 values. Spontaneous ventilation allows the patient's physiologic drive to control minute ventilation and may be safer than an *unreliable* ventilator. The disadvantage of spontaneous ventilation is that patients develop atelectasis and some degree of CO_2 retention due to anesthesia-induced depression of the central response to CO_2.

Emergence From Anesthesia and Tracheal Extubation: When surgery is ending, anesthetists have to prepare for emergence from anesthesia and tracheal extubation. Although the tracheas of pediatric patients may be extubated during deep anesthesia, this may be riskier than awake extubation in pre-adolescent children because children are more likely to develop laryngospasm when going through the second stage of anesthesia during awakening. Weather the trachea is extubated during awake or deep anesthesia; neuromuscular blockade must be fully reversed before removing the tracheal tube. The child's respiration must be regular and her/his tidal volume must be at least 3cc/kg. For painful surgical procedures, a somewhat slower and deeper respiratory pattern is often a good clue that the patient has adequate narcotic analgesia. If more narcotic is needed, additional drug can be *titrated* to provide adequate pain relief without significantly depressing the respiratory rate.

For deep tracheal extubation, the pharynx is suctioned. The tracheal tube is removed while the patient is breathing anesthetic and 100% oxygen and is in the third stage of anesthesia. Airway adjuncts, such as oral or nasal airways, may be helpful after tracheal extubation but have the potential for inducing laryngospasm or bleeding. Once the trachea is extubated, the anesthetic is discontinued and the patient is allowed to awaken while breathing oxygen.

For an awake tracheal extubation, the inhaled gas is discontinued and 100% oxygen is provided. When the patient opens his or her eyes and the pharynx has been carefully suctioned of secretions and blood, the tracheal tube is removed. After tracheal extubation, the anesthetist must ensure that the patient's airway in unobstructed, that he/she is spontaneously breathing well, and that the oxygen saturation is above 97% before removing the monitors and leaving the operating theater. Following tracheal extubation, positioning patients on their side (when possible), rather than supine, often relieves airway obstruction. In this position the tongue tends to fall away from the posterior pharynx.

Sometimes anesthetists leave tracheal tubes in place and transport anesthetized patients to the post-anesthesia care unit (PACU - recovery room), where a nurse or the anesthetist removes the tracheal tube when the patient is awake and breathing adequately. This practice improves operating room turnover, but it should only be done when the anesthetist or nurse can provide an adequate, effective airway if the airway becomes obstructed. Emergence from anesthesia is just as critical as the induction of anesthesia and is usually best conducted in the operating room where the anesthetist has good control of the airway and the patient is fully monitored.

Post-Operative Airway Monitoring and Maintenance: During the post-operative period, pediatric patients are no longer under the continuous care and observation of the anesthetist, yet many problems can and do occur in the post anesthesia care unit (PACU) and on the ward. A well-planned anesthetic ideally results in an awake and comfortable patient at the end of surgery. Despite the best planning though, airway compromise still occurs and is the most common serious complication in the PACU and on the patient wards. Skilled nursing care in both locations is essential for patient safety. Anesthetists provide additional oversight in the PACU, but following some procedures, pediatric patients require intensive care, particularly if they have ongoing airway problems.

Both anesthetic medications and metabolic derangements occurring during surgery and anesthesia exacerbate preexisting apnea in neonates and very young infants. Premature birth, a history of apneic episodes, anemia (Hbg <10g), hypothermia, acidosis, hypocalcemia, and hypoxia are risk factors for post-operative apnea. Babies with any of these problems must be carefully observed in an inpatient setting for at least 24 hours after anesthesia. Some of them require continued tracheal intubation and mechanical ventilation, especially after extensive surgery. Premature babies ($\leq$37 weeks gestation) are prone to post-operative apnea until they reach 44 weeks gestation. Many hospitals and anesthetists require them to be >65 weeks gestation before

they will send them home the day of surgery. Elective procedures should probably be delayed in premature infants until after their brain has matured (>65 weeks gestation).

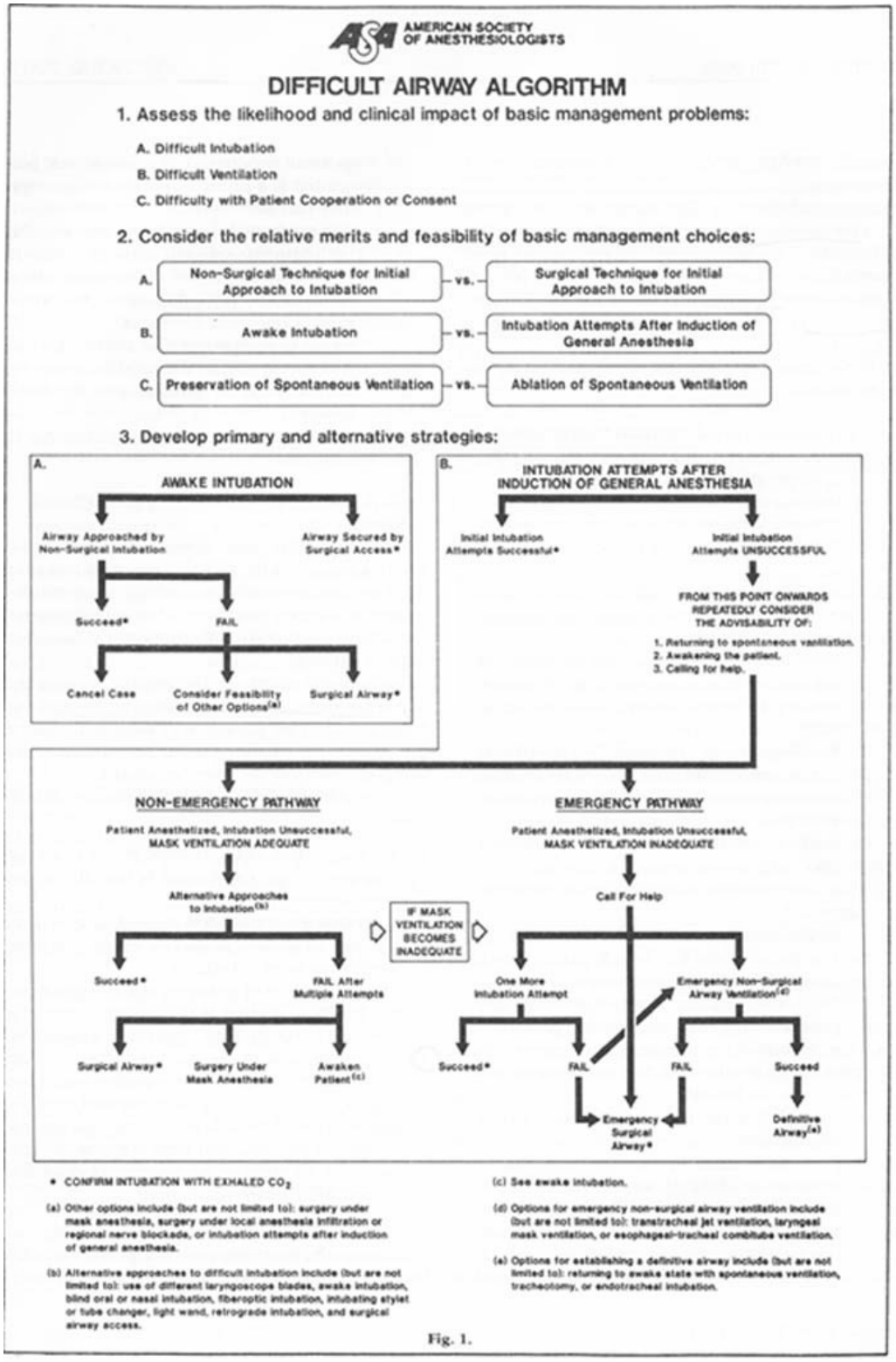
Pediatric patients should be monitored in a PACU with pulse oximetry; oxygen should be available for administration if needed. Electrocardiogram (EKG) and blood pressure monitoring are also helpful. PACU nurses must have the ability to recognize and support a compromised airway (**See Chapter 22**), because airway obstruction and aspiration (blood, secretions) can be life threatening. Sometimes PACU nurses begin giving oral fluids to patients after oral or facial surgery, such as cleft lip and cleft palate repair.

Once patients meet criteria for discharge from the PACU (airway and cardiovascular systems stable, alert, comfortable, pain and nausea controlled) they may be shifted to the ward, or possibly discharged home (**See Chapter 22**). Ward nurses must be vigilant and not leave the entire care of the patient to the parents. Airway status and oral intake should be checked and documented by the nurses at least every four hours. If patients are sent home after their stay in the PACU, they should receive clear instructions on what to do and where to go for help if the child has an airway of other complication.

Difficult Airway Management

The pre-operative history and physical examination require special consideration because they are the best way to identify potential problems. Congenital abnormalities, such as Pierre-Robin syndrome, Treacher-Collins syndrome and acquired abnormalities (hydrocephalus, trauma), will be discussed below.

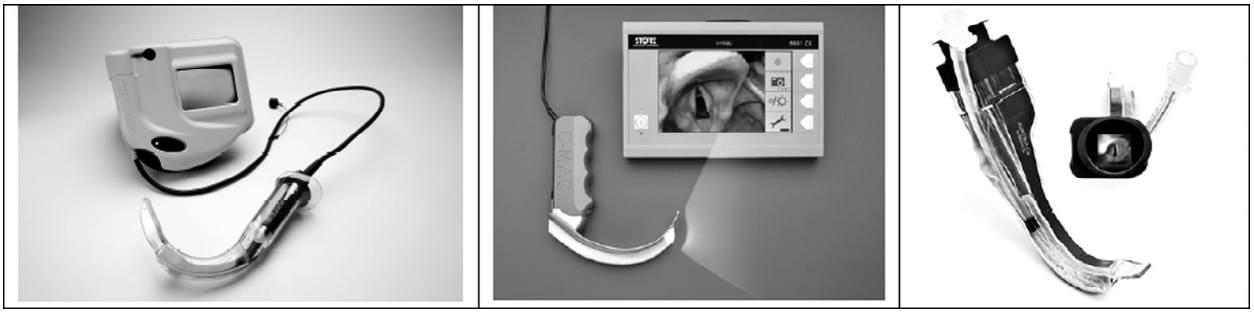
Figure 6-19: The Difficult Airway Algorithm



http://www.greggordon.com

If a difficult airway is identified prior to anesthesia, the anesthetist must consider several options (**Figure 6-19**). Awake tracheal intubation is less likely to be successful in young patients because they lack understanding of what is happening and are unable to cooperate. A key question the anesthetist must answer is whether an anesthesia mask will fit the child's face and provide an effective airway and a means of oxygenating and ventilating the patient's lungs. If not, the surgeon and anesthetist must discuss the need for, the benefits of, and the risks of establishing a surgical airway (tracheostomy). It may be safer to delay the surgery until the patient is older or the patient's transient airway problems have resolved. If, on the other hand, a mask airway appears likely to be effective, anesthesia can be induced with inhalation agents and spontaneous breathing. An intravenous line should be in place. If induction of anesthesia is successful, the next step is to determine whether positive pressure ventilation is possible. If positive pressure ventilation is possible and the depth of anesthesia is adequate, laryngoscopy is attempted, with or without muscle paralysis. One or more of the following modes of visualizing the vocal cords is used: 1) direct laryngoscopy with standard laryngoscope blade, 2) video-assisted laryngoscopy with Glidescope, Cmac laryngoscope, or Airtraq laryngoscope (**Figure 6-20**), that allows the anesthetist to "see" the glottis with indirect optics (see video) 3) lighted stylet or fiberoptic stylet (**Figure 6-21**), 4) fiberoptic laryngoscopy, and 5) blind nasotracheal intubation. Each method requires good airway skills and some require additional, expensive equipment, which may not be available in many operating theaters. Familiarity with each device is important, however. An intubating introducer is a simple but very effective device for tracheal intubation (see video). It consists of a semi-rigid bougie with its distal tip bent at 30 degrees. The introducer can be passed behind the epiglottis and into the trachea, even when the vocal cords are not visible on laryngoscopy. A tracheal tube that is as small as 4.0mm internal diameter can be passed over the smallest sized intubating introducer.

Figure 6-20: Video Assisted Devices for Tracheal Intubation



The picture on the left is of a Glidescope. The curve of the blade makes it easier to see the glottis, which is shown on the screen (See middle picture). The middle picture is of a C-MAC laryngoscope. The one on the right is of an Airtraq device. On the latter device, the viewing scope is on the end of the laryngoscope blade and provides a much smaller picture. All of them work when it is not possible to visualize the glottis with a regular laryngoscope, but at times even these devices fail to allow tracheal intubation.

Figure 6-21: Fiberoptic Stylet (Lighted Stylet) for Tracheal Intubation

This lighted stylet allows a tracheal tube to be placed over the aluminum light wand, which can be bent to make it easier to pass the lighted stylet and tracheal tube into the trachea. When the tube and stylet are in the trachea, a circular, small ball of light is seen in the midline of the neck in a dark room. If the lighted stylet is in the esophagus, diffuse light is seen in the neck. This device is passed blindly. It often helps to have an assistant grasp the patient's tongue with a gauze pad and pull the tongue out of the mouth. This frequently makes it easier to insert the tracheal tube into the trachea.

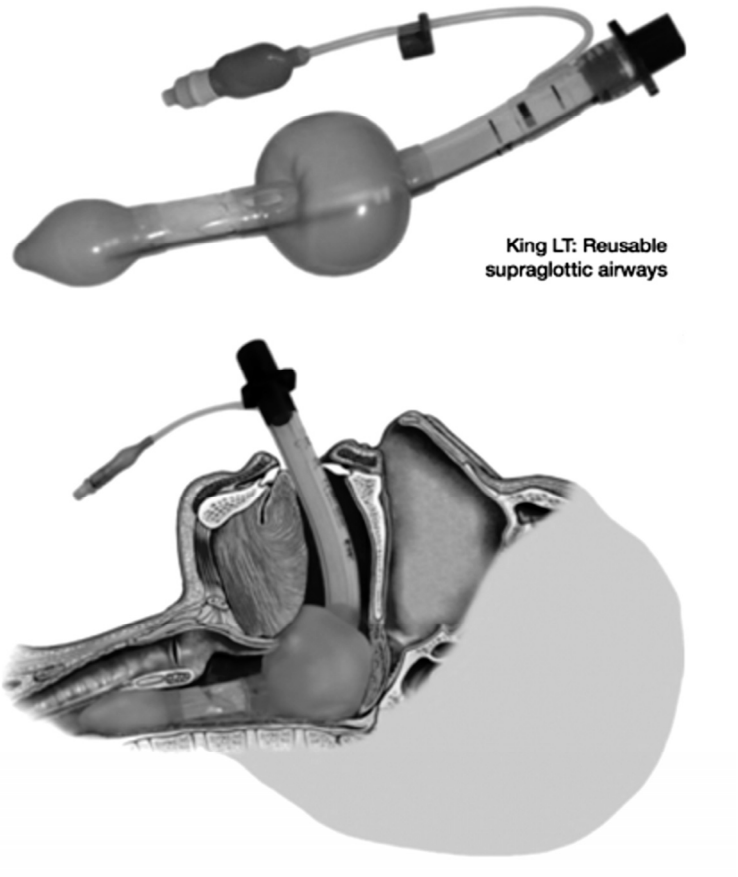
In addition to determining which of these modes of intubation is available and likely to be successful, the anesthetist and surgeon should consider having an emergency instrument tray and nurses ready so an emergency tracheostomy can be performed if it becomes necessary. Emergency needle cricothyrotomy can be considered but is a riskier procedure in small children than in adults. A child's neck landmarks are less well defined and the airway is smaller than those of adults. This usually makes surgical tracheostomy the emergency procedure of choice.

Laryngeal mask airways are used at several points in the difficult airway algorithm and have become the first line airway device for difficult ventilation and difficult tracheal intubation. If the standard mask airway is marginally effective for oxygenation and ventilation, an LMA can be placed as soon as the patient is deeply enough anesthetized to tolerate pharyngeal stimulation. It can also be placed if laryngoscopy is unsuccessful. The LMA has two advantages compared to a face mask airway for the duration of the anesthetic: the LMA does not require hands to hold it and it is more securely positioned than a face mask. LMA's can also be used for fiberoptic or "blind" tracheal intubation. The bronchoscope or TT is passed through the mask and into the trachea. Special techniques are required to advance a relatively short TT through the LMA and between the vocal cords. It is sometimes difficult to remove a LMA following successful tracheal

intubation without unintentionally removing the tracheal tube. LMA's that are specifically designed to facilitate tracheal intubation are quite useful but are not made in smaller sizes. LMA's are also used during preparation for urgent tracheostomy.

A *Combitube* (**Figure 6-22**) is another supraglottic airway that has a distal balloon that fits into and occludes the esophagus. This prevents ventilation of the stomach and aspiration of gastric contents. This device is passed blindly into the airway; unfortunately, its use has a higher complication rate, and it is not made in sizes appropriate for young children.

Figure 6-22: The Combitube for Securing the Airway



The combitube is a device that is placed blindly into the pharynx to provide emergency ventilation of the lungs. It consists of a distal tube with a balloon that is passed into the esophagus; the balloon of the distal tube is then inflated to reduce the possibility of aspirating gastric contents. The proximal tube with its balloon is in the mouth near the glottis. Ventilation is done through the proximal tube, which has an opening near the glottis. The advantage of this tube over a LMA is that higher airway pressures can be achieved with a combitube. http:www.cathlabdigest.com

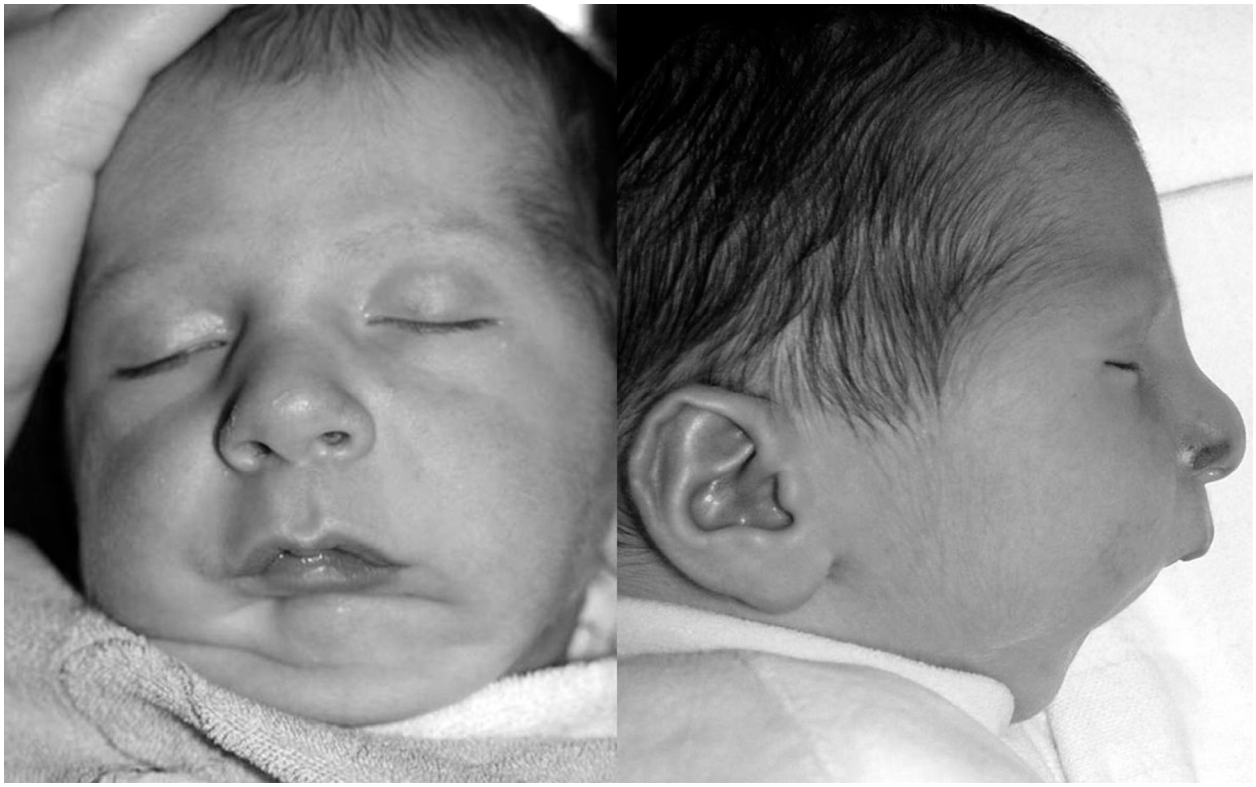
When the anesthetist encounters an unexpected difficult airway, he/she must rapidly decide if the devices immediately available to her/him will allow rapid securing of the airway. If advanced airway tools are not available, the wisest course of action is usually to awaken the patient. If bag-

and-mask ventilation is effective but the trachea cannot be intubated, the anesthetist can continue mask ventilation until advanced airway equipment (when available) can be obtained. Making multiple attempts at laryngoscopy with the same laryngoscope blade is not wise because it usually causes trauma without any benefit. The anesthetist should try different airway tools on subsequent intubation attempts and should be as gentle as possible. If a fiberoptic laryngoscope is available, it should be used fairly quickly. However, using these devices in an emergency is not appropriate or successful in most cases if the anesthetist has not used them before. Experience should first be gained with them in normal patients with normal airways.

When advanced airway devices are unavailable and emergency tracheal intubation is required, it is often possible to intubate the trachea by placing the second and third fingers of one hand into the patient's mouth and lifting the epiglottis (which is somewhat stiffer than the surrounding tissue). The TT is guided below the fingers and epiglottis through the vocal cords and into the trachea (digital intubation).

A difficult airway should be anticipated in children who have one of several congenital syndromes. The underdeveloped mandible of patients with Pierre-Robin syndrome (**Figure 6-23**) pushes their relatively large tongues against the palate and posterior pharynx. In utero, abnormal positioning of the tongue may cause a cleft palate. After birth the airway is often obstructed. In very young babies with airway obstruction, awake tracheal intubation may be successful, especially if a second person grasps the patient's tongue with a sponge and pulls it out of the mouth. LMA's provide temporary relief of the obstruction in the delivery room while fiberoptic intubation and/or a tracheostomy are being organized for long-term care. Some patients with Pierre-Robin syndrome do not require tracheostomy during infancy but may present for various surgical procedures later in childhood. These patients require careful consideration of their airway management. As patients with Pierre-Robin syndrome grow older, their mandibles grow, which improves their airway symptoms. Mask ventilation and tracheal intubation also become easier.

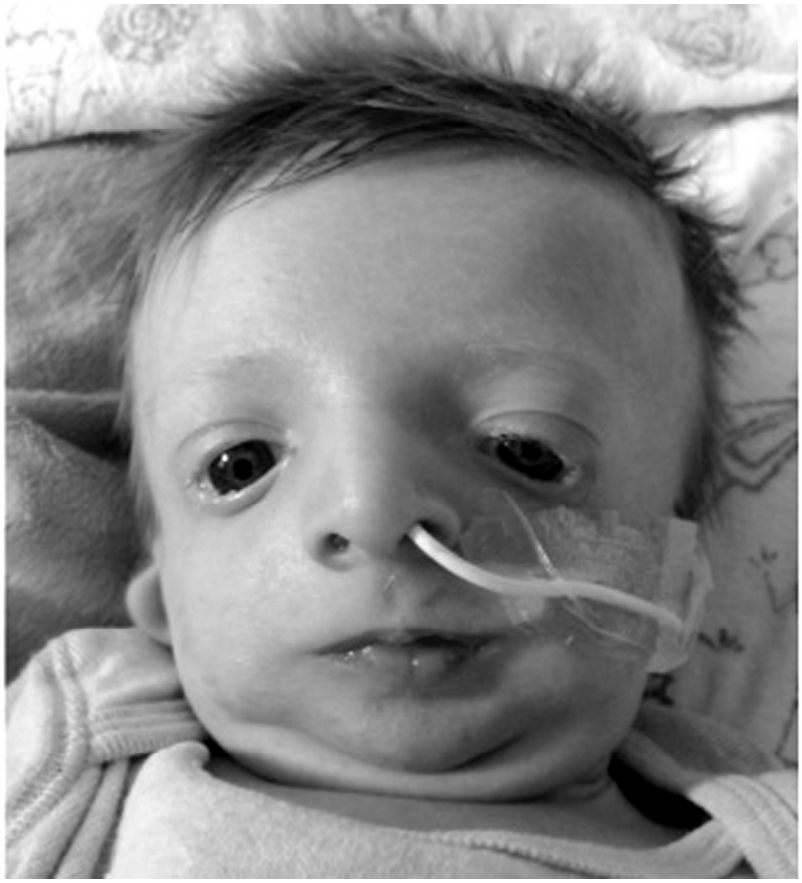
Figure 6-23: Pierre-Robin Syndrome



When looked at from the front, the patient's chin does not look grossly abnormal. But when looked at from the side, there is marked retronathia (posterior position of the mandible). This will make it difficult or impossible to intubate the trachea by standard means. See above. http://www.ijaponline.net

Patients with Treacher-Collins syndrome (**Figure 6-**24) also have mandibular hypoplasia, a cleft palate, and airway obstruction; they should be managed in the same manner as patients with Pierre-Robin syndrome.

Figure 6-24: Treacher-Collins Syndrome



This baby with Treacher-Collins syndrome has a mandible that is similar in size to that of the patient above with Pierre-Robin Syndrome. This baby also has orbital and ear abnormalities, including absent ear canals and low set ears.

Babies with Goldenhar syndrome (**Figure 6-25**) often have unilateral mandibular hypoplasia and limited, asymmetric oral opening that further complicates airway management. The glottis may not be in the midline, even when the trachea appears to be in its normal midline position. The glottis is often far off to one side of the pharynx or the other.

Figure 6-25: Goldenhar Syndrome

The right side of this child's face is normal. The left side of the mandible is smaller than normal and the left ear is nearly absent. The glottis may not be in the midline, making tracheal intubation more difficult. If the glottis cannot be seen, it usually will come into view when the hyoid bone is pushed from one side to the other. The tracheal tube should only be removed at the end of surgery when the child is awake. http://www.ccakids.com

Repair of cleft lips and palates in non-syndromic children is a relatively common surgical procedure (**See Chapter 16**). Their airways are usually not difficult, although the anatomy of their mouth and pharynx provides a very different view during laryngoscopy. Closure of the palate may cause post-operative airway obstruction. By closing the palate, the oral space is reduced in size, and this reduces the amount of space for the tongue, which is often swollen after surgery due to pressure exerted by a retractor during surgery. At the end of surgery, the surgeon should place a suture deep in the tongue tissue to allow the nurses and others to pull the tongue forward away from the posterior pharyngeal wall and open the airway if necessary. Another critical and potentially lethal problem following cleft or other oral surgery is inadvertently leaving a throat pack in the airway at the end of surgery. When this occurs, the airway may be totally occluded by the throat pack after the trachea is extubated. ***It is the responsiblility of all members of the surgical team to assure that the throat pack is removed at the end of surgery and before tracheal extubation!***

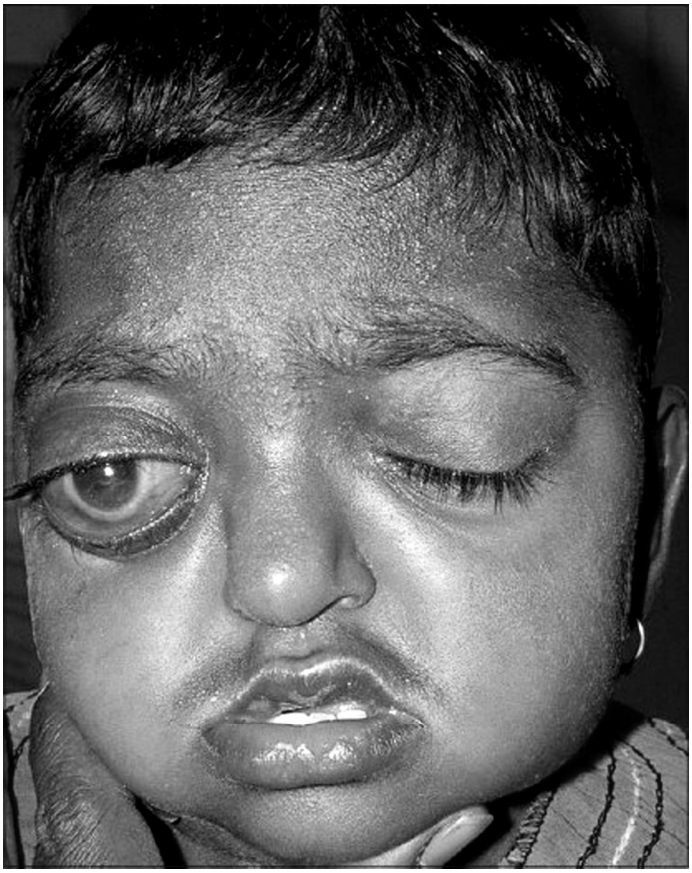
Down syndrome is more common than Pierre-Robin or Treacher-Collins syndromes and is often associated with an abnormal airway. These patients have mid-face hypoplasia with a depressed

nasal bridge, a small mouth, a large tongue, tonsil and adenoid hypertrophy, hypotonic pharyngeal muscles, and a small caliber trachea and tracheomalacia. All of these features contribute to airway obstruction. If the airway obstruction is long-standing, older children may have obstructive sleep apnea, central hypoventilation, and pulmonary hypertension. Patients with Down syndrome may also have lax ligaments between the first and second cervical vertebrae, putting them at risk for subluxation of these vertebrae and injury to the spinal cord if the neck is extended during anesthesia. Because children with Down syndrome are often overweight, it may be difficult to obtain intravenous access. Airway management of children with these problems is challenging. A careful history and physical examination help determine the degree of difficult airway. Anesthesia is usually induced with inhalation agents and a mask airway. For patients with severe airway obstruction, IV insertion prior to anesthesia induction is safer. The head must be maintained in a neutral position, even during laryngoscopy, because of the possibility of spine instability. After insertion of an oral or nasal airway, mask ventilation is usually satisfactory and the glottis is easily visualized during laryngoscopy. The anesthetist should select a 0.5-to-1mm smaller TT than usual for age. Post-operative observation in a recovery area should be extended for patients with possible obstructive sleep apnea. Patients with long-standing obstructive sleep apnea should be evaluated for pulmonary hypertension prior to surgery and anesthesia (**See Chapter 8**).

Other disorders associated with large tongues are Beckwith-Wiedeman syndrome and the mucopolysaccharidoses (see below).

Craniosynostosis, particularly the syndromic forms [e.g., Cruzon's (**Figure 6-**26) disease or Apert's syndrome], may also have airway obstruction due to their mid-face hypoplasia. Many of these patients have obstructive sleep apnea. Some have hydrocephalus. They may have severe enough airway obstruction to warrant tracheostomy in infancy. The anesthetist should be prepared to use devices for difficult airway management in these patients.

Figure 6-26: A Child With Cruzon's Disease

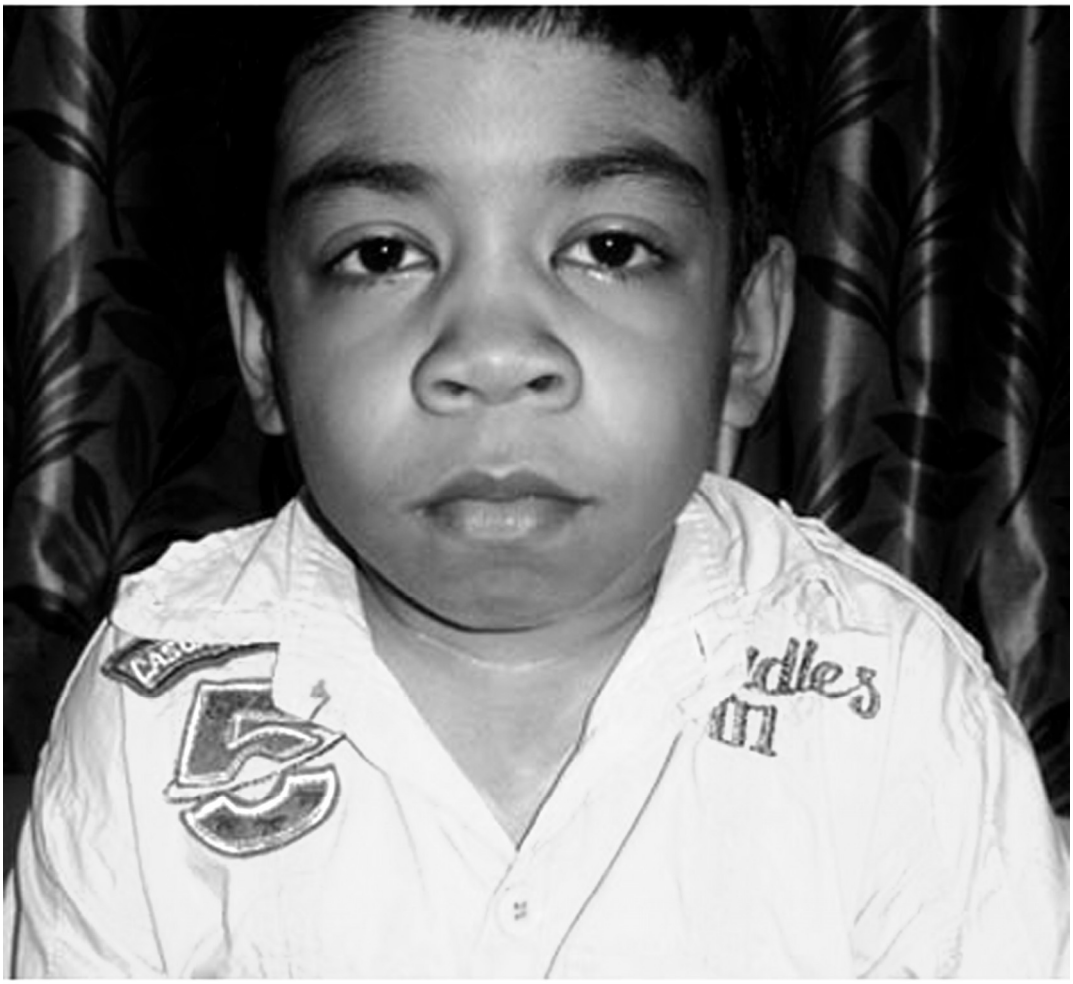


This child with Cruzon's disease has exophthalmos (bulging eyes), mid-face hypoplasia, and craniosynostosis, a hypoplastic maxilla and a "protruding" mandible. Tracheal intubation is usually not difficult. http://www.goohealthhub.com

Severe hydrocephalus, as well as head, face, and neck tumors, can distort and obstruct the airway from the mouth down to the trachea. Careful examination of the supine patient and a careful history of the patient's respiratory pattern aid the anesthetist in planning anesthesia and airway management.

Mucopolysaccharidoses are a group of metabolic disorders in which glycosaminoglycans are stored in cells throughout the patient's lifetime. As a result, these patients may have diseased cardiac valves, developmental delay, airway obstruction, sleep apnea, frequent respiratory infections, micrognathia, macroglossia, glaucoma, and hepatospleenomegally (**Figure 6-**27), Types I and II mucopolysaccharidoses, formerly called Hunter's and Hurler's disease, have continued deposition of glycosaminoglycans and progression of their symptoms. Thus, their airways become more difficult with increasing age.

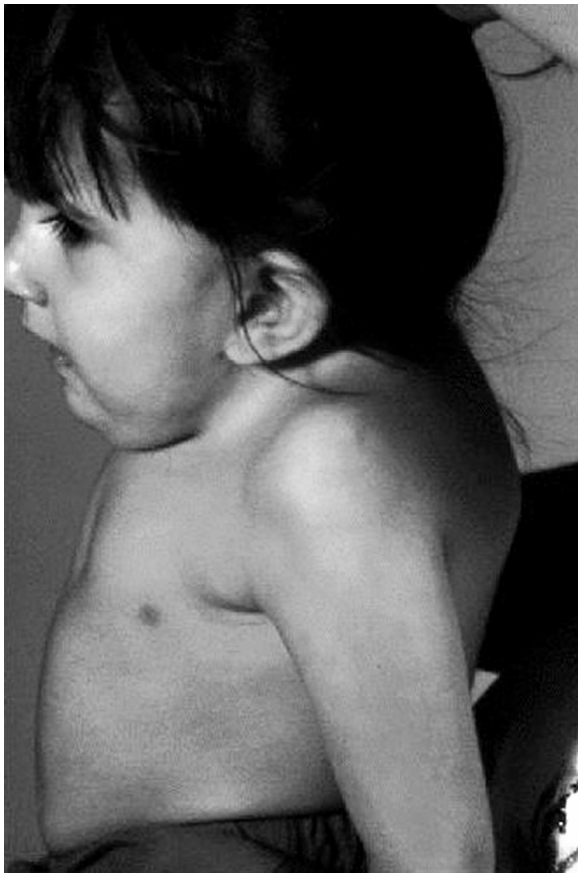
Figure 6-27: A Child With Mucopolysaccharidoses



This patient with a mucopolysaccharidosis has the typical distinct facial features, which include flat facies, depressed nasal bridge, bulging forehead, and short stature. http://www.static.dnaindia.com

Klipel-Feil syndrome (congenital fusion of any two of the seven cervical vertebrae) causes decreased neck mobility and may make positioning during anesthesia induction and tracheal intubation difficult (**Figure 6-28**).

Figure 6-28: Klippel-Feil Syndrome

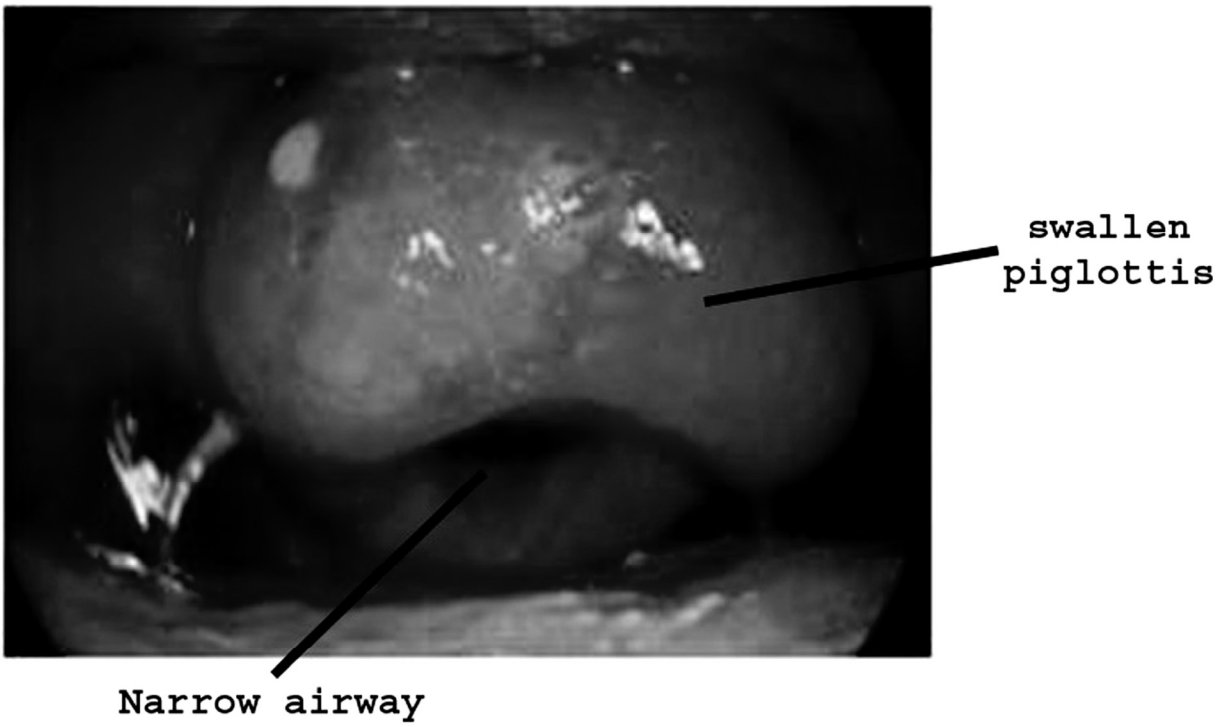


This child with Klippel-Feil Syndrome has a non-mobile neck, making it difficult to intubate her trachea. She also has a very short neck and a low hairline. http://www.syndrome.org

Acute airway infections, including tonsillar and retropharyngeal abscesses, may cause airway obstruction (**See Chapter 17**). A careful inhalation induction allows the anesthetist to determine the degree of airway obstruction and to address it in a step-wise manner, as described above. Care must be taken to avoid opening the abscess during airway manipulations, especially prior to tracheal intubation, as this might permit purulent material to enter the lungs and cause pneumonia or abscesses.

Epiglottitis is an acute infection caused by Hemophilus influenza or other bacteria (**Figure 6-29**). The epiglottis, vocal folds, and posterior arytenoids may swell enough to completely occlude the airway.

Figure 6-29: Epiglottitis



Note the very swollen epiglottis and the narrow airway, which at times can cause difficulty when intubating the trachea. http://www.dromktb.com

Stridor is common; these patients often appear very anxious. Until the infection has been treated with antibiotics and the edema has receded, the anesthetist must be extra careful when intubating the trachea. Parental presence during induction of anesthesia may help calm the patient and improve breathing. Having an IV in place during inhalation induction of anesthesia is safest for the patient. If the airway becomes more obstructed, induction of anesthesia can be stopped and the patient awoken. A surgeon who is able to perform an emergency tracheostomy and all the equipment required to do so must be immediately available in the operating room, in case the anesthetist cannot relieve the airway obstruction. A smaller than usual TT should be used.

For patients with infectious croup or tracheitis that is severe enough to cause edema of the sub-glottic airway, it is important to use a much smaller TT than usual for the patient's size and age.

Acute airway problems, such as burns, trauma, foreign body aspiration and anaphylaxis, can also produce a difficult airway (**See Chapter 19**). Face and neck burns cause acute local swelling; smoke inhalation also causes acute airway swelling. Patients who have burns of their face and upper airway should have their tracheas intubated as soon as possible, before maximal swelling occurs. For severe facial burns, tracheostomy may be the safest airway for long-term

management. Similar considerations arise for patients with face and neck trauma. It is best to anticipate airway compromise and secure the airway before edema makes tracheal intubation impossible.

For patients with *head trauma* and brain injury, early tracheal intubation and mechanical ventilation prevents hypoxemia and hypercarbia that often occur when the airway is unsecured. Careful induction of anesthesia is required to prevent increasing intracranial pressure and to prevent aspiration of stomach contents. A modified rapid sequence induction, with the patient well anesthetized at the time of tracheal intubation, is usually the safest choice.

Retrieval of a foreign body from the airway or esophagus is a common emergency procedure in toddlers. Almost all patients with foreign bodies have full stomachs when anesthesia is induced and some have stridor. Neck and chest X-rays help determine the location of the foreign body. A careful inhalation induction of anesthesia with an intravenous line in place is indicated. Depending on the location of the foreign body, cricoid pressure may worsen spontaneous or positive pressure breathing. Tracheal intubation may push a foreign body deeper into the trachea. These issues must be taken into consideration when the anesthetist and surgeon discuss the safest approach for removing the foreign object. Communication between surgeon and anesthetist is essential for providing optimal care for patients with foreign bodies.

Anaphylaxis occurs in response to many allergens. During surgery these allergens include antibiotics, neuromuscular blocking agents, and latex. Bronchospasm, a common component of anaphylaxis, usually requires prompt tracheal intubation. The anesthetist must determine the safest and quickest method of airway management. Edema of the upper airway and trachea occur rapidly. If the patient's airway is not intubated when first seen, the anesthetist may wish to intubate the trachea before edema formation occurs. Once the trachea is intubated, the tracheal tube should be left in place until the airway edema subsides. This is the safest course of action. While the anesthetist is preparing for tracheal intubation, epinephrine should be administered subcutaneously or intramuscularly 0.3-0.5ml of 1:1,000 epinephrine. The dose for children is 0.01mg/kg. The drug can also be infused as an IV drip (1mg in 500ml of D5W). The infusion should start at 1mcg/min and be increased as needed every 3-5 minutes.

SUMMARY

Many of the complications occurring in the operating room and PACU are due to the airway. Consequently, it is important to understand normal airway development and the differences occurring with age. This understanding is especially important when patients have a difficult airway. When this occurs, providing an airway for these patients is a team effort on the part of the anesthetist, surgeon, and nurses. Only when all members of the team understand what needs to be done will patients do well. Thus, it is often important to have "dry runs" and simulate an airway emergency so everyone knows what they are to do in an emergency.

Chapter 7

INDUCTION AND MAINTENANCE OF ANESTHESIA

Latha Kampalath, MD

INTRODUCTION

Differences in anatomy and physiology make administering anesthesia to children unique. However, these differences can also make the anesthesia induction challenging and unpredictable. Thus, it is important to carefully evaluate patients, understand the history of their problem(s), evaluate their laboratory tests, and do a thorough physical examination before taking them to the operating room (**See Chapter 1**).[1]

Providing anesthesia for children in any part of the world requires an understanding of the advantages and disadvantages of the local healthcare system. In many areas of the world children are faced with natural calamities, famine, war, social unrest, and communicable diseases.[2] Malaria, human immunodeficiency virus (HIV) and tuberculosis are common in many of the world's poorest countries. The World Federation of Societies of Anesthesiologists published standards for safe anesthesia in 1992, but anesthetists in some countries cannot comply with these guidelines for lack of equipment, drugs, and training. In most of these countries, there are few trained anesthesiologists. Nurse anesthetists or Clinical Officers, not physicians, provide most anesthetics in these countries, often under difficult conditions.

EVALUATION AND SELECTION OF PATIENTS

Children in some of the poorest countries on earth are frequently free of congenital anomalies because those with congenital syndromes and genetic diseases do not survive early life.[2] However, those who do survive often experience anemia, malnutrition, and parasitic or other infectious diseases. When possible anemia and infections should be treated prior to surgery. However, this is usually not possible when emergency surgery is required. While it may sometimes be difficult to obtain an appropriate history of the child's health, it is usually possible to do a through physical examination. Anomalies of the airway (congenital or acquired), heart (rheumatic heart disease), hepatosplenomegaly (malnutrition, malaria, sickle cell disease) are a few examples of problems that can be detected by a thorough physical examination. Pulse oximetry (SpO_2) and knowledge of the patient's hemoglobin concentration are important. If

bleeding is anticipated, it is probably safest for the patient if her/his hemoglobin concentration is ≥10gm/dl at the start of surgery.

PREPARATION OF OPERATING ROOM

Functional and properly serviced anesthesia equipment is important for delivery of safe anesthesia.[3,4] All equipment needed to deliver anesthesia should be available and tested before anesthesia is induced to assure that the equipment functions normally. A continuous supply of electricity is needed to power electrical equipment, including monitors and ventilators. Whenever possible there should be a backup generator that quickly and effectively provides electricity to the operating rooms when the local electricity source fails. If no backup generator is available, the anesthetist, surgeon, and nurses must have backup plans for how they will monitor the patient's vital signs, suction secretions and blood, and provide enough light for the surgeon to operate and control bleeding. If intravenous (IV) cannulas, tracheal tubes (TT), and laryngoscopes must be reused, they should be sterilized between patients to prevent inadvertent infections and deaths. Before inducing anesthesia, the anesthetist and surgeon must determine the type and quantity of IV fluids available and the availability of blood or blood products for transfusion should they be required.

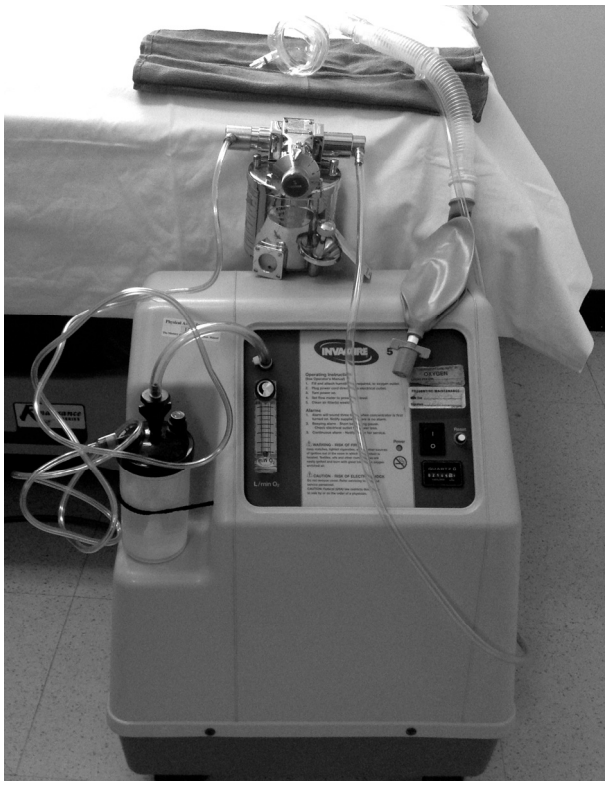
Oxygen Supply

When cylinders provided oxygen, it is important to have an alarm system that alerts the anesthetist to the fact that the oxygen cylinder is running out of oxygen. E-cylinders only contain 625 liters of oxygen when full.[5] If the inspiratory gas flow is 10 liters/minute, the tank will empty in about one hour. Pressure in the oxygen tank is proportional to the amount of oxygen left in the tank. Full G-cylinders contain 5,300 liters and H cylinders contain 6,900 liters of oxygen respectively. Thus, it takes much longer for the tank to empty. Oxygen from the wall commonly comes from G and H cylinders, and the tanks must be changed frequently. It is good practice to have an E-cylinder and its pressure-reducing valve immediately available in the operating room for times when larger tanks unexpectedly run out of oxygen during surgery. Having a self-filling Ambu™ type bag immediately available will allow the anesthetist to ventilate the patient's lungs with room air in case of a sudden loss of oxygen or electricity. If nothing else is available, the anesthetist can provide mouth-to-mouth breathing. This is more effective if oxygen is available and a tube from the oxygen source is placed in the anesthetist's mouth to increase the inspired oxygen concentration.

Oxygen concentrators are used to extract oxygen from air when compressed oxygen is unavailable (**Figure 7-1**).[6] The Zeolite in these devices adsorbs nitrogen from the air, leaving 85-90% oxygen in gas breathed by the patient. Concentrators provide a cheap, reliable source of oxygen and are unlikely to cause fires. Oxygen cylinders are usually painted white to comply with World Health Association (WHO) standards.[5] Pressure regulators and gas flow meters, integral

parts of oxygen delivery systems, must be serviced often to assure they are functioning correctly and accurately.

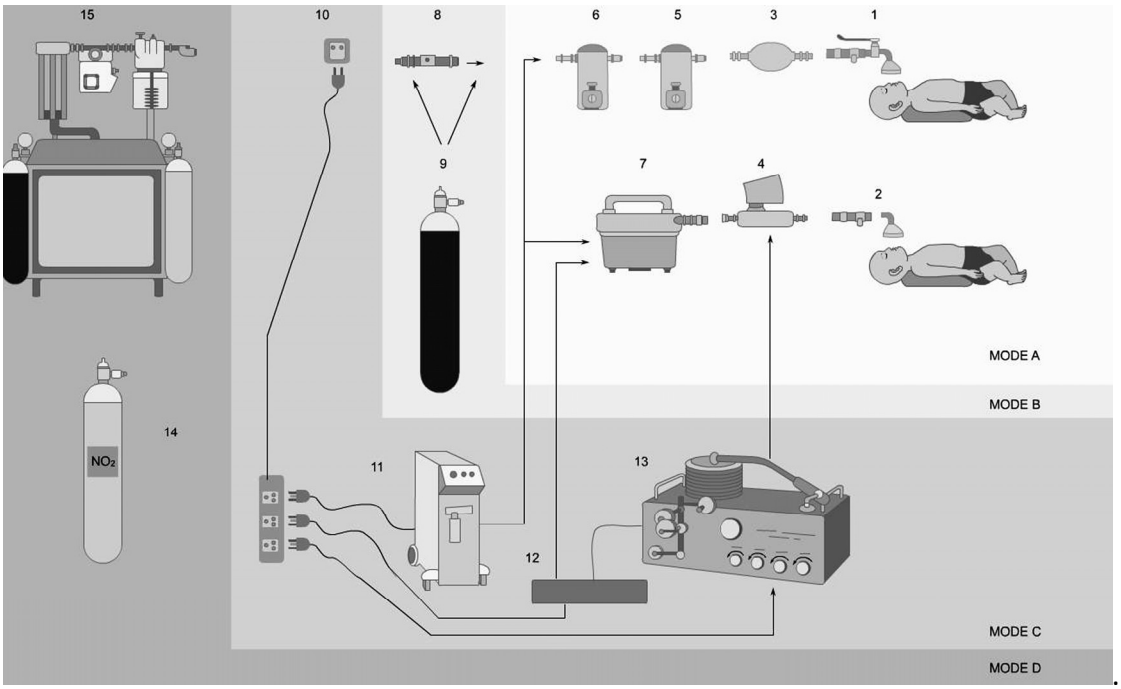
Fig 7-1: Oxygen Concentrator Connected to Vaporizer with Jackson-Rees Circuit



Anesthesia Delivery Systems

Anesthesia delivery systems should be inexpensive, safe, and easy to assemble (**Figure 7-2**). They should also work when compressed gas is unavailable. Two of these systems, EMO (Epstein Macintosh Oxford) and OMV (Oxford Miniature Vaporizer), are widely used for inhalational anesthesia when there is no secure source of compressed gas. These draw-over vaporizers work well with oxygen concentrators. The negative pressure created by the patient's inspiratory efforts draws air from the atmosphere, which is used as the carrier gas for the anesthetic.

Figure 7-2: Scheme by Ell Ashy et al for Inhalation Anesthesia in Developing Countries. (Adapted from Bissonnette) [7,8]



Mode A *– provides basic inhalation anesthesia with air, spontaneous ventilation, or self-inflating bags*

Mode B *– provides oxygen enrichment but requires an oxygen source (concentrator or cylinder)*

Mode C *– requires electricity to power the oxygen concentrator, air compressor and/ or ventilator*

Mode D *– requires a Boyle machine and nitrous oxide cylinders*

T-piece with reservoir tube and face mask

Ambo™ pediatric valve

Self inflating bag

Oxford inflating bellows

Oxford miniature vaporizer with halothane

OMV with trichloroethylene

Epstein Macintosh Oxford vaporizer with ether

Farmer's entrained with an oxygen cylinder

Oxygen cylinder

Electrical power source

Oxygen concentrator

Air compressor

Manley ventilator

Nitrous oxide

Boyle's apparatus

Oxygen cylinders or oxygen concentrators can be connected to the supplementary inlet of the vaporizer to increase the oxygen concentration of the delivered gas. When EMO and OMV systems are used for pediatric anesthesia, the recommendation is to use them with Jackson-Reese system for greater patient safety.

Copper Kettle or Vernitrol devices are nonspecific vaporizers that can be used with most inhaled anesthetic agents. The concentration of anesthetic delivered to the patient depends on the vapor pressure of the anesthetic liquid, gas flow through the device, and total gas flow in the system. When providing anesthesia with draw over vaporizers at high altitude, it is often necessary to supplement the air with some oxygen to prevent hypoxemia.

The Ohmeda 885A machine has a universal, non-agent specific vaporizer that allows safe delivery of different inhaled agents and has been used for wartime medical care as well as medical missions.[9] This machine conveniently and safely provides anesthesia in remote locations (**Figure 7-3**). It consists of a simple circle system and a carbon dioxide (CO_2) absorber. It is lightweight, compact, and can be used with either compressed oxygen or medical air. Because nitrous oxide (N_2O) cannot be used with this machine, it is difficult to deliver a hypoxic mixture of gas. Standard halothane, isoflurane and sevoforane vaporizers work with this machine. The vaporizers are temperature and pressure compensated, making their output relatively constant, assuming the vaporizers are serviced at least once a year.

Figure 7-3: The OBAMED Portable Anesthesia Machine

This photograph shows an OBAMED portable anesthesia machine with a sevoflurane vaporizer, anesthesia circuit and bag, anesthesia, flow meters for oxygen and nitrous oxide, and a CO_2 absorber.

The Kambatta anesthesia system is another simple, low technology way to deliver anesthesia and oxygen (**Figure 7-4**).[10] It includes a pressurized oxygen supply, a flow meter connected to a freestanding vaporizer, a portable disposable sealed carbon dioxide absorber, and a breathing circuit. This system can be assembled in a few minutes.

Figure 7-4: The Khambatta et al Anesthesia System.

Inhaled anesthetics can be interchanged in non pin-indexed standard vaporizers. A pin-indexed vaporizer is one that has pins that only allow a drug-specific vaporizer to be connected to the anesthesia machine and to deliver one inhaled anesthetic at a time. When the pin indexing system is bypassed, the anesthetist must pay close attention to the output of the anesthetic vaporizers and the patient's condition, because it is possible to administer too much or too little anesthetic to the patient. Halothane and isoflurane have roughly the same vapor pressures. Consequently, they can be used with either a halothane or isoflurane vaporizer without significantly affecting the vaporizer's accuracy and output. It is advisable to empty all residual volatile anesthetic from the vaporizer before filling it with fresh liquid anesthetic to avoid mixing anesthetics and delivering a mixture of anesthetics to the patient. Monitoring end-tidal anesthetic concentrations is the best way to detect the presence of anesthetic mixtures.

It is advisable to scavenge anesthetic gases from the operating room to prevent their inhalation by surgeons, nurses, and anesthetists. This can be done with portable anesthesia machines by running corrugated tubing from the exhaust valve of the breathing system to an outside window or a suction device.

Old exhausted soda lime may not change color when exposed to CO_2. Consequently, when exhausted soda lime is used, patients develop hypercarbia and respiratory acidosis. When soda lime is unavailable or in limited supply, a non-rebreathing circuit (e.g., Mapleson or Bain circuit) is a good option because the patient's $PaCO_2$ will not rise if the gas flow in the system is at least

twice the patient's minute ventilation [tidal volume (7m/kg in all patients) X respiratory rate]. The disadvantage of these systems is that that they require a high total gas flow to prevent CO_2 rebreathing and this wastes precious oxygen and anesthetic.

Monitors

Intraoperative monitoring of oxygen saturation (SaO_2), arterial blood pressure (BP), body temperature, and the electrocardiogram (ECG) provides early warning of impending cardiopulmonary problems (**See Chapter 2**). Monitoring of end-tidal CO_2 is also very helpful. If CO_2 is present in the expired gases, the tracheal tube is in the trachea (not the esophagus) and ventilation is occurring.

A precordial stethoscope allows the anesthetist to determine the presence of breath sounds and the quality of the heart tones. Breath sounds indicate that the patient's airway is patent and he/she is breathing. Heart tones, on the other hand, are an indication of cardiac function. Crisp, normal heart tones suggest normal myocardial function. Diminished and less crisp heart tones suggest decreased myocardial function. Changes in heat tones during anesthesia indicate improving or worsening myocardial function.

INDUCTION OF ANESTHESIA

NPO Guidelines and Preoperative Sedation

It is important to determine preoperatively if a patient has drunk or eaten anything recently. [11, 12] The standard Society of Anesthesia (ASA) guidelines for when to stop eating and drinking before elective surgery [Nothing By Mouth (NPO)] (**See Table 1**), reduces the incidence of vomiting and aspiration of gastric contents in the perioperative period, a common cause of death in many patients.

Table 7-1: NPO Guidelines

Type of food/liquid	Hours before inductions of anesthesia
Clear liquids	2 hrs.
Light meal	6 hrs.
Full meal	8 hrs.

Clear liquids are fluids one can see through (water, apple juice without pulp, etc.); a light meal consists of toast and liquid (e.g., coffee). NPO – nothing by mouth.

Infants and young children who live in warm climates and have fasted for many hours are at risk for developing hypovolemia and becoming hypotensive during anesthesia induction. Hence every patient without a reason not to drink should be encouraged to take in clear liquids up to 2-3 hours prior to surgery. They should be allowed to drink as much as they want before being made NPO because large volumes of fluid empty from the stomach faster than small volumes of fluid.

Oral premedication is often used to sedate children before surgery. This often allows the anesthetist to place an IV catheter preoperatively, which he/she can use to induce anesthesia. The amnesia and sedation provided by preoperative medication often reduces the child's anxiety and agitation when he/she is taken from her/his parents.

Midazolam (Versed) syrup (0.5 – 0.75mg/kg; maximum dose 10mg), when given 15-20 minutes before the induction of anesthesia, usually produces a calm child during the induction. Midazolam sedates most children and produces amnesia for preoperative events. If midazolam syrup is unavailable, the intravenous form of this drug can be given orally. However, midazolam, which has a high pH, taste awful. The taste can usually be hidden in a sweet, acid solution (e.g., 30ml of Coca Cola or clear fruit juice). If an elixir of acetaminophen is available, midazolam can be mixed with the elixir to hide midazolam's bitter taste. To get the desired effects of oral midazolam, the drug must be given 15-20 minutes before entering the operating room. Midazolam is sometimes given intra-nasally 0.2-0.3mg/kg to uncooperative children, but this produces a bitter taste and stings when the drug comes in contact with the nasal mucosa. The intravenous dose of midazolam is 0.05mg-0.1mg/kg. Half of each dose of oral midazolam is metabolized when venous blood from the stomach passes through the liver (first pass effect). Thus, half of an oral dose never reaches the central circulation and brain. If the surgery is short, premedication with midazolam may delay discharge from the recovery room, but not by more than a few minutes.

Ketamine (Ketalar) is another drug that is often used for premedication because it produces a dissociative state that allows smooth transfer of combative children to the operating room. The oral dose of ketamine is 5-10mg/kg (maximum dose 10mg/kg). It too has a bitter taste that must be hidden in something sweet (see above). Ketamine can also be given intramuscularly 2-5mg/kg (max dose 6mg/kg), but ketamine produces significant pain and burning on injection. Despite this, intramuscular ketamine is often the preferred route for sedating combative children or those who will not drink midazolam or ketamine. Delayed awakening from anesthesia and euphoria during recovery from anesthesia are the chief side effects of oral ketamine. Combining oral ketamine with oral benzodiazepines (ketamine 3mg/kg + midazolam 0.5mg/kg) provides effective preoperative sedation and significantly reduces the incidence of postoperative hallucinations. Rectal ketamine 4-6mg/kg can be given once or one dose of intranasal ketamine 0.75mg/kg provides good sedation. The long half-lives of *diazepam* and *lorazepam* make them less attractive as premedication drugs.

ANESTHETIC TECHNIQUES

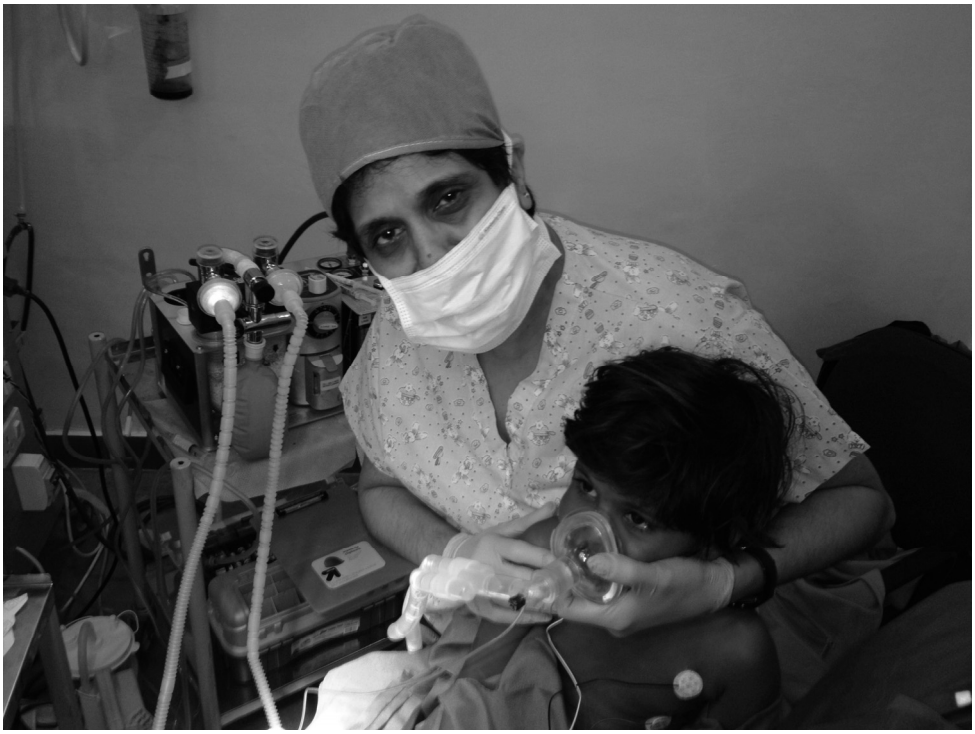
The method by which anesthesia is induced depends on the age and health of the child and on the type and urgency of the surgery. *Inhalation induction* is the most common method used in children in most countries because children seldom like being stuck with needles to start an IV. Inhalation induction of anesthesia occurs quickly due to the fact that the uptake and distribution of inhaled agents occurs more rapidly in children than in adults. This happens because children have increased minute ventilation and a decreased functional residual capacity (MV:FRC). They also have lower blood-gas partition coefficients for inhaled anesthetics than older patients. All of these factors work together to increase the rate at which the anesthetic concentration rises in the lungs. This makes more anesthetic available to pulmonary blood. The larger cardiac output/kg of body mass and the relatively higher blood flow to vessel rich tissue groups provides more anesthetic to the brain and central nervous system in a shorter period of time.

Halothane and sevoflurane are commonly used for inhaled induction of anesthesia because they cause less airway irritation than other drugs; however, in higher concentrations halothane and sevoflurane can also produce coughing, breath holding, and laryngospasm, especially when the anesthetic concentration is increased rapidly.[6] Children frequently have respiratory infections and are seldom free of a cough and congested airways. These infections increase the risk for coughing if anesthetic concentrations are rapidly increased. For these reasons it is usually best to gradually increase the anesthetic concentration (halothane 0.5–1% every four breaths; sevoflurane 2% every 2–4 breaths) to reduce the incidence of coughing and breath holding. Nitrous oxide is a good analgesic and amnestic; its administration shortens induction times when administered with sevoforane or halothane. If proper safety mechanisms are not in place, it is possible to inadvertently deliver a hypoxic mixture of gases when using nitrous oxide.

When a child is combative and crying uncontrollably, anesthesia induction can be rapidly accomplished with a high concentration of anesthetic (*single-breath induction*). The anesthesia circuit is first filled with high concentrations of the gas (halothane 5%; sevoflurane 8%) and the gas flows through the circuit increased to 8-10L/min. The y-connector on the anesthesia circuit is occluded and the gases are allowed to circulate and equilibrate in the anesthesia system for 2-3 minutes. The mask is then placed on the child's face and he/she is allowed to breathe the gas mixture. The Adjustable Pressure Limiting (APL) valve is kept wide open to make it easer for the child to exhale. A good seal between the mask and face is necessary to prevent dilution of the anesthetic gases and ensure rapid induction of anesthesia. As the child cries, he/she will take several deep breaths of the anesthetic/oxygen mixture and quickly become unconscious. At this point, it is important to reduce the anesthetic concentration and gas flow to normal. Sometimes it is necessary to assist the child's breathing. Failure to decrease the anesthetic concentration sufficiently rapidly will cause rapid depression of respiratory and cardiac function.

Children can be placed in one of several positions for induction of anesthesia. They can sit on the operating room table **(Figure 7-5)** while the anesthetist holds the child close to her/his chest. This often works well for children who refuse to lie down.

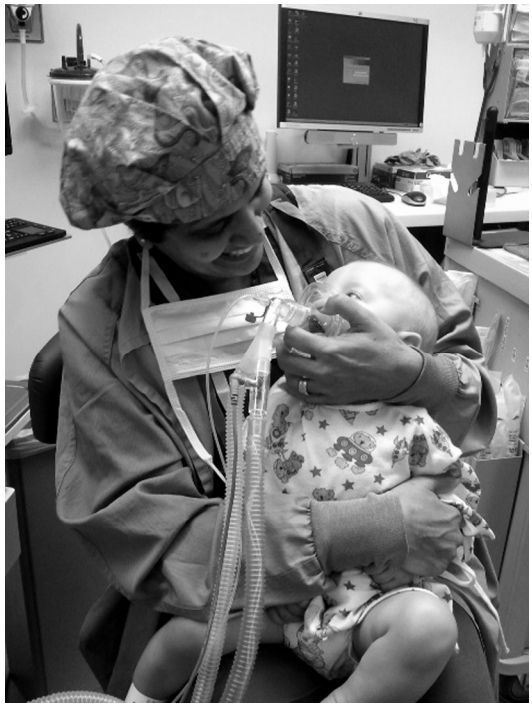
Figure 7-5 Mask Induction of Anesthesia



This photograph shows the author holding a young child while he is sitting on an operating table and leaning backwards against the anesthetist during the induction of anesthesia. This is easy to do, calming for the child, and gives the anesthetist better control of the situation.

The child can also sit in the mother's or the anesthetist's lap (**Figure 7-6**). Singing to the child or telling her/him a story often distracts the child and makes the induction smoother. The inside of the facemask can be smeared with different flavors (orange, bananas, strawberry, bubble gum) to hide the pungent odor of the anesthetic vapor. It is important not to get this food flavoring on the rim of the mask, as it may stain the child's face for days.

Figure 7-6: Mask Induction of Anesthesia in an Infant.



The child is held in the anesthetists lap. It is important not to apply excessive pressure on the abdomen because this will obstruct breathing.

Halothane has long been successfully used to anesthetize children and is still available in many countries. It depresses airway reflexes, causes bronchial dilatation, and provides good depth of anesthesia for tracheal intubation without the need for muscle relaxants. It causes some peripheral vasodilatation and myocardia depression. Therefore, its concentration should not be increased too rapidly.

Sevoflurane is less pungent than other inhaled agents and usually allows for a rapid, smooth induction of anesthesia, even when high doses (8%) are used. Because loss of consciousness occurs more quickly, children struggle less with sevoflurane than they do with halothane. Sevoflurane is associated with less cardiac depression and fewer cardiac arrhythmias than halothane. It is also less likely to sensitize the myocardium to epinephrine. Rapid recovery from sevoflurane reduces the child's length of stay in the PACU, unless he/she develops emergence delirium, which occurs more frequently in younger children.

Intravenous induction of anesthesia is usually preferred for older children. If necessary, the child can be given oral premedication to help her/him tolerate insertion of an IV. Breathing a 50/50 percent mixture of nitrous oxide (if available) and oxygen provides sufficient sedation and pain relief to allow insertion of an IV. For emergency surgery intravenous induction of anesthesia is usually preferred, as it is for the child who is predisposed to aspirate her/his gastric contents. The

most common drugs used for intravenous induction of anesthesia are propofol and ketamine. Pentothal (Thiopental Sodium) can also be used if available.

If the child's sympathetic nervous system is not maximally stimulated, *ketamine* preserves airway reflexes and breathing. It also maintains hemodynamic stability in moderately hypovolemic patients. Ketamine does *not*, however, prevent hypotension in severely hypovolemic patients. Ketamine is also a good analgesic and an effective drug for supplementing inhaled anesthetics during maintenance anesthesia. One problem with ketamine is that it often causes excessive airway secretions. Combining atropine (10-30mcg/kg – maximum dose 1mg) or glycopyrolate (8-16mcg**/**kg – maximum dose 200mcg/dose) with ketamine prevents the excessive secretions. When intravenous access is difficult and an inhalation induction of anesthesia is not a good choice (bleeding after tonsillectomy or full stomach), ketamine 5-8mg/kg can be administered into the deltoid or triceps muscles. Drugs are absorbed twice as rapidly from these muscles as they are from the legs or buttocks. Ketamine injection can be followed by intramuscular succinylcholine 4mg/kg – maximum dose 100mg. A better solution is to combine ketamine with atropine or glycopyrolate and succinylcholine in the same syringe. Doing this usually results in an anesthetized patient and good intubating conditions in about 45 seconds. Oxygen should be given for two minutes before injecting this drug mixture to avoid causing hypoxemia. Ketamine 100mg/ml is used for IM injections (when available) because its used reduces the total volume of fluid injected and injection-induced pain.

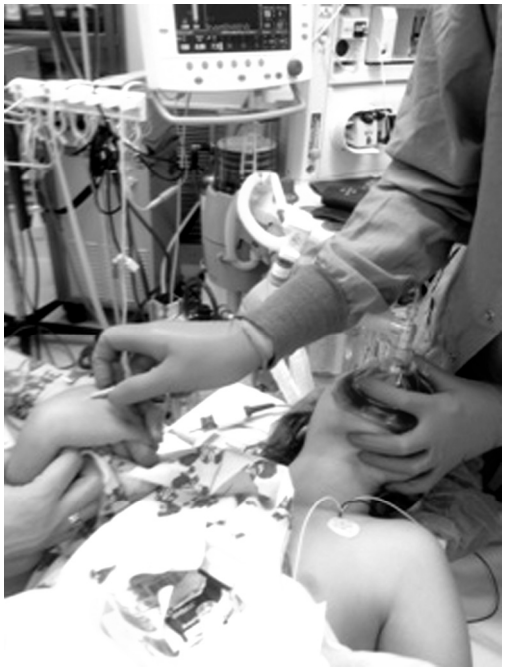
Propofol 2-3mg/kg IV is widely used as an induction agent for pediatric anesthesia. However, IV propofol causes burning and pain in the vein unless lidocaine 1mg/kg and/or fentanyl 1mcg/kg is injected before giving the ketamine. When lidocaine is injected into the vein, the arm circulation should be occluded for a couple of minutes before injecting the propofol. Propofol should *not* be given to patients who are allergic to eggs, as this may induce an allergic reaction.

Thiopental 4-5mg/kg IV is an ultra short-acting barbiturate that is also used for IV induction of anesthesia. However, its use may delay emergence from anesthesia if the surgical procedure is short.

Methohexital 30mg/kg, *Ketamine* 4-6mg/kg, *Thiopental* 5-8mg/kg and *midazolam* 0.75mg/kg can be given rectally for induction of anesthesia, but this is seldom done due to poor and erratic drug absorption from the rectum.

Once the child is unconsciousness, IV access is obtained. This can be challenging in children who have been fasting for many hours in a hot environment. Fortunately, most inhaled anesthetics dilate peripheral veins, which makes starting an IV easier. At times it is necessary to insert an IV while simultaneously assuring a good mask fit and adequate ventilation of the patient's lungs (**Figure 7-7**). If a second person (surgeon, nurse) is available who is skilled at starting IVs, he/she can start the IV while the anesthetist maintains the airway.

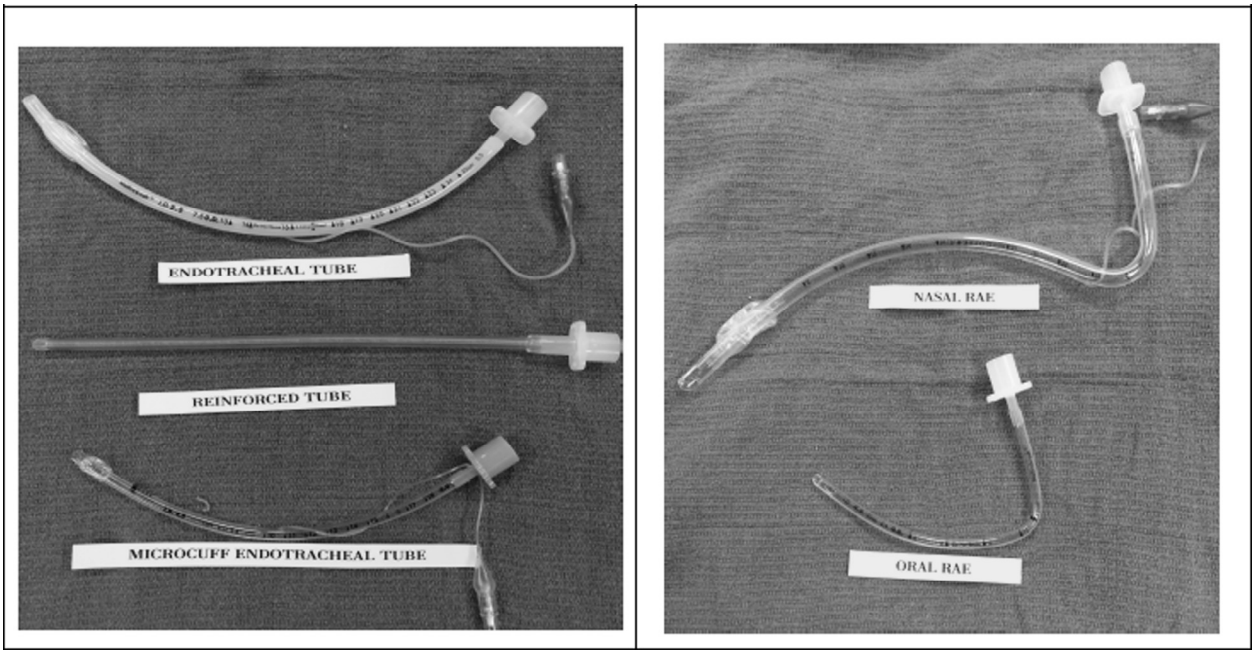
Figure 7-7. An Anesthetist Starting an IV While Maintaining a Good Mask Fit and an Open Airway



Note that the anesthetist is holding a mask and providing a jaw thrust at the same time she is starting an IV. A nurse placed a tourniquet on the arm and is holding the hand in position for the anesthetist. After the needle is in the vein, the nurse connects the IV to the intravenous catheter and secures the IV with tape.

The tracheas of most children are usually intubated under deep inhalation anesthesia. Administering a dose of IV lidocaine 1mg/kg, propofol 2-3mg/kg or fentanyl 1mcg/kg just before laryngoscopy and inserting the tracheal tube reduces coughing and breath holding that can occur when the anesthesia level is too light. A single dose of succinylcholine 1mg/kg often makes tracheal intubation easier, but it may also lead to malignant hyperthermia in susceptible patients, especially when administered with halothane. Cardiac arrest and death can also occur when succinylcholine is administered to patients who have known or unknown muscle disease. Despite this, a syringe of succinylcholine should be immediately available for injection into an upper extremity if laryngospasm and upper airway obstruction occur and the patient has no IV. If the patient has severe hypoxia and bradycardia, it may not be possible to relieve the laryngospasm by I.M. injection of succinylcholine because both conditions severely reduce blood flow to peripheral muscles. Consequently, the amount of time required to absorb the drug may be very long. Under these circumstances, it is often better to inject succinylcholine into the base of the tongue through a 25 or 30-gauge needle, because blood flow to the head is better maintained during hypoxemia and acidosis than it is to peripheral muscles. After an intra-lingual injection, the vocal cords usually relax sufficiently in about 10 seconds to permit ventilation of the lungs with oxygen. Using larger needles for intra-lingual injection of drugs (unless absolutely necessary to prevent death) may cause bleeding into the tongue and worsen airway obstruction.

Fig 7-8-Left: Types of Tracheal Tubes; Figure 7-8- Right: RAE Tubes

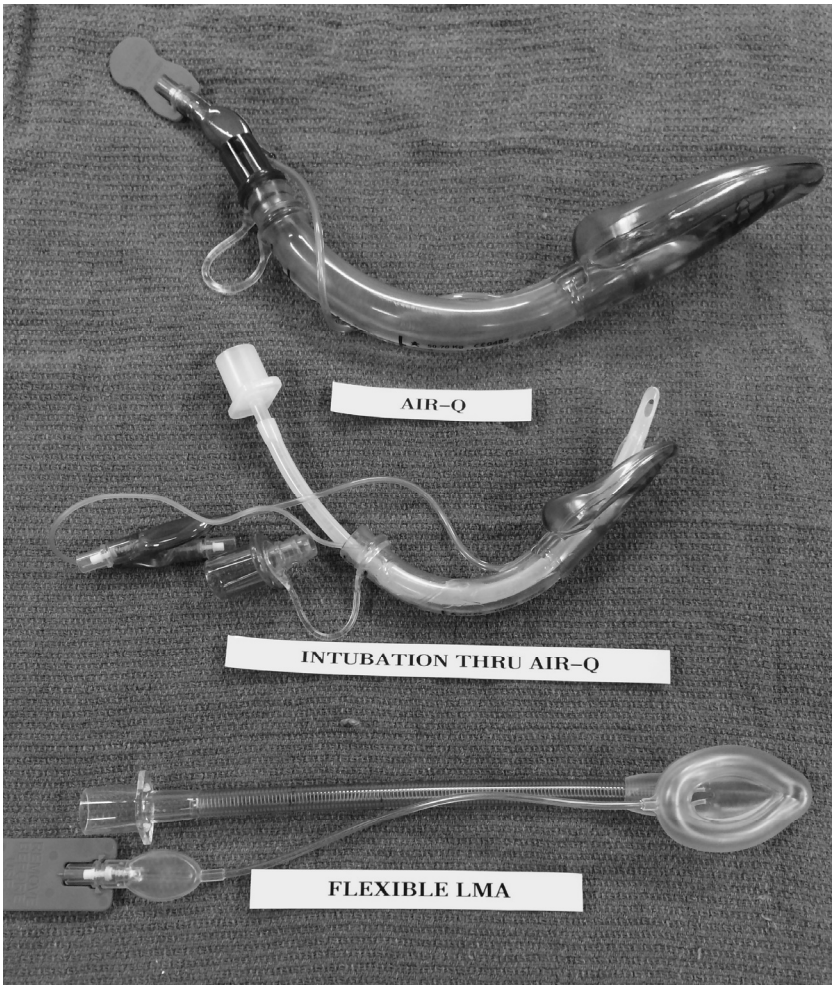


Left – Standard tracheal tubes. Right - RAE tubes. They have a preformed bend that allows the proximal end of the oral tube to be directed downward to allow the surgeon better access to the mouth and face. The nasal RAE tube has a preformed bend that allows the proximal end of the tube to be directed towards the top of the head, which potentially reduces injury to the nose during nasal intubation. Care must be taken to assure there is no upward tension on a nasal tracheal tube to prevent ischemia of the nares.

Whether to use a tracheal tube (TT – **Figure 7-8**) or a laryngeal mask airway (LMA) to secure the airway depends on the type and duration of surgery. Tracheal tubes are either cuffed or uncuffed. Cuffed TTs are ideal for abdominal and airway surgery, for preventing aspiration of gastric contents and oral secretions, and for providing more effective mechanical ventilation for patients with poorly compliant lungs. However, non-cuffed tracheal tubes are commonly used for children less than six months of age because cuffed tubes can interfere with blood flow to their tracheal mucosa and cause airway damage. When either type of tube is used, gas should be heard to leak from between the tube and the trachea when 20cmH_2O inspiratory pressure is applied to the airway. This reduces the chance of tracheal injury. Tracheal intubation is the preferred method for securing an airway for complex and/or prolonged procedures. Oral RAE tubes (**Figure 7-8 - Right)** are used for surgery on the face, mouth, throat, and neck, including repair of cleft palates and cleft lips. These tubes reduce the likelihood of the tube kinking during surgery. They also move the tracheal tube out of the surgeon's way, making it easier for her/him to see and operate. Age appropriate RAE tubes might be too long for the airways of children whose necks are shorter than average, or they might be too short for children who necks are longer than average. If the tube is too long for a given patient, the tip may enter the right mainstem bronchus or it might stimulate the carina and induce coughing. If the tube is too short, it may come out of the trachea during surgery, especially when the head is extended.

Laryngeal Mask Airways (LMAs) are often used for hernia repair, orchidopexy, and orthopedic procedures on extremities (**Figure 7-9**). However, it must be remembered that LMAs can become malpositioned or dislodged easily, especially in infants, and that when this occurs it can obstruct the airway. They add a considerable amount of dead-space to the child's airway (about 100% in neonates), and this elevates her/his $PaCO_2$ and causes respiratory acidosis. It is *inappropriate* to use an LMA in patients who have or may have increased intracranial pressure (e.g., head trauma).

Figure 7-9: Types of LMA's



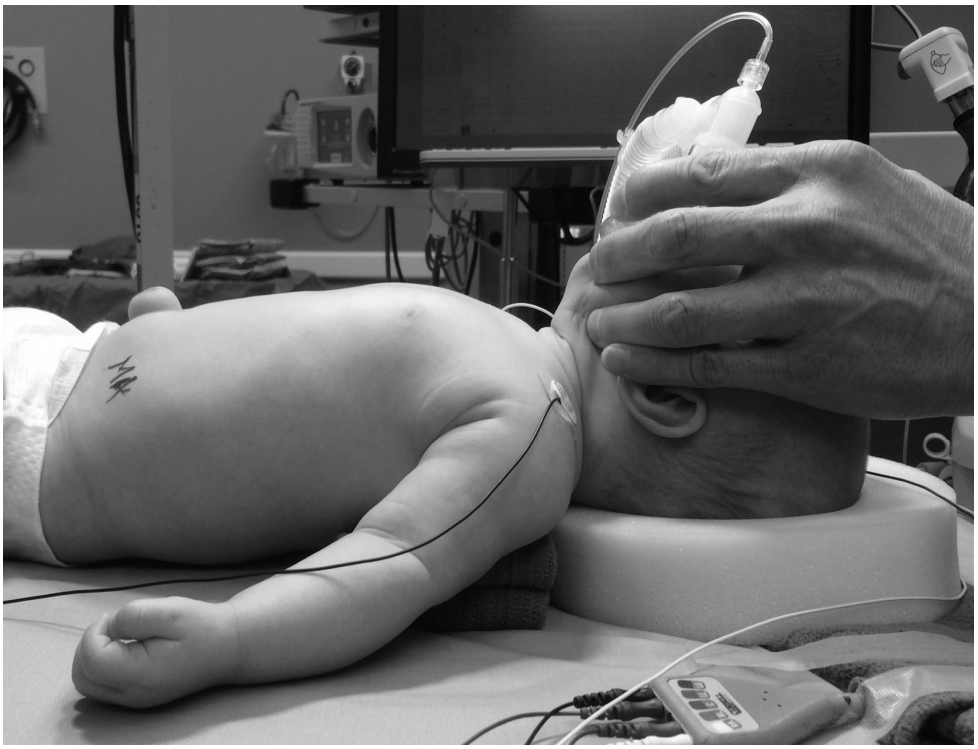
LMAs have a cuffed opening that fits over the glottis of a patient through which the patient breathes. The mask is connected to a wide-bore tube that has a 15mm connector that allows connection to an anesthesia circuit or ventilator. It is seldom possible to generate more than 20cmH_2O inspired pressure without causing gas to leak around the mask.

Complications During Induction of Anesthesia[11,12]

Several things can go wrong during the induction of anesthesia. If coughing and breath holding occur, they can quickly lead to oxygen desaturation. Low levels of inhaled anesthetics block tone in the genioglossis and hyoglossis muscles, muscles that normally keep the tongue forward and

away from the posterior pharyngeal wall to maintain a patent airway. Loss of tone in these muscles often causes upper airway obstruction, especially in young children. Enlarged tonsils and adenoids can also interfere with breathing (**See Chapter 17**). Airway secretions and regurgitation of stomach contents are frequent causes of laryngospasm. Respiratory infections, oral secretions or blood in the oral cavity, attempts at tracheal intubation during light levels of anesthesia, or painful stimulae during the second stage of anesthesia also cause laryngospasm. It is best not to examine children while anesthesia is being induced.

Figure 7-10: Sniffing Position During induction of Anesthesia and Tracheal Intubation



Note that the anesthetist's fingers are on the bone of the mandible, not in the soft parts of the chin. Placing the fingers in the soft parts of the chin pushes the tongue posteriorly and obstructs the airway. The child's face is looking forward and the head is not flexed or extended. This give the best conditions for intubation of the trachea and for ventilation of the lungs of a baby with a bag-and-mask.

Properly positioning patients during induction of anesthesia is important for the prevention of upper airway obstruction and for improving conditions for tracheal intubation. Placing a small roll under the shoulders of small children and having their heads rest on a small pillow keeps their head in a neutral position and reduces airway obstruction. **(Figure 7-10)** This improves conditions for mask ventilation and tracheal intubation. A proper fitting facemask is necessary for good mask ventilation, especially in patients who have abnormalities of their head, face, and neck. A combination of chin lift, jaw thrust, and the application of 10 cm of H_2O continuous positive airway pressure (CPAP) helps overcome upper airway obstruction in spontaneously breathing children. Once an appropriate level of anesthesia has been reached, it is possible to insert an oral

airway, if needed. The heads of small children should not be hyperextend, as this narrows the tracheas and decrease air movement. The anesthetist's fingers should be placed on the mandible, not in the soft tissue of the submental triangle of the chin. The latter pushes the tongue into the pharynx and obstructs the airway.

Laryngospasm, while a frequent occurrence in children, only occurs during light levels of anesthesia or when the patient is awakening. It does not occur in deeply anesthetized patients. Failure to recognize laryngospasm and intervene immediately can result in hypoxemia, central nervous system (CNS) injury, and death. During severe laryngospasm, the true and false vocal cords reflexly close, causing complete closure of the glottic opening and absence of air movement. Milder laryngospasm causes inspiratory noise (stridor) when the child attempts to breath through a partially closed glottis. With worsening laryngospasm, the initial suprasternal and infra-sternal retractions progress to rocking chest movements. Once the glottis is fully closed, attempts at breathing cease, and severe hypoxia and bradycardia occur. Without rapid, appropriate treatment, cardiac arrest follows. Initial treatment includes applying a tight-fitting mask to the face and creating a end-expiratory pressure of 15-20cm of H_2O plus applying jaw thrust and gentle positive pressure ventilation. This often breaks the laryngospasm. If ventilation of the lungs is successful, the depth of anesthesia is deepened. If this does not break the laryngospasm and the anesthetist has IV access, propofol 1-2mg/kg can also be administered. Succinylcholine 1-2mg/kg IV or 2-4mg/kg IM or intralingual can be used to facilitate tracheal intubation if the laryngospasm persists (see above). Gentle compression of the chest has been known to occasionally break laryngospasm, but this is not routinely recommended.

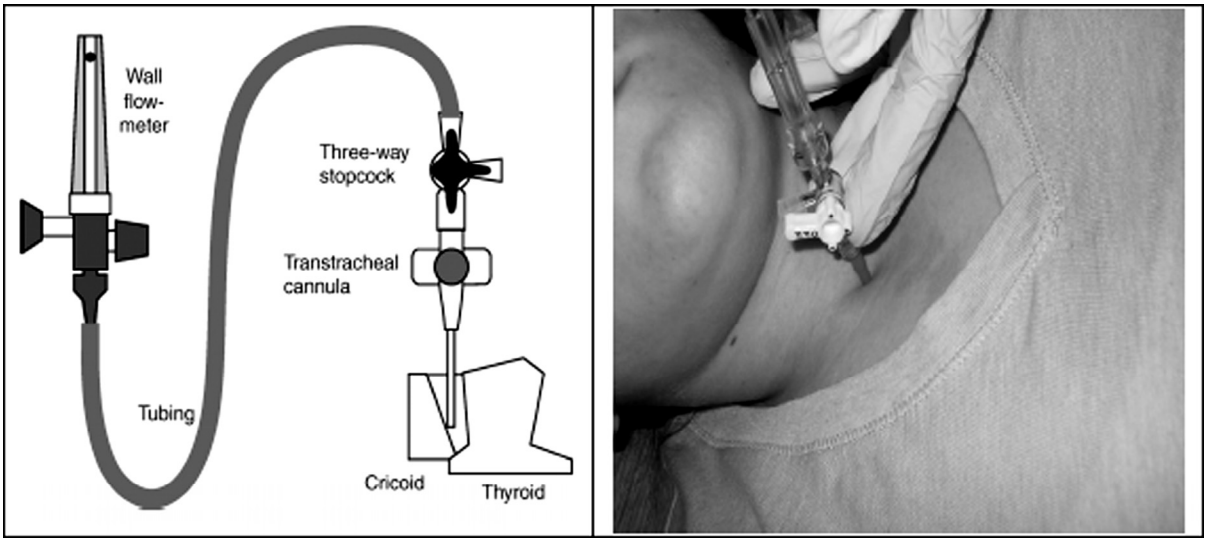
Hypotension (a >30% decrease in preoperative systolic blood pressure) is common during the induction of anesthesia in children. Both volatile anesthetics and propofol produce hypotension because they depress cardiac function and induce vasodilation. Induction-induced hypotension is more pronounced in children who have fasted for many hours or who have had vomiting or chronic diarrhea. Administering boluses (10-20ml/kg) of a balanced salt solution [e.g., lactated Ringer's solution (LR) or normal saline (NS)] often restores blood pressure to normal. It must be remembered, however, that only 20% of each bolus of fluid given remains in the intravascular space; the remainder is lost into the intra- and extra-cellular spaces. Thus, more than one bolus of LR or NS may be required to treat hypovolemia. Hemorrhage and/or sepsis are frequent causes of hypovolemia and hypotension. Ketamine is a good choice for induction of anesthesia in moderately hypovolemic patients because it causes the release of catecholamines, and this raises the arterial blood pressure.

Bradycardia has many causes, including hypoxemia, succinylcholine, and halothane. Since young children mostly depend on heart rate for cardiac output, it is important to quickly restore their heart rate to normal when it is low. Reducing the anesthetic concentration is one way of increasing the heart rate, but this usually takes a few minutes. Atropine 20-30mcg/kg or

glycopyrolate 4-10mcg/kg, on the other hand, rapidly increases the heart rate to normal. To prevent succinylcholine-induced bradycardia, atropine is often given just before or along with the injection of succinylcholine.

A *difficult airway* is defined as the inability to ventilate the patient's lungs with a bag and mask or the inability to intubate the patient's trachea by direct laryngoscopy (**See Chapter 6**). Anatomic abnormalities of the face and/or neck (short mandible, large tongue, small mouth) or the presence of a congenital syndrome or of an intraoral or pharyngeal mass should alert the anesthetist to the fact that it may be difficult to maintain a patent airway. It may be possible to determine how difficult it will be to ventilate the patent's lungs by giving a breath by bag-and-mask each time the patient inspires. Using an airway adjunct, such as a lighted wand or bougie (when available), is very helpful (**See Chapter 6).** A video laryngoscope or glidescope are other options, but these are very expensive and seldom available. If one suspects the patient has a difficult airway, spontaneous ventilation should be maintained while inducing anesthesia and securing the airway. If the lungs cannot be ventilated and oxygen desaturation occurs, an 18-gauge IV catheter can be inserted into the trachea through the cricothyroid membrane to provide an emergency airway. Doing so may be life saving. (**Figure 7-11)**

Figure 7-11. Insertion of an IV catheter for Ventilation of the Lung in a Patient with a Totally Obstructed Airway.[13, 14]



The figure on the left shows the equipment needed to provide ventilation and/or oxygenation through a catheter inserted into the crycothyroid membrane. The flow meter adjusts the flow of gas through the catheter. Because the resistance of the catheter is so high the gas flow through the catheter is low. By inserting a 3.0 tracheal tube 15mm connector into the proximal end of the tracheal catheter, it is possible to connect a Jackson-Reese breathing circuit to the tracheal catheter and slowly ventilate the lungs. Ventilation must be done slowly because the resistance of the catheter. Gas usually escapes from the lungs during exhalation, but if there is no gas escapes during exhalation, the lungs will become over inflated, which may interfere with pulmonary blood flow or cause a pneumothorax. While the lungs are being oxygenated, every effort to relieve the airway obstruction must be undertaken. The figure on the right shows a catheter through the neck and cricoid membrane of a child with airway obstruction. [16,17]

MAINTAINENCE OF ANESTHESIA[11,12]

Inhalational agents (isoflurane, sevoflurane, halothane) are commonly used to maintain anesthesia during surgery. Halothane is still commonly available and used in many hospitals throughout the world. An important problem with halothane is its ability to sensitize the myocardium to exogenous epinephrine, which results in dysrhythmias. No more than 10-15mcg/kg of epinephrine should be injected when infiltrating tissues, and the dose of epinephrine should not be repeated more often than every 20 minutes. Prolonged recovery from anesthesia is another drawback to using halothane for maintenance of anesthesia, especially if rapid turn over of cases is anticipated.

Inhaled anesthetics can be supplemented with intravenous analgesics (fentanyl 1mcg/kg, morphine 100mcg/kg) to reduce the amount of inhaled anesthetic administered. Narcotics are inexpensive and provide good intra- and postoperative analgesia. Large doses of intraoperative narcotics may delay emergence from anesthesia. *Ketorolac* 0.5 mg/kg – maximum dose 30mg - given intraoperatively provides good analgesia, but fear of platelet dysfunction and of postoperative bleeding deters some surgeons from using it. Either rectal 40mg/kg or intravenous or oral acetaminophen 15mg/kg can also effectively relieve postoperative pain.

Use of neuromuscular blockade to facilitate surgery requires a means of artificially ventilating the lungs, either a mechanical ventilator or an AMBU™ type bag during surgery. Ventilating the patient's lungs by hand often distracts the anesthetist and ties up her/his hands, making it difficult to perform and/or concentrate on other tasks. However, when the lungs must be ventilated by hand, it is important to occasionally give a breath that is twice the patient's normal tidal volume to prevent atelectasis form developing.

Regional anesthesia (**See Chapter 21**) and neuraxial blockade are great adjuncts to general anesthesia. They can also provide adequate anesthesia for surgery if general anesthesia is unavailable or cannot be performed for some reason. Sedating children with small amounts of narcotics or benzodiazepines helps calm them while the blocks are being performed. It is important that someone other than the person doing the block monitor the child's heart rate, respiratory rate, and blood pressure while the block is being performed. Regional and neuraxial blocks are cost effective and provide good postoperative pain relief as well. However, learning to successfully provide these blocks requires time and training. Infra-orbital and nasal nerve blocks provide effective pain relief for repair of a cleft lip and reduce the need for opioids, especially in babies.[15] Caudal epidural blocks are usually adequate for lower abdominal and urological surgeries. Extremity blocks effectively reduce postoperative pain for patients undergoing orthopedic and burn procedures.

Total intravenous anesthesia (TIVA) using propofol, narcotics, benzodiazepines and ketamine is more difficult to use without an infusion pump because it is difficult to control the rate of

infusion. Muscle relaxants are often used during TIVA. Before adding muscle relaxants, it is important to assure that the patient is adequately anesthetized to prevent her/him from being awake, paralyzed, and in pain during surgery. Propofol lacks analgesic properties, but it reduces postoperative nausea and vomiting. After the initial bolus dose, lower infusion rates of propofol are used for maintenance of anesthesia in babies because they clear the drug more slowly than older patients. Prolonged infusion of propofol may not be a good idea, because prolonged infusion of propofol has caused the propofol infusion syndrome (lactic acidosis and death) after several days of use in intensive care units.

Ketamine based general anesthesia is popular and effective in countries where the availability of tracheal tubes, masks, anesthesia machines, and inhaled anesthetics is limited. This drug provides dissociative anesthesia and can be administered by one of several routes (IV, IM, rectally); in most patients ketamine use preserves cardiovascular stability and airway reflexes. When using this technique, it is still necessary to assure that the airway is patent and that the patient is breathing adequately. It may also be helpful to add midazolam 0.1-0.2mg/kg IV to the initial dose of ketamine to prevent the unpleasant hallucinations experienced by some patients during recovery from ketamine anesthesia. Because ketamine increases the production of airway secretions, it is helpful to give a single dose of glycopyrrolate (8-16mcg/kg or atropine 10-30mcg/kg) with the initial dose of ketamine to block the production of secretions.

Normal saline and lactated Ringer's solutions are commonly used as maintenance IV fluids during surgery. However, giving large volumes of normal saline may lead to hyperchloremic acidosis. Albumin or plasmalyte are frequently used for rapid expansion of the intravascular volume. The anesthetist should be sure that blood products are immediately available if significant blood loss is expected during surgery.

Maintaining the patient's body temperature near normal is important. Covering exposed body surfaces with plastic sheets, clothing, and surgical drapes is a simple, inexpensive ways to do this. If possible, the surgeon should use warm, sterile irrigation solutions during surgery to help conserve the patient's body heat.

Standard ASA monitoring (arterial blood pressure, ECG, heart rate, body temperature, and SaO_2) should be used when available. (https://www.asahq.org/For-Members/Standards-Guidelines-andStatements.aspx). These monitors provide early warning of impending problems and save lives. (**See Chapter 2**)

EMERGENCE FROM ANESTHESIA

Awakening from anesthesia requires the anesthetist to anticipate the conclusion of surgery, reduce the anesthetic concentration being used, and allow the child to breathe spontaneously. If muscle relaxants were used, their effects should be reversed. The concentration of more soluble

anesthetics (e.g., halothane) should be reduced earlier to allow the patient to awaken from anesthesia at the end of surgery. Once spontaneous respiration, tidal volume, and respiratory rates are adequate, secretions are suctioned from the oral cavity and throat and the tracheal tube is removed while the lungs are fully expanded (deep breath). Otherwise any secretions present in the mouth may be sucked onto the vocal cords with the next inspiration and cause laryngospasm. The dose of narcotics or other analgesics should be given in sufficient quantities to block pain but not to such an extent that they interfere with breathing.

Tracheal extubation can be done when the patient is fully "awake" and has return of her/his airway reflexes or while he/she is still anesthetized. A fully awake child is one who is breathing adequately, opens her/his eye on request, grimaces, and makes purposeful movements. Anything less than this means the child is still partially anesthetized. This degree of awakening usually occurs at end-tidal halothane concentrations below 0.15% or sevoflurane concentrations below 0.6%. It is probably safer to extubate the tracheas of most patients when they are awake. Awake extubation is necessary for patients with delayed gastric emptying (due to drugs, trauma, or systemic illness), a difficult tracheal intubation, or respiratory problems.

Deep tracheal extubation occurs when the TT or LMA is removed from a spontaneously breathing patient who is still anesthetized. This method is recommended for children with asthma and those with elevated intraocular pressures. A surgical level of anesthesia is maintained with a volatile anesthetic (e.g., sevoflurane 1.64%). After suctioning the mouth and pharynx to remove any secretions, a large breath is given and the tracheal tube is removed while the lungs are fully expanded. This causes the patient to exhale before inspiring, which reduces the likelihood of pulling secretions onto the vocal cords or into the lungs with the next inspiration. This maneuver often induces a cough, which also helps expel any secretions. The volatile anesthetic agent is then turned off and the patient allowed to breathe 100% oxygen through a facemask while awakening form anesthesia. It is sometimes helpful to insert an oral airway for a few minutes to prevent upper airway obstruction during awakening. The child can then be taken to the recovery area (**See Chapter 22**).

The choice of deep or light tracheal extubation depends on the preference and training of the anesthetist, as well as on surgical needs. Oxygen saturations are higher during the first five minutes after deep tracheal extubation, but there is no difference in the incidence of airway complications or oxygen requirements after that. One advantage of deep tracheal extubation is that turnover of the room between cases is quicker than it is following awake extubation.

When to remove an LMA after surgery has been studied extensively. The usual practice is to remove it during deep anesthesia because this reduces the incidence of laryngospasm and oxygen desaturation.

Emergence from anesthesia can be delayed for one of several reasons, including the patient having received large doses of narcotics or being more sensitivity to anesthetic vapors. Hypothermia and acidosis prolong the effects of muscle relaxant, which can also delay recovery. When the child is ready to be transferred from the operating room to the recovery area, he/she can be transferred breathing room air if the SaO_2 is normal or while breathing 100% oxygen if it is not. The SaO_2 of about 15% of children is below 95% when they breathe room air during transport. At high altitude (where the oxygen concentration of air is reduced) it is often helpful to moderately hyperventilate the patient with 100 percent oxygen for 3-4 minutes before rapidly transferring the patient to the recovery room where he/she should again breath oxygen if possible. The hyperventilation reduces breathing during transport of the patient and maintains a more normal oxygen saturation.

Complications During Recovery

Postoperative *nausea and vomiting* are common complications during recovery from anesthesia. Prophylactic anti-emetics, such as ondansetron (Zofran- 0.1mg/Kg) and dexamethasone (4-8mg), reduce these symptoms when these drugs are given near the end of surgery. Nausea and vomiting are more common after tonsillectomy or bowel or eye surgery. Pain and dehydration also increase the incidence of nausea and vomiting. When patients are adequately hydrated and free of pain, their recovery from anesthesia is smoother.

Emergence delirium is common in 2-6 year old children, mostly after they have been anesthetized with sevoforane or desflurane.[16] Many times the symptoms are self-limited but pharmacological intervention may be needed if the symptoms persist. Small intravenous doses of propofol 0.5-1mg/Kg, midazolam 0.02-0.1mg/Kg, dexmedetomidine 0.5mg/Kg, or fentanyl 1-2mcg/Kg can be given.

Post extubation stridor is common if tracheal, laryngeal, or vocal cord edema develops during surgery and narrows the airway. Gas flow through the constricted area is turbulent and this causes the stridor (a harsh, high-pitched inspiratory sound). Stridor is more common in smaller infants and children due to the fact that their airways are smaller and that a small amount of edema significantly narrows these small airways. The amount of respiratory distress caused by this obstruction may be severe. Treatment of the edema includes humidified oxygen, dexamethasone, and nebulized racemic epinephrine (0.25ml of racemic epinephrine diluted in 2.5ml of normal saline). When racemic epinephrine is unavailable, epinephrine 0.5 ml per kg (maximal dose: 5 ml) of L-epinephrine 1:1,000 can be mixed with saline and given via a nebulizer.

Negative pressure pulmonary edema (NPPE) is a rare but potentially lethal problem. It occurs when the patient generates large negative pressures to overcome a totally or partially obstructed upper airway. Pink frothy fluid is often seen coming from the mouth. Administration of 100% oxygen and application of 5-10cmH_2O positive end-expiratory pressure (PEEP) may be required to

treat the edema. A potent diuretic, such as Lasix (1mg/kg in patients <1 year of age – max dose 2mg/kg/dose, 6mg/kg/24hrs. IV; 0.5-1mg/kg IV for older patients – max dose 6 mg/kg/24hrs.) may hasten fluid removal from the lung and improve oxygenation.

CONCLUSION

Safe induction and maintenance of anesthesia requires a thorough understanding of the patient's medical problems and past medical history. All drugs and equipment needed to meet likely emergencies should be available before the induction of anesthesia. If blood or blood products will be required during surgery, they should be available before surgery begins. Preparation should be made to care for the child after surgery (PACU, ICU, home) and for her/his pain relief.

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Chapter 7: INDUCTION AND MAINTAINENCE OF ANESTHESIA

Chapter 8:
ANESTHESIA FOR INFANTS

George A. Gregory, MD

Introduction

For the purpose of this chapter, infants are children between one month and twelve months of age. Most of them are healthy when they require surgery, but some can be quite ill. The function of many organs matures during this first year of life, and this must be taken into account when planning surgery. In some countries, many patients requiring anesthesia and surgery during the first year of life were born prematurely, and their surgery is to correct problems related to prematurity. This chapter discusses some common problems for which patients require surgery during the first year of life.

Overview of Physiologic Changes

The functions of many organs change during the first year of life and may be significantly different at 12-months of age than one-month of age. It is hoped that the information in this section will allow anesthetists to better understand events occurring during anesthesia and treat them appropriately.

Cardiovascular[1]

For the first 6-12 months of life, heart rate is the major component of cardiac output. As the infant ages myocardial contractility becomes more important and heart rate less important (**See Chapter 12**). The cardiac myocytes of young infants are composed of 50% contractile elements and 50% water and ground glass substance. The contractile elements are arranged differently and contract less effectively. By about a year of age**,** contractile elements make up approximately 75% of myocyte volume; ground glass substance makes up the remaining 25%. Early in life, the excess ground glass substance makes the cardiac ventricles “stiffer” and less compliant. Consequently, giving fluid in excess of volumes needed to produce a normal blood volume only increases CO 15%, whereas in adults, it will increase it more than 100%.[2] Increasing the infant’s heart rate (atropine, glycopyrolate), on the other hand, more than triples the CO of neonates. Drugs that decrease heart rate (fentanyl, propofol, halothane) decrease the infant’s CO to varying degrees. During anesthesia, decreases in CO seem to parallel decreases in oxygen consumption.[3] Despite decreases in CO up to 50%, normal animals show no evidence of acidosis during anesthesia. The arterial blood pressure changes over the first year of life are shown in **Table 8-1**.

Table 8-1: Normal Arterial Blood Pressures and Heart Rates vs. Age

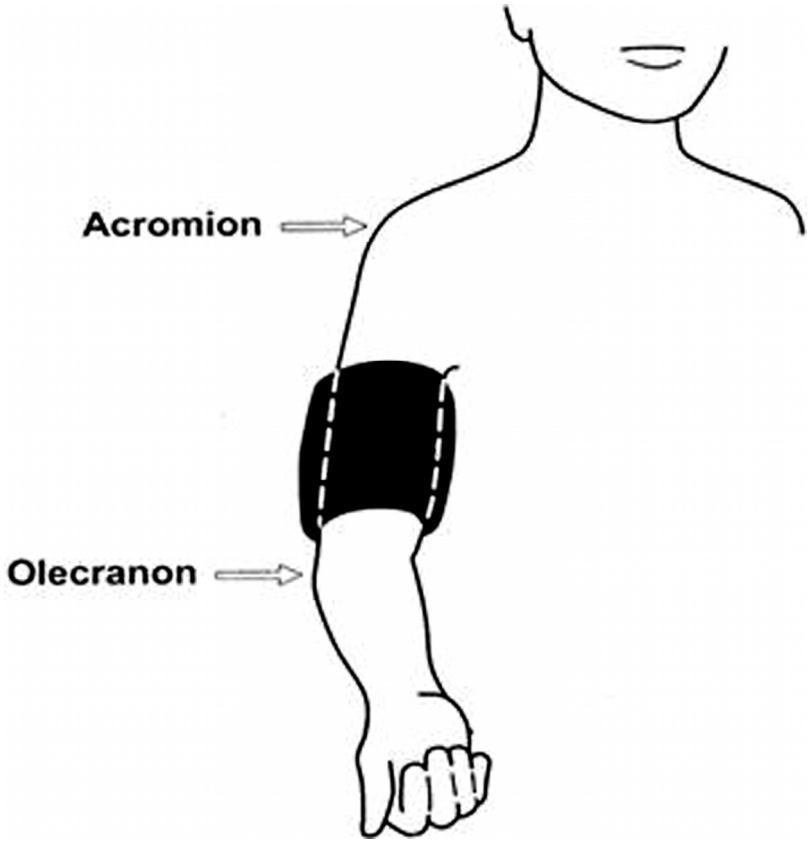
AGE	Systolic (mmHg)	Diastolic (mmHg)	Mean (mmHg)	Heart Rate (BPM)
Neonate	70 ± 9	42 ± 12	55 ± 11	125 ± 25
1 Year	98 ± 4	53 ± 3	68 ± 3	90 ± 20

Of the three arterial blood pressures available (systolic, diastolic, and mean) mean arterial pressure is usually the most helpful in infants because it provides the "average" perfusion pressure to critical organs. Even if the arterial pressure tracing is damped, mean arterial pressure is usually correct. Arterial blood pressure can be measured in several ways (**See Chapter 2**). The easiest method is with an automatic blood pressure device (**Figure 8-1**); they are relatively expensive and sturdy. However, these devices tend to give higher arterial blood pressures, especially diastolic blood pressures, in infants than intravascular measurements.

Figure 8-1: An Automated Arterial Blood Pressure Monitor

This is an automated blood pressure monitor and cuff that measures pulse rate, and systolic and diastolic pressures. It can be fitted with a smaller cuff for use in neonates.

Figure 8-2: Blood Pressure Cuff Position



This figure shows the correct position of a blood pressure cuff in a child. The cuff width should also be 40% of the circumference of the upper arm at the mid point between the acromion and olecranon bones. ***Reprinted with permission from National Kidney Foundation, Inc****.* www.kidney.org

Arterial blood pressure can also be easily measured is with a blood pressure cuff, an inflation bulb, and a manometer. This method works well for most patients, is portable, and can be easily taught to almost anyone. Position of the cuff on the arm or leg is important (**Figure 8-2**). Intra-arterial blood pressure measurements are important for sick infants because they provide beat-by-beat changes in arterial pressure. Intravascular catheters also allow the anesthetist to withdraw arterial blood for blood gases, pH and electrolyte determinations. There is a considerable amount of information available from the arterial pressure tracing besides the systolic, diastolic, and mean blood pressures. The upstroke (first part of the pressure wave) is an indication of myocardial contractility. The straighter (more vertical) the line, the better the myocardial contractility. On the other hand, if the line is bent over to the right (as in myocarditis and aortic valve stenosis), ejection of blood from the heart is delayed and decreased. The position of the dicrotic notch on the downslope of the pressure wave where the pressure increases slightly correlates with closure of the aortic valve at the end of systole. In normal adults and older children, the dicrotic notch appears in the upper one-third of the wave. In young children it is in the upper half. When the blood pressure is low or the peripheral vascular resistance is low, the

notch position is lower on the descending slope of the pressure wave. Several alterations of the pressure wave are associated with *hypovolemia*. The first is narrowing of the pressure wave. The second is lower position of the dicrotic notch on the down slope of the wave. Third is a decrease in the arterial blood pressure with inspiration, especially with mechanical ventilation (**Figure 8-3**). This change in pressure with ventilation is due to inspiratory pressure-induced decreases in venous blood return to the heart. During expiration pressure in the lungs decreases, and arterial pressure increases as blood returns to the heart. All of these features of the arterial pressure tracing can and should be used to determine the adequacy of the intravascular volume when it is possible to measure intra-arterial pressures. The blood volume of term babies is about 90ml/kg and decreases to 80ml/kg by one year of age.[4]

Figure 8-3: Intravascular Arterial Pressure Tracings

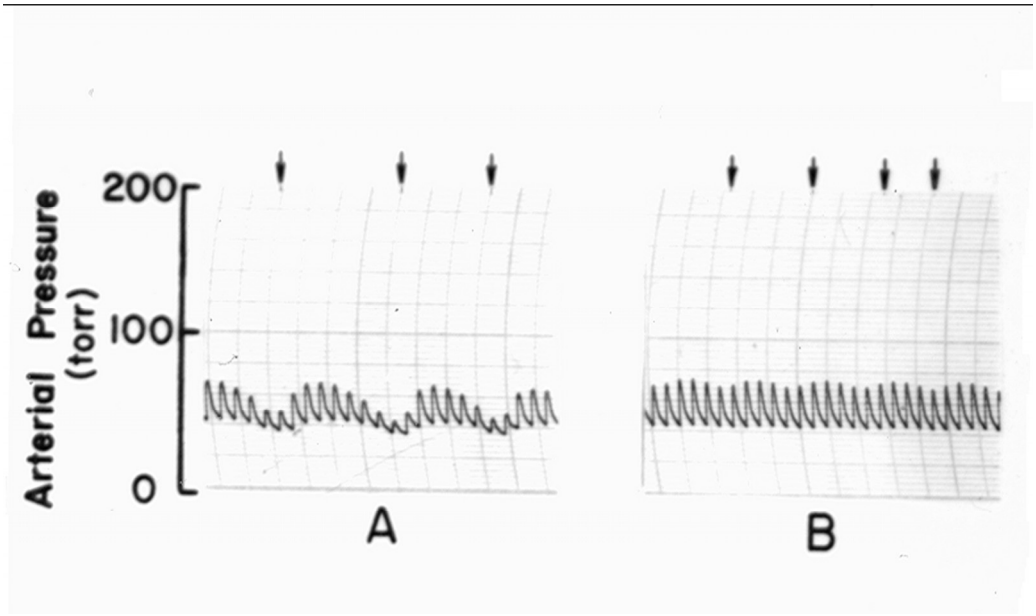


Figure A shows the arterial blood pressure tracing of hypovolemic infant. The arrows indicate inspiration. With each inspiration, the arterial blood pressure decreased >50%. During expiration the pressure was normal. Figure B shows the arterial pressure tracing from a normovolemic patient. Note that there was a <5% decrease in the systolic blood pressure with each breath (arrows).

Pulmonary[5]

The lungs are not fully developed at birth, although they allow adequate oxygenation and ventilation for the baby's needs. At birth there are only 40-50 million alveoli; by one year of age there are about 350 million.[6] The normal respiratory rate at birth is higher than that at one year of age (**Table 8-2**).

Table 8-2: Normal Pulmonary Function: Neonate, Infant, Adult

Variable			
Age	**2-10 Weeks**	**9-12 Months**	**Adults**
Weight (kg)	4.7 ± 1.2	9.6 ± 1.2	70 ± 10
Heart Rate(bpm)	120-150	80-120	60-80
Vt (ml)	21 ± 1.8	23 ± 4.3	500 ± 50
Crs (ml/cm H_2O)	5.5 ± 1.65	11.5 ± 2.4	100 ± 20
Rrscm (H_2O/ml/sec)	0.084 ± 0.021	0.042 ± 0.011	0.5-2.5
FRC (ml)	88.1 ± 26.8	178.4 ± 29.0	3000 ± 600

Kg = kilograms; ml = milliliters; Sec = seconds; Vt = tidal volume; Crs = compliance of respiratory system; Rrs = resistance of respiratory system; FRC = Functional residual volume. These are a compilation of data from Hanrahan JP, Brown RW, Carey VJ, Castile RG, Speizer FE, Tager IB. Am J Resp Care Med1996;670-80; Nguyen TTD, Hoo Ah-F, Lum S, Wade A, Thia LP, Stock J Pediatr Pulmonol 2013;48:370-80.

Because there are fewer alveoli and more connective tissue in the lungs of neonates, their lungs are stiffer, i.e., less compliant. As the infant grows, the lungs become more compliant, which takes less work to breathe. Neonates breathes 30-60 times a minute, and the one-year-old breathes 20-30 times per minute to maintain the same, normal $PaCO_2$. Tidal volume is about the same for everyone, infants and adults, about 7-10cc/kg. Therefore, for the $PaCO_2$ to be the same with a lower respiratory rate, the tidal volume of each breath must increase, which it does. Blood gases change during the first few months of age (**Table 8-3**).

Figure 8-3: Normal Blood Gases and pH -f Neonates and 1-Year Olds

Variable 0	Neonate	Infant
PaO2 (mmHg)	55 - 85	85 - 100
PaCO2 (mmHg)	35 - 45	35 - 45
pH	7.36 - 7.43	7.34 - 7.44
BE (meq/l)	-3 - +3	-2 - +2
SaO2 percent	9 - 100	96 - 100

PaO_2 = partial pressure of oxygen; $PaCO_2$ = partial pressure of carbon dioxide; BE = base deficit (excess); meq/l = miliquivelents/liter. Partially modified from Siberry GK, Iannone R. The Harriet Lane Handbook, Mosby, St Louis, MO, 2000

Because the chest walls of neonates is much more compliant than their lungs, neonates usually have atelectasis for much of the first month of life, and this is reflected in their PaO_2, i.e., the PaO_2 is lower in neonates and increases to adult levels by about a month of life as the ribs begin to calcify and they maintain a negative intrathoracic pressure at end-expiration. The neonate's functional residual capacity (FRC - volume of gas remaining in the lung at the end of expiration) is smaller than that of older children and adults (neonates = 30ml/kg; one year old = 40 ml/kg).

This means that neonates and young infants have less total oxygen in their lungs at end-expiration (25cc in 1mo old; 84cc in 1yr old) [weight in kg x FRC in ml x 0.21% oxygen], if he/she is breathing room air. The infant's oxygen consumption is about 10cc/kg, which means there is only enough oxygen for about 1-2min before they become hypoxic. Cardiac output (250-300cc/kg/min, in neonates and 70-80ml/kg in adults) also affects the rate of developing hypoxemia. Higher COs remove oxygen more quickly from the lung. The net result of a smaller FRC, higher oxygen consumption, and higher cardiac output is more rapid oxygen desaturation in young infants (often in about 1-2min). Oxygen desaturation occurs commonly in infants during the induction of anesthesia due to inadvertent hypoventilation or the administration of narcotics or inhaled anesthetics.

Renal

The urinary tract consists of two kidneys, two ureters, a urinary bladder and a urethra. Urine is produced by 10-12 weeks gestation **See Chapter 3** for development of the urinary system and function. In utero, urine produced is excreted into the amniotic fluid. Failure to make or excrete urine and maintain a normal amniotic fluid volume causes compression of the infant's chest and underdevelopment of the lungs. This often results in respiratory distress at birth and necessitates prolonged mechanical ventilation. Multiple sites in the urinary tract are prone to develop abnormalities and obstruction during fetal life. These include the urethra (urethral valves), the ureterovesico junctions (i.e., where the ureters enter the bladder), and the ureteropelvic-junction (where the ureters connect with the kidneys). In neonates, symptoms caused by urine blockage include: fever, not eating, irritability, diarrhea, vomiting, frequent urination, and the presence of dark, cloudy or bloody, foul-smelling urine. Untreated urinary tract obstructions can cause developmental abnormalities of the kidneys, or if severe enough, to not function at all.

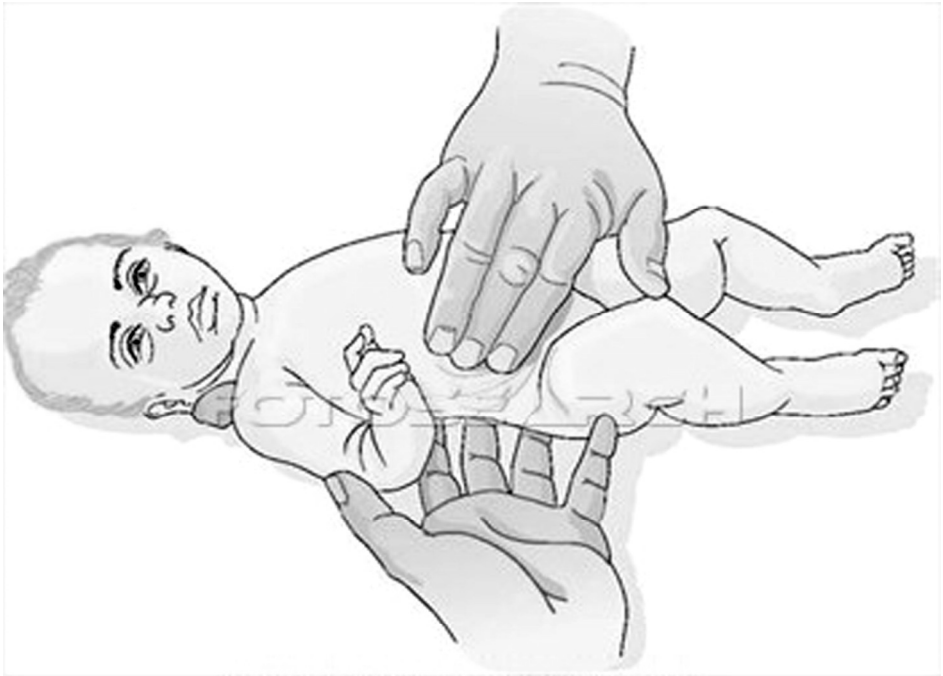
Developmental genitourinary anomalies often require surgical repair during the first year of life. These include posterior urethral valve, ureteral obstruction, vesicoureteral reflux, cayliceal abnormalities, renal and adrenal tumors, and extrophy of the bladder. The following is a discussion of the types of surgery commonly seen in patients between one month and one year of age.

Posterior urethral valves (PUV) are present in about 1:8,000 live births, primarily in males. PUVs are the result of an abnormal membrane that crosses the opening from the urinary bladder to the urethra. This is the primary cause of bladder outlet obstruction in neonates. Symptoms include: low amniotic fluid volume, a distended bladder, and in some patients respiratory failure from hypoplastic lungs. The diagnosis of posterior urethral valves is frequently made by an ultrasound examination of the abdomen, which shows the distended bladder, hydronephrosis, a thick bladder wall, and bladder diverticula. A surgeon makes the diagnosis at cystoscopy when he/she visualizes the posterior uretheral valve. The treatment of posterior urethral valves is relatively

simple. The abnormal tissue is ablated surgically during cystoscopy, which opens the urethra to urine flow.

Ureterovesical-junction (UVJ) obstruction is due to insufficient lengthening of the ureter, which prevents normal formation of the ureterovesical valve at the junction of the ureter and the bladder. When this occurs, the open valve allows reflux of urine from the bladder into the ureter. This prevents normal kidney growth and functioning, placing the kidney and ureter at risk for developing postnatal urinary tract infections (UTIs). During physical examination, it may be possible to palpate an enlarged kidney. Some times, it is possible to palpate an enlarged ureter. The infant's kidney is palpated by placing one hand behind the upper abdomen and the other on the abdomen just below the right costal margin (**Figure 8-5**). By moving the front hand back and forth and pressing gently downward, one can usually feel the kidney as a globular mass. If it is enlarged, it is easy to feel the kidney. An ultrasound of the abdomen will show the kidney and the obstruction. Treatment may include daily administration of antibiotics to prevent urinary tract infections, but surgery is the only definitive treatment of this lesion.

Figure 8-5: Palpation of the Infant Kidney



See text above for description of kidney palpation. With Permission.

Ureterocele is another form of UVJ obstruction. A portion of the ureter fails to develop normally where it enters the bladder, and this abnormality bulges into and obstructs urine flow into the bladder. As a result, the ureter dilates, and the kidney develops hydronephrosis. Surgery is the

primary treatment of this lesion.

Ureteropelvic junction (UPJ) obstruction is a condition in which blockage of the junction between the ureter and the kidney reduces/prevents urine flow from the kidney to the ureter. This results in hydronephrosis (distention of the kidney pelvis and calices with urine) and abnormal kidney maturity. The diagnosis is ultimately made by ultrasound, but can be suspected during physical examination by feeling a large kidney (See above). When available, radionuclide scans can be helpful, as is ultrasound. Antibiotics are usually given preoperatively to reduce the incidence of UTI.

Diagnosis of urinary tract obstruction is most easily made by ultrasound. It is also important to search for abnormalities in serum electrolytes. Whether electrolyte abnormalities occur or not depends on whether one or both kidneys are obstructed. Compensation by a non-obstructed kidney may lead to normal serum electrolytes. If both sides of the urinary tract are involved (e.g., posterior urethral valves), both kidneys may be abnormal. These patients may well have electrolyte abnormalities. Acidosis or hyperkalemia (K^+ >5meq/l) increase both the number of arrhythmias during anesthesia and the risk of cardiac arrest. The likelihood of cardiac arrest happening increases when serum K^+ concentrations exceed 6meq/l. Hypernatremia (elevated sodium concentration) occurs when patients cannot excrete normal amounts of sodium (Na^+) due to the renal failure and blockage. Hypermagnisemia may also occur. If it does it may affect the central nervous system, muscles, heart and blood vessels (hypotension, bradycardia), and in severe cases it may causes coma and cardiac arrest. If possible, electrolyte abnormalities should be corrected preoperatively. However, this is not always possible.

Definitive treatment of urinary tract anomalies is usually surgical, usually when the patient is less than one year old. Because of their young age, these patients require general anesthesia, even though some bladder procedures can often be done endoscopiclly. When the patient has no IV preoperatively, one can be inserted after the induction of anesthesia with inhaled anesthetics (halothane, sevoflurane). If the patient has an IV, anesthesia can be induced with oxygen, propofol 2-3mg/kg or sodium pentothal 4-6mg/kg IV. Because these procedures are frequently lengthy, it is better to intubate the trachea and control ventilation during surgery when possible. Surgical retraction and positioning of the patient for surgery often make spontaneous breathing ineffective.

Intraoperative noninvasive arterial blood pressure, ECG, and SaO_2, and temperature are adequate monitoring for most of these patients. However, it may be necessary to measure electrolyte concentrations intraoperatively in some patients. Temperature measurement is important because manipulation of infected kidneys, ureters, and the urinary bladder may lead to hyperthermia with temperatures of >40°C. When this occurs, the anesthetists must quickly determine if the fever is due to seeding the circulation with bacteria or to malignant hyperthermia (**See Chapter 23**).

Fluid and electrolyte administration can be complicated for these patients, especially if they have fluid retention or electrolyte abnormalities. Some of these infants have fluid retention and edema because there urine output is low. It is important to determine this preoperatively so that fluid administration can be adjusted to compensate for any hypoervolemia present (**See Chapter 14**). An enlarged liver suggests right ventricular failure. This should lead the anesthetist to cautiously give intraoperative fluids and not cause further volume overload and worsening right ventricular failure. Fluids should be limited to only those amounts that are necessary to maintain normal perfusion and arterial blood pressure.

If the child has hyperkalemia, mild hyperventilation may reduce the serum K^+ concentration slightly, but may also increase the likelihood of a seizure, especially if the patient is hypocalcemic. The pH should be maintained within the normal range when possible. On the other hand, hypoventilation will make existing hyperkalemia worse, because extracellular hydrogen ion (H^+) is exchanged for intracellular potassium, which raises serum potassium concentrations even higher. If the serum ionized calcium concentration is reduced, it may be necessary to give calcium gluconate or calcium chloride to correct the abnormality. Hypocalcemia plus hyperventilation may lead to seizures. Hyperventilation must be avoided. Being able to measure end-tidal CO_2 is very helpful for maintain $ETCO_2$ concentrations.

Anesthetists should plan for treatment of postoperative pain (**See Chapters 20 and 21**). The simplest and most effective method of relieving pain is with a caudal or epidural anesthetic that is placed after the patient is asleep. As long as the patient is cared for in a monitored unit postoperatively, a catheter can be placed in the caudal or epidural spaces and the patient can be given repeated doses of local anesthetic and preservative free morphine or fentanyl (**See Chapter 21**) in the intensive care unit (ICU). If this cannot be done, pain can be relieved with narcotics, if available, nonsteroidal ant-inflammatory drugs (NSAIDs), or acetaminophen, tramadol (**See Chapters 18 and 20**).

Ear Nose and Throat

Tonsillectomy and/or adenoidectomy (T & A) are two of the most common ear, nose, and throat procedures done in infants. They are usually performed because the infant has chronic tonsillitis or enlarged tonsils and adenoids that obstruct her/his airway. Infants with severe obstruction may develop sleep apnea and may go on to develop pulmonary hypertension.

Sleep apnea occurs in two percent of infants with enlarged tonsils.[7] Patients with this condition pause their breathing or stop breathing all together for varying amounts of time or have shallow or infrequent breathing when asleep. Apnea is defined as cessation of breathing for 20 seconds or more. Periodic breathing occurs when breathing stops for <15 seconds. Repeated apneic periods may lead to hypoxemia and acidosis, pulmonary vasoconstriction, and eventually pulmonary hypotension. In rare cases it may lead to right ventricular failure. If it is believed that

the patient has pulmonary hypertension, the anesthetist must determine the degree of pulmonary artery obstruction present and whether it is fixed or not. If it is fixed, the pulmonary vessels fail to dilate with breathing 100% oxygen or nitric oxide, or with respiratory alkalosis. There are three types of apnea: central (CSA), obstructive (OSA), and mixed.

CSA is characterized by a lack of respiratory effort due to lack of central nervous system signaling to initiate breathing. This is the least common type of OSA. Ondine's Curse (failure to breath when asleep) is one form of CSA and is usually fatal.

OSA is relatively common and is caused by relaxation of the pharynx and the genioglossis and hyoglossal muscles. This causes airway obstruction that is made worse during inspiration and leads to snoring. As with most upper airway obstructions, the tongue "falls back" in the throat and partially obstructs the airway. During inspiration, the patient attempts to overcome the obstruction by generating more negative pleural pressures. The increased negative pressure "pulls" the tongue against the pharyngeal wall, increasing airway obstruction. Because there is little or no air movement, $PaCO_2$ increases and SaO_2 decreases. The increased CO_2 awakens the patient and he/she begins to breath. Patients with OSA often make respiratory motions (the chest moves) during inspiration, but little or no gas moves into the lungs. Children with OSA have excessive sleepiness during the day, difficulty with school (if they are school age) and enuresis (bed wetting). They are often belligerent and hard to get along with. Measuring and recording SaO_2 throughout a night is useful for the diagnosis of obstructive sleep apnea. Hypoxemia, sleep depravation, obesity, exposure to cigarette smoke makes OSA worse. Treatment may include decreasing exposure to cigarette smoke, weight loss, sleeping upright or on side, nasal continuous positive airway pressure (CPAP), or other methods of keeping the pharynx open during sleep. Tonsillectomy and adenoidectomy are often the best choice. Patients with sleep apnea will usually not be immediately better after T & A. It may take months for their sleep apnea to go away. Thus, the patient must be observed carefully for signs of OSA, and if the patient was using CPAP preoperatively, he/she should continue to do so after surgery.

Mixed sleep apnea is characterized by the presence of both OSA and CSA simultaneously. Many of these patients have abnormal response to CO_2 and may not awaken as easily form apnea.

History and Physical Examination

Performing an effective history and physical examination is important in all patients, but is especially important in those undergoing T & A. What symptoms is the patient having? Why is the patient having surgery? Is it because he/she has repeated tonsillitis, or is it because he/she has sleep apnea (**See above**)? Does he/she have difficulty drinking/eating and swallowing? Does he/she have difficulty exercising (if old enough)? If so, is it due to airway obstruction? Can the patient open her/his mouth – how far? Are the tonsils enlarged (touching each other)? Can the patient breath through her/his nose when their mouth is closed, or are her/his adenoids so large

they obstruct the airway? In infants, closing the child's mouth and placing a few strands of cotton in front of a nostril helps evaluate whether the child can breath through her/his nose. If the cotton moves with breathing, the nose is not obstructed. Then test the other nostril. Is the second heart sound louder than the first sound? This occurs when there is an element of pulmonary hypertension. Is the liver enlarged? It should normally be 1-2cm below the right costal margin. If it is larger than this, it may be due to an element of right ventricular failure that is the result of pulmonary hypertension. Infants with pulmonary hypertension seldom have enlarged neck veins, as adult do. Are the pulses normal or are they decreased? This could indicate some degree of myocardial failure. Is the child well hydrated and making urine (**See Chapter 1**)? If the patient has some heart failure and is hypovolemic, cardiac output will be inadequate when the patient is anesthetized. Is the patient's blood pressure normal for age (**See Appendix 1**)? Are the breath sounds normal or are they decreased due to airway obstruction?

Anesthesia is usually induced with inhaled anesthetics and the concentration of anesthetic increased over several minutes. A positive end-expiratory pressure of 5-10cmH_2O is used to open the pharynx and prevent the tongue for obstructing the airway. CPAP prevents the airway from completely collapsing and causing total airway obstruction at very light levels of anesthesia. If necessary an oral airway can be inserted, assuming the anesthetist can get it between the enlarged tonsils. Inserting a nasal airway is usually is *not a good idea* in patients who have enlarged adenoids because doing so may dislodge a portion of the soft, infected adenoids. When this occurs, there may be significant bleeding that will make ventilation of the patient's lungs more difficult and may make it more difficult to see the glottis and intubate the trachea. An oral RAE tube (**See Chapter 7**) is used when available, because this removes the tube from the surgeon's field and makes it easier for her/him to see. The surgeon must take care not to touch the tracheal tube with a cautery to avoid initiating an airway fire.

Three things are need for an *airway fire* to occur: 1) Oxygen >30% or nitrous oxide; 2) Something that will burn (tracheal tube or sponges; 3) A spark (cautery).[8] All of these are present during a T & A. Use of a cuffed tracheal tube reduces the amount of oxygen leaking around the tracheal tube and decreases the concentration of oxygen in the posterior pharynx to that of room air. If there is a leak of anesthesia gases around the tracheal tube during ventilation, the concentration of oxygen in the pharynx may increase when more than room air (21% oxygen) is used for the carrier gas, and this will be a potential source of combustion. If possible the inspired oxygen should be kept below 30 percent throughout surgery because this reduces the likelihood of fire. In all cases, the surgeon should not touch the tracheal tube with an electrocautery. If a fire occurs, **Figure 8-6** shows actions that should be taken. Survival of these patients depends on rapid treatment of the problem and on the extent of the burn.

Table 8-6: Outline for Action During a Tracheal Fire

This plan of treatment should be understood by everyone in the operating room and be initiated immediately.[8]

Young children are frequent victims of otitis media and require incision and drainage of the middle ear and/or placement of ear tubes to equalize the pressure inside the middle ear with that in Eustachian tubes. Both procedures are quick and are easily done with an inhaled anesthetic delivered through a facemask. It is important that the head not move while incisions are made in the eardrum or when tubes are being inserted. This is especially important if the surgeon is performing the procedure with the aid of a microscope. Very slight movements are magnified by the microscope, which makes it difficult for the surgeon.

Tracheostomy is sometimes required in infants. If possible, a tracheal tube or LMA is inserted to control the airway during surgery because the complication rate of tracheostomy is lower when this is done. Infants have small necks. Thus, there is a greater chance of injuring the carotid artery or jugular vein during tracheostomy. For this reason, it is important to have a good IV for this procedure. Occasionally, air dissects along the pre-tracheal fascia and causes a pneumothorax or a pneumomediastinum.

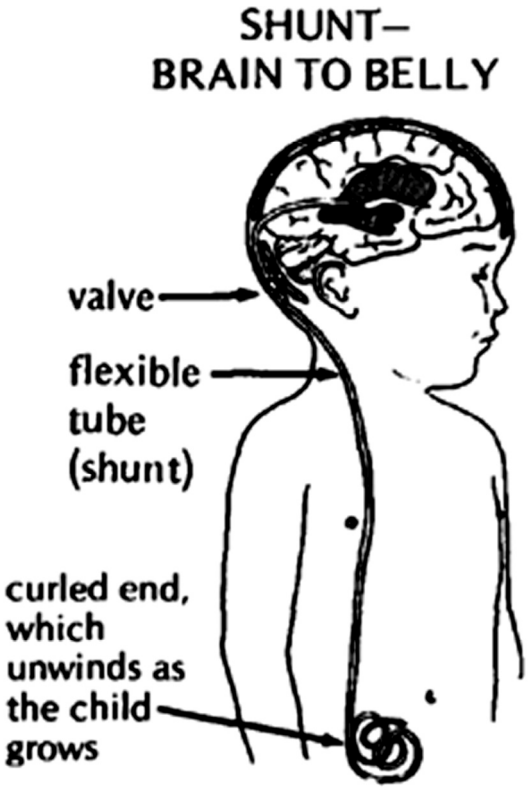
Stridor is common in infants due to abnormalities of their larynx and trachea. Stridor is a high-pitched musical breath sound that is caused by turbulent airflow in the larynx, trachea, or bronchi. It is the result of narrow or obstructed airways and can be inspiratory, expiratory, or both. However, it is usually heard during inspiration, often when a patient has croup. Epiglottitis, a foreign body in the airway, or uncommonly laryngeal tumors can be the cause of stridor in infants. Glossoptosis (a tongue that is too large for the mouth) or a small mandible (Pierre Robin Syndrome) may also cause Stridor.

Neurosurgery

Neurosurgery is not commonly required during the first year of life, except in a few instances (**See Chapter 10**). One of these instances is treatment for hydrocephalus.

Hydrocephalus occurs when there is a disturbance in the balance of cerebral spinal fluid (CSF) production and removal. Under normal circumstances the balance between these two variables results in a relatively constant volume of CSF and a relatively constant intracranial pressure (ICP). When this balance is disrupted, the volume of CSF and the ICP increase. If the increase in pressure is excessive, it interferes with cerebral blood flow (CBF) and with tissue oxygenation and nutrition. Symptoms of increased ICP in an infant include irritability, inability to console the infant, vomiting, and when the pressure is high, somnolence. If this goes on for a period of time, the baby may stop breathing and have a cardiac arrest. Removal of excess CSF from the brain is the only treatment of hydrocephalus. However, the excess CSF should not be removed by spinal tap, as a sudden decrease in ICP below the brain may cause the increased pressure in the brain to force the brainstem through the foramen magnum, causing apnea, unstable cardiac conditions, and death. Placing an intraventricular to peritoneum shunt is the definitive treatment for hydrocephalus. This requires a small craniotomy to place a pressure relief valve into the ventricle through the brain. The external portion of the pressure valve is connected to a catheter that is passed subcutaneously into the peritoneal cavity where excess CFS is drained. This CSF is absorbed from the peritoneal cavity. As a temporizing measure, the neurosurgeon can place a catheter transdermally into a cerebral ventricle. The catheter is connected to a sterile bag. A stopcock in the system is intermittently turned to drain CSF.

Figure 8-7: Ventriculoperitoneal Shunt



This figure shows the ventriculoperitoneal shunt system in a young child. See above for description. From: www.dinf.ne.jp

Anesthesia is usually provided by sevoflurane or by intravenous narcotics and propofol. Both drugs can depress respiration and increase the $PaCO_2$, which will increase the ICP further. Therefore, it is prudent to ventilate the patient's lungs to maintain a normal $PaCO_2$. When available, the best way to maintain the $PaCO_2$ normal is by measuring end-tidal CO_2. If the child's $PaCO_2$ was elevated for some time, he/she may have compensated for the increased CO_2 by accumulating sodium bicarbonate in the blood. If this occurred, it is easy to hyperventilate these small children and make them very alkalotic, which will markedly reduce cerebral blood flow. If the alkalosis goes on for very long, CNS injury may occur. Care must also be taken keep the arterial blood pressure at or near the preoperative pressure to help maintain adequate CBF. Having an elevated ICP and a decreased arterial blood pressure will cause marked cerebral hypoperfusion and CNS injury. The SaO_2 should be kept above 96 percent by increasing the inspired oxygen concentration (FiO_2) if needed. Sudden decreases in ICP may lead to hypotension, for reasons not clearly identified. At the end of surgery, removal of the tracheal tube occurs when the patient is breathing adequately and maintaining a normal (for the patient) end-tidal CO_2 or $PaCO_2$. Pain is usually not a significant component following a VP shunt and can

be treated with NSAIDS or acetaminophen. It is better to avoid administering narcotics to these patients for pain because it may increase the incidence of apnea after surgery. The surgeon can place long-lasting local anesthetics (Marcaine 0.25%) in the scalp and abdominal incisions.

Unfortunately, VP shunts become obstructed and require replacement. Catheter infections are also common, which necessitates removal of the shunt, placement of an external drain, and treatment with antibiotics until the infection has cleared. Then a new VP shunt can be paced.

Arterial-venous malformations (AVM) are relatively uncommon lesions in infants. Abnormally developed intracranial vessels, both arterial and venous, cause these malformations. At times blood flow through these abnormal vessels is so extensive that patients develop high-output heart failure (gallop rhythm, hepatomegaly, and a murmur or bruit heard over the anterior fontanel. The echocardiogram of patients with high-output failure, if available, will shows cardiac ventricular dysfunction and dilatation of the right atrium. The heart rate is elevated and the pulses are often diminished, due to the heart failure. Pulmonary edema may occur. Treatment of AVMs includes injecting material into the vessels to occlude them and reduce the high output failure.

Anesthesia for patients who have an AVM and high-output failure requires close attention to the patient's cardiovascular system. How is the heart failure being treated? Is the patient receiving diuretics, and is her/his fluid intake being limited? If either is true, the patient may be intravascular volume depleted and in heart failure. Is the patient receiving vasoactive drugs, such as dopamine hydrochloride, to improve myocardial function? If so, the drug should be continued during anesthesia and the dose adjusted as needed. When the arterial-venous malformation is partially or totally occluded by the procedure, cardiac output will decrease towards normal and the dose of dopamine or other vasoactive drugs may have to be decreased rather rapidly. Inserting an arterial catheter to measure beat-to-beat variation in arterial blood pressure is important and should be done when possible. This catheter not only allows measurement of arterial blood pressure but also permits the anesthetist to obtain blood for determining acid-base status and blood gases. Arterial blood gases should be maintained as normal as possible because acidosis or alkalosis may worsen any heart failure present. It is common for radiologists to use large volumes of "flush" solution and dye during the procedure, which may worsen the heart failure before the vessels are occluded. It is important to keep track of the volume of blood taken out and of fluid and dye given. Continuous measurement of urine output is also important, as an excess of dye can either increase urine output and cause hypovolemia or decrease renal function. It is usually necessary to mechanically ventilate the patient's lungs during and after surgery. Using a PEEP of 5-10cmH_2O usually improves oxygenation.

Tethered spinal cord is a common complication of spina bifida and is often repaired during the first year of life (**See Chapter 10**). A tethered spinal cord occurs when tissue attachments prevent the spinal cord from moving in the spinal column. As the child grows, this lack of movement

stretches the spinal cord, and this may cause neuologic symptoms; including pain and incontinence; it may also cause scoliosis. Patients with a tethered spinal cord frequently have dimpled skin over the lower spine; some children have fatty tumors on the lower back. Deformities of the feet are common, as is progressive leg weakness.

Excision of spinal tumors and correction of meylomeningocel are uncommon in this age group (**See Chapter 10**).

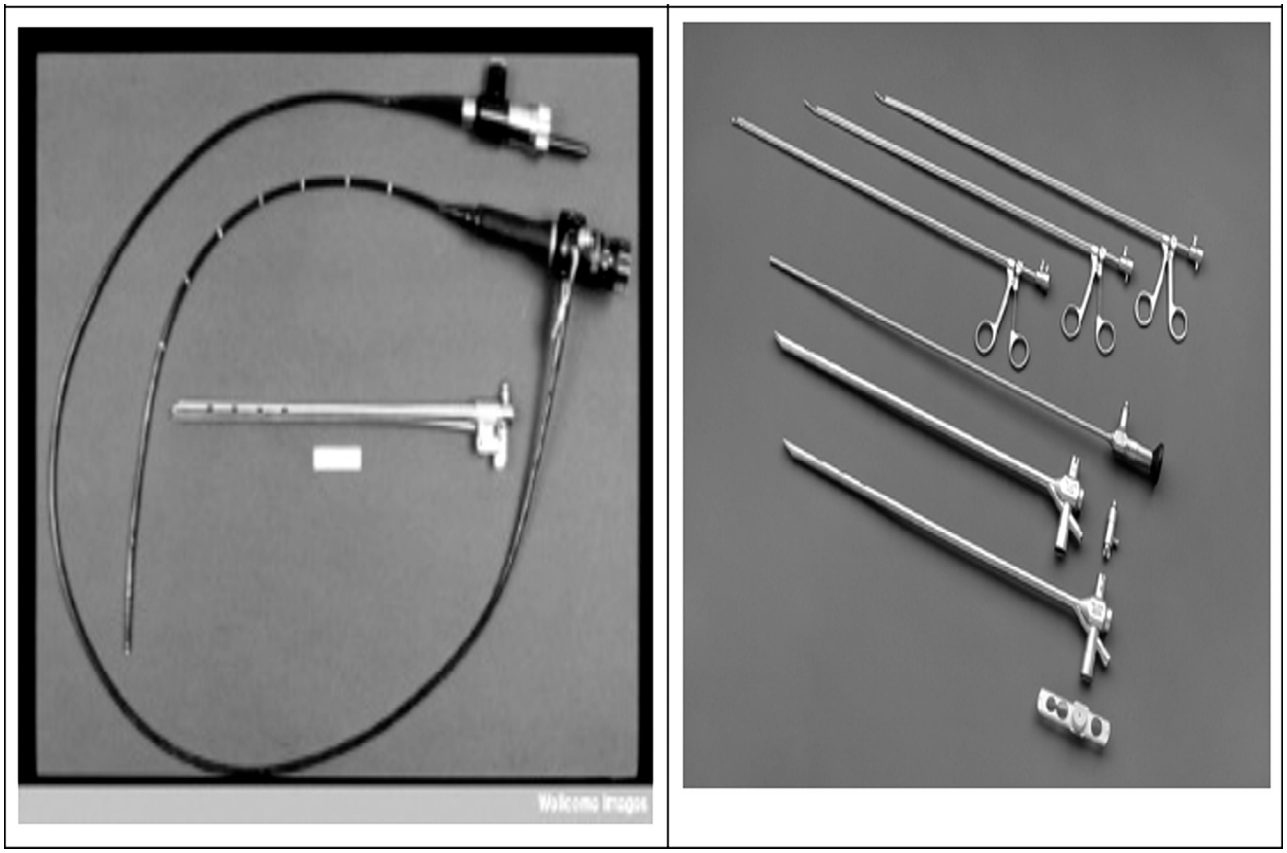
Pulmonary

There are a few reasons that infants require anesthesia for thoracic surgery during the first year of life. These include: Bronchogenic lung sequestration, congenital lobar emphysema, and bronchogenic cysts, which will not be discussed here (**See Chapter 11**).

At times, infants require *bronchoscopy* for diagnosis of tracheal or laryngomalacia, to remove foreign bodies, and for tumors. Patients requiring bronchoscopy commonly have respiratory distress, including intercostal, substernal, sternal, and suprasternal retractions; tachypnea; grunting respirations; wheezing; and occasionally cyanosis. The retractions are evidence of increased work of breathing. Grunting respiration is evidence of loss of FRC and occurs because the baby is attempting to increase FRC by rapidly taking in a breath and exhaling slowly through partially closed vocal cords. Grunting is heard during exhalation. Tachypnea, rather than deeper inspirations, occurs because it is less work for a child with a relatively unstable chest wall to breath rapidly and shallowly than it is to take deeper breaths. Taking deep breaths tends to increase chest distortion and decrease effective breathing. Rapid breathing also helps maintain FRC by decreasing the amount of time available during expiration to empty the lung of gas. This improves oxygenation.

There are two forms of bronchoscopy, fiberoptic and rigid (**Figure 8-8**).

Figure 8-8: Types of Bronchoscopes



*On the left is a **fiberoptic bronchoscope**, which is compared to a rigid scope (middle of image). The fiberoptic scope easily passes through an appropriate size tracheal tube. The light is very bright and the optics provide very clear images of the airways. This device is useful for observing movement of the airways during breathing. On the right are images **of rigid bronchoscopes** with three graspers above for obtaining tissue or removing foreign bodies from the airways. Below (in order) are the light source and two bronchoscopes of different sizes. There is a connector for delivering oxygen or connecting a ventilator to the device to breath for the patient during bronchoscopy.*

Fiberoptic bronchoscopy (**Figure 8-8 on left**) is often used to evaluate dynamic changes in the airway. The simplest way to do this is to insert an LMA and place the bronchoscope through the LMA while the patient inhales anesthesia and oxygen. The vocal cords are easily identified through the laryngeal end of the LMA and this allows the bronchoscopist to visualize the vocal cords and determine if they move normally during spontaneous breathing. Once the vocal cords have been examined, local anesthesia (2% lidocaine sprayed through an atomizer – max dose 7mg/kg with epinephrine and 4mg/kg without epinephrine) can be sprayed on the vocal cords and the bronchoscope can then be advanced through the vocal cord into the trachea to determine if the airway moves normally during inspiration or collapses.

Rigid bronchoscopy (**Figure 8-8 on right**) uses a rigid metal tube and a light source. It is especially useful for biopsying tumors and for removing foreign bodies from the airway. Aspiration of foreign bodies occurs in this age group of patients because they place things into their mouths and have relatively poor control of swallowing. Peanuts are common foreign bodies that infants aspirate. Once aspirated, peanuts swell and obstruct airways. They may not be easy to remove in tact because they disintegrate when touched with a grasper.

Anesthesia for bronchoscopy is provided in one of several ways. Inhalational anesthesia is often used because it tends to depress respiratory reflexes and reduce coughing, gaging, and laryngospasm when the scope is inserted. Because a fairly deep level of anesthesia is required to block airway reflexes, hypotension may occur and may require treatment with drugs such as ephedrine (100-200mcg/kg). The dose may have to be repeated. To get around this problem, some anesthetists give muscle relaxants and paralyze the patient. While paralyzed, the patient's lungs must be ventilated through the bronchoscope. This is accomplished during rigid bronchoscopy by adding oxygen to the side port of the bronchoscope and intermittently occluding the end of the bronchoscope with a finger. When the bronchoscope is occluded, the oxygen flowing into the lungs expands the chest and gives a "breath". Removing the finger from the end of the bronchoscope allows exhalation. During fiberoptic bronchoscopy, the bronchoscope is inserted through the LMA or tracheal tube and the lungs are ventilated with a mechanical ventilator or Jackson-Reese system. A biopsied tumor may cause airway bleeding. If this occurs it may be necessary to quickly advance a tracheal tube into the opposite main stem bronchus and provide one-lung ventilation.

*Laryngotracheal polyps a*re benign airway lesions that can obstruct the larynx, trachea, and bronchi, depending on their location. They may be due to the human papilloma virus or airway trauma (tracheal intubation, tracheostomy). The lesions are often recurrent and can severely obstruct the airway, especially when more than 50% of the airway is occluded. Symptoms of obstruction include: dyspnea (difficult or labored breathing), occasionally cyanosis, inspiratory retractions, use of accessory muscles of breathing (intercostal muscles, nasal flaring), wheezing or decreased breath sounds, stridor.

These polyps are usually removed surgically. In developed countries, this is done with a laser and then covered with podophllin to suppress polyp regrowth. When no laser is available, they are removed with a cautery and then threated with podophllin. If it is necessary to intubate the trachea to treat these lesions, the tracheal tube should be taped with aluminum foil tape to reduce the possibility of an airway fire; <30% oxygen should also be used during laser or cautery use (**See Above**). Induction of anesthesia in children with airway polyps can lead to total airway obstruction. Many practitioners permit these patients to breath spontaneously throughout the procedure once the airway is secured. If possible, the tracheal tube should not pass through the polyps (except in an emergency) or the polyps may be pushed deeper into the airway and seed

new areas. Positive pressure ventilation may do the same. If it is possible for the surgeon to remove a large portion of the polyps, there will be less airway obstruction at the end of surgery. If so the tracheal tube can be removed before the patient awakens from anesthesia.

Abdominal Surgery[9]

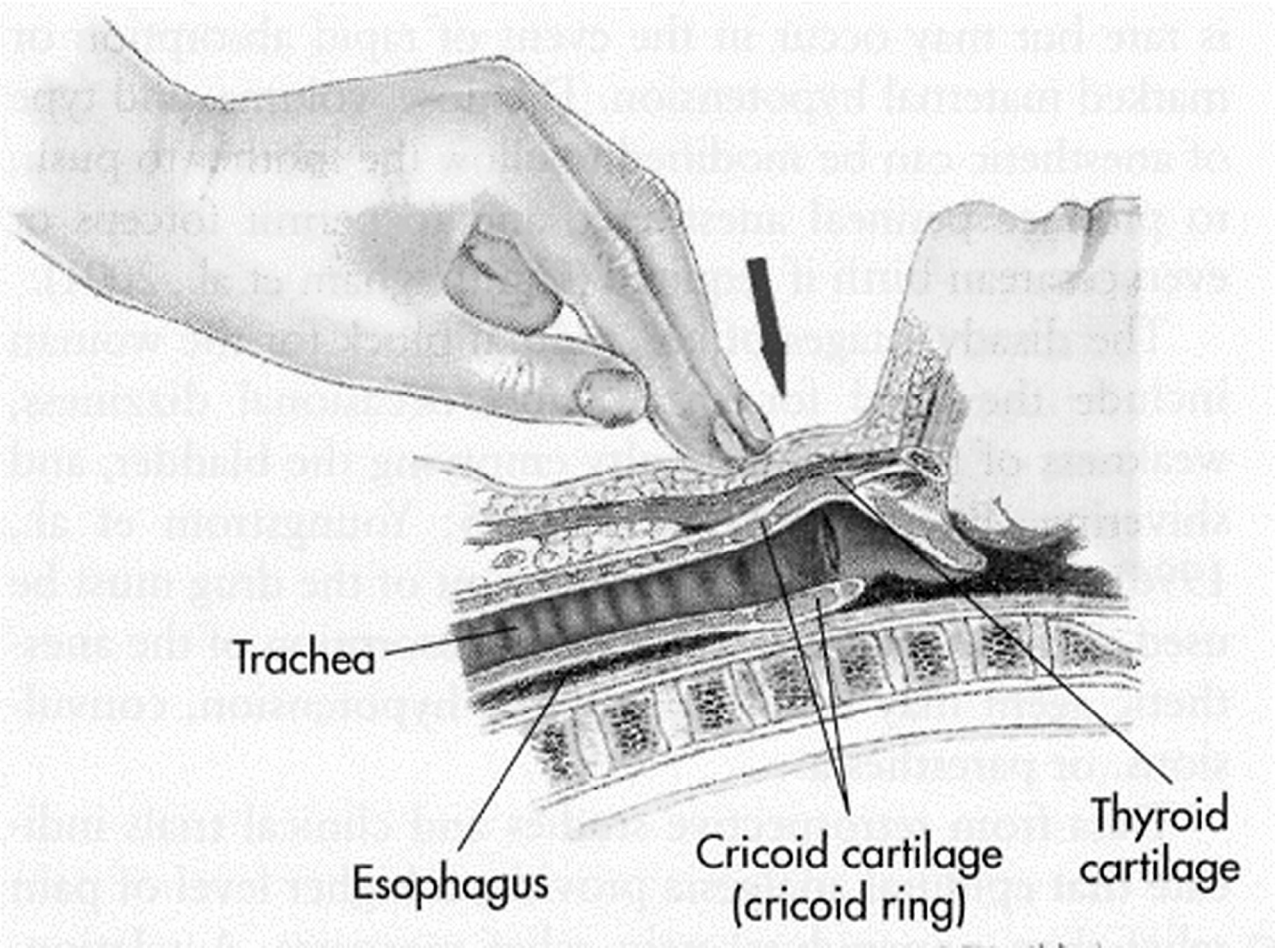
Infants often undergo abdominal surgery for inguinal hernia repair, urology procedures, necrotizing enterocolitis (NEC), imperforate anus, Hirschprung's disease, duodenal atresia, bowel obstructions, and pyloric stenosis.

Inguinal Hernias

Inguinal hernias occur in 0.8% - 4% percent of children born at term and about 10% of those born prematurely.[10] Hernias are more common in males. Fifteen-twenty percent of inguinal hernias are bilateral.[11] They can be either symptomatic or asymptomatic. An inguinal hernia is diagnosed when a bulge is found in the labia, scrotum, or groin that gets larger with crying, upright position, or moving. If a hernia cannot be reduced (incarcerated) or the blood supply is obstructed (strangulated), the hernia is accompanied by pain. Incarceration occurs in about 15% of all patients with hernias and in nearly 30% of infants with hernias. Strangulated hernias are often associated with a bowel obstruction. Surgeons try to reduce hernias (i.e., return them to the abdomen) to rule out the presence of strangulation or to prevent it from happening. Narcotics are given to treat the pain, and the child is placed in a head down (Trendelenburg) position while gentle pressure is applied over the hernia. This reduces the hernia into the abdomen in about 75% of instances. Children with strangulated hernias often have evidence of bowel obstruction (vomiting, abdominal distention, fever, and/or abdominal pain). If the hernia cannot be reduced, urgent surgery is required to relieve the obstruction and prevent bowel death.

Anesthesia for patients with strangulated or incarcerated inguinal hernias is not different from that for patients with other bowel obstructions. They should be considered to have a full stomach and to be at risk for vomiting and aspiration of gastric contents. Sufficient suctioning should be available to quickly remove any material found in the mouth. If possible, a nasogastric or orogastric tube should be inserted before inducing anesthesia to remove as much gastric fluid and material as possible, although suctioning will not remove everything from the stomach.[12] When possible, anesthesia should be induced in a head up position while holding cricoid pressure. Pressure is applied over the crycothyroid cartilage in a backward, upward direction (**Figure 8-9**).

Figure 8-9: Cricoid Pressure



Cricoid pressure is applied over the cricoid cartilage in a backward and upward direction. This figure shows a completely occluded esophagus with this maneuver. However, cricoid pressure fails to occlude the esophagus in about 15% of patients due to anatomic variations in the position of the esophagus. From: intranet.tdmu.edu.ua

If excessive cricoid pressure is exerted, the soft trachea may also be obstructed, making it more difficult to breathe for the patient or intubate the trachea. In addition, excessive pressure may make it difficult or impossible for the patient to breath spontaneously. There is continuing debate about whether or not anesthetists should ventilate the patient's lungs when cricoid pressure is being applied. If there is a delay in accomplishing tracheal intubation, the patient's lungs should be gently ventilated with oxygen by bag-and-mask to maintain oxygenation. If excessive cricoid pressure prevents insertion of the tracheal tube, cricoid pressure is relaxed slightly to allow the tracheal tube to be inserted.

There is debate about whether anesthesia should be induced by mask or by rapid-sequence if the inguinal hernia is incarcerated or strangulated. If the patient has an IV, he/she can be oxygenated for 3-5 minutes while breathing spontaneously, following which propofol 3mg/kg and

succinylcholine 1-2 mg/kg are given intravenously. The succinylcholine should be preceded by 30mcg/kg of atropine to prevent succinylcholine-induced bradycardia and decreased cardiac output. As soon as the patient stops breathing, a tracheal tube is inserted and the lungs are ventilated with oxygen. When the anesthetist is certain the tracheal tube is in the correct position and the lungs can be ventilated adequately, cricoid pressure can be released. If the patient has no evidence of bowel obstruction, anesthesia can be induced via a mask and an IV can be inserted.

Caudal anesthesia can also be used to provide excellent pain relief after surgery in patients who have hernias without a bowel obstruction (**See Chapter 21**). Spinal anesthesia has been used with great success for infants with inguinal hernias. However, the pain relief provided by spinal anesthesia lasts a shorter time than a caudal anesthetic. When a spinal or epidural anesthetic is used, it is often helps to have the infant suck on a nipple stuffed with cotton and soaked with glucose during surgery. This prevents her/him from crying, straining and pushing bowel out through the wound, making it difficult for the surgeon to perform the surgery. The child can also be sedated during the surgery.

At the end of surgery, the patient can be awakened and the tracheal tube removed. For postoperative pain relief, ilioinguinal nerve blocks (**See Chapter 21**) can be preformed or the surgeon can infiltrate the wound with local anesthetic. NSAIDS can also be used. Postoperative narcotics are seldom needed.

Either open or endoscopic surgery is used to repair inguinal hernias. Endoscopic repairs usually cause less postoperative pain, but they have a higher rate of recurrence. One advantage of an endoscopic procedure is the ease with which the surgeon can explore the opposite side to see if there is a hernia on that side also. This is important because up to 60% of patient have a patent processus vaginalis. For either repair, it is common to insert an LMA or intubate the trachea of patients undergoing inguinal hernia repair.

Appendectomy

Appendicitis is uncommon in infants, but when it occurs, it has potentially serious consequences for the patient because the appendix has ruptured in 80% of <1 year-old infants at the time of surgery. The younger the patient, the more likely perforation is to have occurred. This is due to the fact that infants cannot communicate their symptoms to their parents or surgeons and because their symptoms are more vague than those of older children. The most common presenting symptoms in infants are fever, diarrhea, and abdominal tenderness rather than the classic symptoms of appendicitis in older patients (anorexia, nausea and vomiting, periumbilical pain followed by right lower quadrant pain). However, the infant's symptoms can also be caused by intussusception of the bowel, respiratory illness, infections, and gastrointestinal disorders. The vagueness of these symptoms delays the diagnosis and causes the high (85%) incidence of

perforated appendix. It also leads to an approximately 20% rate of normal appendix at appendectomy surgery.

Management of appendicitis includes hydration (they frequently do not drink sufficient fluids), broad-spectrum antibiotics to cover both gram positive and gram-negative organisms, and treatment for pain (if needed). Antibiotics are usually given after surgery until the child is afebrile for two days. In many countries appendectomies are done laparoscopically, although 10-20% of these procedures must be converted to open laparotomy.

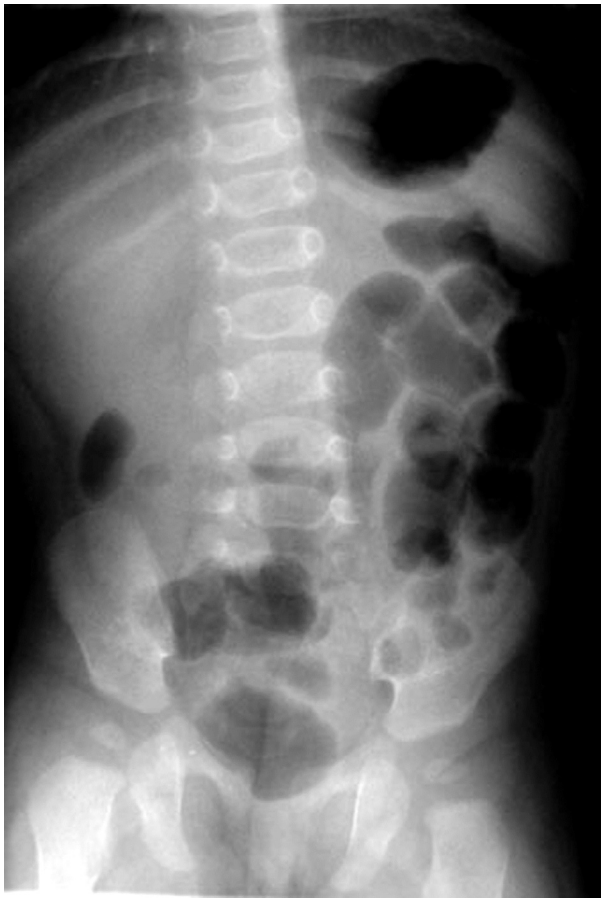
Because these children have a "full stomach" and are vomiting, anesthesia is induced by rapid sequence induction when possible. It can also be induced by mask induction while holding cricoid pressure, remembering that cricoid pressure is ineffective in 15-20% of patients. If possible, a nasogastric tube should be inserted to drain as much gas and fluid from the stomach as possible before inducing anesthesia. Suctioning the stomach may reduce the risk of gastric aspiration but does not remove all of the gastric fluid. Some liquid and solid food (if there is any) may remain in the stomach and can be aspirated. Since many infants have a perforated appendix, they may be septic and require a significant amount of fluid administration to maintain a normal arterial blood pressure. Occasionally vasopressors may also be required. Surgical manipulation of infected material may make sepsis worse. Postoperative pain can be treated with narcotics, but usually they are treated with narcotics and Tylenol or Ketorolac to reduce the total dose of narcotics given. Epidural anesthesia/analgesia can be used, but there is concern about doing so in septic patients.

Meckel's diverticulum is a congenital small sac off of the distal ileum. While they relatively common and can be found in two percent of people, they are usually asymptomatic. If symptoms occur, they usually do so before two years of age and consist of painless rectal bleeding, intestinal obstruction, volvulus, and intussusception of the bowel. At times the symptoms are difficult to differentiate from those of appendicitis. When available, the diagnosis is made by a technetium-99 scan, or it can be made at colonoscopy. Ultrasonography and CT scan may also be helpful. When symptomatic, surgery is the treatment for a Meckl's diverticulum. The anesthesia considerations are similar to those for appendectomy.

Intussusception is a condition in which one segment of intestine (usually large bowel, especially in infants) turns into (invaginates) the intestinal lumen next to it causing bowel obstruction (**Figure 8-10**). However, intussusception does occur in small bowel, especially when a gastrojejunal tube that is too long is in place. The tip of the tube serves as a lead point for the intussusception. Intussusceptions usually occur in the first year of life, although it can occur later.[13] If treated early the outcomes are very good. If treatment is delayed, intussusception can be fatal. Small bowel intussusceptions tend to self-correct if not caused by a gastrojujunal tube; large bowel obstructions do not. About 30% of infants with intussusception have concurrent a viral upper respiratory tract infection (URI).

These patients often arrive at hospital with bilious vomiting if the patient has a bowel obstruction; colicky, intermittent pain; current jelly type stool (blood, mucus, sloughed cells); and diarrhea. They may be dehydrated from vomiting and lack of fluid intake (**See Chapter 1**). On physical examination, a sausage shaped mass can be felt (between spasms) in the right lower quadrant of the abdomen in about 40% of patients. Treatment of intussusception is either nonsurgical or surgical. Nonsurgical treatment consists of filling the bowel with water, barium, or gas to force the intussuscepted bowel out of the other bowel and corrects the obstruction. If this does not work, the patient must undergo surgery to relieve the obstruction. Occasionally a portion of bowel must be resected. After air reduction, one study found a 10% incidence of recurrence of intussusception. Over all, >90% of nonoperative reductions of intussusceptions were successful. Sedation is required for this procedure because distending the bowel with air is very painful.

Figure 8-10: Intussusception of the Bowel



This abdominal X-ray shows small bowel dilatation and little gas in the right lower and upper quadrants of the abdomen. The sausage shaped, air filled bowel in the right lower quadrant is the intussusception. Courtesy of Hanmin Lee, MD

Malrotation and Volvulus of the Bowel. At 8-10 weeks gestation the bowel returns to the abdomen from the umbilical coelom, rotates counter clockwise and fixes in place in the right and

left side upper quadrant. If this does not happen, the bowel is free in the abdomen. This allows malrotation of the bowel, which allows the bowel to be rotate around a narrow mesentery, and this puts the child at risk for volvulus (twisting of the bowel) and occlusion of the bowel's blood supply. This is one of the few true emergencies in pediatric surgery. Failure to correctly diagnose the patient's condition and appropriately treat it is often fatal.

Despite the fact that approximately 1in 500 people have malrotation of their bowel, only a small portion of them develops a volvulus. When they do, it usually occurs in the first few weeks of life. Volvulus can be divided into acute and chronic. The acute form is of most concern for the anesthetist because the entire midgut may be ischemic. Failure to unwind the bowel leads to intestinal necrosis and death. Patients with a volvulus appear with bilious vomiting, metabolic acidosis, lactatemia, oliguria, haematochezia, hypotension and shock with progressing ischemia. However, there may be no acidosis preoperatively if the vessels are obstructed and acid cannot get into the circulation. When the bowel is untwisted and blood flow returns, however, the patient may suddenly have severe metabolic acidosis and may undergo cardiac arrest and death.

Anesthesia for infants with torsion of the bowel is similar to that all patients with a bowel obstruction and a full stomach. These infants may also be quite intravascular volume depleted from decreased fluid intake and extravasation of fluid into the bowel. Careful preoperative evaluation of intravascular volume and appropriate correction of preoperative hypovolemia is necessary to prevent hypotension and death with the induction of anesthesia. Sometimes large volumes of fluid >40ml/kg are required to correct volume losses. Many of these patients also have clotting abnormalities from diffuse intravascular coagulation (DIC). When possible, clotting studies should be done pre- and intra-operatively. Monitoring of these patients during anesthesia and surgery includes frequent determination of arterial blood pressure (with an arterial line if possible – **See Chapter 2**), ECG, SaO_2, and body temperature. Once the torsion is relieved, the patient may become severely hypotensiive and require additional volume and vasopressor administration. Some patients develop hypocalcemia and require calcium administration, especially when there is evidence of reduced cardiac output and tissue perfusion. Because fluid and glucose intake have been compromised, some patients may be hypoglycemic. Consequently, it is important to measure blood glucose concentrations during surgery and initiate appropriate treatment when needed. If hypokalemia is detected, it should not be treated until the acidosis is controlled, unless there is evidence of hypokalemia on ECG (decreased T-wave amplitude, ST-segment depression and T-wave inversion, presence of a U wave). If sodium bicarbonate is given to partially correct metabolic acidosis, this should be done carefully while ventilation is increased to eliminate the CO_2 produced. Remember, each milliliter of bicarbonate produces 25cc of CO_2 when fully reacted with acid. This must be removed, in great part by the lungs.

Inhaled anesthetics are more likely to cause severe hypotension in these sick infants than divided doses of fentanyl 30-50mcg/kg or morphine 200-300mcg/kg. Since these patients are paralyzed

with muscle relaxants during surgery, use a narcotic based anesthetic is appropriate. This combination of drugs tends to better maintain cardiac output, arterial blood pressure, and tissue perfusion in sick infants. Mechanical ventilation will probably be required postoperatively. While it is possible to use spinal or epidural anesthesia for these cases, this is probably not a good idea because the patients are often septic. Postoperative pain relief is required and can usually be accomplished with low dose fentanyl or morphine (**See Chapter 20**).

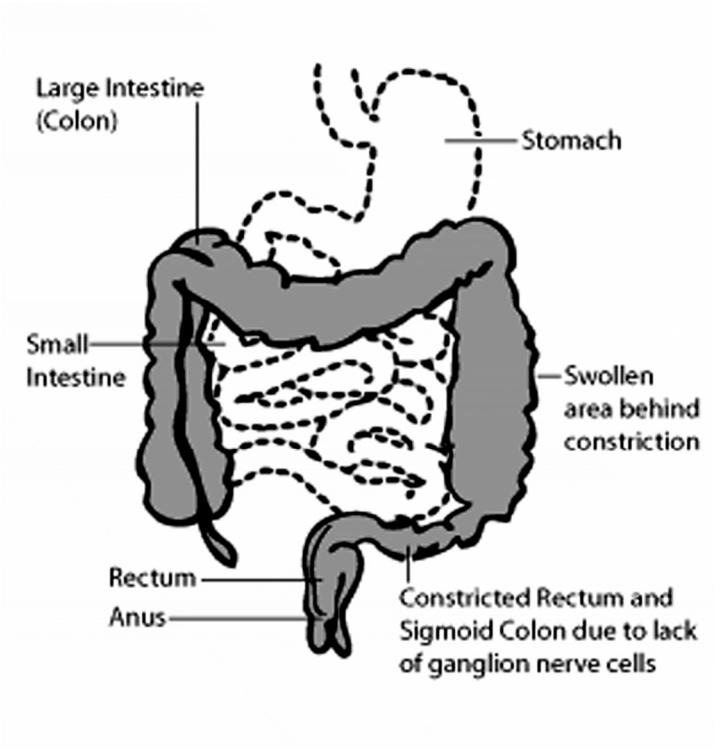
Pyloric Stenosis: Pyloric stenosis is found in 2:1000 live births and usually occurs during the first two months of life. It is due to thickening of the pyloric muscle. During contraction of the stomach and pylorus muscle, the pylori's contracts and narrows, causing a bowel obstruction. The most common symptom is non-bilious forceful vomiting, often several feet. The diagnosis is easily made by ultrasound when available. In about half of the patients, it is possible to feel and "olive" sized firm mass just to the right of the midline near the costal margin. Peristaltic waves can be seen moving across the upper abdomen of some infants after feeding. Despite frequent vomiting, these patients usually appear hungry and eat well. The vomiting leads to inadequate fluid intake, dehydration, lethargy, decreased number of bowel movements, and constipation in sicker patients whose diagnosis is delayed. If not diagnosed and treated early, vomiting causes the loss of Na^+, K^+, $Hall^-$ (Hydrochloric acid) □ alkalosis and dehydration □ Na^+ and fluid retention in exchange for K^+ □ paradoxical academia □ alkalosis and dehydration □ more loss of Na^+, K^+, $Hall^-$ with additional vomiting. If these electrolyte abnormalities are present, a nasogastric tube should be inserted and connected to intermittent suction when possible. The electrolyte abnormalities should be corrected before proceeding with surgery. Care should be taken to avoid hyperventilating these already alkaloid patients because this will worsen their alkalosis, decrease cerebral blood flow more, and cause hypotension. Correction of pyloric stenosis is not an emergency. The surgery should only be done when the patient's condition is stable.

Pyloric stenosis is done either open or endoscopic ally. In many countries almost all of these repairs are done endoscopic ally because it shortens hospital stay. However, both types of surgery require tracheal intubation for airway protection. All of these patients require an IV preoperatively. This allows the anesthetist to do a rapid sequence induction of anesthesia while cricoid pressure is being held (**See Above**). If an open procedure is done, a caudal anesthetic is helpful for the treatment of postoperative pain (**See Chapter 21**). If the procedure is done endoscopic, this is not necessary. Pain in the latter group of patients is easily controlled with Tylenol, ketorolac, etc.

Hirsch rung's disease is a disorder of the bowel (mostly large intestine) that occurs in about 1:5000 births (**Figure 8-13**). It is caused by abnormal development and absence of the nerve supply (ganglion cells) to parts (or rarely all) of the colon that prevents affected colon from relaxing and pushing stool through. As a consequence, patients have severe constipation. The obstruction usually occurs in the colon closest to the anus but can occur anywhere in the colon or

the entire colon may be involved. It occurs four times more often in males and is more common in white than in non-white babies.

Figure 8-11: Hirschprung's Disease



This figure shows the underdeveloped area of colon that lacks ganglion cells and relaxes poorly. The area of bowel above it is dilated. Courtesy of Hanmin Lee, MD

Diagnosis of Herschprung's disease is based on both clinical and biopsy information. Failure to have a meconium stool within the first 48h after birth suggests this diagnosis, as does presence of megacolon on X-ray. The enlarged segment of bowel is found proximal to the aganglionic segment of bowel. Following digital examination of the child's rectum, there may be an "explosive" stool. Vomiting may also occur. The gold standard for diagnosis of Hirschprung's disease is biopsy of the affected segment of colon. The diagnosis is made when no ganglion cells are found in the biopsied material.

Treatment consists of performing a colostomy to relieve the bowel obstruction and to allow the child to grow, although some surgeons now do corrective surgery in very young infants. When those caring for the infant think the patient is in the best condition possible, the surgeon resects the abnormal portion of bowel and connects normal bowel to the anus by one of several techniques (Swenson, Soave, Duhamel, Boley). Fifteen percent of patients do not receive full bowel control from these procedures and must have enemas and other procedures each day to have a stool. Anesthesia for these patients is similar to that for any patient with a bowel

obstruction (see above).

In infants, *abdominal masses* can be of many origins (**Table 8-7**).

Table 8-7: Causes of Abdominal Masses in infants

Neoplastic Tumors	Infectious	Other
Hepatocellular Carcinoma	Hydatid Cyst	Impacted Feces
Hepatoblastoma	Toxic Megacolon	Mesenteric Cysts
Neuroblastoma	Retroperitoneal Intra-abdominal Abscess	Intussusception Volvulus
Wilms Tumor		
Teratomas		
Lymphoma		
Rabdomyosarcoma		

Modified from Gregory GA, Andropoulos DB (eds.). Gregory's Pediatric Anesthesia, 5th Edition. Wiley-Blackwell, Oxford UK; 2012, pp. 2012.

Neuroblastomas are neural crest tumors in the adrenal glands and paraspinus ganglia. They are the most common abdominal tumor of infancy and can be life threatening. Slightly more boys have this tumor than girls. Upwards of 75% of patients have metastasis to bone marrow, liver, lymph nodes, and skin. Presentation of neuroblastoma depends on site of the primary tumor, the presence of metastasis, and tumor size. Some patients present early with pain and malaise, but most, especially those with a sizable mass, present with abdominal pain, weight loss, abdominal distention, anemia, and failure to thrive. About one fourth of patients have catecholamine producing tumors and hypertension. Intra-thoracic neuroblastomas may cause Horner's syndrome (ptosis, miosis, enopthalmos, anhydrosis) on the affected side. Some patients have diarrhea, hypokalemia, and occasionally hypovolemia.

The diagnosis of neuroblastoma is primarily made when high levels of serum and urine catecholamines are present and an abdominal mass is present on physical examination, plain X-rays of the abdomen, CT scan, ultrasound, and MRI. Treatment of neuroblastoma includes surgery and chemotherapy. Surgery is effective if the tumor is a localized and has not metastasized. However, about half of the tumors have metastasized at the time of presentation. Tumors that have metastasized are first treated with chemotherapy to reduce tumor size and then with surgery.

Following a complete history and physical examination, evaluation of laboratory data (especially the Hgb and K^+), standard monitoring of heart rate, arterial pressure, ECG, and SaO_2, general

tracheal anesthesia is induced. If there is evidence of vomiting or upper bowel obstruction, induction of anesthesia should be by rapid sequence. Adequate intravenous access is needed to treat the excessive blood loss seen with tumors that involve major blood vessels or are very large. If significant blood loss is thought to be likely, intravascular arterial pressure monitoring is very useful (**See Chapter 2**). Depending on the site and size of the incision, patients can have significant pain after surgery. Epidural analgesia is often very helpful. Postoperative pain can also be treated with narcotics and NSIDS.

Conclusion

Infants have varied problems from uncomplicated to very complicated that must be understood before the induction of anesthesia to prevent problems during anesthesia. Appropriate correction of intravascular volume and electrolytes before the induction of anesthesia is important. Monitoring aids in detecting changes early and providing therapy to prevent untoward events.

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Chapter 8: ANESTHESIA FOR INFANTS

Chapter 9
ANESTHESIA FOR CHILDREN AND ADOLESCENTS[1]

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Introduction

In this chapter, children are defined as individuals between one year of age and puberty; adolescents are defined as those between puberty and 18 years of age. Many children in these age groups have chronic diseases that were either present at birth or developed as young children and progressed. Multiple developmental changes occur as teenagers go from being children to adults. Chronic disease may affect the rate of their developmental. Consequently, some children with chronic disease are at very different stages of development. In many countries, children must function as adults to survive and are quite mature for their age. The rate at which they develop and mature affects their ability to deal with difficulties related to surgery.

Development and Behavior

Adolescence is the time when children become adults. Along the way there is cognitive development, greater emotional liability, erratic behavior, and increased risk taking. The latter is associated with increased mortality from trauma, automobile accidents, homicide, and suicide. In some areas of the world this also includes death and injury from war. Substance abuse is a form of risk taking and is common in children and adolescents throughout much of the world.

Use of illicit drugs in the United States has increased over the past few years. http://www.-samhsa.gov/data/NSDUH/2012SummNatFindDetTables/Index.aspx Alcohol use (often to excess) is rampant in the world's children (>59% of USA 15-16 year olds) drink alcohol and more than 30% use drugs, including marijuana, cocaine, and methamphetamines. With the daily development of "designer drugs" [drugs that were initially discovered (often in illegal laboratories) that have their structure and activity altered to produce a new drug for specific psychic effects] it is often hard to determine what the interaction of these drugs will be with medications used to produce anesthesia. Illegal use of prescription and nonprescription narcotics is increasing worldwide.

[1] This chapter is a modification of a chapter co-authored one of us (DBA) for Gregory's Pediatric Anesthesia, Wiley-Blackwell, 2012

Children using these drugs have a potential for becoming apneic when anesthesia is induced. Chronic use of narcotics and other central nervous system depressants may increase drug requirements, which may make the treatment of postoperative pain more difficult. When this occurs, it is often necessary to titrate narcotic doses to effect, i.e., provide sufficient pain relief without depressing breathing, rather than giving a set dose of narcotic every four hours. Use of illicit drugs during growth and development can lead to permanent CNS alterations.[2] If cocaine and alcohol are abused before 14-15 years of age, portions of the brain may be underdeveloped. In some patients, chronic use of marijuana as a youth produces psychosis later in life. These CNS changes may affect perioperative pain therapy. Higher or lower doses of narcotics may be required. It is not possible to know until narcotics are administered.

If the anesthetist has concerns that the patient has recently used illicit drugs, it is important to obtain as good a history as possible from the patient and to perform a complete physical examination. Possible drug use should be discussed with the patient without the parents being present. MAC (minimum anesthetic concentration) is reduced by acute use of CNS depressants. On the other hand, chronic use of depressants, such as alcohol, increases MAC.[3]

Teenage Cigarette Smoking

Recent studies showed that about 20% of American teenagers smoked tobacco once in the previous month and 8% smoked it more often.[4] The use rate may be higher or lower in other countries. In many Southeast Asian countries, the rate is about 12%. Boys usually smoke more often than girls, but not always, especially when girls use cigarette smoking to control their weight. The younger the age a person begins smoking, the more likely he/she is to damage her/his health. Malaysian children who were exposed to second hand smoke (usually at home), who used drugs and alcohol, who did not attempt to quit smoking, who were involved in fighting, and who were being bullied had the highest incidence of current tobacco smoking.[5] Other factors in tobacco use included parent's marital status (divorced) and parental (especially maternal) smoking. The study by **Hisam** and colleagues pointed out that about 165,000 children die of respiratory diseases caused by cigarette smoke each year.[6] Most of these are in Africa and South East Asia. Fifty-six percent of Pakistani children are exposed to second hand smoke (SHS) each day, usually from family members.[6] Symptoms due to SHS included headache, rhinorrhea, asthma attacks, coughing, earache, and nausea and vomiting. Chronic airway irritation from SHS increases the incidence of bronchospasm and "asthma". SHS also increases the likelihood of bronchospasm following placement of a tracheal tube, especially when the depth of anesthesia is light. Twice as many respiratory events occurred in 16-year old smokers, including laryngospasm and hypoxemia, than in nonsmokers. Cigarette smoking increases the amount of carbon monoxide (CO) in hemoglobin, which displaces oxygen from hemoglobin and lowers the oxygen content of blood. Even low levels of carbon monoxide reduces oxygen content because CO is 200 times more avidly bound to hemoglobin than oxygen, and this lowers the oxygen content of

hemoglobin. Despite the reduced oxygen content, the reading on the oximeter is high because the oximeter reads CO as oxygen. Administering 100% oxygen displaces CO from hemoglobin and improves the oxygen carrying capacity of Hbg. Therefore oxygen should be used in smokers during anesthesia, if possible.

Pregnancy and Reproductive Issues in Adolescents

Teenage pregnancy has decreased in many Developed countries over the past 20 years. Worldwide, teenage pregnancy rates vary between 143 per 1000 live births in some sub-Saharan African countries to 2.9 per 1000 in South Korea.[7,8] The risks of low birth weight, premature labor, anemia, and preeclampsia are higher in teenage pregnancies, even after controlling for differences in whether the mother received prenatal care or not. The worldwide incidence of premature birth and low birth weight is higher among adolescent mothers. In West Bengal, 15-19 year old mothers had more anemia, preterm delivery, and low birth weight than older mothers.[9] In many countries, teenage pregnancy is accompanied by poor health care and lack of prenatal care.

Teenage females require emergent and non-emergent surgery for the same reasons male teenagers do, appendicitis, trauma, tumors, etc. Because of the widespread suggestion that anesthetics, including halothane, sevoflurane, nitrous oxide, benzodiazepines, affect organ formation during the first trimester of pregnancy, it is important to determine whether the patient is pregnant when she arrives for surgery, especially in countries where the incidence of teenage pregnancy is high. This has led many hospitals to perform a urine pregnancy test in all menstruating females before inducing anesthesia. If the test is positive, this result should be discussed with the patient and then with the parents if the patient gives her consent. Since it is believed that the greatest risk for drug-induced teratogenicity occurs while organogenesis is taking place (15^{th}-56^{th} day of gestation),[10] consideration should be given to delaying non-emergent surgery for pregnant females until six weeks after delivery of her baby, or at least until the second or third trimester of pregnancy. If the surgery is emergent, anesthesia and surgery must proceed, but drugs that have little chance of injuring the fetus should be used when possible. http://ceaccp.oxfordjournals.org/content/6/2/83.full Consideration should be given to using spinal or epidural anesthesia or a peripheral nerve block when possible (**See Chapter 21**). This plan should be followed despite the fact that there is little evidence in humans that inhaled anesthetics (N_2O, halothane, sevoflurane), benzodiazepines, local anesthetics, opioids, muscle relaxants, or the drugs used to induce anesthesia (pentothal, propofol) cause genetic defects when used in normal doses. Congenital anomalies occur in about three percent of human babies born without exposure to anesthesia. Despite the lack of data, anesthesia should be limited in pregnant women during the first trimester of pregnancy until we have more information.

Chronic Disease

Many children and teenagers require surgery for treatment of chronic diseases. Many of them have undergone anesthesia and surgery multiple times before. If they had a "bad experience", it will be a problem for the anesthetist. If they had a "good experience", it will be easy. Either way, it is often better to premedicate them with oral or intravenous drugs before taking them to the operating room. Midazolam 0.5-1.0mg/kg orally (maximum dose 15mg) or 0.02-0.03mg/kg IV (maximum dose 2mg) provides effective premedication for the majority of patients. Most importantly, it provides some memory loss for the perioperative period. However, it takes 15-20 minutes for the desired effects of oral midazolam to take effect. The following pages will discuss anesthesia for several chronic diseases.

Cystic Fibrosis

Cystic fibrosis (CF) is an autosomal recessive disease that occurs in people on all continents but is more common in those of Hispanic (1:12,000) or African (1:15,000) decent. Defective chloride (Cl^-) secretion and excessive sodium (Na^+) resorption reduce the amount of liquid in the airways. This liquid is necessary for transporting mucus from the lower airway to the mouth to be swallowed or expectorated (spit out). Because surface fluid is lacking, mucus cannot be moved and secretions become inspissated (thick, dry, viscus), which blocks the airways. Chronic infection and other entities produce a profound inflammatory response, which progressively damages the lungs. Inflammation is responsible for most CF-induced morbidity and mortality. Fifty percent of asymptomatic children have the bacterium P Aruginosa in their lungs. Aspergilus fuingatus and Streptococcus aureus are also commonly found there. These and others organisms are responsible for the inflammation, bronchiectasis and bronchomalacia that occur. Chest X-rays demonstrate hyperinflation of the lungs, flattened diaphragms, bronchiectasis, and cystic formations (blebs). Evidence of progressive pulmonary fibrosis is also present in most patients. Increased airway reactivity is another feature of CF that may or may not improve with administering bronchodilating drugs. Some patients actually get worse after bronchodilator administration because, when inhaled, these drugs cause further mismatching of ventilation and perfusion. CF patients have very "floppy airways (due to loss of cartilaginous support), and exhaling (especially forced exhalation) can compress the airways and worsen the airway obstruction. When this occurs gas is "trapped" in the lungs, which increases the residual volume and FRC (volume of gas in the lung at end-expiration). This eventually leads to hypoxemia, CO_2 retention, pulmonary hypertension, and cor pulmonale. Depending on the degree of cor pumonale, surgery and anesthesia-related morbidity and mortality may increase. Some patients require oxygen at home, especially when sleeping. If they do, it is very important that they receive oxygen during surgery and in the post-anesthesia recovery room.

Most patients develop hyperplasia of the nasal mucosal, chronic sinusitis, and nasal polyps, all of which obstruct breathing. Irrigation of the nose and sinuses with hypertonic saline helps loosens and removes excess secretions and improves breathing.[11] Nasal intubation or insertion of a nasal airway *should be avoided* because doing so may remove the polyps and inflamed tissue and cause severe bleeding. Bleeding is uncommon with use of oral airways and oral tracheal tubes.

About 90% of patients with CF have decreased pancreatic exocrine function from plugging of their pancreatic ducts, which induces inflammation and auto digestion of the pancreas. Plugging of the pancreatic ducts plus the auto-digestion reduces lipase secretion, which prevents absorption of fat and vitamins A, D, E, and K. Vitamin deficiency can increase the numbers of fractures, bleeding and neurologic abnormalities, and cause a decline in cognitive function. Ingesting enteric-coated pancreatic enzymes on a regular basis improves all of these abnormalities. Progressive loss of pancreatic islet cells leads to diabetes mellitus.

Liver failure occurs in some patients, the presentation of which varies, depending on the degree of liver involvement. Liver regeneration may cause multiple palpable nodules on an enlarged liver. Once patients develop portal hypertension, their spleens enlarge and esophageal varices develop. This often results in hemorrhage and hematemesis (vomiting of blood). In severe cases, patients develop ascites. The ascetic fluid presses against the diaphragm, causes atelectasis, and worsens oxygenation and ventilation (é CO_2). Reduced bile secretion leads to a small gallbladder in about 25% of patients. The diagnosis of liver disease in CF patients is frequently made on physical examination when an enlarged nodular liver, with or without an enlarged spleen, is palpated. There may be, however, hepatomegaly without abnormalities of liver function tests and clotting in some patients. Platelet counts may be below 50,000/mm^3. Ultrasonography and magnetic resonance imaging help determine the presence of hepatomegaly, fatty infiltrations, fibrosis, and portal vein enlargement.

Treatment of the pulmonary effects of cystic fibrosis entails inhalation of hypertonic saline, chest physiotherapy, and postural drainage to help clear pulmonary secretions.[12] While inhalation of N-acetylcysteine (NAD) has been used for years to treat patients who have CF, the drug has recently been shown to be detrimental because it increases the amount of inflammation. Inhaled antibiotics (e.g., tobramycin, aztreoman) reduce the quantity of P. aeruginosa in the lungs. Oral azithromycin may also reduce the pro-inflammatory effects of pulmonary bacteria and to slowly improve some aspects of lung function (forced vital capacity in one second). Chronic use of azithromycin use may also reduce the amount of P. aeruginosa. Pulmozyme® is a deoxyribonuclease that attacks white blood cell that accumulate in the mucus. Pulmozyme's effects on WBC's makes the mucus more liquid and easier to cough up and remove. Its use effectively reduces atelectasis and lung hyperinflation over several days, improving oxygenation.

Sinus Surgery for Cystic Fibrosis Patients

Sinus infections are common in CF patients and are the source of many of the pulmonary infections, especially with P. aeruginosa. Thus, improving sinus wellbeing improves pulmonary health. Thick upper airway mucus interferes with cilliary function and obstructs sinus drainage, leading to infection, pain, and sinusitis.

Diagnosis of rhinosinusitis is primarily clinical and is diagnosed when there is inflammation of the nose and paranasal sinuses plus two of the following:[13]

- Blockage and/or congestion
- Nasal discharge
- Facial pain or pressure
- Loss of smell
- Polyps
- Mucopurulent discharge from the middle meatus
- Obstruction of the middle meatus

The incidence of nasal polyps increases with age. Chronic infection of the nasal mucosa (usually with Staphylococcus aureus and Pseudomonus aeruginosa) is thought to cause polyposis. Impaired mucosal ion transport leads to thickened secretions that block glandular ducts, which causes them to dilate. This makes it easier for organisms to grow in the mucus and for inflammation to develop.

The outcome following treatment of nasal polyposis (NP) varies. However, it is clear that systemic steroids have no effect on NP and may cause diabetes mellitus. One relatively effective treatment is nasal lavage with normal saline. It is simple to do, reduces the amount of thick secretions, and makes breathing easier. Topical steroids reduce inflammation. Betamethasone reduces the size of polyps.[14] While it is clear that inhalation of antibiotics (tobramycin, colistin, aztreonam) reduces pulmonary symptoms, there is little or no beneficial effect from inhaled antibiotics on nasal polyps. High-dose ibuprofen reduces neutrophil activation in the lungs of CF patients and slows progression of lung disease.

Surgical treatment of nasal polyps is used when more conservative medical measures fail. Surgery does not cure the polyposis, but it relives some of the symptoms, making life easier for the children.

Anesthesia management of patients who have CF requires a definite understanding of the patient's lung disease and its severity, which can range from very mild to life threatening. A major problem is assuring an airway during the induction of anesthesia, because mucus and polyps often block the nose. Therefore, closing the patient's mouth during induction of

anesthesia completely obstructs the airway. If this occurs, oxygen desaturation may occur quickly because of the patient's abnormal lung function. Keeping the mouth open during the induction of anesthesia is the solution to this problem. An oral airway is often helpful, but it is best not to insert anything (nasal airway, suction catheter, tracheal tube) into the nose during the induction of anesthesia because doing so may cause hemorrhage and worse airway problems.

Portal Systemic Shunts

Patients with CF who have liver disease, portal hypertension, hepatospleenal hypertropyy, and esophageal varacies and bleeding may require surgical creation of a portosystemic vein shunt to relieve the portal hypertension. These patients are very ill and often have clotting abnormalities, making surgery and anesthesia more difficult. Without very specialized care, such patients seldom survive.

Anesthetic Care of Patients With Cystic Fibrosis

Preoperative evaluation of patients with cystic fibrosis is very important because during this time the anesthetist can determine which of the multisystem diseases the patient has and how these diseases may be affected by surgery and anesthesia. Most importantly, the patient's pulmonary disease must be understood. What is the patient able to do? How far can he/she walk without becoming short of breath or hypoxemic? Are the patient's toenails or fingernails clubbed? If they are, this suggests the patient has both liver disease and hypoxemia because the liver metabolizes the normal amounts of vascular endothelial growth factor (VegF) made each day. Failure to metabolize VegF leads to abnormal vessel growth (clubbing, pulmonary shunts). Patients with clubbing usually have serious hypoxemia. What is the respiratory pattern? Is the patient tachypneic? How much work is he/she doing to breath? What is her/his SaO_2 or PaO_2? Does the patient require increased ambient oxygen continuously to maintain a SaO_2 above 94%? Is the patient using her/his accessory muscles to breath (intracostal muscles)? Are there rhonchi or rales? What kind and color of secretions does he/she produce? Are they green (P. aruginosa) or yellow (Staphylococcus aureus). White or clear secretions are usually not infected. Are the breath sounds decreased? Are they the same throughout the chest or are they decreased in some lung areas? Does the patient have petechiae or bruising? If so, is there other evidence of bleeding (rectal, oral, pulmonary)? Is there evidence of cardiac disease? Is the liver enlarged? Is the spleen enlarged? Does he/she have a history or evidence of diabetes mellitus? Does he/she require insulin or other anti-glucose treatment? Is the patient well nourished or not? Poorly nourished patients may not heal well after surgery and may have an increased number of infections?

Physical Examination

Once a complete history and physical examination is done and the anesthetist understands the patient's problems, the anesthetist can plan the anesthetic. Whatever the plan, the goal is to have the patient breathe spontaneously at the end of surgery and to be breathing sufficiently adequately to allow removal of the tracheal tube and maintain adequate oxygenation and ventilation (CO_2 removal). Once a tracheal tube is in place, it is sometimes difficult to remove it at the end of surgery. However, if a TT is needed, a TT should be inserted and plans made to possibly mechanically ventilate the patient's lungs postoperatively. When a tracheal tube is required, it should be as large as possible without injuring the trachea and glottis. Larger tubes make it easier to remove the copious amount of secretions from the lungs during surgery. The inspired gases (anesthetics, oxygen) should be warmed to near body temperature and fully humidified to aid in removing pulmonary secretions.

Anesthesia can be induced by any of the standard methods (**See Chapter 7**). However, an inhaled induction may be better in <10yr old children because it allows the anesthetist to have better control of the rate of anesthetic induction and because drugs used for inhaled induction of anesthesia also tend to dilate the airways.The latter may improve or worsen ventilation/perfusion ratios by blocking hypoxic pulmonary vasoconstriction and increasing blood flow to poorly ventilated (shunt) areas of lung. Consequently, the anesthetist must be prepared to rapidly intubate the patient's trachea, administer oxygen, and control ventilation if this occurs. Mismatching of ventilation/perfusion can also make inhaled inductions of anesthesia longer than they are in patients with normal lungs. Muscle relaxants can be used to intubate the trachea and for some types of surgery (abdominal, intracranial, pulmonary) but should be avoided when possible because many patients with CF use their intercostal muscles to breath and rely on muscle tone to maintain patent airways. Residual muscle relaxant at the end of surgery may make it more difficult for them to breath.

Before tracheal extubation, nebulization of normal saline into the lung may loosen secretions, making them easier to remove by suctioning. However, it must be remembered that suction devices can produce significant negative pressures (100-200cmH_2O) and remove part of the lung volume along with the secretions. Consequently, the lungs should be re-expanded with a twice-normal sized breath and airway pressures should be held for 3-5 seconds for several breaths to open up atelectatic areas of lung. When this is done, more secretions may be released from atelectatic lung and require additional suctioning and lung re-expansion. Patients with CF require close observation after tracheal extubation (preferably in an ICU) until it is certain that he/she can maintain adequate ventilation and oxygenation. Non-invasive support of ventilation [continuous positive airway pressure (CPAP) by mask or BiPhasic Positive Airway Pressure (BiPAP)] may help these patients make the transition form tracheal intubation to extubation. Postural drainage[12] and inhalation of saline may help remove pulmonary secretions.

One factor that aids the transition from tracheal intubation to extubation is providing adequate pain relief after surgery. While there are many ways to do this, continuous epidural or caudal analgesia relieve the patient's pain and makes it easier for her/him to breath. It also improves oxygenation and ventilation. Ibuprofen administration not only provides pain relief, it also decreases inflammation and reduces the amount of secretions.

Congenital Heart Disease (See Chapter 12)

Congenital heart disease is common throughout the world. Many of these patients survive into their teenage years and adulthood and require surgery and anesthesia for the same non-cardiac procedures as their "normal" peers. Before providing anesthesia, it is important to obtain the latest information about the patient's cardiac status. This includes a list of current medications and their purpose. Whether these patients require antibiotics for surgery depends on the lesion and the type of surgical repair he/she underwent (**See Chapter 12**). It is important to determine if the cardiac surgery they underwent was corrective or palliative. If it was the palliative, the circulatory system is still abnormal, and the patient may well be hypoxemic. Hemoglobin concentrations, sometimes to above 20g/dl, increase to compensate for the hypoxemia, which increases blood viscosity and the possibility for clotting and occluding the blood supply of some organs (brain, kidney). Clotting is more likely to occur in hypotensive polycythemic patients. Consequently, it is important to maintain normal arterial blood pressures and blood volumes. Infusing 5-20ml/kg of normal saline just before induction of anesthesia helps reduce blood viscosity. Many anesthetic drugs dilate the circulation, which improves blood flow in normotensive patients.

Before inducing anesthesia, the anesthetist and surgeon should determine what postoperative care will be required and determine how and where it will be provided. Adequate pain relief is very important because pain limits breathing and this causes acidosis and alteration in blood flow through shunts, if they exist. **See Chapter 12** for more information.

Cancer

Cancers are relatively common in older children and teenagers and include leukemia's, lymphomas, central nervous system (brain, spinal cord), bone tumors, and soft tissue sarcomas. Many cancer patients undergo repeated surgeries. Many require insertion of central lines for chemotherapy and bone marrow aspirations to follow the progress of their cancer treatment. When available, many of these children require radiation therapy, which necessitates anesthesia or sedation to prevent the patient from moving during the treatment. MRI's and CAT scans are needed to follow the progress of their cancer. Younger children must be sedated for these procedures; teenagers usually can usually have their scans without sedation.

Chemotherapeutic agents successfully treat many cancers. However, these drugs often produce serious consequences of their own. They suppress the immune system and reduce the number of white and red blood cells and platelets, causing anemia and bleeding. Cardiotoxicity is common with doxorubicin and epirubicin. Many cytotoxic drugs cause liver damage ranging from hyperbilirubinemia to liver fibrosis. With more serious involvement, clotting is depressed and hemorrhage is common. Tumor lysis and the drugs themselves may initiate renal failure. Some drugs (bleomycin, methotrexate, carmustine) and radiation therapy cause pulmonary fibrosis in some patients. Thus, the anesthetist must be aware of any chemotherapy-induced heart or lung injury present. Some patients undergo cranial radiation for treatment of leukemia and CNS tumors. This has occasionally been associated with developmental delay.

Sickle Cell Disease

Sickle cell disease (SCD) is caused by substitution of a single amino acid (valine for glutamate) on the β-chain of hemoglobin. It most commonly affects people from Africa. Central and South America, the Caribbean islands, and countries around the Mediterranean area. About 50% of infants born with AS or SS are born in Nigeria, India, and the Democratic Republic of Congo. Sickle cell disease (SS) causes changes in red cell configuration (sickling) when exposed to hypoxia, cold, and or acidosis. The changes in shape make it harder for red cells to pass through capillaries, which cause vascular obstruction. The life span of the red cells is shorter (10-20 days) in patients who have SS disease and this leads to anemia (Hbg 7-9g/dl) in many patients. Greater than 80% of their hemoglobin is abnormal. Heterozygous patients, i.e., those with a single copy of the S gene, are usually asymptomatic and have no sickling during anesthesia and surgery.

Sickle cell disease is a multi-organ disease that varies in severity from patient to patient and usually worsens with increasing age. Clinical manifestations of Hbg SS (homozygous form) include chronic pain, acute pain crises, priapism, stroke, gallstones, and avascular necrosis of the joints, especially the hip joints. Pain, either chronic or acute, is present in most patients. This pain is usually the result of small vessel occlusion by sickled red cells and by impairment of tissue oxygenation and nutrition. These vaso-occlusive episodes consist of severe, acute pin, inflammation, and ischemia.[15] They occur anywhere in the body. The problem most feared by anesthetists is acute chest syndrome (ACS) because this is a major cause of death in SCD patients, especially after they are two year of age.[16] ACS is due to pulmonary capillary vaso-occlusion, which leads to fever, hypoxia, and new infiltrates on a chest X-ray. Exchange transfusions are sometimes used to reduce sickling. Some patients with ACS go on to develop pulmonary hypertension, which, if severe enough, may result in death during anesthesia and surgery. Asthma is also a common finding in patients with SCD. It too is a predictor of mortality and should be appropriately treated to prevent an asthma attack (bronchoconstriction) during surgery.

It is now understood that the problems with SCD are not only related to sickling. They are also related to increased RBC "stickiness" and adhesion of RBCS and WBCs to injured blood vessel walls, which occludes vessels and interferes with tissue oxygenation. A large portion of the disease is the result of inflammation. As Firth and Head stated,[17] "the symptoms stem predominately from chronic endothelial damage rather than from acute erythrocyte deformation". They suggest, "acute changes in endothelial regulation of flow and hemostasis are key steps in the initiation and progression of vaso-occlusion". These changes are activated by inflammatory cytokines and other factors. One scenario for vaso-occlusive (VOC) disease is vasoconstriction, leukocyte adhesion, platelet activation, adhesion of cells, and increased coagulation. Whether hypoxia induces the symptoms associated with SCD is unclear, as there are data that patients with SCD tolerate moderate to sever hypoxia without the onset of VOD.

Clinical characteristics of SCD include evolving organ damage, periodic bouts of severe pain, and worsening pulmonary disease. Central nervous system bleeding, pulmonary disease, and infections are major causes of death associated with this disease. Acute chest syndrome, is defined as a new infiltrate on X-ray, hypoxia, chest pain, pyrexia, tachypnea, cough, and wheezing. Other problems include pulmonary emboli from bone marrow. There is also worsening lung disease with aging. This includes airway obstruction and hyper-reactivity in younger patients; in adults it includes pulmonary fibrosis. Pulmonary function decreases and pulmonary hypertension develops. This eventually leads to right ventricular failure and death in the worst cases.

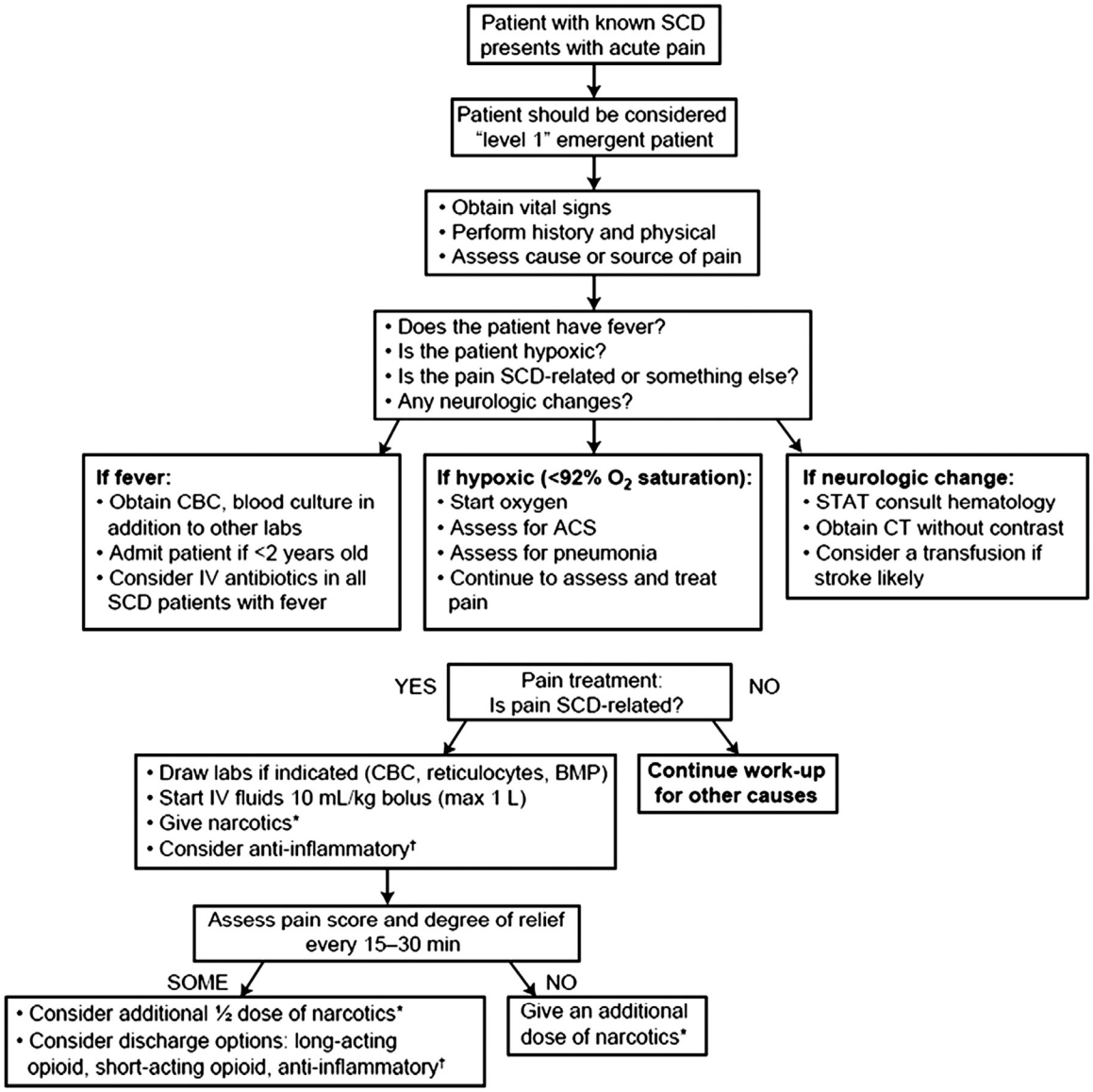
Stroke occurs in many patients with SCD (10% of <10-year old children) due to arterial disease, not venous occlusion. With repeated strokes, patients loose intellectual function. The silent infarct form of the disease produces lesions consistent with infarcts on magnetic resonance imaging that may not be associated with clinical symptoms. They are however, linked to intellectual impairment, poor educational success, and risk of later infarcts.

Renal disease includes glomerular disease and papillary necrosis. It presents with varying amounts of proteinurea and hematuria.[18] The ability to concentrate urine is often lost (hyposthenurea). Because of these abnormalities, one might expect patients with SCD and renal lesions to have worse outcomes after surgery.

Other complications of SCD include fever, infections, bleeding, thrombosis, embolism, and death from causes besides SCD.

Treatment of SCD is often symptomatic. It is very important to determine the cause of and treatment required for pain, as this is the problem that brings most SCD patients to the attention of health care providers. While pain produced by vaso-occlusive disease is common, the anesthetist must always search for other causes of pain and treat them. **Figure 9-1** is one schema for evaluating the pain of patients who have SCD.[19]

Figure 9-1: Scheme for Evaluation of Patients With Pain and SCD



This figure provides a scheme for evaluating and treating pain in patients with pain due to SCD and other causes of pain.. Morphine 0.1 mg/kg or hydromorphone 0.015–0.02 mg/kg.† Ketorolac (Toradol®) 0.5 mg/kg (max of 30 mg) or ibuprofen(10 mg/kg). ACS, acute chest syndrome; BMP, basic metabolic panel; CBC, complete blood count; CT, computed tomography; i.v., intravenous; SCD, sickle cell disease; STAT, immediate. With permission* mailto:KanterJ@musc.edu

Infection is a major cause of death in resource poor countries. Infections may occur in association with strokes, acute chest syndrome, pulmonary embolism, splenic and hepatic sequestration, gallstones, and many other conditions or they may occur without the presence of any of these other problems. However, all of these conditions should be looked for in febrile patients. While *blood transfusion* may improve the condition of patients with pain crisis, transfusion should not be done repeatedly because doing so imposes a significant risk of iron overload and all of the

problems associated with it. Penicillin prophylaxis and anti-pneumococcal vaccination decrease life-threatening infections in children with SCD.[20,21]

Hydroxyurea is the only know pharmacologic agent that treats and prevents recurrent vaso-occlusive disease[19]**.** It is said to work by increasing the number of hemoglobin-F containing red blood cells and decreasing the number with hemoglobin-S. It may also reduce leukocyte and reticulocyte production and vaso-occlusion. Chronic use of hydroxyurea has decreased vaso-occlusive events, hospitalizations, organ damage, chronic hypoxemia, and stroke without causing significant side effects.

Blood transfusion is proven therapy for reducing stroke and bone marrow suppression in patients with HbgS disease.[22] The goal of transfusion is to reduce the amount of HbgS containing RBC's to <30% of the total RBC volume, which reduces the incidence of stroke and bone marrow suppression. However, transfusion therapy should only be used sparingly because it causes iron overload, which leads to cirrhosis of the liver, diabetes mellitus, cardiomyopathy, arthritis, and joint and bone pain. Transfusion therapy is often limited to patients with life threatening conditions, including acute chest syndrome, aplastic sequestration, and cerebral infarction. Packed red blood cells or exchange transfusion is used for the purpose of raising the concentration of non-sickle hemoglobin, but exchange transfusion more effectively reduces HbgS concentrations over a short period of time. However, it requires technology and people. Blood for transfusion should be cross-matched for ABO and Rh compatibility, should not contain HbgS containing cells, and should be leukocyte poor, if possible. It should also be cross matched for C, D, e, e and Kell antigens to reduce the risk of alloantibody sensitization, especially since these patients may receive multiple blood transfusions over their lifetimes. The only true "cure" for this disease is stem cell transplantation, which is not available everywhere and is very technology driven. Finding appropriate donors is often difficult.

Anesthetic management of patients with SCD has changed over the past ten years. It was believed that most of the complications of SCD were due to deformation of the red blood cells, occlusion of capillaries, and ischemia. Since then it has become clear that it is more complicated than that (**See above**).

Preoperative evaluation is very important because patients with SCD often have multisystem disease. As in other patients, the goal is to define existing and potential problems associated with anesthesia and surgery and prevent them from occurring. The anesthetist's goal is still to prevent sickling, acute chest syndrome, pain crisis, stroke, and real failure, among other things. It is important to know if the patient's disease has worsened recently and what therapy he/she has received to treat SCD. There is controversy whether transfusion is necessary preoperatively if patients have anemia and whether exchange transfusion or simple transfusion of packed red blood cells to an Hbg concentration of 10g/dl is sufficient. Since there appears to be no difference in outcomes with the two methods, simple transfusion is easier and associated with

fewer problems (e.g., hypocalcemia), simple transfusion to a Hbg concentration of 10g/dl is often preferred.[23] Strict attention must be paid to hydration, oxygenation, temperature (warm IV fluids when possible), prevention of acidosis and hypovolemia, although there is question about whether these variables cause the complications of SCD.

Several factors are related to the incidence of postoperative complications in patients with SCD. Recent hospitalizations for treatment of SCD, an increased number of hospitalizations in the past year, increased age, pregnancy, infections (urinary, pulmonary), and organ failure are among the reasons for postoperative complications.[24,25]

How to prevent perioperative complications is controversial. For low risk procedures, it may not be necessary to transfuse these patients. For larger procedures preoperative transfusion to a Hbg concentration of 10g/dl may be appropriate. Since many of these patients will require multiple surgeries, it is probably better to transfuse them only if needed, not just to decrease the amount of HbgS. Each hospital will have its own requirements. Oxygen therapy has been a mainstay of therapy for patients with SCD. However, data that support using oxygen for this purpose are missing. Increasing the oxygen carrying capacity by blood by transfusion may be more important in severely anemic patients (**See Chapter 1**). Breathing 100% oxygen adds little oxygen to the blood but transfusion to a Hbg of 10g does (1.3cc/g of Hbg). No data exist in surgical patients that show hypoxemia causes sickling of HbgS containing cells. Sickle cell hemoglobin shifts the oxygen dissociation curve to the right, making more oxygen available to the tissues at any given PaO_2. Pulse oximetery underestimates the true SaO_2 of patients with SCD by about 2% due to the large amounts of methemaglobin present in the patient's blood.[26] Rather than treat the patients by some formula, it seems appropriate to use the same criteria for transfusion used for other anemic patients (**See Chapter 4**).

Another hallmark of therapy for SCD during surgery has been to prevent *dehydration* from occurring. However, diuretics and hypertonic contrast media have been used without increased sickling and generous hydration has not prevented sickling.[18] Patients should probably receive an appropriate amount of fluid for the surgery being done, not an amount based on having SCD (**See Chapter 3**).

Cold is also thought to incite sickling, but again, evidence to support this thought is lacking. Patients have been cooled during cardiac surgery without adverse effects on their red blood cells. It is probably safe to maintain the temperatures of patients with SCD as we would for other patients. Keep them in a normal range (neither hot nor cold) when possible.

Acidosis also is said to precipitate complications in patients with SCD without supportive data. When sickling occurs, it is unclear whether it occurs because of the acidosis or the underlying condition that caused the acidosis. Certainly alkalization (sodium bicarbonate) does not prevent pain crisis following surgery in patients who have SCD.[27] Once again, the goal when treating

patients who have SCD should be to maintain their pH in the range acceptable for any other patient.

Intraoperative anesthetic management is usually similar to that for other patients. Both general and conduction anesthesia (epidural, spinal, block) have been used without untoward problems. Most of this information comes from women in labor, not from children. In one study, the use of epidurals was not associated with postoperative complications following cholecystectomy. Oxygenation improved significantly following epidural analgesia for pain.[28] In general, only case reports are available in children. Care should be taken to prevent atelectasis during surgery, as this will worsen any lung disease. Body temperature, acid-base status, oxygenation, and fluid administration should be maintained, as they would be for other patients.

Postoperative care should be similar to that provided for other patients undergoing similar surgery: adequate analgesia, early ambulation, and maintenance of a normal SaO_2. If acidosis occurs, its cause should be sought and treated, as it would be in any other patient. Since the pain during a pain crisis is often severe, it must be treated effectively and quickly.[15] Patient controlled analgesia (PCA) should be used when available. Fentanyl patches are also effective, especially when PCA is not available. A patch and PCA should *not* be used at the same time, as their effects may be synergistic and depress breathing. Acetaminophen and nonsteroidal anti-inflammatory drugs are effective adjuncts to narcotic administration, provide additional pain relief, and decrease the total amount of narcotic given.

Because acute chest syndrome occurs about three days after surgery, patients with SCD must have access to appropriate medical care, even if they have been discharged from hospital following surgery. Those who live far from a hospital should remain in the vicinity of the hospital for about a week following surgery, if possible. Early ambulation, incentive spirometery, bronchodilators, analgesia, and broad-spectrum antibiotics may reduce ACS in patients with SCD.

Diabetes Mellitus

Most children have type I diabetes (insulin requiring), but the increase in worldwide obesity, lack of exercise, and sedentary lifestyles (television, computers) have increased the incidence of type II (non-insulin dependent) diabetes in children. Maternal obesity during pregnancy is also a factor in this increase. Type I diabetes is caused by autoimmune destruction of pancreatic beta cells, which requires insulin for its treatment. Type II diabetes is caused by resistance to the effects of insulin on cells and mostly occurred in adults over 40 years of age in the past. During the past 20 years this has changed. Now many children also have type II diabetes. These children are often obese and have fat deposits in their abdomens. The also have non-alcoholic liver disease. Treatment of type II diabetes is by diet, exercise, and drugs (metformin, sulfonylureas, etc), but exercise and diet alone usually fail to solve the problem. About 10% of children with type II diabetes require insulin. Increasingly more children with diabetes are undergoing surgery to

reduce the size of the stomach (See below) to control their weight. Fortunately, few children have the renal and ophthalmologic complications that occur with diabetes in older people.

Preoperative evaluation of diabetic patients must include several questions. Does the patient have any of the complications of diabetes? How is her/his diabetes being treated? How much insulin is the patient taking each day? Has the patient been hospitalized recently for treatment of her/his diabetes? Is the glucose hemoglobin A1C elevated (>7.5%), indicating poor control of blood glucose concentrations?

When children with diabetes mellitus require surgery, they should be the first case of the day to avoid prolonged fasting. Diabetic patients only need to fast the same amount of time as non-diabetic patients. If their surgery must be done in the afternoon, they can have a light breakfast and take clear glucose containing fluids (apple juice without pulp) up to two hours before anesthesia. If patients are taking insulin two or more times per day, they should take 50% of their intermediate insulin dose in the morning and have an IV started. An infusion of 5% Dextrose in ½ NS or 5% Dextrose and NS is run at maintenance rates. Insulin is added (1 unit of regular insulin per ml of fluid) and infused at 0.025-0.1 units of insulin/kg/hr. Blood glucose concentrations are monitored at least every hour while insulin is being infused and maintained between 90 and 180mg/dl. It may be necessary to administer potassium (20ml/l of fluid) along with the insulin infusion to maintain a relatively normal serum potassium concentration.

Anesthesia requirements of patents with diabetes mellitus are usually the same as for other patients undergoing similar surgery. However, the responses may be altered with advanced neuropathy. Minimum anesthetic concentration (MAC) is reduced 10-23% in the in diabetic rats.[29] These data have not been confirmed in humans.

Inflammatory Bowel Disease

About 25% of patients with inflammatory bowel disease (Crohn's Disiease, ulcerative colitis) are children and teenagers, who often require hospitalization and (in some cases) surgery. Crohn's disease is a transmural inflammatory process found in many parts of the entire bowel but usually in the small bowel. Ulcerative colitis on the other hand, is a disease of the colon, often the lower colon, and is not transmural. Diarrhea, fever, ileus, blood per rectum, and abdominal pain occur in varying degrees with both forms of inflammatory bowel disease (IBD). Those with Crohn's disease tend to have more abdominal pain (**Table 1**). These autoimmune diseases occur more commonly in people of Northern European ancestry, but they also occur in people from all other sections of the world. Joint and skin involvement occurs commonly. Treatment for both forms of IBD is similar: corticosteroids, 6-mercapto purine or azathioprine, maesalamine, and methyltrexate. Many patients are often treated with chronic antibiotics (metronidazole or ciprofloxacin).

Table 1: Presenting Symptoms with Inflammatory Bowel Disease

Crohn Disease	Ulcerative Colitis
Abdominal Pain	Rectal bleeding
Weight loss	Diarrhea
Growth failure	Urgency/tenesmus
Anemia	Abdominal pain
Diarrhea	Anemia
Perianal disease	Weight loss
Fever	Fever
Arthritis	Arthritis
Skin lesions	Skin lesions

Reconfigured From: Rabizadeh S, Dubinsky M, Rheum Dis Clin N Am 2013;39:789-799. ***With permission***

Surgery is commonly needed for failed medical management or for perforated bowel, abscesses, bowel obstruction, toxic megacolon, and perirectal abscesses or fistulae. Patients with IBD are often well known to anesthetists because these patients have required repeated surgeries. As their disease worsens, an ileostomy or colostomy may be required. Total colectomy and ileostomy may also be needed for progressive ulcerative colitis. Many children require repeated upper and lower endoscopy to evaluate the effectiveness of their therapy and follow the progression of their disease. Anesthetists must anesthetize or sedate these patients often and must take into account the fact that these patients often vomit, have full stomachs, and are on steroids, etc. Postoperative wound infections and other complications are widespread.

Preoperative evaluation of patients who have IBD should determine the state of the patient's disease. Is it progressing or stable? What is the patient's nutritional status? Is he/she loosing weight, or is her/his weight stable or increasing? Those with poor nutrition are more often prone to postoperative complications, such as wound dehiscence and wound infections. What medication does the patient take? Is he/she taking steroids every day? What are the doses of steroids taken? Does he/she require steroid coverage for the anesthetic? Are there complications of chronic steroid intake (e.g., hip necrosis).

If the anesthetist takes into account the above considerations, either general or conduction (spinal, epidural) anesthesia can be used, although the anesthetist must consider whether it is appropriate to do a nerve block or conduction anesthesia in a patient who is potentially infected. Because many of these patients have chronic diarrhea, her/his serum potassium concentration must be determined; it may be quite abnormal (low). If so, consideration should be given to replacing potassium preoperatively, especially if the surgery is not emergent.

Obesity

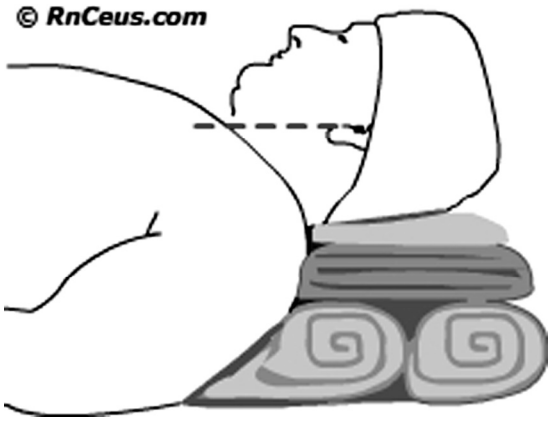
Childhood obesity is a worldwide problem that is increasing every year. As countries become wealthier and food becomes more available, people eat more. The food eaten is often high in fat and calories. Many people have the belief that a "fat child is a healthy child". This is not the case. Obese babies go on to be obese teenagers and adults and develop diabetes mellitus and hypertension. Eventually they develop renal and cardiac disease.

Overweight is defined as a body mass index [body weight in kg/height in meters squared (kg/m^2)] between the 85^{th} and 94th percentile for age. Obesity is defined as being at the 95^{th} percentile for age or above.[30] In the United States and Europe, at least half or all children are overweight or obese by these criteria. This epidemic is due to excessive intake of calories without increasing the number of calories burned by work or exercise. Obesity leads to insulin resistance and to the metabolic syndrome, which consists of abdominal fat deposition, hyperinsulinemia, arterial hypertension, and hypertriglyceridemia. Obese children also have pulmonary (asthma, obstructive sleep apnea, atelectasis, hypoxemia), cardiovascular (systemic hypertension, left ventricular hypertrophy, pulmonary hypertension, right ventricular failure), endocrine (insulin resistance and type II diabetes), and gastrointestinal (delayed gastric emptying, esophageal reflux and aspiration, non-alcoholic fatty liver disease). While diet, exercise and drugs are often used in an attempt to help obese children loose weight, this seldom works long-term.

During preoperative evaluation, the anesthetist must search for associated diseases; including tibia vera (Blount's disease) slipped capital-femoral epiphysis, cholecystitis, tonsillar hypertrophy, and sleep apnea. It is especially important to determine if the patient has obstructive sleep apnea, because, in severe cases, OSA may be associated with pulmonary hypertension and right ventricular failure. Patients who have pulmonary hypertension have greater mortality and morbidity, especially if they also have right heart failure. Some patients with OSA use CPAP or BiPAP during sleep to maintain a relatively normal FRC and oxygenation. Pulmonary hypertension and the need for nighttime CPAP are concerning and should be investigated thoroughly before inducing anesthesia. Patients requiring either CPAP or BiPAP should have whichever form of respiratory support they use reinstituted postoperatively while recovering from anesthesia. It is safer to observe patients with OSA in hospital for at least one night, in and ICU when possible.

For induction of anesthesia, the patient's upper body and head should be placed on a "ramp" (**Figure 9-2**) to improve breathing (bowel falls away from the diaphragm).

Figure 9-2: Obese Patient Positioned for Induction of Anesthesia



Ramp position aligns the patient's auditory canal with the sternal notch. With permission from: www.rnceus.com

Anesthesia can be induced with either inhaled or IV medications, depending on the patient's condition. An inhaled induction of anesthesia takes longer in obese patients, especially when more fat-soluble drugs (halothane) are used, but the advantage of an inhaled induction is the ability to more easily awaken the patient if problems arise. Either form of anesthesia induction relaxes pharyngeal muscles and allows the tongue to "fall back" and obstruct the airway. As soon as the depth of anesthesia permits, inserting an oral airway will improve breathing. Maintaining a CPAP of 5-10cmH_2O by mask will also help maintain an open airway. When the level of anesthesia is deep enough, a tracheal tube can be inserted. If succinylcholine is used (and it should be used with caution), the dose should be the dose needed for a normal weight patient of similar age. The dose should not be based on the patient's total body weight. Basing the dose on total body weight often provides an excessive dose of succinylcholine. Tracheal intubation is usually not difficult. However, obtaining vascular access may be very difficult because the patient's veins are buried in fat. Fortunately, veins on the volar surface of the wrist are usually visible and large enough to insert an appropriate sized IV. Fat does not deposit there. It is sometimes easier to find veins of appropriate size after anesthesia is induced, as most anesthetics tend to dilate veins. At times it may be necessary to insert a small IV for the induction of anesthesia and a larger one following induction. Obese patients should be considered to have a "full stomach", even if they have not eaten for more that six hours because obesity delays gastric emptying. Cricoid pressure should be held during induction of anesthesia and until the tracheal tube is confirmed to be correctly placed in the trachea.

How to dose drugs in obese patients is often unclear, due to lack of data. This lack makes it hard to know whether to base drug requirements on the patient's actual weight and her/his ideal body weight. It is usually best to give drugs based on ideal body weight and determine the effect of the

drug. If more drug is required, give it. However, drug dosages based on the ideal weight may be inadequate.

Bariatric surgery is done to reduce both the size of the stomach and food intake. This usually results in a significant loss of many kilograms of body weight, but over time some of the lost weight is regained. However, the weight loss often reverses the comorbidities (hypertension, type II diabetes, OSA, non-alcoholic fatty liver disease) and improves the patient's wellbeing. Surgery reduces the gastric volume by one of two methods. First, a Roux-en-Y can be created as an open procedure or endoscopically. This creates a 10-30ml gastric pouch that is connected to one limb of the Roux. The second method places a balloon around the top of the stomach to create a small pouch just distal to (below) the gastroesophageal junction. The advantage of this method is that the balloon volume can be adjusted later to alter the amount of food accepted in the pouch. Endoscopic procedures are associated with fewer complications, faster recovery from surgery and anesthesia, and shorter hospital stays.

Conclusion

Chronic diseases are common in children and teenagers, and most of these diseases have a worldwide incidence that may vary, but nonetheless the anesthetist may encounter. Neurodevelopmental and behavioral changes, cystic fibrosis, congenital heart disease, cancer, sickle cell disease, diabetes mellitus, inflammatory bowel disease, and obesity all produce important changes that may affect the conduct of an anesthetic. In adolescents, reproductive issues and substance abuse are also common problems encountered by the anesthetist. A clear understanding of these problems greatly assists the anesthetist in planning a safe anesthetic and improves outcomes of anesthesia and surgery.

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Chapter 10
ANESTHESIA FOR NEUROSURGICAL PROCEDURES IN CHILDREN

Marisol Zuluaga Giraldo, MD, Bruno Bissonnette, MD

Introduction

Recent advances in neurosurgery, neuromonitoring, and neurointensive care have dramatically improved outcomes of patients with surgical lesions of the central nervous system (CNS). To reduce morbidity and mortality, anesthetists caring for children requiring neurosurgical procedures should have a thorough knowledge of neuroanatomy, neurophysiology, normal cognitive development, and the effects of anesthetics on the developing central nervous system, the fundamental differences between children and adults, and the implications of the surgical approaches. The objective of this chapter is to review fundamental principles related to the perioperative anesthesia management of pediatric patients undergoing surgery to treat neurological pathology.[1,2]

Anesthetists in developing countries may not see some of the patients and procedures described in this chapter or be able to provide some of the care discussed, but by understanding the principles discussed in this chapter, they will be better able to provide better care for all of their patients, neurosurgical and others.

Neurophysiology

At birth, the central nervous system (CNS) is incompletely developed. There are significant differences in the physiology of the cerebrovascular system and in cranial bones development during different stages of development. The CNS undergoes many structural and physiological changes over the first two years of life, and these changes have major implications for the anesthetist's preoperative evaluation and perioperative management of neurosurgical patients.[3]

The skull is an enclosed space that is occupied by brain tissue (80%), cerebrospinal fluid (10%), and blood (10%) in normal children. These percentages may change significantly with the presence of space-occupying lesions, such as edema, tumors, hematomas, or abscesses. According to the Monro-Kellie doctrine an increase in the volume of one intracranial compartment must be accompanied by a decrease in the volume of one or both of the other compartments to maintain normal intracranial pressure. The skulls of infants and small children can expand to accommodate changes in intracranial

volume because their cranial sutures are open. However, this accommodation occurs slowly and does not allow immediate compensation for acute changes in volume.

Intracranial compliance is defined as a change in intracranial pressure (ICP) with a change in intracranial volume. When the intracranial volume is normal, the ICP is low and the brain can easily accommodate changes in intracranial volume. However, rapid increases in intracranial volume overwhelm the brain's ability to compensate, which leads to intracranial hypertension and deterioration of the patient's condition.[4]

The fontanels and open cranial plates or open sutures of neonates and infants provide space for the cranium to expand. The mass effect of slow-growing intracranial lesions, e.g., tumors and diseases related to increased production or decreased absorption of cerebrospinal fluid (CSF), is often masked by this head enlargement. Slow increases in ICP and intracranial volume are compensated for by expansion of the cranial plates. When an infant presents with signs and symptoms of intracranial hypertension, he/she has significantly advanced disease. The true consequence of not being able to further expand the skull (volume) is a potentially lethal rise in ICP. The volume change necessary to accommodate an acute change in ICP is very limited even in presence of opened fontanels and mobile cranial plates. Furthermore, the non-elastic nature of the duramater prevents further a rapid expansion of the volume to limit the change in pressure. Once the sutures and fontanels are closed, the intracranial compliance of children is less than that of adults. The posterior fontanel usually closes at two months of age; the anterior fontanel closes a few months later.[5]

The ICP of normal neonates and infants is 2-to-6mmHg. In older children, it is <15 mmHg, which is lower than that of adults. In children the choroid plexus produces 0.35 ml/min, or about 500 ml/day, of CSF, which is resorbed through arachnoid villi and ependymal lines in the ventricles. Reabsorption of CSF increases with a slow increase in ICP. However, some pathological processes may obstruct the arachnoid villi or alter CSF flow within the brain and spinal cord, and this decreases absorption of CSF. Pathological processes, such as intracranial hemorrhage, inflammation, infection, tumors or congenital malformations, may decrease CSF reabsorption. If this occurs, the increase in intracranial volume increases ICP.

The cerebral circulation is tightly regulated by a number of homeostatic mechanisms. The main factors influencing CBF are the systemic blood pressure, CO_2, O_2, blood viscosity, and cerebral autoregulation. Adenosine and nitric oxide also affect CBF by altering coupling between metabolism and cerebral blood flow. Cerebral blood flow is directly coupled to metabolic oxygen demand, and both increase immediately after birth. A full-term newborn will autoregulate her/his mean arterial blood pressure between 20-60 mmHg. This narrow range of autoregulation makes infants vulnerable to ischemia and intraventricular hemorrhage. Thus, it is important to monitor arterial blood pressure to detect and treat high or low pressures that could increase the risk for intraventricular hemorrhage or cerebral ischemia and increase perioperative morbidity.[6,7]

In normal adults CBF is approximately 55ml/100g of tissue per minute. Almost 15% of the cardiac output goes to an organ (brain) that is only 2% of body weight. CBF in children is approximately 100ml/100g/min, which is 25% of their cardiac output. CBF in both preterm and term infants is lower, 40-50ml/100g/min. The cerebral metabolic rate for oxygen ($CMRO_2$) is one determinant of cerebral blood flow. The $CMR0_2$ and glucose requirements are higher in children than in adults (5.8 versus 3.5ml of oxygen/100g of brain tissue/min). The child's metabolic rate for glucose is 6.8mg/100g of brain tissue/min and the adult's is 5.5mg/100g of brain tissue/min.[8]

The coupling of $CMRO_2$ to CBF is probably mediated by the local hydrogen ion concentration in cerebral vessels. Conditions that cause acidosis and hypoxemia dilate cerebral vessels and increase CBF and cerebral volume. When autoregulation is impaired, CBF is determined by factors other than metabolic demands. If CBF exceeds metabolic requirements, hyperemia occurs. Finally, infants and children have larger heads in relation to body surface and a larger percentage of their cardiac output directed to their brains. The increased head size and increased amount of blood directed to the brain can contribute to hemodynamic instability during neurosurgery in children.[9]

Neuropharmacology

A major goal of neurosurgical anesthesia is to ensure adequate brain and spinal cord perfusion pressures while providing adequate surgical conditions (e.g., a relaxed brain). If drugs or the anesthetic technique used during surgery are not administered appropriately, pre-existing abnormal intracranial conditions can worsen and the risk of further CNS injury will be increased. Some anesthetics and anesthetic techniques protect brains exposed to metabolic and surgical stress. Thus, knowledge of anesthetic effects on the cerebral circulation, metabolism, and intracranial pressures in normal and pathological conditions is necessary. It is also very important to know the effects of drugs and techniques on patients undergoing functional neurosurgery and minimally invasive procedures (e.g., awake craniotomy, stereotactic surgery, identification of epileptic foci, and interventional neuroradiological procedures).

Some studies suggest that the lethal dose for some medications (LD50) is significantly lower in human neonates and children than in adults. The sensitivity of infants to sedatives, hypnotics, and narcotics is significantly higher, probably due to immaturity of the central nervous system (incomplete myelination, immature blood-brain barrier) and increased permeability of the brain to some medications. The effect of inhaled anesthetics also varies with age. The minimum alveolar concentration [(MAC), i.e., the concentration of inhaled anesthetic at which 50%of patients respond to a skin incision] is much lower for neonates (0-31 days of age) than for 1-6 months old infants. Although infants have increased anesthetic requirements, their margin of safety (i.e., the difference between adequate anesthesia and profound cardiovascular depression)

is much less than in adults. Therefore, drug doses should be carefully calculated and their pharmacologic effects carefully monitored to avoid adverse effects.[10]

Intravenous Anesthetics

All intravenous anesthetics decrease CBF and $CMRO_2$. These decreases are caused by depression of neuronal function that lessen brain metabolism. Ketamine is the only intravenous anesthetic that increases CBF, $CMRO_2$, and ICP.

Barbiturates

Barbiturates bind to the alpha subunit of the GABA receptors, which causes sedation and amnesia. They also reduce epileptiform activity. Sodium Thiopental is a neuroprotective agent that causes a dose dependent decreases in CBF, cerebral blood volume (CBV), and $CMRO_2$. Sodium thiopental also reduces ICP while maintaining cerebral autoregulation and reactivity of cerebral blood vessels to CO_2. It also attenuates ischemia-induced release of glutamate and inhibits intracellular release of calcium, which protects the brain during hypoxia/ischemia events.

Sulfhydryl molecules provide additional cerebral protection by scavenging free radicals, and this reduces the extent of brain damage during focal cerebral ischemia. High, nonclinical doses (10-to-55 mg/kg) of thiopentone decreased $CMRO_2$ by 50% when used to produce an isoelectric EEG. It may not be necessary to cause complete EEG suppression to achieve neuroprotection.[12] Barbiturates are also used to prevent increases in ICP associated with laryngoscopy and tracheal intubation. Production and absorption of CSF is unaffected by barbiturates. A significant problem when using barbiturates for cerebral protection is that they depress myocardial contractility and systemic arterial blood pressure, which decreases cerebral perfusion pressures.[11] Clinical doses of phenobarbital decrease the size of an infarct zone in rats following focal cerebral ischemia. Barbiturates have been used to reduce ICP and provide neuroprotection in patients undergoing neurosurgical procedures if cardiovascular stability can be maintained. The slow metabolism of barbiturates causes them to accumulate in the body.

Propofol

Propofol has similar properties to barbiturates, i.e., they decrease ICP, CBF, and $CMRO_2$. Cerebrovascular autoregulation and cerebrovascular responses to alterations in arterial blood pressure and $PaCO_2$ are preserved. The decrease in brain metabolism leads to decreases in CBF. However, several studies have shown that CBF decreases more than $CMRO_2$, suggesting that propofol has direct vasoconstrictor effects on cerebral vessels. In some patients (e.g., Moyamoya disease), large doses of propofol could cause cerebral ischemia. In animals, propofol has antioxidant activity, activates GABA receptors, attenuates excitotoxicity-mediated glutamate release, prevents mitochondrial swelling, and has endo-cannabinoid interactions. All of these

features protect the brain. Even low doses of propofol were shown to provide brain protection.[13] Patients at risk for intracranial hypertension and decreased cerebral perfusion had better brain protection with propofol than with inhaled anesthetics, at least until the dura matter was opened. Some studies suggest that prolonged administration of propofol (usually days) causes metabolic acidosis, hyperlipidemia, progressive heart failure, and death in pediatric patients (Propofol Infusion Syndrome). Therefore, prolonged use of this drug is contraindicated in children. If for some reason propofol must be used for days, the patient's condition must be closely monitored in an intensive care unit.[14]

Benzodiazepines

Benzodiazepines bind to GABA receptors to produce amnesia and anxiolysis. These drugs are said to decrease CBF by 25% and to decrease $CMRO_2$ and ICP, while elevating the seizure threshold. Flumazenil™, a benzodiazepine antagonist, reverses the beneficial effects of benzodiazepines on CBF, CMRO2 and ICP. Consequently Flumazenil should not be given at all or should be given cautiously to patients with intracranial pathology that could increase ICP and to patients who are predisposed to seizures.[15, 16]

Etomidate

Etomidate, a barbiturate-like drug, progressively decreases $CMRO_2$ until an isoelectric electroencephalogram (EEG) is produced. The marked decrease in CBF suggests a direct vasoconstrictor effect on cerebral vessels. Clinical doses of Etomidate reduce CBF and $CMRO_2$ by 35-50%. By decreasing cerebral blood volume, Etomidate effectively reduces ICP. It either has no effect on cerebral perfusion pressure or increases it. CO_2 vascular reactivity is maintained. Despite Etomidate's advantages, particularly its lack of cardiovascular depression, its use is limited by its capacity to suppress the adreno-cortical axis and by its ability to induce severe myoclonic activity, involuntary muscle movements, and myoclonic activity.[17,18]

Ketamine

Ketamine increases CBF (60%) and $CMRO_2$. The increased CBF may elevate ICP, especially in patients with intracranial pathology. The vasodilator effects of ketamine are due in part to its metabolic stimulant effects, direct vasodilator effects, and to cholinergic mechanisms. Although it has been suggested that ketamine has cerebral protective effects, recent studies in young animals showed significant neuronal apoptosis (cell death), even in the absence of brain injury. However, the ketamine-induced apoptosis may have been related to the high doses of drug used and the prolong duration of exposure to it in young animals. Under these experimental conditions, ketamine, other sedatives, and inhaled anesthetics are neurotoxic, but the extent of the neurotoxicity in humans is unknown.[19,24]

Dexmedtomidine

Dexmedtomidine is a highly selective alpha-2 adrenergic agonist with good sedating, anxiolytic, and analgesic properties. Evidence suggests that it protects the heart, brain, and kidneys from ischemic and hypoxic injury.[25] Dexmedtomidine decreases sympathetic transmission in the locus ceruleus, located in the brain stem, which produces sedation. The analgesic effects of this drug are due to activation of alpha$_2$ adrenergic receptors located within the dorsal horn of the spinal cord. This action prevents the release of substance P.[26] Dexmedtomidine has been used for premedication, as an anesthetic adjunct, and as treatment for postoperative delirium in children. Because of its moderate effects on the cardiovascular system, it is widely used for analgesia in pediatric intensive care units. There is evidence that it protects the developing brain.[27,28]

The cardiovascular effects of dexmedetomidine are mediated by central and peripheral nervous system adrenergic receptors. Small doses of this drug suppress the sympathetic nervous system, which decreases arterial blood pressure and heart rate. Hypotension and bradycardia have been reported when a loading dose of dexmedtomidine is given to children. Infusing the drug at 0.3-to-0.7 mcg/kg/h reduces the unwanted side effects. A great advantage of dexmedtomidine is its ability to maintain spontaneous ventilation and airway reflexes, even when high doses are used.

Dexmedtomidine decreases CBF and $CMRO_2$ by similar amounts in adults. It has no effect on ICP, CSF pressure, CPP, or on the reactivity of cerebral vessels to CO_2. Data suggest that dexmedtomidine provides brain protection during cerebral ischemia[29] in the developing brain of animals by blocking activation of the pro-apoptosis caspase-3 and by expressing tyrosine kinase, which is important for cellular plasticity.[30]

Dexmedtomidine preserves motor and sensory evoked potentials, making it a good drug for surgeries requiring motor and sensory monitoring. When used in combination with opioids and/or propofol, dexmedtomidine facilitates neurophysiological monitoring for scoliosis surgery and for placement of deep brain electrodes in pediatric patients.[31,32] Because sedation with dexmedtomidine acts through adrenoreceptors in the locus ceruleus, effects that mimic normal sleep, the drug is particularly useful for sedating children requiring EEG studies.

Localized tumor or seizure focus resection near areas important for language or movement (motor cortex) requires patient cooperation during surgery. Many drugs besides dexmedtomidine have been used to allow proper intraoperative monitoring, however, most cause respiratory depression, airway obstruction, hemodynamic instability, vomiting, dysinhibition of the CNS, and pain. Because it produces sedation, anxiolysis and analgesia without causing respiratory depression, dexmedtomidine is commonly used for awake craniotomy in older children and adolescents.[33,37]

Inhalational Anesthetics

Volatile anesthetics dilate cerebral vessels and increases CBF. Their effect on the ratio of CBF/$CMRO_2$ could increase brain volume and ICP. Volatile anesthetics cause more cerebral vasodilation in children than adults. They increase CBF in the following order: halothane > desflurane > isoflurane > sevoflurane.[38]

In children, halothane-induced increases in CBF persist despite reducing or discontinuing the drug. This phenomenon (i.e., cerebrovascular hysteresis) does not occur with isoflurane. At equipotent doses, isoflurane and sevoflurane decrease $CMRO_2$ less than halothane. Desflurane is the most powerful cerebral vasodilator of the modern inhalational anesthetics, while sevoflurane has the least effects on CBF and CBV in both adults and children.

Below 1.5 MAC, sevoflurane has little effect on the CBF of children and adults. Cerebral autoregulation is maintained at sevoflurane concentrations below 1 MAC. Cerebrovascular reactivity to CO_2 is also maintained. The cerebrovascular response to CO_2 is lost in children when their $PaC0_2$ exceeds 45-50mmHg. This does not occur in adults. Despite lower blood gas partition coefficient and more rapid recovery from general anesthesia, the effects of desflurane on the cerebral vasculature make it a less desirable agent for neurosurgery than either sevoflurane or isoflurane, especially for patients who are neurologically compromised.[39,40]

Nitrous Oxide

Nitrous oxide (N_2O) dilates the cerebral vessels of both adults and children when used alone or in combination with halogenated anesthetic agents or propofol. It increases cerebral blood flow in gray matter, especially in the supratentorial region. The exact mechanism for this increase is unknown; however, there is evidence that it is mediated by activation of ischemic mitochondria and by adrenal sympathetic stimulation. Cerebral autoregulation is affected when N2O is used alone or in combination with sevoflurane. In conclusion, N_20 alters cerebral autoregulation, cerebrovascular reactivity to CO_2, increases CBF and $CMRO_2$, and increases ICP while decreasing the threshold for ischemia in infants and children. It is recommended that N_2O not be used for patients at risk for having abnormal cerebral perfusion pressures and neuronal ischemia.[41,42]

Opioids

Opioids are thought to have few or no effects on CBF, $CMRO_2$, and ICP. Cerebral vascular reactivity to CO_2 and cerebral autoregulation are preserved. By blocking pain-induced release of catecholamines, opioids may indirectly decrease CBF. Opioids prevent hemodynamic responses during direct laryngoscopy, especially in patients with increased ICP or cerebrovascular disease. Cerebrovascular CO_2 reactivity and cerebral autoregulation are normal in patients given fentanyl. Fentanyl has no effect on CSF production, but it reduces reabsorption of by 50%.

Remifentanyl is an ultra short acting synthetic opioid that shares some similarities with fentanyl and alfentanyl, however its analgesic effect is about 65 times that of alfentanyl.[43] The extremely short half-life (3-5 min) and rapid recovery after remifentanyl makes it ideal for use in neurosurgical patients. Its metabolism by plasma and tissue esterases is independent of renal and liver function.[44] Remifentanyl decreases the $CMRO_2$ of both animals and humans.[45] Even very high doses (over 3 mcg/kg/min; clinical dose 0.1 to 0.5mcg/kg/min) of remifentanyl have no effect on $CMRO_2$, making remifentanyl useful in neurosurgical patients.[46]

In humans, infusion of remifentanil increases regional blood flow in a dose-dependent fashion in areas of the brain that process pain. Low-dose remifentanil (0.05mcg/kg/min) significantly increases regional CBF.[47] Several animals and human studies found no effect of remifentanil on CSF production or its reabsorption. Cerebrovascular reactivity to CO_2 is preserved. Morphine and fentanyl, but not remifentanil, disrupt cholinergic neurotransmission. This causes postoperative delirium and impaired memory. Both mean arterial pressure and heart rate are reduced by propofol-remifentanil anesthesia without causing any effects on CBF, implying that cerebral blood volume (CBV) and autoregulation are preserved.[48]

Minimally invasive neurosurgical techniques and functional procedures are now common. Anesthetics that maintain adequate brain relaxation, reduce interference with electrophysiological monitoring, ensure rapid neurological recovery after surgery, and provide neuroprotection are needed. Remifentanyl meets all these requirements.[49] Remifentanil plus an intravenous hypnotic or volatile anesthetic are commonly used. Quick recovery from anesthesia is one of the advantages of using remifentanil during neurosurgery. This allows rapid neurologic evaluation of the patient following surgery, more rapid tracheal extubation, and a better state of consciousness.[50]

Although no single anesthetic agent meets all neurosurgical anesthesia objectives, attempts have been made to identify an anesthetic or anesthetic technique that maintains coupling between CBF and oxygen metabolism, maintains cerebrovascular autoregulation, and does not increase CBV and ICP. Propofol and remifentanil are commonly used for these reasons, especially in patients who have intracranial hypertension. However, as with all patients with intracranial hypertension, a normal or slightly low $PaCO_2$ must be maintained. In patients without evidence of intracranial hypertension, sevoflurane-remifentanil is a good alternative to the use of propofol. Patients with space-occupying lesions, increased ICP, and decreased intracranial compliance do well with a propofol-based anesthetic.[51,52]

Depolarizing Neuromuscular Relaxants

Succinylcholine

Succinylcholine is the only easily available nondepolarizing muscle relaxant. Whether there is an intracranial space-occupying lesion or not, it increases the ICP of both animals and humans. This increase has been associated with muscle fasciculation, increased activity of afferent fibers on muscles, and increased cerebral blood flow. Neck muscles contractions compress the jugular veins and this is thought to increase ICP. The increase in ICP can be prevented or diminished by prior administration of small doses of a non-depolarizing muscle relaxant. Succinylcholine has caused life-threatening hyperkalemia, especially in patients with subarachnoid hemorrhage, traumatic brain injury, cerebral hypoxia, stroke, and paraplegia.

Non-depolarizing Neuromuscular Relaxants

Some muscle relaxants and their metabolites affect cerebral circulation through histamine release. Clinical doses for atracurium appear to have significant effects on CBF, $CMRO_2$, and ICP. Laudanosine, a metabolite of atracurium, easily crosses the blood-brain barrier and may induce seizures in seizure-prone patients. Seizures seldom occur in other patients. Cisatracurium releases less laudanosine and histamine than atracurium and has weaker CNS effects than atracurium.[53] Pancuronium, vecuronium and rocuronium have minimal effects on CBF, $CMRO_2$, and ICP. The increase in blood pressure and heart rate produced by these drugs might further increase the ICP of patients who have intracranial hypertension, especially if they have disordered cerebral autoregulation. Vecuronium does not induce histamine release or change heart rate and blood pressure. Because rocuronium has a rapid onset of action and none of succinylcholine's adverse effects, it is often used for rapid sequence induction of anesthesia and tracheal intubation. Like other neuromuscular relaxants, rocuronium has no effect on CBF.[54,55]

Preoperative Evaluation

Complete assessment and preparation of the pediatric patient for anesthesia and neurosurgery are essential to minimize perioperative morbidity and mortality (See Chapter 1). Preoperative evaluation of neurosurgical patients should include a complete review of the patient's medical conditions and the conditions for which the procedure is being done.[56] (**Table 10-1)**. **Table 10-2** gives some of the special concerns for pediatric patients with neurologic problems. The cornerstone for evaluation of brain function remains clinical history and physical examination.

TABLE 10-1: General Perioperative Concerns in Infants and Children

Condition	Anesthetic Implications
Prematurity	Postoperative apnea
Congenital heart Disease	Hypoxia, arrhythmia, cardiovascular instability, paradoxical air embolism
Gastrointestinal reflux	Aspiration pneumonia
Upper respiratory tract infection	Laryngospasm, bronchospasm, hypoxia, pneumonia
Craniofacial abnormality	Difficulty with airway management

TABLE 10-2: Common Perioperative Concerns for Infants and Children with Neurological Lesions

Condition	Anesthetic Implications
Denervation Injuries	Hyperkalemia after succinylcholine Resistance: nondepolarizing muscle relaxants Abnormal: response to nerve stimulation
Chronic anticonvulsant therapy Arteriovenous malformation Neuromuscular disease	Hepatic and hematologic abnormalities Potential congestive heart failure Malignant hyperthermia, respiratory failure Sudden cardiac death
Arnold-Chiari malformation Hypothalamic / pituitary lesions	Apnea, Aspiration pneumonia Diabetes insipidus, Hypothyroidism Adrenal insufficiency

Chapter 10: ANESTHESIA FOR NEUROSURGICAL PROCEDURES IN CHILDREN

Preoperative Evaluation of the Neurosurgical Patient Includes:

1) Evaluation of the patient's neurological status includes looking for the presence of intracranial hypertension and abnormal vital signs, assessing the respiratory and cardiovascular systems and looking for abnormalities in these systems that can affect neurological processes in the brain and spinal cord, assessing the level of consciousness, and evaluating motor and sensory neurological deficit and cranial nerve involvement.
2) Determination of the presence of raised ICP and associated neurological deficits. **(Table 10-3)** The presentation of patients with increased ICP varies, depending on the duration of ICP elevation. A sudden increase in ICP frequently leads to coma, whereas in less acute raises does not. Patients who awaken with a headache each morning may have hypercapnia during sleep, increased CBV, and decreased intracranial compliance. Neonates and infants who have increased ICP usually have a history of irritability, decreased appetite, and lethargy. Bulging anterior fontanels, dilated skull veins, and an enlarged deformed skull are common signs of increased ICP in neonates and infants. Other signs include double vision (diplopia) caused by oculomotor palsy (rising sun sign) and strabismus (cranial nerve VI palsy). Older children who have increased ICP often vomit in the morning. Papilledema and absent venous pulsation are seen in the eyes when the ICP is elevated.
3) Detection of electrolyte disturbances. Electrolyte disturbances can occur with decreased consciousness, vomiting, bulbar dysfunction, use of osmotic or loop diuretics to lower ICP, and prolonged fasting. Inappropriate secretion of antidiuretic hormone, the brain-induced salt loosing syndrome, and diabetes insipidus may cause disordered sodium and water balance.
4) Detection of hyperglycemia. Hyperglycemia is common in patients with neurologic diseases, especially those who are treated with steroids. Hyperglycemia often causes osmotic diuresis, hypovolemia, and electrolyte disturbances. Patients with a craniopharyngioma frequently have pituitary dysfunction. Proper pre-surgical evaluation of their endocrine system is mandatory.
5) Detection of allergies to foods, drugs, or radiological contrast material. The presence of a latex allergy must be sought, particularly in patients who have a myelomeningocele or others who require regular bladder catheterization.
6) Detection of toxicity to chronic anticonvulsant use. Patients with seizures are often on high doses of anticonvulsants that may cause hematologic disorders, liver dysfunction, or both. Children on chronic seizure therapy often require larger doses of sedatives, nondepolarizing muscle relaxants, and opioids because their anti-seizure medications have increased the enzymes that metabolize these drugs.[57]
7) Evaluation of X-rays, MRIs, and brain computed tomography (CT) studies. All of these studies must be reviewed before surgery to confirm the location of the primary lesion, the

presence or absence of hydrocephalus, compression, cerebral edema, and the anticipated patient position required for surgery. Laboratory tests should be based on the patient's condition and the proposed surgical procedure. Due to the risk for massive bleeding during neurosurgery, the anesthetist should evaluate the patient's hemoglobin (Hb.) concentration, hematocrit (Hct), platelet count, partial thromboplastin time (PTT), prothrombin time (PT), and fibrinogen concentration and should make sure there will be sufficient blood available when needed.

8) Assessment of the airway. It is of great importance that the airway be evaluated thoroughly because some patients with neurological diseases, mainly those with craniofacial malformations, have difficult airways (See Chapter 7). It is vitally important that these abnormalities are recognized and planed for preoperatively.

TABLE 10-3: Signs of Intracranial Hypertension in Infants and Children

Infants	Children	Infants and Children
Irritability	**Headache**	**Decreased consciousness**
Full Fontanels	**Diplopia**	**Cranial Nerve (III and VI) palsies**
Widely separated cranial sutures	**Papilledema**	**Loss of upward gaze (setting sun sign)**
Cranial enlargement	**Vomiting**	**Signs of herniation; Cushing's triad**
Pupillary changes		

Premedication

Premedication of patients undergoing neurosurgery can cause respiratory depression, hypercapnia, loss of protective airway reflexes, and sudden increases in ICP that can alter the patient's state of consciousness, hemodynamic stability, and can increase her/his risk for death. Therefore, preoperative sedatives and narcotics should be avoided in these patients unless the drugs are given in a monitored environment under the supervision of an anesthetist.

Patients with normal ICPs scheduled for endovascular procedures are often sedated to reduce anxiety, prevent systemic hypertension, and prevent rupture of intracranial vascular lesions. Oral midazolam (0.5mg/kg) is particularly useful for relieving anxiety, producing amnesia, and reducing the risk for seizures in some neurosurgical patients. The taste of this bitter drug must be hidden in a sweet solution (e.g., 5ml of Coca Cola). Emotional preparation of the patient, when properly done, is often the only "premedication" needed for children who are older than six years of age.

This type of "premedication" helps reduce preoperative anxiety and helps avoid potentially deleterious intracranial cerebrovascular changes that raise ICP**.**

Perioperative Considerations

Knowledge of potential complications of neurosurgical procedures allows the anesthetist to develop strategies to prevent these complications and to initiate prompt treatment for them if they occur. Both early recognition and early treatment of complications reduces the morbidity and mortality related to these procedures. Factors such as emergency surgery, severe co-morbidities, age (prematurity), complications with sedation, increased difficulty in placing vascular accesses, massive bleeding, and massive transfusion are potential complications that increase the morbidity and mortality of infants and childen.[58-61]

The goal of neuroanesthesia is to ensure that the child has adequate cerebral perfusion pressure and that the ICP does not increase during induction of anesthesia by preventing hypoxemia, hypercapnia, and hypertension. An understanding of the patient's preoperative status, coexisting conditions, and the presence of an elevated ICP allow the anesthetist to choose appropriate drugs for induction and maintenance of anesthesia. IV induction with thiopental 5-8 mg/kg or propofol 2-4 mg/kg, an opioid (fentanyl and remifentanil), and a short acting neuromuscular relaxant (succinylcholine, rocuronium), facilitates rapid tracheal intubation of children who have intracranial hypertension and are at risk for pulmonary aspiration. If the use of succinylcholine is contraindicated by its effects on CBF and ICP or the patient has a spinal cord injury, prolonged standing, burns, and subacute paretic limbs, rocuronium is often used rather than succinylcholine. Patients with the problems listed above can develop sudden hyperkalemia and die when given succinylcholine.

Children without an elevation of ICP and no intravenous access or those in whom obtaining IV access will be difficult can have anesthesia induced with sevoflurane and oxygen while ventilation is controlled to prevent unwanted increases in CO_2 and ICP. After placement of an IV, sodium thiopental or propofol can be given to prevent the increases in ICP associated with laryngoscopy and tracheal intubation. During the induction of anesthesia, moderate hyperventilation (PCO_2 30) via facemask reduces the increases of ICP produced by inhaled anesthetics, the administration of opioids and hypnotic agents, and by laryngoscopy and tracheal intubation. However, sevoflurane and hyperventilation can induce seizures, particularly in children with know epilepsy. At times sevoflurane can cause seizures in children who have no history of seizures.[62] Ketamine should not be used to induce anesthesia because of its detrimental effects on CBF, $CMRO_2$ and ICP.

Airway Management

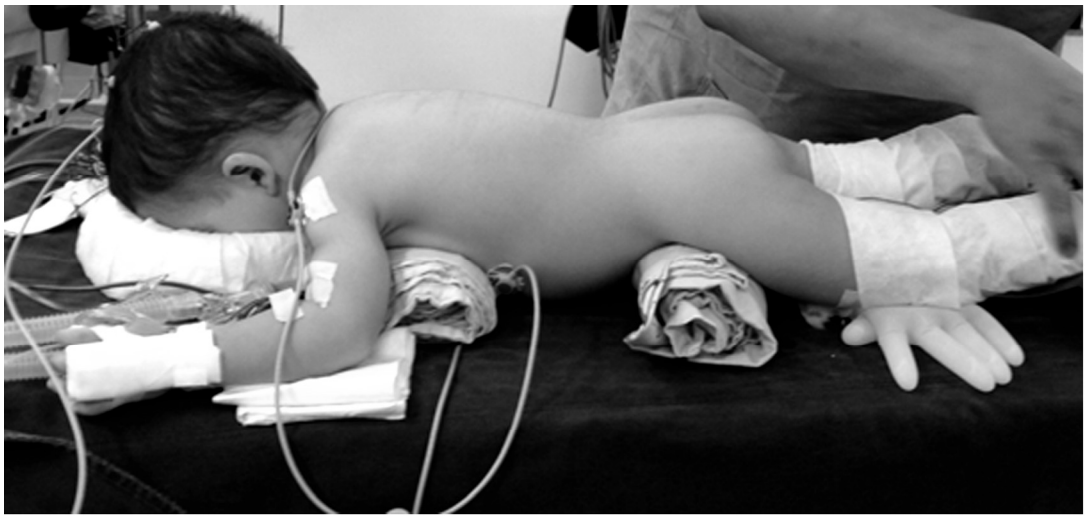
Developmental changes in the pediatric airway have significant impact on anesthetic management. Because the trachea is relatively short, the tracheal tube can easily migrate into the right bronchus when the head is flexed, especially when the child is placed in the prone position with her/his neck flexed. Extension of the head may cause accidental tracheal extubation. Care should be taken to secure the tracheal tube to the patient's face. Nasotracheal

intubation may provide greater stability if the tube is fixed in place by a placing a suture through the columella and the edge of the tracheal tube and then tying the suture around the tube. Oral tubes can be sutured to the teeth. Nasal tubes may be more comfortable after surgery for patients requiring postoperative tracheal intubation and mechanical ventilation. Nasotracheal intubation is used for surgery done in the prone position, when the airway will be inaccessible during surgery, and when the patient is very young. Contraindications for nasal intubation include choanal stenosis, procedures to repair basal skull fractures, and sinusitis. Tracheal tubes can bend and obstruct when they curve around the base of the tongue. Use of a pre-formed tracheal tube (e.g., RAE) prevents this kinking.

Patient Position During Surgery

Part of the preparation for anesthesia includes preparing the operating table and having appropriate equipment to prevent injury to the patient induced by the positioning during surgery. Artificial tears/ointment should be placed in the eyes before sealing the eyelids closed with a suture or tape. Pressure on the globe during surgery can cause retinal ischemia and postoperative blindness. Furthermore, in prone position the face and other sensitive areas must be padded to prevent injury from excessive pressure. To avoid compromising ventilation in the prone position, a "U-shaped" bolster is placed under the child to allow free movement of the abdomen and diaphragm. In smaller patients, small rolls can be placed under the upper chest and the pelvis. Placing a roll under the patient's abdomen would increase intra-abdominal pressure, compress the inferior vena cava and epidural veins, and increase bleeding during spinal surgery. **(Figure 10-1)**

Figure 10-1: Prone Position for Spinal Tumor Resection.



Elevating the head 10 degrees improves cerebral venous return and reduces venous congestion. This reduces sagittal sinus pressure, which may increase the risk for venous air embolism after the

skull is opened. Rotating the head to the side compress the jugular veins, reduces venous return, and increases ICP. If the head must be rotated, rotating the patient's trunk to maintain axial alignment can prevent obstruction of venous return.

During any surgical procedure, it is important for the anesthetist to maintain access to the tracheal tube, connections to the tube, and the anesthetic circuit so he/she can be easily inspect them. Furthermore, it is desirable to have a hand or foot visible during surgery to assess peripheral perfusion, skin color, and neuromuscular relaxation. Significant facial and airway edema occur in patients who are placed in the prone position for long periods of time and/or are given large volumes of IV fluids. If significant facial edema occurs, it is advisable to leave the child's trachea intubated during the immediate postoperative period and wait for the edema to subside. Postoperative blindness has occurred during spinal surgery done in the prone position. The usual causes are blood loss (hypovolemia) or pressure on the eye. Pressure of the eyeball, anemia, and systemic hypotension must be avoided if blindness is to be prevented.[63,64]

Vascular Access and Hemodynamic Monitoring

Because of the risk for massive intraoperative bleeding and because it may be difficult obtain additional vascular access during neurosurgical procedures, two large peripheral venous catheters should be inserted before the patient is positioned for surgery. In small infants, blood transfusion should be initiated sooner rather than later because it is so easy to fall behind on volume. If possible, blood should be brought to the operating room and stored in an ice chest before the procedure begins. Blood transfusion should be performed via a peripheral IV when possible and not via a central venous catheter to reduce the risk of potassium induced cardiac arrhythmias.

Monitoring during anesthesia includes a precordial or esophageal stethoscope, ECG, noninvasive blood pressure, pulse oximetry, capnography and temperature. Urine output should be monitored during lengthy procedures, especially when osmotic diuretics are used.[65] Patients scheduled for major craniotomies and spine surgery are at risk for hemorrhage and hemodynamic instability cause by air embolism, manipulation of cranial nerves, and herniation of the brain. The high risk for cerebral injury during neurosurgery justifies inserting an arterial line (**See Chapter 2**) to monitor the patient's hemodynamic status, intravascular volume, acid-base status, electrolytes, blood sugar and lactic acid concentrations, and hematocrit. Increase variability of the arterial pressure waveform with positive pressure ventilation is an excellent indicator of intravascular volume deficit.

Arterial catheters are often percutaneously placed into a radial, posterior tibial, or femoral artery.[66,67] The utility of central venous access is controversial in pediatric patients. However, its use should be considered when intravenous access is difficult, when assessment of right ventricular filling pressures might be helpful, and when it may be necessary to infuse inotropes or vasopressors during surgery. Central venous catheters are most often inserted into the

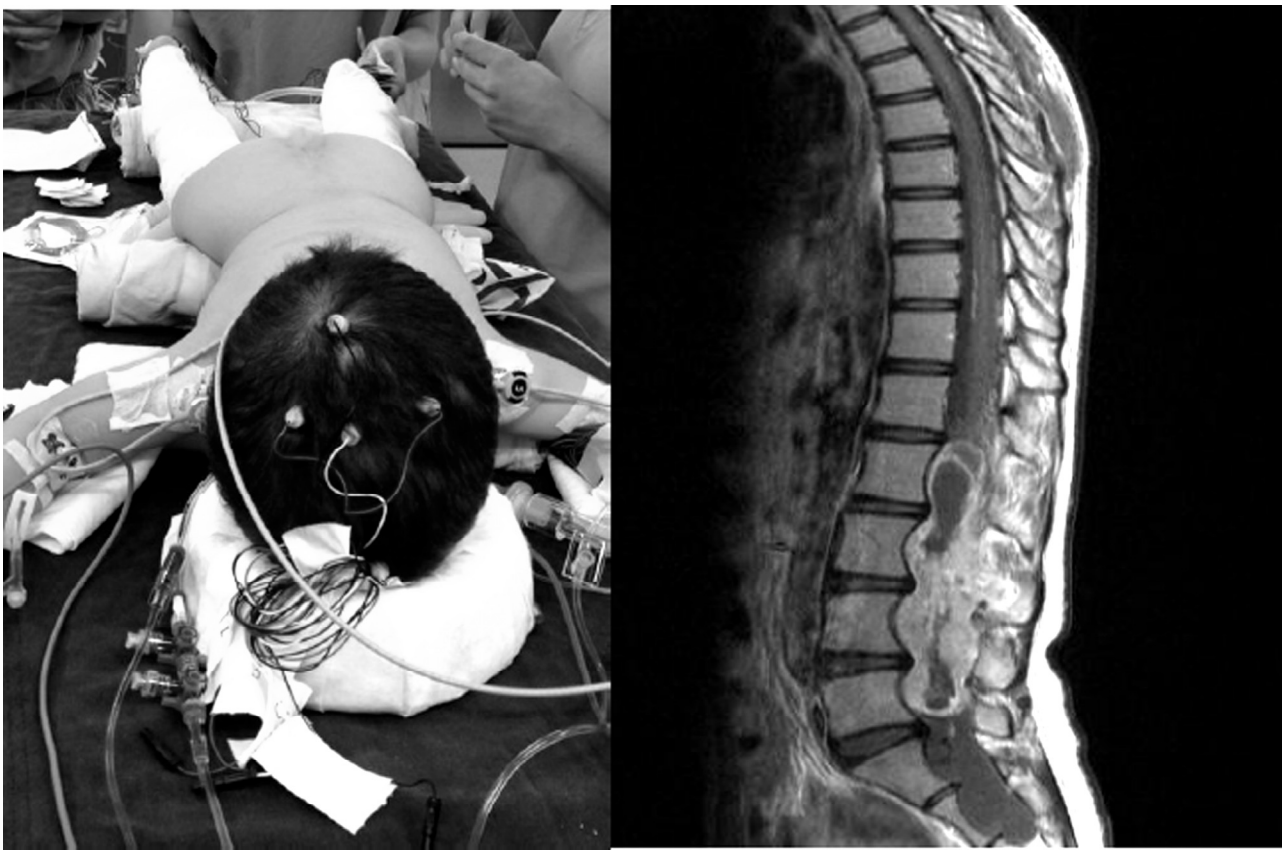
subclavian and femoral veins (**See Chapter 2**). Femoral vein catheters are usually more easily available to the anesthetist during the surgery, but they should be removed as soon after surgery as possible to avoid complications from their use. The internal jugular route is seldom for insertion of central lines in patients undergoing neurosurgery because they compromise cerebral venous return, especially in young children. In patients undergoing neurosurgery, a precordial Doppler should be used routinely, along with capnography and an intra-arterial catheter, to detect air embolism before hemodynamic instability develops.[68,69]

Neurophysiological Monitoring

Recent advances in neurophysiological monitoring have improved the safety of brain and spinal cord surgery. In developed countries, intraoperative neurophysiological monitoring (IONM) is used for most spinal and cranial surgeries. IONM provides the surgeon with valuable information about the integrity of the spinal cord, nerve roots, and peripheral nerves during critical moments in the surgery.[70]

Preoperative evaluation should include deciding which anesthetics are appropriate for the type of neurophysiological monitoring that will be used. Some intravenous and inhaled anesthetics have effects on intraoperative neurophysiologic recordings. Neurosurgical procedures that benefit the most from IONM are procedures involving the corticospinal tracts, dorsal columns, and the cranial nerves and nerve roots. These include surgery for anterior and posterior spinal fusion, release of a tethered spinal cord, dorsal rhizotomy, craniotomies for tumor resection, and posterior fossa decompression. **(Figures 10-2A-2B)**

Figure 10-2 A: (Above) Preparation for Neurophysiological Monitoring in Spinal Tumor Resection. Figure 10-2 B: (Below) Spinal Tumor Resection



Monitors used during neurosurgery include electromyography (EMG), somatosensory evoked potentials (SSEP), motor evoked potentials (MEP), electroencephalography, electrocortigraphy (EEG-EcoG), auditory evoked potentials of the brain stem (PEA), and EMG for monitoring cranial nerves VII , IX , X , XI , XII. Cerebral oximetry (NIRS), EEG, jugular bulb catheters, and transcranial Doppler have been used to monitor cerebral oxygenation during surgery.[71-73]

Electromyography (EMG)

During surgery, the EMG is often used to monitor activity of muscles. This type of monitoring provides real-time information on the state of conduction through nerve roots and peripheral nerves. Peripheral nerves can be irritated by surgical manipulation, inadvertent retraction or compression of nerves or spinal cord, stretching of a nerve, or by ischemia from inappropriate positioning of the patient for surgery. This can be detected by EMG monitoring. Muscle relaxants should not be used during EMG monitoring because they prevent muscles from responding to normal signals from nerves. Facial muscles innervated by the seventh nerve are especially sensitive to neuromuscular relaxants.

Somatosensory Evoked Potential (SSEP)

SSEPs are generated by action potentials in the nuclei of the central nervous system and by primary sensory cortical neurons in response to peripheral nerve stimulation. The nerves most often stimulated include the median or ulnar for the upper limbs and the posterior tibial nerve for the lower extremities. The signal generated travels via the stimulated ipsilateral peripheral nerve and ascends to the cuneiform nuclei (upper limb) or graciles nuclei (lower extremities and trunk) of the dorsal spine. From there the signal crosses to the contralateral side and travels to the sensory cortex via the thalamus.

SEPs provide valuable information about sensory tract integrity from the peripheral nerve to the sensory cortex. The amplitude and latency of the waves produced by the electrical stimulus must be evaluated before surgery to determine whether the signal is normal or abnormal. During surgery SSEPs are compared with the signals obtained prior to surgery to detect acute intraoperative changes. The surgeon and anesthetist should be notified immediately of a greater than 50% decrease in signal amplitude or of a 10% increase in conduction latency from pre-incision values.

Blunt trauma of the spinal cord typically causes immediate changes in SSEPs, whereas with spinal cord ischemia the changes to occur later. Acute loss of SSEPs indicates loss of neurological function and requires immediate action by the surgical team. Blood loss that is sufficient to reduce blood flow to the cuneiform and gracilis nuclei, thalamus, or primary sensory cortex alters SSEPs. SSEPs disappear when the cortical blood flow is below 15-18ml/100g /min. It is important to monitor the effects of patient position on SEPs prior to surgery because nerve compression or tissue ischemia affects the signals. Decreased ulnar SSEPs are often seen during traction on the brachial plexus. Changes in SSEPs should never be ignored because they may indicate nerve injury and a potential for increased postoperative morbidity. Over time, prolonged infusion of propofol can lead to gradual decreases in SSEPs as propofol accumulates in the tissues. The effects of anesthetics should always be distinguished from pathological effects. If the anesthetist is familiar with the pharmacokinetics of drugs used during neurosurgery, he/she can adjust the

infusion rates (doses) of the drugs during long procedures to reduce unwanted complications and to shorten wake up times. Neuromuscular blocking drugs do not directly influence the SSEP. In fact, they may improve the quality of SSEPs by eliminating muscle artifacts. Inhalation anesthetics reduce the amplitude of SSEPs.

Motorsensory Evoked Potentials (MEP)

The major drawback to SSEP monitoring is that it only evaluates the integrity of the ventral motor portion of the spinal cord cortical tracts. MEPs are acquired during surgery by applying a high voltage, short duration stimulus to the motor cortex via the scalp. The amplitude, latency, and morphology of the signal are used to assess the integrity of motor conduction. In general, all inhalational anesthetic agents, except desflurane, have significant negative impact on MEPs, even at low doses. Total intravenous anesthesia (TIVA) with propofol and remifentanil reduces or prevents alterations of MEPs. Muscle relaxants block MEPs and should no be used when MEPs are being monitored. A history of seizures is a relative contraindication to the use of MEPs because the electrical stimulation used to test for MEPs can induce intraoperative seizures that might go unnoticed. If this happened, detrimental effects on the brain might occur. Ventriculoperitoneal shunts, skull fractures, and implanted metallic devices can affect placement of scalp needles and interfere with the electrical stimulation and monitoring of the MEPs. The presence of a cochlear implant is an absolute contraindication to the use of MEPs.

Electroencephalography (EEG)

EEGs are used intraoperatively to evaluate depth of anesthesia, adequacy of the cerebral regional and global perfusion, assess electrical patterns following cortical stimulation, and determine the presence of seizure activity. Continuous EEG monitoring can detect cerebral ischemia. The EEG pattern deteriorates when CBF is <18ml/100g of brain/min or approximately 40% of normal. During surgery for MoyaMoya disease, EEG monitoring is used to determine which $PaCO_2$ provides the best CBF. It is also used to determine the adequacy of blood volume and the effects of temperature on the balance oxygen of supply and demand. EEG monitoring is also used in these patients to determine the onset of ischemia.

EEG Monitoring is also helpful during repair of cerebral aneurysms and arteriovenous malformations.[74] A decrease in CBF is accompanied by a decrease in EEG amplitude. In the absence of medications that potentially produce an isoelectric signal, the presence of an isoelectric EEG suggests brain death. Asymmetry in the amplitude of the EEG between the right and left hemispheres indicates an imbalance in the CBF between the two hemispheres, possibly due to an hematoma or ipsilateral vascular insufficiency.

Auditory Evoked Potentials (AEP)

AEPs are divided into short latency, average latency, and long latency signals. During surgery short-latency responses are resistant to the effects of anesthetics; the others are not. AEPs are used to assess the integrity of the eighth cranial nerve or the ascending auditory pathway up to the level of the inferior colliculus. AEPs are also useful during resection of acoustic neuromas, meningiomas, or pontomedullary tumors.[75]

Spinal fusion surgery to correct scoliosis is perhaps the most common use of IONM in children. Before IONM was available, the wake-up test was used intraoperatively to determine if the motor system was intact. This test has been abandoned where IONM is available because many patients woke up with nerve deficits despite a normal wake up test deficits during surgery. Other complications occurred, including inadvertent tracheal extubation in prone position, accidental removal of the venous catheters and arterial line, and an increased risk for neurological injury. The problem with this test was that it only provided valuable information during the short period when the patient was awake. No information is provided when the patient was anesthetized.

Acute complete loss of SSEPs or MEPs during spinal surgery is a major concern for the surgeons because it indicates trauma to the spinal cord. This injury may be the result of direct nerve injury, vascular insufficiency, or vascular ischemia from systemic or local hypotension. The loss of MEPs or SSEPs should trigger an immediate response by the anesthetist and surgeon to restore spinal cord perfusion as quickly as possible by raising the perfusion pressure.

The anesthetist's response should include increasing the inspired oxygen concentration when possible, decreasing the amount of anesthetic being administered, and increasing intravascular volume with boluses of crystalloid fluids or RBCs. Vasopressor or inotropic drugs are used to increase the perfusion pressure when the above measures fail to correct the problem. The surgeon must look for surgical causes for the altered MEPs and/or SSEPs. If the MEPs or SSEPs cannot be restored within 15 minutes, it is essential to undo the surgical correction and prepare to awaken the patient to determine if spinal cord damage has occurred. If awakening the patient confirms the findings of the MEPs and SSEPs, a spinal cord injury protocol should be initiated immediately to prevent further cord damage and restore function to the spinal cord. Several institutions have recommended quickly giving a bolus of methylprednisolone 30mg/kg followed by a continuous infusion of 5.4mg/kg/hour of methylprednisolone for the next 24 hours. Methylprednisolone stabilizes cell membranes by decreasing the release of free radicals during ischemia, which decreases the risk of neuronal injury during spinal cord reperfusion. Hypothermia, hypotension, hypoxemia and hypocapnia also affect neuromonitoring.[76]

Maintenance of Anesthesia

General anesthesia is maintained with inhaled or intravenous anesthetics or with both. The ideal medications for this purpose would decrease $CMRO_2$ and ICP while maintaining normal CBF. Remember, all anesthesia agents are vasodilators and have the potential for increasing CBF, CBV and ICP in patients with decreased intracranial compliance. Low concentrations of isoflurane or sevoflurane (less than 1 MAC), when combined with a continuous infusion of fentanyl or remifentanil and sufficient positive pressure ventilation to maintain normocapnia have minimal effects on CBF and ICP. N_2O is usually not used for maintenance anesthesia during neurosurgery because of its detrimental effects on $CMRO_2$, CBF, ICP and its increased risk of causing postoperative nausea and vomiting. Moreover, N_2O has unwanted effects on SSEPs and MEPs. Neuromuscular blocking agents should be used (when appropriate) during the perioperative period to prevent inadvertent movement and occasionally fatal neurological consequences. Chronic preoperative use of anticonvulsants increases the doses of muscle relaxants and opioids required because anticonvulsants increase activity of the liver enzymes that metabolize these drugs. When muscle relaxants cannot be used during surgery, dexmedetomidine provides effective sedation and analgesia without affecting ventilation, calm awakening from anesthesia, and neuroprotection. Consequently, dexmedetomidine is widely used during electrophysiological monitoring and awake craniotomy in children.[33]

Fluid Management

Intraoperative fluid management has many implications for anesthetists during pediatric neurosurgical procedures. These patients experience rapid changes in intravascular volume from bleeding, administration of osmotic diuretics, or diabetes insipidus. The goals of fluid management include maintaining CPP, preserving intravascular volume, and preventing cerebral edema by maintaining an isovolemic, isotonic, and isooncotic state.

The Starling equation describes the factors that govern movement of fluids between intravascular and extracellular spaces. Unlike other tissues, the brain and spinal cord respond to changes in intravascular volume differently than most other tissues because the brain and spinal cord are isolated from the intravascular compartment by the blood brain barrier (BBB). The BBB is composed of astrocytic foot processes and endothelial cells that form tight junctions. These tight junctions limit movement of molecules between the intravascular space and the CNS. The very small pore size at these junctions (7-to-9 A°) limits free movement of many molecules, including electrolytes and proteins, into and out of the brain. The BBB acts as a semipermeable membrane. Water moves freely through this membrane in response to the relative concentrations of impermeable solutes on each side of the membrane. Glucose and amino acids require energy and transporters to cross the membrane. In muscle, lung, and other tissues, the pore size of capillary endothelium is about 65 A°, which allows many small molecules and ions (Na^+, Cl^-) to

move freely across the membrane. Large molecules, such as proteins, do not move freely. Electrolytes easily pass from the capillary lumen to the extracellular space of muscle etc. Movement of water between the intravascular and extravascular spaces of peripheral tissues is governed by the oncotic gradient created by the plasma concentration of large macromolecules.

Conversely, fluid moves in and out of the CNS through osmolar gradients that exist between plasma and extracellular fluid. These gradients are created by the relative concentrations of all osmotically active particles, including most electrolytes. These differences between the brain and other tissues explain why administering large volumes of iso-osmolar crystalloid causes peripheral edema (by dilution of plasma proteins) without increasing the amount of water in the brain or increasing ICP. Giving hypo-osmolar fluids allows more water to enter the brain. This causes cerebral edema and increases ICP. On the other hand, giving hyperosmotic fluids, such as mannitol or hypertonic saline (3%), removes water from the brain and reduces the cerebral edema, and ICP. If the BBB is intact, plasma osmolality is the major determinant of water movement between the CNS and the intravascular space.[77,78]

Fluid therapy for neurosurgical patients requires knowledge of three properties of blood, osmolarity, oncotic pressure, and hematocrit. Anesthetists have a variety of commercial solutions available for infusion during surgery that are best categorized by their osmolarity, dextrose content, and oncotic pressure. Normal saline is commonly used as maintenance fluid during neurosurgery because it is slightly hyperosmolar (308mOsm/L) and minimizes cerebral edema. However, hyperchloremic acidosis occurs when more than 60 ml/kg of normal saline are administered. Saline-induced hyperchloremic acidosis does not increase morbidity and mortality. In general hypo-osmolar solutions, such as Ringer's lactate solution plus dextrose, are not used during neurosurgery because they increase the amount of free water available for transport into the brain. Metabolism of the dextrose also produces free water and will directly contribute to enlarge the interstitial volume and edema.

To maintain adequate intravascular volume, cardiac output and tissue perfusion, ongoing blood loss, insensible water loss (e.g., through ventilation), and urine output should be replaced with an iso-osmolar crystalloid solution. Excessive fluid resuscitation should be avoided. There is strong evidence showing that monitoring of DYNAMIC cardiovascular variables (changes in arterial pulse pressure, changes in systolic blood pressure during positive pressure ventilation) makes it easier to maintain a stable blood volume than does monitoring STATIC hemodynamic parameters (right atrial pressure, central venous pressure).[79]

It is very difficult to quantify blood loss during neurosurgical procedures because much of the blood is confined to the surgical field and hidden. Furthermore, the surgeons use of large volumes of irrigating solution throughout surgery that mix in the suction with blood, making it difficult to quantitate true blood loss.

A large percentage of blood loss in pediatric patients occurs during dissection of the scalp. Infiltration of the skin and scalp with local anesthetic with epinephrine just prior to surgery reduces blood loss. The risk of significant blood loss during craniotomy and the resulting decrease in blood volume should lead anesthetists to routinely calculate each child's predicted blood volume and determine how much blood he/she will allow the patient to loose before initiating blood transfusion. The decision to transfuse or not transfuse a patient should not only depend on the patient's Hb. concentration and HCT but also on the patient's hemodynamic status and the adequacy of tissue perfusion. During acute bleeding, the recommendation is to maintain a Hb. concentration of 8g/dl.[80] More than four-month-old hemodynamically stable infants who are not actively bleeding can have an Hb concentration of 7g/dl before they are transfused. Infants <4 months of age and those with cyanotic or other congenital heart disease, chronic lung disease, and hemoglobinopathies should have their Hb. concentrations maintained at 10g/dl.[81,82]

When massive bleeding is anticipated, the blood bank should be consulted prior to surgery to ensure that there will be a reserve of less than two-week old blood available for the procedure. Blood older than this duration of storage time has high potassium concentrations, and rapid transfusion of this blood has caused cardiac arrests. The volume of blood to be transfused should be calculated based on the formulas given above. Each 5cc/kg of packed red blood cells (Hct 70%) raises the hemoglobin concentration by 1g. Whether to transfuse a patient or not depends on the expected blood loss during the remainder of the surgery and on the expected postoperative blood loss from drains, frequent laboratory examinations, surgical re-intervention, etc. It is safer to transfuse blood to a patient from a single donor than from multiple donors.[83,84] **Table 10-4** provides the steps and the objectives for management of perioperative bleeding in neurosurgical patients**.**

TABLE 10-4: Steps and Goals for Perioperative Bleeding Management in Neurosurgical Patients

Steps
Preoperative
Calculate child's blood volume in function of the age
Calculate allowable blood loss
Reserve blood products in accordance to the patient needs and the surgery
Order fresh red blood cells (maximum one or two weeks of storage)
Keep available the red blood cells in OR when risk of massive blood loss is present

TABLE 10-4: (Continued)
Intraoperative
Install the hemodynamic monitoring according to the hemodynamic changes and predicted blood loss.
Install a central vascular access according to expected blood loss
Secure two large bore peripheral venous access
Control regularly blood loss
Monitoring acid base, electrolyte, lactic acid, glycemia, platelets, TP, TPT, Hb, Hct
Goals
Maintain cerebral perfusion pressure and tissue perfusion within normal values
Maintain diuresis at 0.5-1 ml/kg/h
Maintain blood pH between 7, 35 and 7,45
Maintain calcium around 1.15 mmol/l
Maintain normothermia and normocapnia at all time

Glucose-containing solutions should not be used in patients with CNS or spinal cord pathology. As stated above, metabolism of the glucose releases free water, which reduces the serum osmolality and increases the brain water content and ICP. Many studies in humans and animals have reported that glucose administration increased CNS damage and worsened focal ischemic insults by causing tissue acidosis. The mechanism by which hyperglycemia does this is believed to be through increased lactic acid production. The increased intracellular lactate and acid have neurotoxic effects that cause neuronal death and cellular necrosis. Hyperglycemia also increases glutamate release, which increases neuronal injury and worsens outcomes.[85] Pediatric patients, particularly young infants, are at risk for developing hypoglycemia if they have low glycogen stores. The potential for developing hypoglycemia is increased by the fact that they have limited ability to make more glucose via gluconeogenesis. Therefore, it is often necessary to continuously infuse sufficient glucose to maintain normoglycemia. The current recommendations is to give 120mg/kg/hour or 2mg/kg/min of glucose to preterm and to term babies who had glucose infused preoperatively, to children with liver disease or metabolic disorders, to children receiving total parenteral nutrition, to malnourished children whose weights are below the 3rd percentile for age, and to children treated with beta blockers. The only way to know if a patient is normoglycemic, hypoglycemic, or hyperglycemic is to frequently measure her/his blood glucose concentration.[86-90]

Management of the Intracranial Pressure

During neurosurgical procedures cerebral edema can have devastating consequences. Causes for cerebral edema include increased permeability of the blood-brain barrier with accumulation of osmolytes in the interstitium of the brain; this causes lysis and necrosis of cells and cerebral ischemia. Hyperventilation ($ETco_2$ 25-30mmHg) is sometimes used to prevent eminent herniation of the brain, but hyperventilation itself can also injure the brain by reducing CBF and cerebral

perfusion pressure. For every mmHg decrease in $PaCO_2$, brain blood flow decreases by two percent. Elevating the head 10° and using hyperosmolar therapy should be instituted early.[91]

Administration of hypertonic saline (3%) (3-5ml/kg bolus over 30-60 min, continuous infusion 0.1-1.0ml/kg/h) produces an osmotic gradient, which allows transfer of brain interstitial water to the intravascular space. This significantly decreases cerebral edema and ICP. Hypertonic saline is thought to also decrease production of CSF.[92]

The beneficial effects of hypertonic saline include reduction of ICP and reduction of cerebral edema without compromising intravascular volume, arterial blood pressure, and CPP. Hypertonic saline probably reduces ICP by restoring cell membrane potentials, causing the release of atrial natriuretic peptide, by anti-inflammatory effects, and by stimulating cardiac output. Unwanted side effects include rebound increases in ICP, pontine myelinolysis, renal failure, subarachnoid hemorrhage, and hyperchloremic acidosis.[93, 94] Children who received 10ml/kg of 3% saline had significant reductions in their ICP, whereas those who received the same volume of normal saline 0.9% did not.[95]

Mannitol 20% (osmolality 1,098) is the osmotic diuretic most commonly used during neurosurgery. Small doses 0.25-to-0.5g/kg increase osmolarity by 10 mOsm, which is enough to reduce brain edema and ICP within 10-to-15 min. The effects of Mannitol last about two hours. Mannitol should not be given faster than 0.25-to-0.5g/kg over a period of 20-to-30 minutes to avoid hemodynamic instability. Mannitol administration has a biphasic effect on the ICP. It temporarily increases ICP by initially increasing the intravascular volume and cerebral blood flow. Water is then rapidly withdrawn form the intracellular and interstitial spaces of the brain, which reduces ICP. Administering repeated doses of mannitol has led to hyperosmolality, renal failure, and cerebral edema. Acute tubular necrosis and renal failure from high plasma osmolarity (320 mOsm) and excretion of unmetabolized mannitol in the urine have been observed in adults.[96,97]

Furosemide and ethacrynic acid (loop diuretics) cause diuresis, decreased CSF production, less cerebral edema, and improved intracellular water transport. The recommended dose of these diuretics is 0.2-to-0.3mg/kg when administered with mannitol or 0.5-to-1mg/kg when administered alone. Furosemide prevents mannitol-induced rebound cerebral edema.[98]

Steroids

Corticosteroids are used in a variety of diseases in children, particularly neurological conditions, such as brain tumors. Steroids inhibit tumor-induced angiogenesis, cerebral edema, and CSF production. They also decrease the release of free radicals.

Temperature Homeostasis

Anesthesia-induced alterations of skin blood flow and exposure to a cold operating room, cause perioperative hypothermia in pediatric patients. Neonates and infants are especially prone to hypothermia due to their large surface-to-volume ratio. Hypothermia has effects on the pharmacokinetics and pharmacodynamics of many anesthetic agents. It prolongs the effects of volatile agents, intravenous anesthetics, and neuromuscular relaxants. Hypothermia increases perioperative blood loss, inhibits activation and aggregation of platelets, increases release of heparin-like anticoagulant substances, inhibits the synthesis of coagulation factors, and alters fibrinolytic activity. All these changes increase the need for blood transfusion. Other effects of hypothermia include depression of myocardial function, impaired hypoxic pulmonary vasoconstriction, increases risk of hypoglycemia, and increased infections. Hypothermia also increases oxygen consumption and pain. The patient's temperature should be monitored during surgery and measures should be taken to keep the temperature within normal limits. During the induction of anesthesia and placement of intravenous and invasive monitoring lines, children should be covered with cotton blankets, lie on a thermal mattress (if available), have a convective air system over them when possible, be placed under a radiant heat lamp that is no closer than three feet from the surface of the patient's skin (to avoid burns), and have warm intravenous fluids administered.[99-103]

Air Embolism

Air embolism occurs commonly during intracranial procedures. This is because air enters the central circulation through open veins in the scalp and skull during spontaneous breathing when the pressure inside the open veins is lower than atmospheric pressure. Placing the surgical site above the level of the heart (e.g., during a semi-sitting craniotomy for posterior fossa surgery) or having a low CVP increases the pressure gradient between the surgical sit and the heart. This facilitates venous air embolism (VAE). The problem is made worse by the fact that the bony venous sinuses in the skull are held open by their attachment to bone and cannot collapse. Other possible sites for air entry include the bridging veins and epidural venous sinuses. When air enters the central circulation, blood flow in the right ventricle and pulmonary artery is blocked. This causes sudden pulmonary hypertension, decreased pulmonary artery blood flow, decreased left ventricular preload, cardiovascular collapse, and in some cases cardiac arrest. The severity of the patient's symptoms depends on the speed with which air enters the central circulation and on the volume of gas in the heart and vessels. Intracardiac shunts (e.g., a patent foramen ovale) often allow passage of air from the venous to the arterial circulation, causing paradoxical air embolism. As little as 0.1cc of air in a coronary artery can be fatal to a young child. The incidence of air embolism during craniosysnostosis surgery is high (>80%), even when the operating table is kept completely flat. [104] An air embolus is more likely if there is hemorrhaging and the CVP is low. A low CVP increases the pressure gradient between veins in the head and the heart.

Echocardiography (either transthorasic or transesophageal), precordial Doppler, a pulmonary artery catheter, and capnography can be used to detect air emboli. When used, the precordial Doppler is placed in the right 4th or 5th interspace near the sternum. This device is easy to use, non-invasive, and inexpensive. Proper positioning of a Doppler is confirmed by listening for the characteristic sound of air in the venous circulation with rapid injection of a few milliliters of normal saline, which always contains micro bubbles of air.[105] Although transesophageal or transthoracic echocardiography detects small emboli more effectively, these devices are not easy to use, the data from them is not easy to interpret during neurosurgical procedures in children, and they are very expensive. *Capnography* can detect air emboli, but its sensitivity is low. ECG changes, alteration in the heart rate, sudden decreases in blood pressure, and sudden increases in CVP may also suggest the presence of air embolism.[106]

Measures to prevent air embolism include minimizing pressure gradients between the surgical site and the heart, preventing hypovolemia, and using positive pressure ventilation throughout surgery. Treatment of air emboli includes: 1) informing the surgeon immediately of the problem and having her/him flood the surgical field with saline to prevent further air entry. All exposed bony surfaces are covered with bone wax; 2) administering 100% oxygen, 3) discontinuing inhalational anesthetics to limit further cardiovascular depression; 4) placing the child in Trendelenburg position (i.e., put the surgical site below the heart), and 5) placing the child in the left lateral position to increase venous return of blood; Doing these things favors pulmonary perfusion and mobilization of air in the apex of the right ventricle, 6) withdrawing as much air as possible through the central venous catheter (if there is one), and 7) preventing further air entry by administering bolus of fluid and vasopressors to increase intravascular volume and blood pressure. If a child has a cardiac arrest in the prone position, the anesthetist should immediately begin cardiac compressions from the back and continue doing so until the patient can be turned to the supine position.[107]

Emergence From Anesthesia

At end of surgery, tracheal extubation can be done in either the operating room or the intensive care unit, but where ever it is performed every effort should be made to prevent coughing, staining, systemic hypertension, and hypercarbia. This could potentially increase ICP, arterial blood pressure, and reduce venous return from the CNS. The tracheal tube is only removed when patients have protective airway reflexes, adequate spontaneous breathing, and are fully alert (to protect the airway). However, if these criteria are not met in the operating room or if there is a high likelihood for postoperative intracranial hypertension, the tracheal tube should be left in place and the patient taken to the intensive care unit for monitoring and support of ventilation.

The incidence of nausea and vomiting in neurosurgery patients is high, due to the irritant effect of blood on the ventricles and to the use of opioids. Antiemetic drugs should be given before the

end of surgery to prevent vomiting. The effects of muscle relaxants should be reversed at the end of surgery because their residual effect will decrease ventilation, cause hypercapnia, and interfere with the postoperative neurological assessment of the patient.

Pain after a craniotomy is usually not severe and can be managed with oral or intravenous acetaminophen in most cases. If the pain is moderate or severe, titrated doses of opioids are recommended. Anti-inflammatory drugs, such as dexmedtomidine, can be used to reduce pain if the drug chosen has no significant effects on platelet function. However, dexmedtomidine, by its sedative effects, may make it difficult to assess the patient's neurologic status. Dexmedtomidine is often used during neurosurgery to provide sedation and analgesia because it does not cause respiratory depression. Giving dexmedtomidine before the end of surgery reduces the need for postoperative opioids.[108]

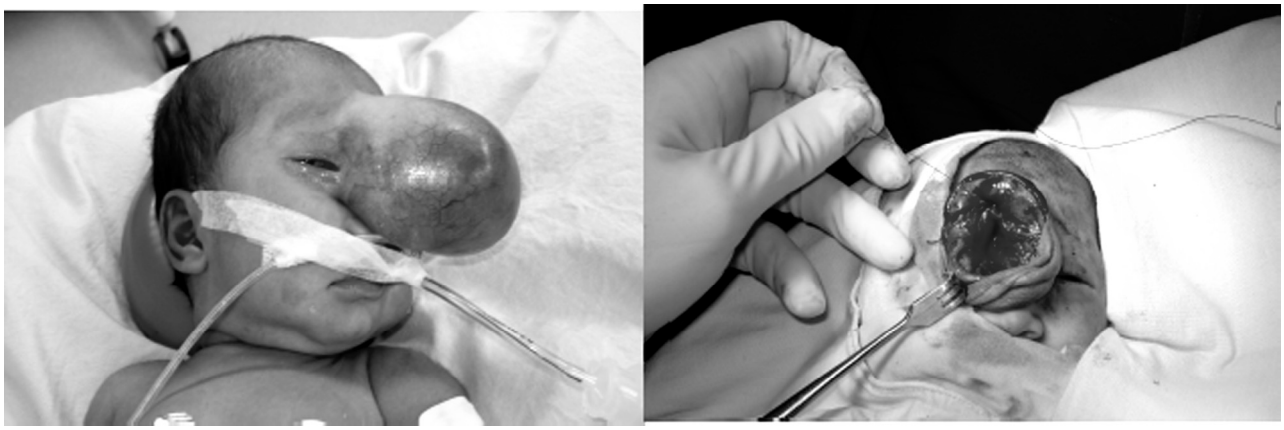
Anesthetic Considerations for Specific Neurosurgical Procedures

Neural Tube Defects

If the neural tube fails to close during fetal development, a broad range of malformations, from spina bifida to anencephaly, occur. Failure of the vertebral arches to fuse during fetal development causes spina bifida. When only a dural sac filled with cerebral spinal fluid bulges through the bony defect, it is called a meningocele. If the bulging sac also contains neural tissue, it is called a myelomeningocele. In children, these two defects are the two most common conditions in the lumbosacral area that require neurosurgical care. While both lesions can cause neurologic symptoms, meningoceles cause fewer and less severe neurologic symptoms. Paralysis, when present, is often mild and incomplete. Myelomeningoceles, on the other hand, often have severe bladder dysfunction requiring frequent bladder catheterization. They also may have severe bowel dysfunction and complete paralysis of their lower extremities. Meningoceles and meylomeningoceles require surgical correction within 24 hours of birth to minimize the risk of dural sac rupture and infection. Most patients who have a myelomeningocele have hydrocephalus 3-5 days after closure of the myelomeningocele. When this occurs, they require a ventriculoperitoneal shunt to relieve pressure in the brain. Patients with hydrocephalus may also have an Arnold Chiari malformation type II of the skull. Some myelomeningoceles are now being corrected in utero, which has the potential for reducing the incidence of hydrocephalus and the need for ventriculoperitoneal shunting.[109]

Encephaloceles are neural tube defects that usually occur on the head but can occur anywhere along the neural tract. These defects are sometimes associated with head enlargement, making ventilation with a facemask and tracheal intubation difficult and unpredictable. (**Figures 10-3A-10-3B**)

Figure 10-3 A: Newborn with Frontal Encephalocele Figure 10-3 B: Surgical Resection.



Anesthetic considerations for neural tube defects include:

1. Having a thorough knowledge of the general principles of managing children for anesthesia in the neonatal period, especially their fluid requirements, metabolic flux of glucose, temperature management, and the differences in their cardiovascular, respiratory, and renal systems is very important. A complete preoperative evaluation and understanding of coexisting congenital defects is mandatory. Infants are at high risk of perioperative morbidity and mortality if they were born prematurely or have congenital anomalies besides the neural tube defect. Their risk is also increased by the immaturity of most of their systems and by the narrow margin for error for administration and dilution of medications, difficulty with airway management, and intravascular access.[110]
2. *Positioning:* In most cases induction of anesthesia is accomplished in the supine position after the child is positioned on a u-bolster to prevent applying direct pressure to the myelomeningocele sac.
3. *Latex allergy:* Patients with a history of neural tube defects are at high risk for developing latex allergy, because of they require multiple surgical procedures, and frequent bladder catheterization with rubber catheters. Care should be taken to ensure a latex free environment in the operating room.[111]

Children presenting for repair of a myelomeningocele rarely have increased ICP because the dural sac is very compliant or ruptured. However, if the child has an Arnold-Chiari malformation, the intracranial cavity may be isolated from the spinal canal preventing drainage of CSF from the brain. When this occurs, patients have increased ICP. Deficits that occur at about the T-4 level often cause paraplegia.

In some infants with meylomeningocele, evaporation of water from the myelomeningocele sac is high. For this reason, the baby's hydration status must be determined and abnormalities corrected before anesthesia is induced. Special care should be taken to ensure that blood is

available in the operating room before beginning surgery. With large lesions, there can be significant blood loss when the surgeons mobilize tissues to close the defect. When the patient is placed in the prone position, rolls are placed under the chest and hips to allow adequate excursions of the chest and abdomen during breathing and to reduce pressure on the abdomen that might compress the inferior vena cava and epidural veins and increase bleeding.

A *Chiari II malformation* is a boney abnormality of the posterior fossa and upper cervical spine that allows displacement of the cerebellar vermix, fourth ventricle, and brainstem through the foramen magnum. Children with this lesion can have vocal cord paralysis, stridor, respiratory distress, apnea, swallowing disorders, and aspiration of secretions. A tracheostomy and gastrostomy should be considered earlier rather than later.[112] Chiari I malformations more commonly occur in healthy children without myelodysplasia. With this defect, there is caudal displacement of the cerebellar tonsils below the foramen magnum, but the clinical manifestations are usually mild and consist of headache and neck pain. Surgical treatment of a Chiari I malformation includes a decompressive craniectomy and a suboccipital laminectomy. Patients with a *tethered spinal cord* have tissue attachments that prevent the spinal cord from moving in the narrow spinal cannel as the child grows. This condition is diagnosed clinically, or radiologically. The most common signs and symptoms of a tethered spinal cord are muscle weakness of lower limbs, sensory disturbance of the legs, bowel or bladder dysfunction, back pain, and gait disorders.[113] Intraoperative release of a tethered cord requires neurophysiological monitoring when available. Direct nerve stimulation and an EMG aid the surgeon in differentiating neural tissue from other tissue. Observing the anal sphincter response or detrusor function of the bladder to stimulation improves the likelihood of preserving these functions. Muscle relaxants should not be used during tethered cord surgery because they block responses to nerve stimulation. This may cause the surgeon to inadvertently transect some nerves.

Hydrocephalus

Hydrocephalus (water on the brain) is caused by an imbalance between production and absorption of cerebrospinal fluid. This imbalance causes the ICP to rise. Hydrocephalus may be congenital or acquired. **(Table 10-5)** Although some reports suggest that incidence of hydrocephalus in children has decreased in some developed countries, others reports suggest it has increased due to survival of more premature babies. Neonatal infections and neural tube defects are common in some developing countries, which has significantly increased the number of patients with hydrocephalus worldwide. In East Africa there are more than 6000 new cases of hydrocephalus each year. In many low income countries, hydrocephalus is a major cause of morbidity and mortality.[114] Hydrocephalus causes pathological changes in brain morphology and maturation, in the microstructure of the brain, in cerebral blood flow, in brain biochemistry, and in metabolism. **(Figure 10-4)** Although surgical treatment does not always reverse the damage, untreated hydrocephalus leads to progressive CNS damage and death.

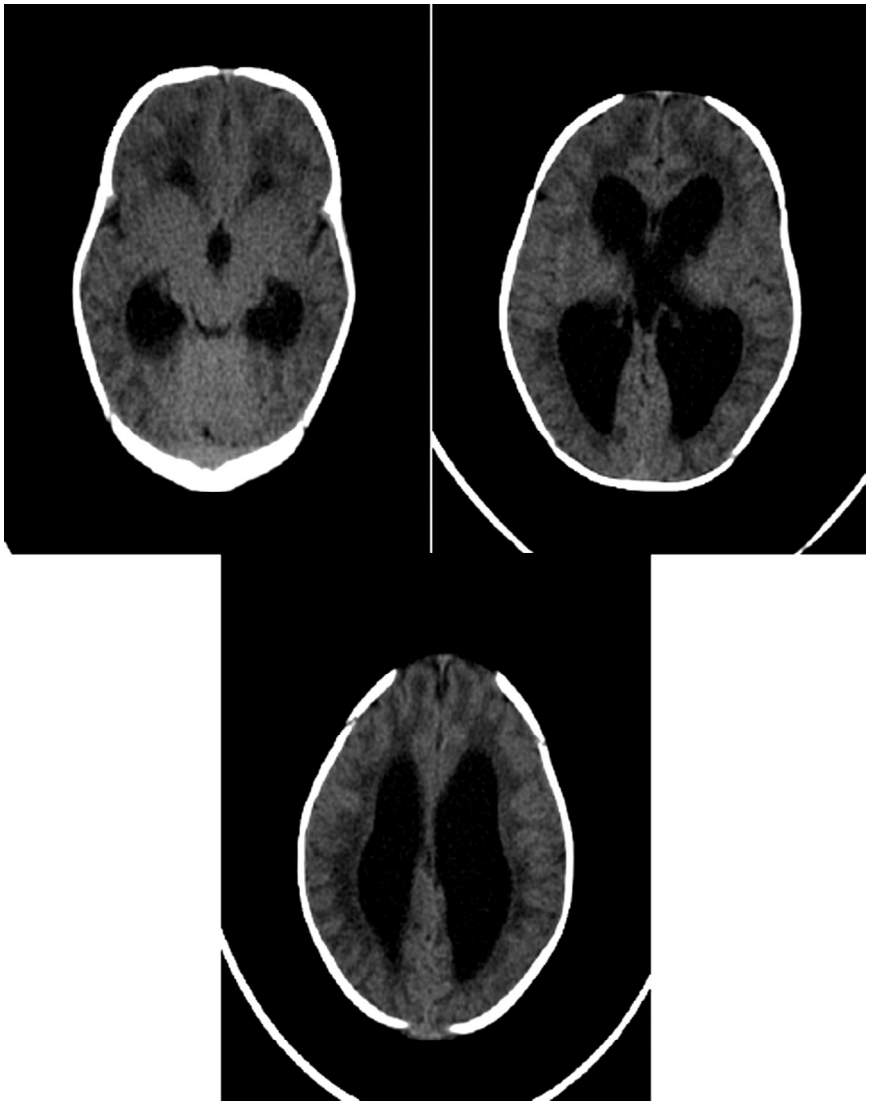
TABLE 10-5: Causes of Hydrocephalus

CONGENITAL CAUSES

Myelomeningocele
Stenosis of the aqueduct of sylvius
Dandy-Walker syndrome
Mucopolysaccharidoses
X-linked hydrocephalus
In-utero intraventricular hemorraghe

ACQUIRED CAUSES
Infections
Intraventricular hemorrhage of prematurity
Space occupying intracerebral cysts
Tumors

Figure 10-4: Acute Hydrocephalus:



Axial CT showing significant lateral and third ventricles enlargement due to transependimal edema secondary to aqueductal stenosis.

The signs and symptoms of hydrocephalus are the result of increased ICP. Increase in ICP during acute hydrocephalus can be fatal unless someone quickly removes some of the excess CSF. The causes of acute hydrocephalus include sudden obstruction of the ventricular system (ventricular hemorrhage in premature infants, hemorrhage into a tumor, or displacement of a third ventricle cyst). In the absence of early treatment, ICP rapidly increases, causing brain herniation and cardiorespiratory arrest. Chronic hydrocephalus occurs with aqueductal stenosis, meningitis, and intracranial tumors. The clinical manifestations of chronic hydrocephalus develop more slowly and include irritability, headache, decreased school performance, confusion, and lethargy. In neonates, widening of the sutures and an increase in head circumference accommodate a slow

progressive increase in ICP up to a point. The symptoms of chronic hydrocephalus in infants may be nonspecific and include include irritability, decreased appetite, and vomiting. Treatment of hydrocephalus is surgical and consists of diverting CSF from the cerebral ventricles to the abdomen (ventriculoperitoneal shunt) or elsewhere. If a ventriculoperitoneal shunt cannot be place, a ventricular-atrial shunt (lateral cerebral ventricle to jugular vein) or ventricular-pleural shunt (lateral cerebral ventricle to the pleural space) can be inserted.[115] Although surgery for hydrocephalus is one of the most common neurosurgical procedures, it is occasionally associated with several catastrophic complications, including bleeding from accidental rupture of a venous sinus, a pneumothorax or hemothorax, injury to neck vessels, and injury to abdominal organs (intestinal perforation, liver laceration). During placement of a ventriculo-atrial shunt, air embolism, ventricular arrhythmias, and vessel injury can occur. A tension pneumothorax is more common with ventriculo-pleural shunts.

Endoscopic ventriculostomy is used in selected cases to create a shunt. This involves inserting a flexible endoscope into the brain and advancing it to the lateral or third ventricle. A hole is then made in the floor of the ventricle to allow CSF to drain into the basal cisterns and subarachnoid space. When the procedure is successful, there is no need for one of the other shunts. Complications of endoscopic ventriculostomy include basilar artery rupture and hypovolemic shock, which requires and an urgent craniotomy. Severe bradycardia occasionally occurs during manipulation of the third ventricle and when cold saline is injected into the third ventricle.[116]

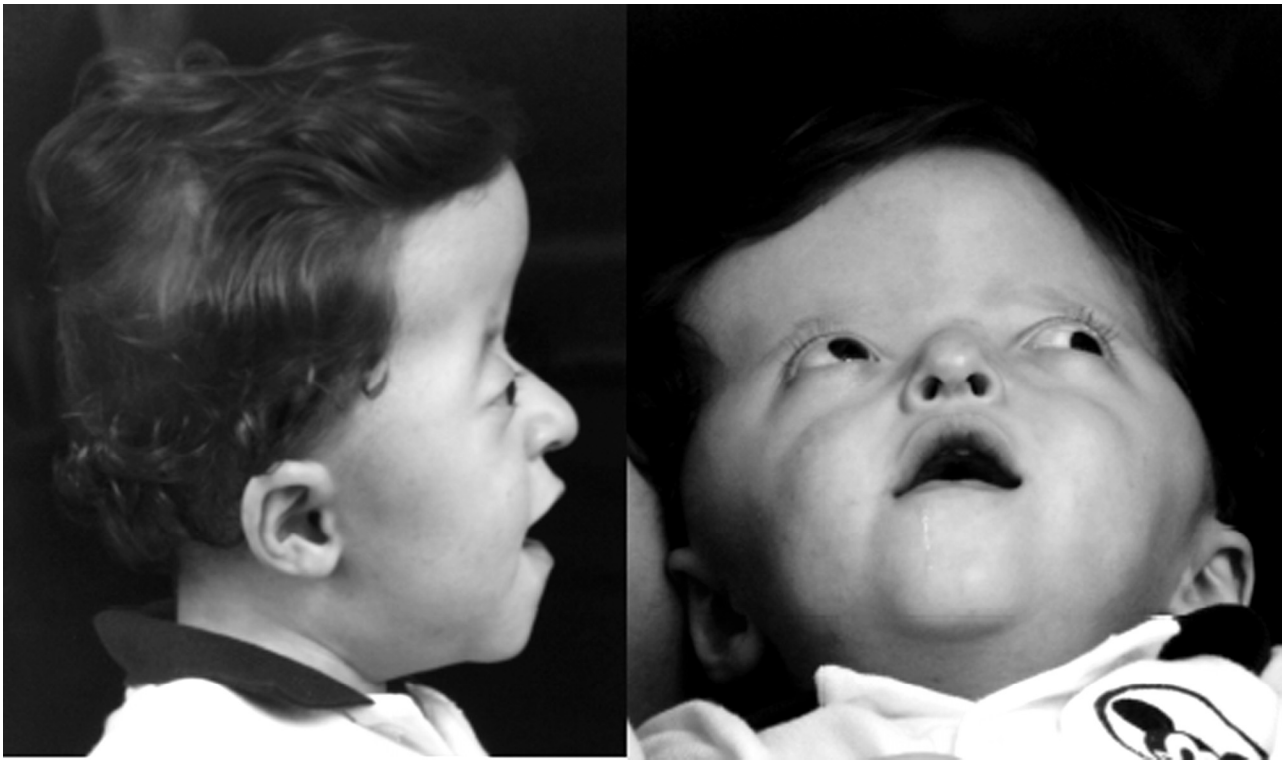
The perioperative management of patients who have hydrocephalus depends on the presence or absence of intracranial hypertension, the cause of the hydrocephalus, and the presence of associated comorbidities. Preoperative sedation should be avoided in patients who have hydrocephalus because it increases the risk of respiratory depression, hypercapnia, and elevated ICPs. Patients with hydrocephalus and history of myelomeningocele must be considered to have latex allergy and should not be exposed to additional latex. Intraoperative monitoring should include non-invasive arterial blood pressure, SaO_2, ECG, $ETCO_2$, body temperature, and continuous measurements of end-tidal inhaled anesthetic concentrations, if possible. If infants and young children have no evidence of increased ICP and have intravenous access, anesthesia can be induced with either inhaled or intravenous anesthetics. But positive pressure ventilation should be instituted as soon possible after giving the drugs used for induction of anesthesia to prevent hypercarbia, cerebral vasodilation, and increased ICP. Induction of anesthesia with inhaled agents should probably be avoided if the patient has intracranial hypertension, because inhaled agents may worsen ICP and increase vomiting and aspiration. Intravenous induction of anesthesia with propofol 3-4mg/kg or thiopental sodium 5-6mg/kg is probably safer for patients with intracranial hypertension. Opioids (fentanyl or remifentanyl) and a muscle relaxant should also be considered for rapid sequence induction of anesthesia and tracheal intubation. Maintenance of anesthesia is done with an opioid (fentanyl or remifentanyl) and sevoflurane or isoflurane. The patient is mechanically ventilated to control CO_2. Postoperative management of

patients of patients with hydrocephalus depends on her/his neurological status and the presence of preexisting co-morbidities.[113,117]

Craniosynostosis

Craniosynostosis is a developmental disorder of the skull that prematurely closes one or more cranial sutures. It occurs in approximately 1 in 2000 live births. In about 80% of cases, it is an isolated anomaly. In the other 20%, it is part of a syndrome or genetic disorder (Crouzon syndrome, Pfeiffer syndrome, Apert syndrome, Muenke, Acrocephalosyndactylia type III). These syndromes are sometimes associated with other craniofacial malformations and extracranial abnormalities. **(Figure 10-5)**

Figure 10-5: Pfeiffer Syndrome.



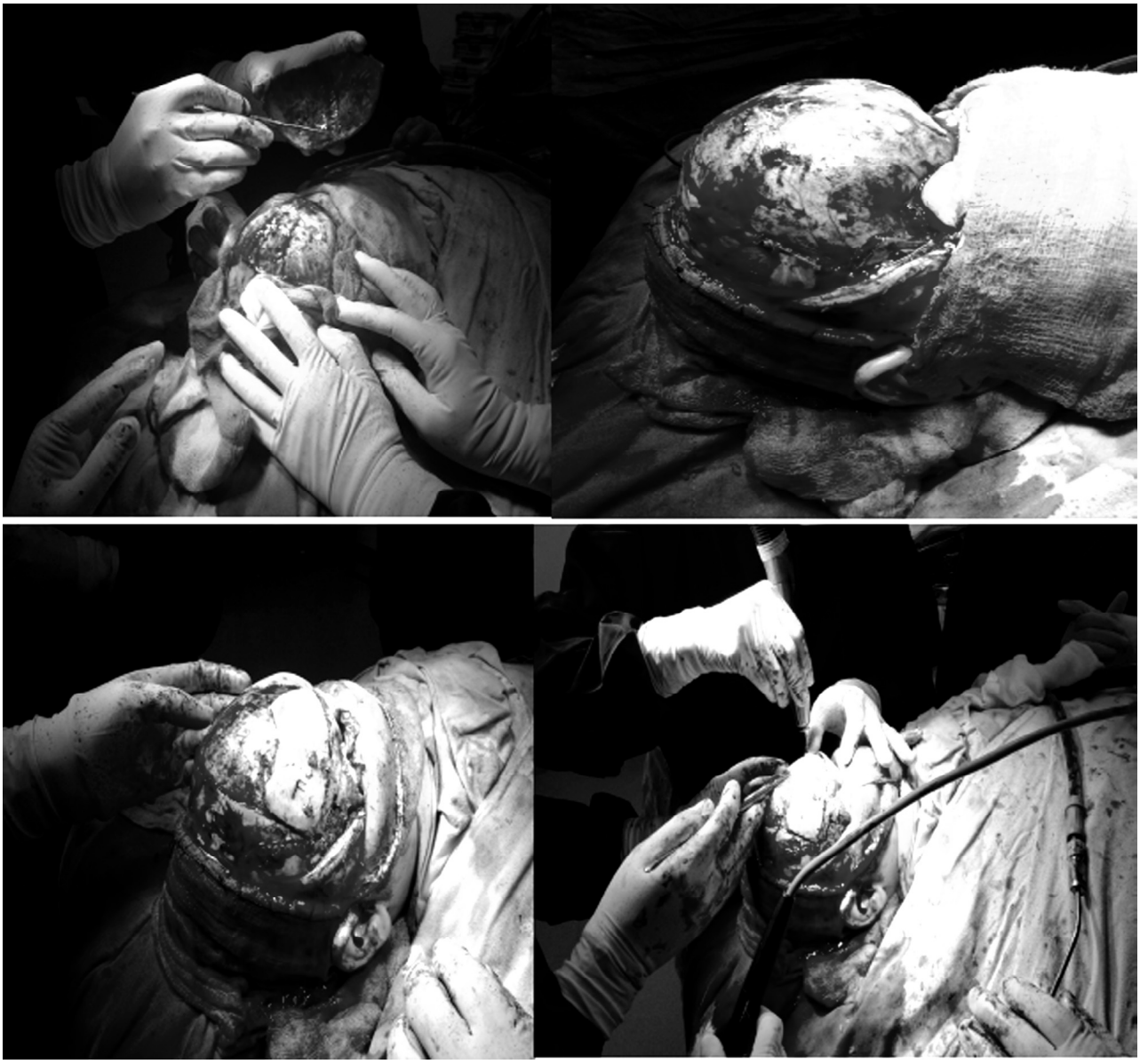
Diagnosis of craniosynostosis is commonly based on the phenotype of the skull deformation. Where available, CT and 3-D reconstruction are used to locate the specific suture that requires repair and to establish a surgical plan. Untreated craniosynostosis can sometimes lead to intracranial hypertension and impaired intellectual and neurological development. Patients with a syndrome and craniosynostosis often have multiple closed sutures and increased ICP. The best cosmetic and neurodevelopmental outcomes occur when surgery to correct these lesions is done during the first year of life.

Laboratory tests for this type of surgery usually include a complete blood count (hemoglobin, hematocrit and platelet count), coagulation tests (PT, PTT, INR), and the cross matching of sufficient blood for transfusion to treat perioperative bleeding if it occurs. The airway should be carefully evaluated preoperatively because some of these children have difficult airway. Each syndrome has its own set of potential complications that must be considered. For instance, children with Apert syndrome have midface hypoplasia and severe proptosis that can make facemask ventilation difficult.[118,119]

When providing anesthesia for craniosynostosis, the anesthetist's main concerns for surgery include the potential for the patient having a difficult airway, massive bleeding, and air embolism. These problems significantly increase the morbidity and mortality of the procedure.[120] The main intraoperative challenge in these patients is hemorrhage, particularly if the patient is less than six months old. Blood loss varies with the number of sutures involved and the surgical technique used (open vs. endoscopic craniectomy).

During scalp dissection a patient can loose 30% of her/his blood volume. Blood loss can be even greater when the periostium is elevated, when osteotomies are done, or when venous sinuses are damaged. Massive bleeding can occur within seconds and may be very difficulty to control. Some studies suggest starting to transfuse blood early during craniosynostosis surgery, since transfusion is almost always required for maintenance of intravascular volume and hemodynamics.[121] **(Figure 10-6)**

Figure 10-6: Severe Perioperative Bleeding in Craniosynostosis Surgery.



This figure demonstrates that it is virtually impossible to measure reliably the amount of blood loss during the procedure.

Perioperative cardiac arrest in children is usually caused by massive hemorrhage and hypovolemia. The reasons for the hypovolemia include underestimation of intraoperative blood loss, inadequate venous access, inadequate monitoring, complications of blood transfusion, complications of massive transfusion (hyperkalemia, hypocalcemia), and coagulopathy.[122] Venous air embolism (VAE) occurs in as many as 83% of patients undergoing craniosynostosis surgery, although most of these episodes have no hemodynamic consequences. VAE also occurs 8% of patients undergoing endoscopic craniosynostosis repairs[123] The possibility of raised ICP should

be confirmed by CT scan, ophthalmologic examination, or by nonspecific clinical findings, such as headache in older children. Forty-seven percent of patients with syndromic craniosynostosis and multiple closed sutures have increased ICPs; the incidence is only 14% when a single suture is stenotic.

The risk of hypothermia during craniosynostosis surgery is high because the large heads of these patients are exposed to room temperatures, cerebral perfusion is high, cerebral vessels are dilated by the anesthetics, and the patients receive large volumes of cool intravenous fluids. Measures must to be taken to prevent hypothermia. The choice of drugs used for induction of anesthesia depends on the child's condition and on her/his airway. Usually an inhaled induction with sevoflurane is appropriate if ICP is not elevated. Many anesthetists induce anesthesia with a volatile anesthetic and enough controlled ventilation to maintain a CO_2 of approximately 30mmHg. Once the anesthetist confirms that he/she can maintain CO_2 in desired range with bag-and-mask ventilation, intravenous medications are administered to facilitate tracheal intubation. The route of tracheal intubation (oral or nasal) is based on the child's position during surgery. However, the authors recommend nasotracheal intubation when possible because it reduces the chance of inadvertent tracheal extubation, especially when the tracheal tube is secured with wires or sutures.

Anesthesia is maintained throughout the procedure with an inhaled agent and oxygen. A dexmedetomidine infusion, when available, helps maintain a stable cardiovascular system. All children should have at least two large peripheral IVs for the surgery. Blood products should be immediately available during heavy blood loss portions of the procedure. Many centers routinely insert central venous catheters for complex reconstruction of the cranial vault. An arterial line is required. A precordial Doppler is used to detect venous air embolism. A urinary bladder catheter allows continuous drainage of urine.

One of the biggest challenges during craniofacial surgery is trying to accurately identifying the amount of blood being lost. Doing so with precision is often impossible because much of the lost blood is hidden under the surgical drapes. Throughout surgery the surgical field is irrigated with large volumes normal saline, which mixes with blood collected from the field, making it difficult to estimate blood loss. Weighing all sponges helps. Each gram increase in sponge weight equals 1ml of blood. Monitoring the morphology of the arterial and venous pressure waves and the trends in arterial blood pressure (up or down) provides useful information about the patient's volume status (**See Chapter 2**). The trend of central venous pressure up or down is more useful than any particular number. As indicated above, urine output, and serial measurements of hemoglobin concentration and arterial blood gases are necessary. Constant observation of the surgical field is still the best way to estimate blood loss.

A decrease in hemoglobin concentration is usually caused by hemorrhage and/or the administration of large volumes of crystalloid, colloid or blood products (dilutional coagulopathy).

Metabolic acidosis, thrombocytopenia, hypocalcemia (from binding of calcium to citrate), and hypothermia also occur. Hyperkalemia, arrhythmias, and even death may sometimes occur when more than two-week old stored red blood cells are administered rapidly. Current recommendations are to administer RBCs that are less than one week old, to wash RBCs before administering them if they are more than two weeks old, and infuse the cells at 1.5ml/kg/min (which is not always possible if blood loss is occurring rapidly).[124-126]

Before beginning surgery, blood products, especially red blood cells and fresh frozen plasma (FFP), should be in the operating room. Much of the blood loss during craniosynostosis surgery occurs with the initial dissection and reflection of the scalp and periosteum. Replacing blood loss with crystalloid or RBCs without FFP causes dilution coagulopathy. Dilutional coagulopathy can be avoided by using a 1:1 mixture of RBCs and FFP (1 unit of RBC and 1 unit of FFP) for transfusion. Whenever possible, a single donor should provide all blood that will be transfused to a given child.[127,128] Fibrinogen levels decreases rapidly during massive bleeding. Administering concentrated fibrinogen 30-50mg/kg or cryoprecipitate 1unit/10kg should be considered when fibrinogen levels are low.[129]

Reducing blood loss during craniosynostosis surgery may improve patient safety and decrease morbidity, mortality, and hospital costs. For these reasons blood saving techniques should be used when possible. Patients found to be anemic several weeks before surgery should be given erythropoietin and an iron supplement to increase their hemoglobin concentrations.[130-132]

Meticulous surgical technique reduces bleeding. Infiltrating the scalp with epinephrine before making the skin incision also reduces blood loss. Since acidosis and hypothermia increase bleeding, efforts should be made to keep the patient's acid-base status and temperature normal. Using restrictive transfusion thresholds (transfusing when the hemoglobin concentration is 7g/dl) reduces the need for transfusion by 44%. However, this is not appropriate if the child is hemodynamic unstable, bleeding profusely, or has severe hypoxia and/or cyanotic congenital heart disease.[133,134] Each institution must decide the hemoglobin concentration at which transfusion will be initiated during these procedures.[135]

Tranexamic acid administration during craniosynostosis surgery decreases perioperative bleeding and reduces the need for blood transfusion.[136,138] The recommended dose of tranexamic acid is 20mg/kg as a bolus followed by an infusion of 10mg/kg/h.[139] This protocol decreased the need for blood transfusion, the number of side effects, and mortality by one third.[140]

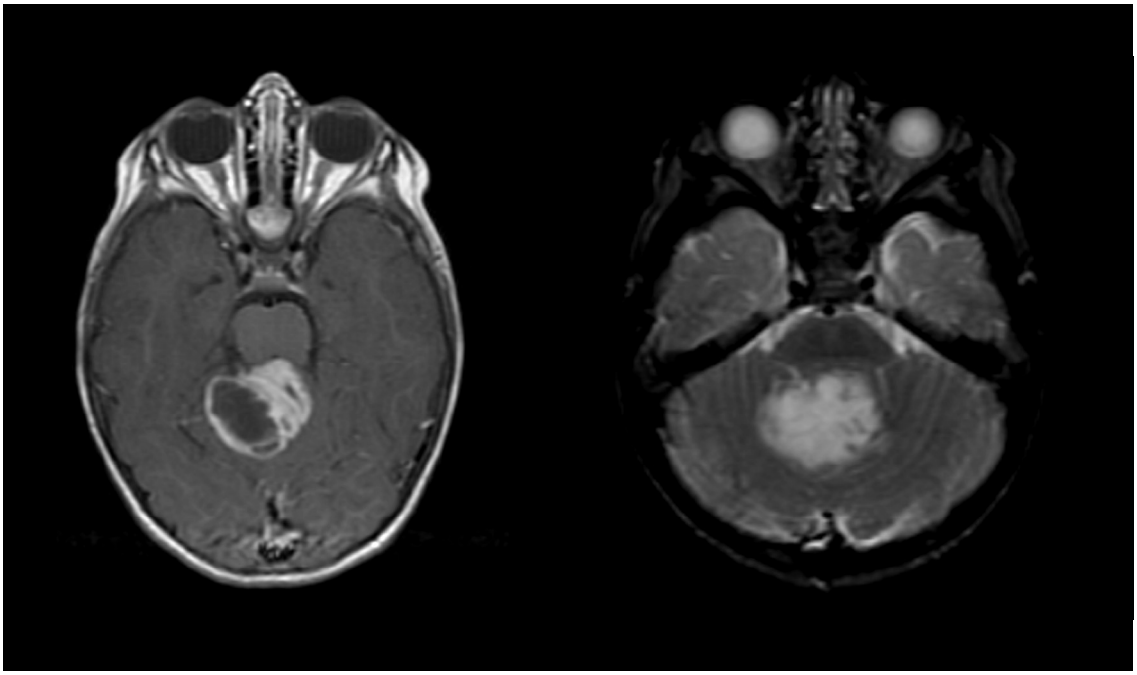
Use of a Cell Saver device has increased in recent years because it decreases the number and volume of allograph blood transfusions required, particularly in children given preoperative erythropoietin. However, the volume of blood recovered must be sufficient to warrant its use.

Children undergoing craniosynostosis repair require intensive care postoperatively. The decision to extubate the tracheal or provide postoperative mechanical ventilation is based on the extent of surgery, the amount of fluid replacement, and the amount of facial and airway edema (particularly in patients who were in the prone position). Postoperative mechanical ventilation may also be required. Patients with Pfiffer syndrome (50%), Crouzon syndrome, or Apert syndroms have a high risk of postoperative apnea if they had preoperative obstructive sleep apnea. Postoperative pain is usually not severe following craniosynsotosis repair and can be managed with intermittent doses of opioids or with oral or intravenous acetaminophen.[141-145]

Cerebral Tumors

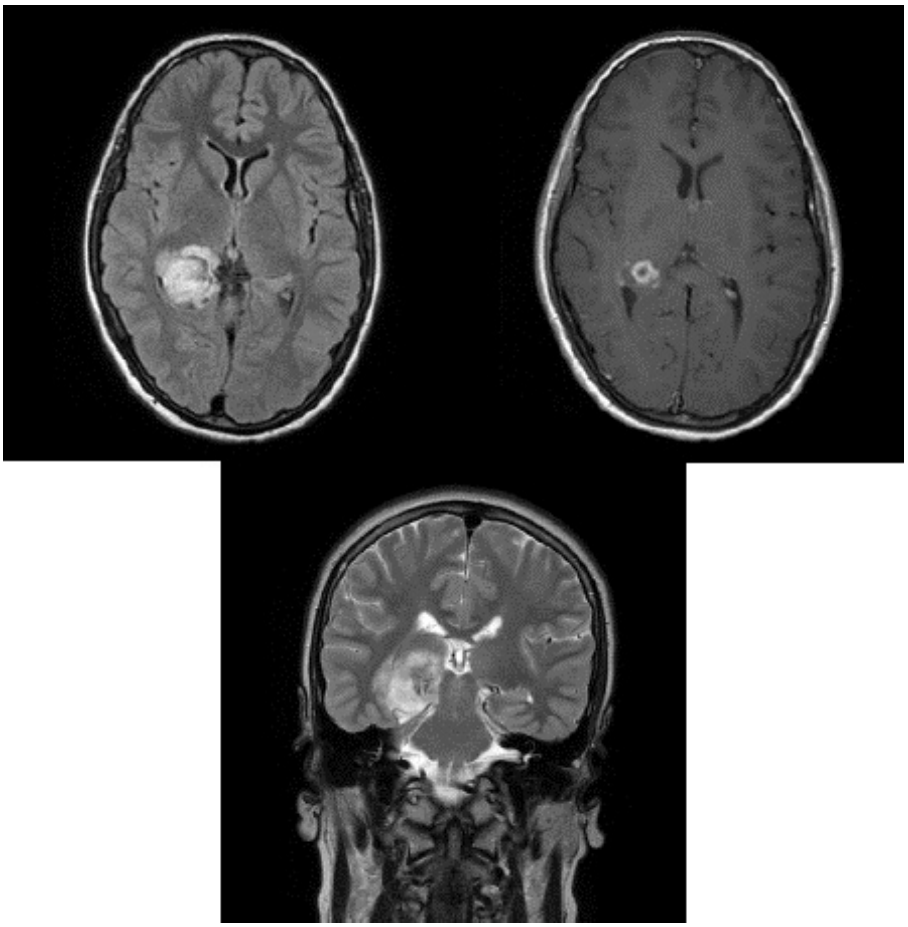
After trauma, cancer is the second most common cause of death in children under 15 years of age. Twenty percent of all pediatric tumors occur in the CNS. Improved diagnostic procedures make it easier to detect CNS tumors.[146] Pediatric brain tumors are conveniently divided into supratentorial and infratentorial. About half of brain tumors occur in each of the two compartments. Depending on tumor growth rate and its anatomical location, the symptoms of supratentorial tumors include neurological deficits, seizures, and/or increased ICP. Twenty-five percent of intracranial tumors (astrocytomas, oligodendrogliomas, ependymomas, and glioblastomas) are located in the cerebral hemispheres. **(Figures 10-7A-10-7B)**

Figure 10-7A: Pylocytic Infratentorial Astrocytoma.



Axial T1-weighted post contrast and axial T2-weighted images showing enhance cerebellar mass with cystic components compressing the fourth ventricle. Enhancement is heterogeneous and includes portion of the wall of the cyst.

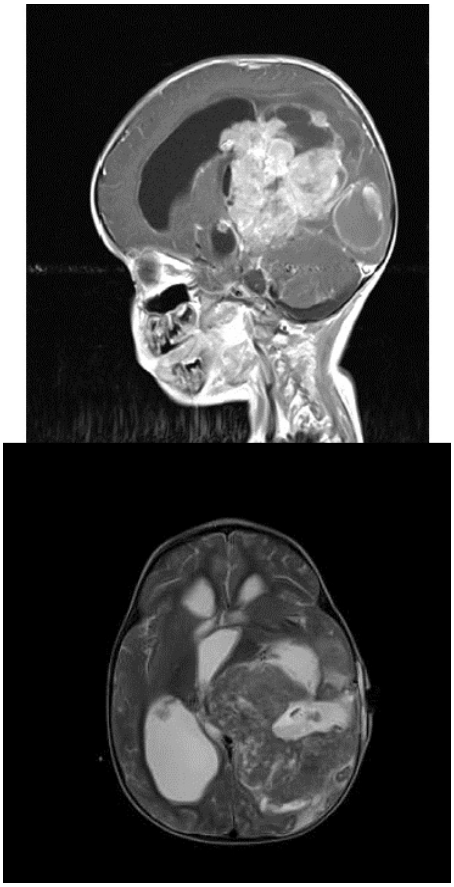
Figure 10-7B: Supratentorial Oligoastrocytoma.



Axial FLAIR, axial T1-weighted post contrast and coronal T2-weighted images showing a heterogeneous cortical and subcortical mass involving the right thalamus and medial temporal lobe.

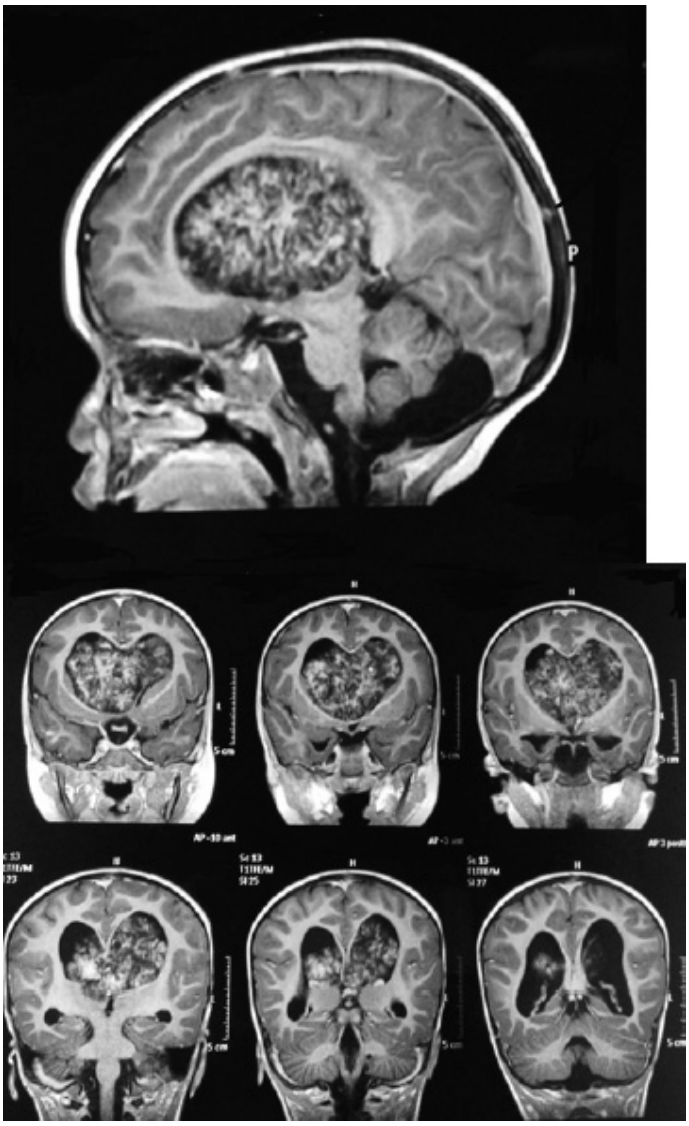
Choroid complexes papillomas are rare and usually occur in <3 year-old children. **(Figures 10-8A-10-8B)** They arise from lateral ventricle choroid plexus. Early hydrocephalus is common with these tumors because they increase production and decrease reabsorption of CSF. They also obstruct flow of CSF from the brain to the spinal cord. Massive intraoperative bleeding is more likely with highly vascular tumors.[147]

Figure 10-8 A: Choroid Plexus Tumor



This 2 years old girl with a large choroid plexus tumor had a gait disorder, irritability and vomiting.

Figure 10-8 B: Intraventricular Choroid Plexus Tumor

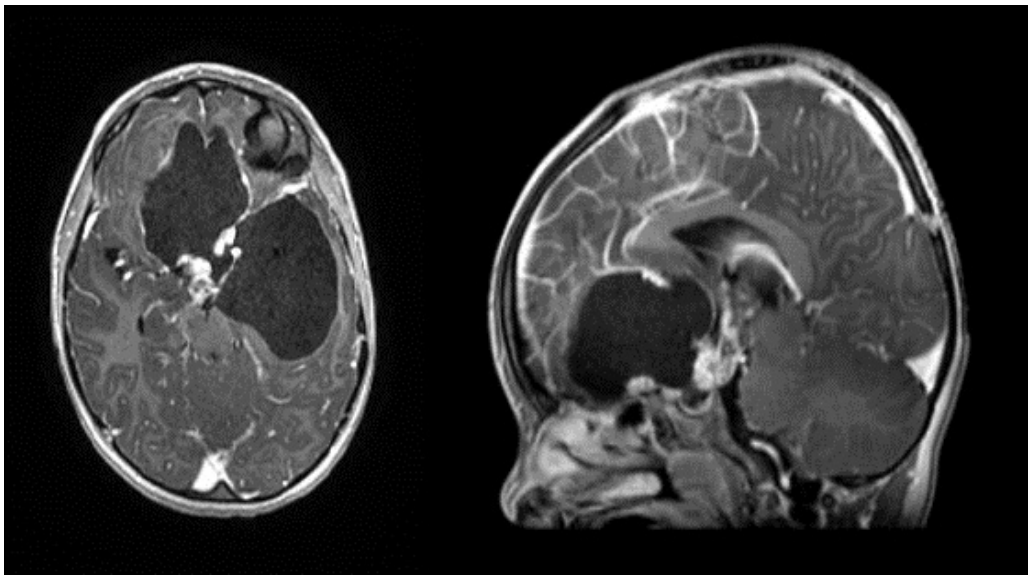


This is the same patient as in Figure10- 8 A.

General anesthetic considerations for resection of supratentorial tumors include increased ICP, electrolyte imbalances, seizures, the effects of anticonvulsants on anesthetic drugs, and the risk of acute and chronic aspiration with abnormal airway reflexes. Due to the high risk of bleeding, blood should be immediately available in the operating room prior to surgery. Two large IVs and invasive hemodynamic monitoring are mandatory. A bladder catheter should be placed when the surgery will be prolonged and when osmotic diuretics or large amounts of IV fluids will be given during surgery. Tumor resection in neurologically sensitive areas of the brain is best done with intraoperative neurological monitoring. Patients older than seven years of age can be considered for awake craniotomy if other forms of intraoperative monitoring are unavailable.

Sellar and suprasellar tumors include craniopharyngiomas, germ cell tumors, hypothalamic tumors, pituitary adenomas, and optic gliomas. Like all patients requiring neurosurgery, the patient must be completely evaluated before surgery, particularly if there are serious endocrine imbalances. Craniopharyngiomas are the commonest parasellar tumors in children and adolescents and are frequently associated with pituitary and hypothalamic dysfunction. **(Figure 10-9)** These children may have endocrine dysfunction, hydrocephalus, and visual disturbances due to compression of the optic chiasm by the tumor. The surgical approach is usually by frontal craniotomy or by the transphenoidal approach.

Figure 10-9: Craniopharyngioma.



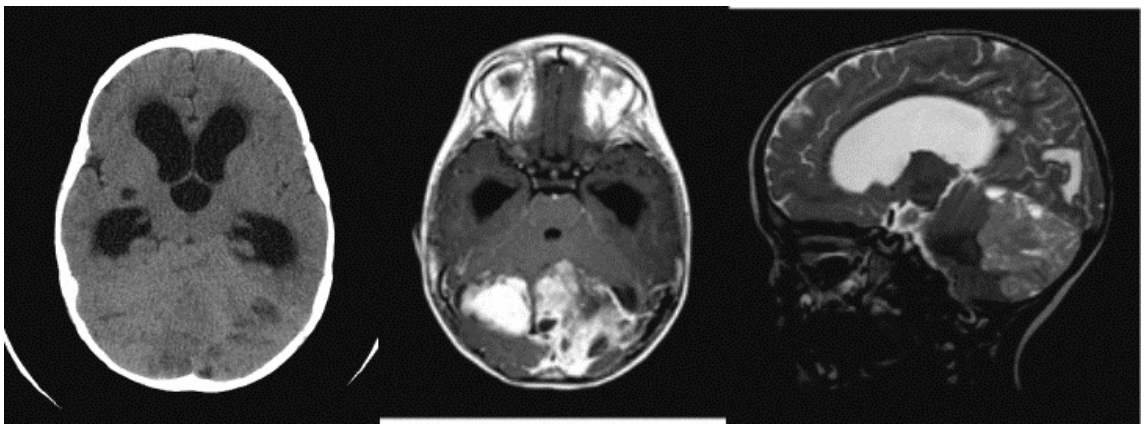
Axial T1-weighted and Sagittal T1-weighted post contrast MRI. Images show a complex partially cystic suprasellar mass with the presence of an enhancing rim and solid components.

Anesthetic considerations for patients with sellar and suprasellar tumors are similar to those for patients with other supratentorial tumors. However, the anesthetist must also look of evidence of endocrine dysfunction (hypothyroidism, growth hormone deficiency, ACTH deficiency, and diabetes insipidus). Specific hormone deficiencies should be replaced. Teenagers and older children can have pituitary adenomas removed by transphenoidal surgery. Patients having sellar and suprasellar tumors removed require the same intraoperative monitoring and vascular access as other patients with brain tumors. Their trachea is usually intubated orally with a pre-formed RAE tube. The anesthetist should always be prepared for massive intraoperative bleeding with these operations. Optic gliomas occur in children with neurofibromatosis. Children with optic gliomas present to hospital with proptosis, increased ICP, and visual disturbances. Hypothalamic dysfunction is usually a late finding. Blood loss may be significant during transphenoidal surgery.

Diabetes Insipidus

Diabetes insipidus (DI) is caused by a deficiency in antidiuretic hormone (ADH), which acts directly on the distal tubules and collecting ducts of the kidney to promote water reabsorption. Brain tumors are often the cause DI, particularly hypothalamic and pituitary tumors. DI also occurs with optic nerve injury and traumatic brain injury. Approximately 75% of patients undergoing transcranial resection of pituitary tumors and in 10-44% of those undergoing transphenoidal resection have DI. Although DI can occur intraoperatively, its onset more commonly occurs 2-to-6 hours after surgery and resolves 2-to-7 days later. Diabetes insipidus is characterized by polyuria (greater than 4ml/kg/h in children and greater than 6ml/kg/h in neonates), increased plasma osmolality (greater than 300mOsm/l) and decreased urine osmolality (less than 300mOsm/l). A urine specific gravity below 1.005 and a serum Na^+ above 145meq/l are characteristic of the hypernatremic hypovolemia associated with DI. The treatment goal for perioperative DI is maintenance of water and electrolyte balance, urine output, and normal hemodynamics. Other causes for the high urine output must be ruled out, including the use of diuretics or mannitol and hyperglycemia. If DI is present prior to surgery, preoperative and intraoperative IV administration of vasopressin should be considered. During surgery careful evaluation of urine output is necessary at least once an hour. Two-thirds of the fluid lost, plus any blood lost, should be replaced every hour to maintain hemodynamic stability. Perioperative fluid management during DI should not include hypotonic solutions. The initial dose of vasopressin 0.5mU/kg/h by infusion is increased every 5-to-10 minutes until the urine output is about 2ml/kg/hour. Vasopressin (DDAVP) 0.5-to-4mcg is given IV as a single dose. DDAVP, a potent antidiuretic with minimal vasopressor effects, can also be used in the perioperative period. A quarter of this dose is also used to decrease bleeding in patients with von Willebrand disease. Vesopressin's effects last 8-12 hours. Serum Na^+ levels must be monitored when using DDAVP.[148,149]

Figure 10-10: **Medulloblastoma**



An axial CT, axial T1-weighted postcontrast MRI and sagittal T2-weighted images showing infratentorial large mass filling and expanding within the fourth ventricle.

Posterior fossa brain tumors include medulloblastomas, cerebellar astrocytomas, ependymomas, and brain stem gliomas. **(Figure 10-10)** They block CSF circulation between the fourth ventricle and spinal canal, which increases ICP. Common signs and symptoms of posterior fossa tumors include morning vomiting, irritability, lethargy, stiff neck, cranial nerve dysfunction, and ataxia. Surgical resection of posterior fossa tumors has important anesthetic considerations beyond those of all pediatric neurosurgical procedures. Care should be taken to fix the tracheal tube securely, since most of these surgeries are done in the prone position. The head is placed in a horseshoe headrest to allow intraoperative access to the airway. Invasive hemodynamic monitoring is indicated, due to the potential for cardiac arrhythmias and sudden change in blood pressure that occur with surgical stimulation of structures in the posterior fossa. There is some risk venous air embolism in the prone position that must be searched for at all times. Most children with posterior fossa tumors have raised ICP and obstructive hydrocephalus. Surgeons often place an external ventricular drain to remove CSF before starting the surgical resection.[150]

Traumatic Brain Injury and Head Trauma

Head trauma is the leading cause of morbidity and mortality in children and adolescents worldwide. Motor vehicle accidents, bicycle accidents, child abuse, and falls are responsible for most of these injuries. Many children with traumatic brain injury (TBI) have minimal neurologic symptoms during their initial assessment. However, symptoms of increased ICP and neurologic deficits develop over time.

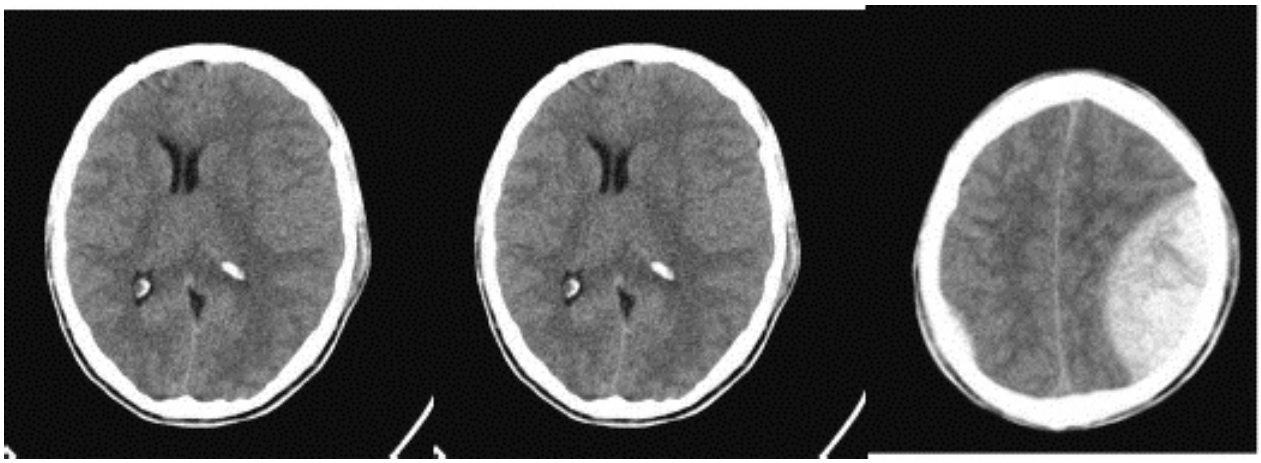
Brain injuries occur in two phases. The first occurs at the moment of impact when the skull, neural tissue, and vasculature are injured. The second occurs when an endogenous cascade of biochemical and cellular events is initiated. This cascade begins within minutes of the initial injury and continues for months, leading to axonal injury and cell death. Cerebral edema, hypotension, hypoxia, hypo- or hypercapnia, intracranial hypertension, and hypo- or hyperglycemia significantly increase secondary brain injury.[151,152]

Physical examination of these patients must search for signs of skull fracture, CSF leak, auditory canal bleeding, periauricular hemorrhage, and foreign bodies. The presence of scalp laceration must be sought because they can cause severe bleeding, hypovolemic shock, and worsen secondary brain injury.

Skull fractures are generally linear and are associated with severe contusion or concussion. Seven-to-ten percent of children with TBI have depressed skull fractures. Surgery to elevate a depressed skull fracture is indicated if the cranial plate is depressed more than the thickness of the bone, if a dural laceration is causing a neurologic deficit, and/or if there is a CSF leak. Few children have basal skull fractures. The signs of basal skull fractures include auditory canal bleeding, nasal bleeding, mastoid ecchymosis, periorbital bruising, and blood behind the tympanic membrane. Complications from a basal skull fracture include a CSF leak, meningitis, and anosmia.

Diffuse brain injuries are the result of rapid acceleration and deceleration of the brain. Efforts should be made to detect focal lesions early, since immediate surgery changes their outcome considerably. Cerebral contusion is common in pediatric patients, and the hairline fractures associated with them are most frequently located in the temporal, frontal, and occipital regions. Bruising occurs during the second phase of the injury. The neurologic effects of cerebral contusion are due to mass effect, edema, or hemorrhage. If there is mass effect, immediate surgery is indicated to prevent or treat increased ICP. Epidural hematomas **(Figure 10-11)** are usually associated with parietal or temporal skull fractures and often with injury to the middle meningeal artery. Signs and symptoms of an epidural hematoma include loss of consciousness that occurs after an initial lucid period. The occurrence of contralateral hemiparesis and ipsilateral mydriasis should lead to immediate drainage of the hematoma and reduction of ICP. Medical management of ICP should be initiated as soon as possible.

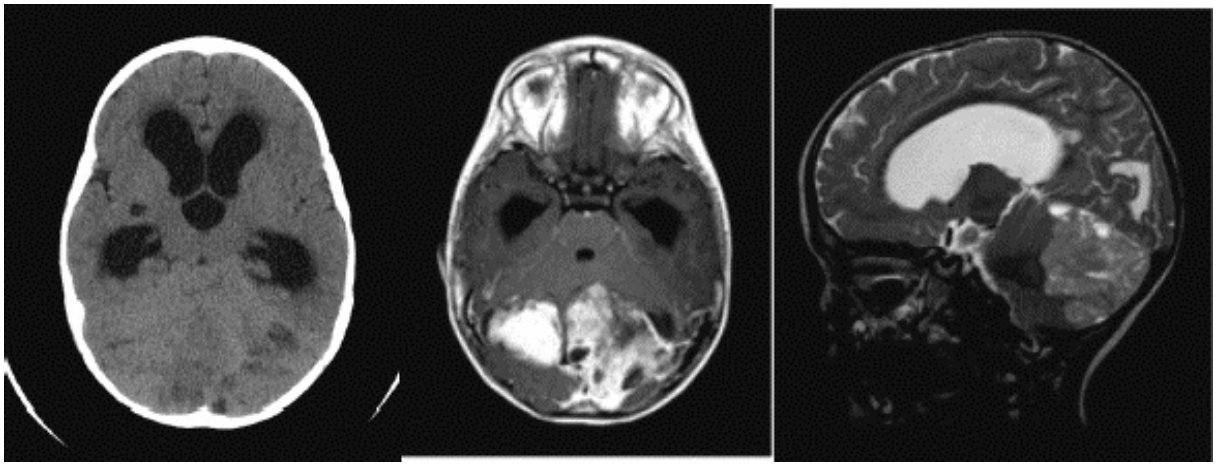
Figure 10-11: Epidural Hematoma:



Axial CT shows a hyperdense, bi-convex epidural hematoma with compression of brain. The internal hypodensity implies an active bleeding.

Acute subdural hematomas **(Figure 10-12)** are associated with cortical damage due to direct parenchymal contusion. There is disruption of the bridging veins between the dura and the cortex. Fractures occur in 30% of older patients and more frequently in those below two years of age. Mortality with subdural hematoma is high. Cerebral edema, increased ICP, and persistent neurological deficits characteristically occur during the postoperative course of patients who survive surgery for subdural hematoma.[153]

Figure 10-12: **Acute Subdural Hematoma.**

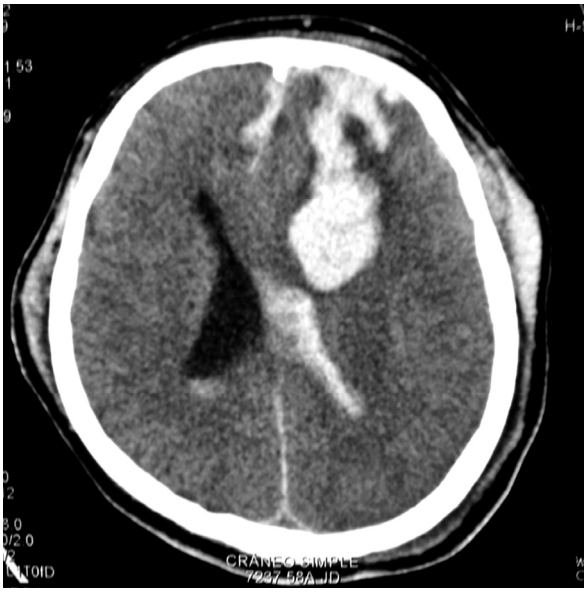


Axial CT images showing a centric, non-homogeneous extra-axial collection with compression and displacement of the underlying brain structures.

Child Abuse (Shaken Baby Syndrome)

Child abuse accounts for 80% of deaths in children who have TBI, a chronic subdural hematoma, and who are less than two years of age. The bleeding is usually caused by acceleration and deceleration forces, direct trauma, and by traction injury to vessels of the brain stem. The child presents with changes in consciousness, lethargy, irritability, vomiting, and seizures. More than 75% of patients with the SHAKEN BABY SYNDROME have retinal hemorrhages. The presence of a subarachnoid hemorrhage on CT, particularly inter-hemispheric blood (subdural hematoma), is a specific sign of the shaken baby syndrome.[154]

Figure 10-13: **Intraventricular Hemorrhage.**



Acute intraparenchymal hematoma and ventricular hemorrhage. Axial non contrast CT shows frontal intra-axial hematoma with mass effect associated to intra-ventricular hemorrhage.

Parenchymal hematomas **(Figure 10-13)** are common in children with severe cortical contusions following TBI. The incidence of parenchymal hematoma is low, but when it is present the prognosis is very poor. Indications for urgent surgery in patients with TBI include an open skull fracture and a depressed fracture causing symptoms. Surgically evacuated intracerebral hematomas are often associated with severe tissue damage. They frequently occur in highly vascularized areas. Due to the poor outcomes and the impact of secondary brain injury on the outcome of TBI, treatment should be directed, not only to the evaluation, diagnosis, and stabilization of the patient, but also at reducing secondary brain injury, which ultimately is the main cause of morbidity and death.

Secondary brain injury is the result of hypotension, hypoxemia, hypo and hypercapnia, hypo- and hyperglycemia, and anemia. Neurologic outcomes with secondary brain injury may be improved by early and adequate resuscitation, [155] triage of the patient to a facility that frequently cares for patients with TBI, and surgery when indicated. The goals of anesthetic management are to secure the airway, prevent secondary brain injury, prevent increased ICP, and to maintain adequate CPP. Positive pressure ventilation is initiated early to maintain a PaO_2 >60mmHg. Positive end-expiratory pressure (PEEP) is avoided because it increases ICP and decreases cerebral venous drainage. Hypoxemia and aggressive hyperventilation must also be avoided. Therapy to prevent cerebral herniation occasionally includes inducing hypocapnia. However, hypocapnia can worsen cerebral ischemia, especially if the arterial blood pressure is low.[156] The status of the brain should be evaluated frequently.

Because of the risk for hypovolemia and hypotension, appropriate amounts of isotonic saline must be given to maintain normovolemia. Anemia, when present, must be corrected. Although specific recommendations for transfusion therapy in pediatric patients with TBI do not exist, there is no benefit to either a liberal or restrictive transfusion protocol.[157] abnormal coagulation often occurs with TBI, but the exact mechanism for this is poorly understood. It is hypothesized that coagulation defects are the result of massive release of tissue factors, disseminated intravascular coagulation, fibrinolysis, hypoperfusion, activation of protein C, and platelet dysfunction (See Chapter 5).[158,159]

The Society of Pediatric Critical Care published evidence-based recommendations for management of severe TBI in children and adolescents in 2012. Anesthetists should be familiar with these recommendations, as they are intended to reduce secondary injury.

Children with severe head trauma and TBI usually die from refractory increased ICP. The following is an outline for treatment of patients with TBI.

Initial Treatment of TBI Should Focus on Management of Increased ICP and Maintenance of the CPP:

1. ICP monitoring should be performed when possible in all children with severe TBI [Glasgow Coma Scale (GCS) <8] because they surely have intracranial hypertension and possibly high ICPs. Early ICP monitoring, when used to guide therapy, reduces morbidity and mortality.
2. ICPs above 20 mmHg should be treated if they persist more than five minutes. ICP monitoring and invasive arterial pressure monitoring are essential to calculate CPP (CPP = MAP-ICP) and guide therapy.
3. The minimum acceptable CPP in children with severe TBI is 40mmHg, the goal being 40-to-50mmHg. For infants CPP might be a lower whereas for teenagers it is higher.
4. Hypertonic saline (3%) is used to reduce intracranial hypertension in children with severe TBI. The recommended dose of **3%** hypertonic saline is 6.5-to-10ml/kg/h. Hypertonic saline can be infused at 0.1-to-1ml/kg/h. It is recommended that the lowest dose of saline that will maintain the ICP below 20mmHg be used, and that plasma osmolality be kept below 360 mOsm/L. There is insufficient evidence to support or reject the use of mannitol in the treatment of intracranial hypertension in children with severe TBI.
5. There is no evidence that hypothermia (32-33°C) is beneficial during the first 24h following TBI and in fact, can negatively effect the outcome in these patients.
6. Prophylactic hyperventilation to a $PaC0_2$ <30 mmHg should be avoided in the first 48h after TBI. The risk on iatrogenically induced cerebral ischemia is very high. When hyperventilation is used to treat refractory intracranial hypertension, neurophysiological monitoring is required to determine the adequacy of cerebral blood flow and the presence of cerebral ischemia.

7. An external ventricular drain is sometimes used to remove CSF and lower the ICP of patients who have severe TBI.
8. Barbiturates can be used to treat hemodynamically stable children who have intracranial hypertension that has not responded to optimal medical and surgical management. The arterial blood pressure should be monitored continuously and the information gained from it used to maintain adequate CPP.
9. Decompressive craniectomy with duraplasty has been used when there are early signs of neurological deterioration, brain herniation, or refractory intracranial hypertension that are not responsive to medical management.
10. Prophylactic treatment with phenytoin may reduce the incidence of early posttraumatic seizures in pediatric patients with severe TBI.
11. Data concerning glycemic control in children with severe TBI are lacking.
12. Steroid use is not recommended for pediatric patients with severe TBI because there is no clinical evidence that it improves outcome.[160]

The ultimate goal in the management of children with TBI is prevention of secondary brain injury. Cardiovascular resuscitation and effective ventilation are important, but other injuries must also be treated. The classic mistake is to focus only on the brain injury and ignore abdominal or thoracic injuries that often contribute significantly to the patient's morbidity and mortality.

Spinal Cord Injury (SCI)

Isolated spinal cord injury is rare in children, and those who have SCI usually have TBI also. The reported incidence of SCI is 5-to-10% in both adults and children. Because pediatric patients have large, heavy heads, 42% of SCIs occurs to the cervical spine, while 31% occur in the thoracic and 27% in the lumbar spine. Automobile accidents, falls, and sports injuries are the usual causes of SCI. SCI is more common in males than females. Due to differences in growth and development of the spinal column, spinal trauma in children is significantly different from that of adults. These differences include larger and heavier heads and greater elasticity and mobility of C1-C3 vertebral bodies in children under eight years of age. Between 8-and-12 years of age, increased mobility is observed between C3-C5; in older children, it occurs at C5-C6. They have of partially ossified vertebrae, more flexible joint capsules, and horizontal, flat vertebral body surfaces .[161,162] Because of these differences, children <8 years of age more commonly have high cervical injuries. The younger the child the greater is the risk for high cervical injury. In children older than 8 years of age, SCI occurs at the lower portion of the cervical spine. Due to the high elasticity and mobility of the pediatric spine, radiologic criteria used to diagnose SCI in children differ from those of adults. Vertebral body dislocation of more than 4.5mm between C2-C3 or C3-C4 in <8 year old children, or subluxation of a vertebra in excess of 3.5mm at any vertebral level in patients >8 years of age is an indication of cervical spine instability. A more than seven degree angulation of one vertebra on another suggests spinal cord injury.

The signs of possible cervical spine injury most often include pain in the posterior midline, peripheral neurologic deficits, decreased level of consciousness, and evidence of cerebral cognitive dysfunctions. However, it must be remembered that the pain may be caused by other trauma and not by SCI. Spinal cord injury in children can be classified into four categories: isolated vertebral fractures, fracture with subluxation, subluxation only, and traumatic spinal cord injury without radiological evidence of injury.

Management of SCI includes: 1) securing the airway while maintaining cervical immobilization in patients who have reduced consciousness, high CSI, and/or decreased airway protection; 2) ensuring adequate oxygenation and proper lung ventilation; 3) restoring the patient's hemodynamics to normal.

SCI has two primary causes: 1) initial injury by mechanical forces and bone fragments that cause direct trauma to the spinal cord. 2) secondary tissue damage that is caused by edema and ischemia from hypotension and hypoxemia. Inadequate resuscitation of a child with unstable SCI may exacerbate the primary lesion and favor development of a secondary injury.

Respiratory failure is the most common cause of death in patients with isolated cervical injuries. Cervical SCI-induced respiratory failure decreases tidal volume and minute ventialtion, increases respiratory dead space, and causes CO_2 retention, all of which worsen secondary brain and spinal cord injury. Thus, it is vital to quickly secure the airway and ensure adequate oxygenation and ventilation. Immobilizing the neck avoids aggravating SCI. Spinal shock, loss of vasomotor tone, bradycardia, and decreased myocardial contractility are common in patients with SCI. However, the anesthetist must also look for other causes for the hypovolemic shock such, as injury to the chest, abdomen, pelvis, and long bones, or obstructive shock secondary to tension pneumothorax or cardiac tamponade.

Fluid management for SCI should begin with normotonic crystalloid solutions. Vasopressors are added if the blood pressure does not respond to fluid administration.

Spinal Cord Injury Without Evidence of Radiologic Abnormalities (SCIWORA)

SCIWORA is a spinal cord injury that occurs without radiologic evidence of vertebral fractures or subluxation. It occurs in 13-32% of pediatric patients with SCI and most commonly affects the cervical spine. SCIWORA is the result of the relaxed spinal ligaments and severe flexion-extension of the spine with trauma. The pathophysiology of SCIWORA is related to punctate hematomas/hemorrhages, edema, and tissue infarction. A MRI helps diagnose soft tissue, ligament, and spinal cord lesions. Treatment of SCIWORA includes appropriate cardiopulmonary resuscitation to ensure proper oxygenation, ventilation, and perfusion. Systemic blood pressure is maintained within normal limits or slightly above normal. The patient's neck is immobilized with a rigid cervical collar. Although still controversial, it is nevertheless recommended that these

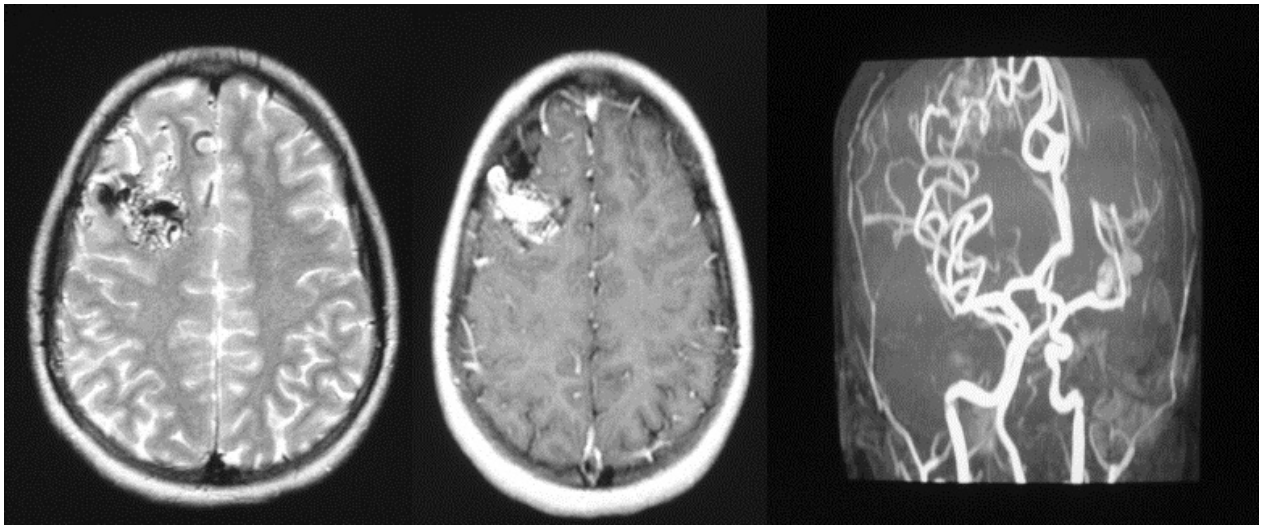
patients be given a bolus of methylprednisolone 30mg/kg and a continuous infusion of 5.4mg/kg/hour of methylprednisolone for 24 hours if the patient arrives during the first three hours after the trauma. If the patient arrives after three hours, the infusion should be continued for 48 hours. Methylprednisolone improves medullary blood flow, inhibits the arachidonic acid cascade, and modulates the immune response. Patients with a transected spinal cord have a poor prognosis (3% recover). Sixty-two percent of those with an incomplete spinal cord transection recover.[163,164]

Vascular malformations

Arteriovenous Malformations (AVMs) and Aneurysms

Arteriovenous malformations and intracranial aneurysms are the most common causes of spontaneous intracranial hemorrhage in children. AVMs occur in 10-to-14/100,000 people, 7-10% of which are children under the age of 18 years.[165] **(Figure 10-14)**

Figure 10-14: Arteriovenous Malformation (AVM).



Axial T2-weighted, axial T1-weighted post contrast and angiographic MRI showing a tightly packed mass looking like a honeycomb distribution of flow. The lack of contrast enhancement represents an AVM nidus in the right frontal lobe.

Bleeding from an AVM frequently presents as intraventricular hemorrhage, sudden onset of seizures, or severe neurological deficits. Intracranial hemorrhage is found in 20-50% of pediatric AVMs. Eighteen-to-twenty percent of AVMs are symptomatic during childhood. Intracranial hemorrhage occurs in 75-to-80% of pediatric patients with AVMs and 50-to-65% of adults.[166,167] Infants and children under two years of age who have AVMs often have congestive heart failure, delayed neurodevelopment (due to blood being shunted away from some parts of the brain by the AVM - so called vascular steal), headache, and a cranial murmur. The risk of rebleeding from

an AVM is high during the first twelve months after the initial bleed. Many posterior fossa and basal ganglia AVMs bleed. The incidence of rebleeding is 25% over the first five years. In children, the location and size of AVMs are risk factors for rebleeding. Deep lesions bleed more often than hemispheric lesions.

Medicosurgical management for AVMs includes endovascular embolization, stereotactic radiosurgery, or as the last recourse, surgical resection. In 70% of cases, endovascular embolization will partially obliterate the AVM, but it does not decrease the incidence of recurrent bleeding. In some cases, embolization is indicated before surgery to reduce flow in the AVM and make clipping the AVM easier. When the approach to an AVM is difficult (brain stem, basal ganglia, thalamus), the lesion is best treated with stereotactic radiosurgery, if available.

Elective embolization of vascular lesions is usually done while the patient is under general anesthesia. Moderate hyperventilation ($PaCO_2$ 30mmHg) improves the radiologist's ability to see abnormal blood vessels because vessels in an AVM do not respond to the vasoconstriction effect (including alkalosis). Thus, hyperventilation increases flow in AVMs making it easier to see the defect. However, as it increases the amount of blood within the lesion, it can affect the ability to deposit the embolizing material and also increase tendency to bleed. Anesthetists should be aware of the material being embolized and of their side effects and any complications they can cause. Close attention must be paid to fluid management, because children with AVMs often have heart failure due to high AVM flows and to the use of contrast medium by the radiologists.

The *vein of Galen*, which drains directly into the straight sinus, is a short but large diameter collection of veins from the deep venous system of the brain. Most malformations of the vein of Galen occur in infants and small children, whereas AVMs are rare in newborns. The clinical presentation of patients with malformations of the vein of Galen depends on the amount of blood shunting through the malformation. Newborns (0-1 month of age) can have multiple fistulas that divert nearly 25% of their cardiac output through them, which greatly increases blood return to the right heart and causes volume-related heart failure. Most newborns with AVMs have congestive heart failure (CHF). One-to-twelve month old infants may present with hydrocephalus that is caused by compression of the aqueduct of Sylvius and also the third ventricle. This prevents normal drainage of CSF from the brain. Children with AVMs may present with compensated congestive heart failure, seizures, or focal neurological deficits. Children over six years of age may present with a subarachnoid hematoma or with intra-parenchymal cerebral bleeding.[168]

Whether therapeutic intervention requires immediate or deferred attention, all children with AVMs require definitive evaluation of their AVM by CT, MRI, and cerebral angiography (if available) after complete assessment of their medical condition. Older children who are alert and oriented may be able to cooperate during these studies, but younger children or those with impaired consciousness require general anesthesia for airway protection and optimization of

conditions for the radiologist. During induction and maintenance of anesthesia, sudden changes in the arterial blood pressure, especially hypertension, can cause intracerebral hemorrhage and raise the ICP. Anesthetics used for maintenance of anesthesia are not different in patients with an AVM from those used for other intracranial procedures (See above). In the absence of congestive heart failure, controlled hypotension has been used during surgical or endovascular closure of AVMs. Because sudden massive bleeding can occur during surgical closure of an AVM, two large bore IVs and an arterial line are needed. Whether the malformation is clipped during surgery or closed endovascularly, the anesthetist must be prepared to treat sudden elevations in intracranial pressure and/or the hyperemic cerebral edema that often occur when the AVM is closed. A hypertensive crisis can be treated with nitroprusside (neonates 0.5-6mcg/kg/min; children 0,5-10mcg/kg/min) or labetalol (0.2-1.0mg/kg intravenous every 10 min, maximum dose 300mg or continuous infusion 0.4-1mg/kg/h). As in all CNS surgery, the anesthetist's goal during closure of an AVM is to maintain hemodynamic stability, cerebral perfusion pressure, control of ICP, provide enough brain relaxation to facilitate surgical exposure of the AVM, and to base fluid administration on blood and urine losses.

Although intracranial aneurysms are rare in children, they are usually large and are located in the posterior cerebral circulation. There are two types of aneurysms: "saccular" (caused by weakness of the muscle wall of the artery) and "mycotic" (caused by damage to arteries by septic emboli). Congenital aneurysms are associated with polycystic kidney diseases, fibromuscular dysplasia, connective tissue disorders (such as Ehlers- Danlos syndrome), and coarctation of the aorta. Congenital aneurysms are usually treated by endovascular embolization.[169-172]

Moyamoya Disease

Moyamoya disease is a chronic vascular condition of unknown etiology that is associated with a severe occlusive cerebral arterial disease. Angiography shows progressive stenosis or occlusion of the terminal portion of the internal carotid and the anterior and middle cerebral and the posterior cerebral arteries. Moyamoya means 'Puff of Smoke', which refers to the angiographic appearance of a puff of 'cigarette smoke' seen beyond the non-vascularized area.[173] Symptoms of Moyamoya disease include transient cerebral ischemia, intracranial hemorrhage, seizures, and headache. Patients with Moyamoya disease have progressive cerebral ischemia from ongoing reduction in cerebral perfusion. Treatment of MoyaMoya disease is surgical revascularization of the affected areas. The goal of revascularization is to promote neo-angiogenesis and induce formation of collateral vessels. In preparation for surgery, the patient should not be fasted for long periods to avoid dehydration. Premedication is indicated to limit anxiety and crying, hyperventilation, decreased $PaCO_2$, cerebral vasoconstriction, decreased CBF, and worsening of cerebral ischemia. There is no evidence that the volatile or intravenous drugs used for induction of anesthesia have effects on the cerebral perfusion of patients with MoyaMoya disease. During surgery, the anesthetist must maintain a balance between the oxygen supply and demand of the

brain. Hypotension, hypoxemia, and changes in CO_2 during laryngoscopy and tracheal intubation are to be avoided. Normocapnia should be maintained during all stages of the procedure. During surgery, EEG monitoring can be used to detect early episodes of cerebral ischemia.[174]

Care should be taken to ensure normal hydration. Hyperosmolar solutions are avoided because they produce diuresis, dehydration, and hypotension. The ideal hematocrit for these patients is 30% because at this hematocrit oxygenation is adequate and tissue blood flow is increased. The increased blood viscosity associated with polycythemia decreases cerebral perfusion and this may lead to cerebral infarction. During surgery, the systemic arterial blood pressure should be maintained slightly above the patient's preoperative pressure. A decrease in mean arterial blood pressure decreases CBF and increases the risk of ischemia or stroke. Cerebral autoregulation is substantially diminished in adults with this disease when compared to children. For this reason, a higher frequency of ischemic symptoms is observed in adults. Asymptomatic, hemodynamically stable patients undergoing a radiological procedure can usually be sent home after the procedure. Those undergoing revascularization procedures require intensive care to ensure proper hemodynamic monitoring and treatment of any abnormalities found. Arterial blood pressure, CVP, urine output, hematocrit, and $Sa0_2$ should be continuously monitored postoperatively. Use of opioids and intravenous acetaminophen for postoperative analgesia reduces the risk of cerebral ischemia.[175]

Surgery for Epilepsy

Epilepsy is a chronic condition that affects 0.5-to-1% of the world's population and is considered to be a public health problem.[176] Despite adequate anticonvulsant therapy and new medications for the treatment of epilepsy; the failure rate of therapy is still 30-40%. Inadequately treated epilepsy decreases the neurocognitive development of children. It increases neuroplasticity of the developing brain. Aggressive surgical management of epilepsy is appropriate for many patients when medical therapy fails to control epileptic discharge.[177]

Epilepsy refractory to medical therapy is defined as inadequate control of seizures despite giving two anticonvulsant medications at their maximum tolerated doses for 1.5-to-2 years. Patients who have adequate seizure control, but suffer unacceptable side effects from the medications, are also considered to have refractory epilepsy. Patients with cortical malformations, abnormal neuronal migration syndromes (Rasmussen, West, Sturge-Weber) frequently have refractory epilepsy. Epilepsy surgery provides anesthetists with two major challenges that are not usually found in other neuroanesthesia procedures: 1) the possibility of the patient's craniotomy being done under local anesthesia and 2) the need for prolonged narcosis. Doing an awake craniotomy in pediatric patients may be difficult. Most of them require general anesthesia. Some anesthetic are avoided during surgery for epilepsy because they affect electrical activity and intraoperative electroencephalographic monitoring.

The Anesthetist's Management of Patients with Epilepsy Includes:

1. A thorough pre-anesthesia assessment of coexisting medical conditions, assessment of side effects of the patient's antiepileptic drugs, and knowledge and understanding of the neurophysiological tests to be performed during surgery.
2. Providing an adequately relaxed brain to facilitate surgery.
3. Avoiding anesthetic drugs that interfere with electrophysiological monitoring.
4. Being aware that it may be necessary to induce seizures during intraoperative electrocorticography.
5. Ensuring adequate CPP and maintaining hemodynamic, respiratory, and metabolic stability.
6. Assuring the absence of awareness throughout the procedure, except when patient cooperation is required to preserve the integrity of vital areas of the brain (speech, motor function).
7. Allowing rapid awakening to allow early neurological assessment of the patient.[178,179]

If neuropsychological testing is available, it can be used to assess the patient's preoperative cognitive function. The results of these tests can be used to predict the impact of the surgery on outcome. EEGs are used to detect seizures, to confirm the presence of abnormal electrical activity, and to guide the surgeon to the area he/she will remove. Intraoperative EEG monitoring helps define the exact location of the seizure focus and its relationship to vital functional areas. The use of EEG monitoring is especially important when the seizure focus is outside the temporal area of the brain. MRI, SPECT and PET scanning have greatly improved localization of the epileptic focus and helped define the relationship of functional areas of the brain to the epileptic focus. Most tests for preoperative evaluation of epilepsy require that the child not move for long periods of time. Children who cannot do so can be sedated with propofol for placement of the EEG wires and recording needles. The effects of propofol on the EEG are short lived once the drug is discontinued. Once the drug is withdrawn, neurologists can obtain baseline EEG recordings. Dexmedtomidine is also be used for this purpose because it causes minimal effects on the EEG.[180]

Longer procedures, such as MRI and SPECT, can be performed with a continuous infusion of propofol. The most common surgical procedures for treatment of childhood epilepsy are resection of the epileptic focus and insertion of a vagal nerve stimulator. Hemispheric resection is indicated for patients with a clear epileptic focus, with abnormal cortical development, hemiplegia, and for Sturge-Weber Syndrome.

Implantation of a vagus nerve stimulator (VNS) is commonly done for intractable epilepsy when it is not possible or desirable to surgically resect the epileptic focus. How VNS works is not well understood, but it appears that it activates the tractus solitaius and other nuclei in the brain stem. This apparently modulates brain excitability by activating both the limbic system and noradrenergic neurotransmitter systems. During surgery the left vagus nerve is exposed, and a

stimulating electrode is placed into the nerve. A signal generator is inserted into the fascia of the pectoral major muscle. The left vagus nerve is used because using the right vagus nerve may cause cardiac pacing. The most common side effects of this procedure are postoperative hoarseness and sore throat. Rare complications include bradycardia, paralysis of facial muscles, and worsening of obstructive sleep apnea. If patients require an MRI after implantation of a VNS, the stimulator should be turned off for the duration of the procedure and turned on once the procedure is completed.

The ideal treatment of intractable seizures is surgical resection of the seizure focus. To do this requires a seizure focus that is unifocal and one that is not close to the motor or language areas of the brain. The area containing the epileptic focus is exposed through a craniotomy. Resection of the epileptic focus is often done in two stages. During the first stage, a craniotomy is done and an array of EEG electrodes is placed directly over the area of the brain believed to contain the seizure focus. The skull is then closed and wires form the EEG electrodes are externalized to allow EEG monitoring of the focus site for approximately 48 hours. During the second surgery, the craniotomy is reopened, and the area identified by the 48 hours of monitoring is resected.[181]

The choice of anesthetic for induction of anesthesia depends on the age of the child, but sevoflurane or propofol are usually used. An arterial line is inserted to allow continuous monitoring of the hemodynamic changes that occur with surgical manipulation. Two large bore IVs are needed for fluid replacement, as in any invasive neurosurgical procedures. Although large blood losses are unusual, it is prudent to have blood available for transfusion. In most cases, balanced anesthesia is appropriate. When intraoperative EEG monitoring is required, volatile anesthetics are not used. Muscle relaxants are not used if EMG monitoring is required. Propofol or dexmedtomidine provide anesthesia during neurophysiological monitoring. At the end of the surgery, the trachea is usually extubated.

Resection of the corpus callosum is done when it is not possible to resect the epileptic focus. This palliative procedure is done to prevent spread of seizure activity to the other hemisphere of the brain. The anesthetic considerations for this procedure are similar to those described above. Patients undergoing corpus callosum resection usually have significant postoperative cognitive impairment. Electrophysiological monitoring is usually not indicated for this procedure.

Hemispheric resection is the most aggressive surgical procedure for epilepsy and is reserved for patients who have intractable seizures that are caused by diffuse hemispheric malformations of cortical development, stroke, childhood hemiplegia syndrome, and Sturge-Weber and Rasmussen syndromes. Anesthetic considerations for this surgery are similar to those for focal resection of an epileptic focus, except there is a greater probability of hemorrhage and blood transfusion. Anesthesia preparation should include two large bore IVs, invasive hemodynamic monitoring, and inserting an urine catheter. Patients usually require several days of intensive care and postoperative mechanical ventilation after this surgery.[182,183]

Deep brain stimulation is rarely done in children, but there are data that suggest stimulation of the thalamic nucleus reduces the frequency of seizures. This procedure is only done in children who have intractable seizures and are not candidates for other surgical procedures, including VNS.[184,185]

Awake and Functional Neurosurgical Craniotomy

Awake craniotomy has limited utility in pediatric patients and young children but is done in older children and adolescents for epilepsy, implantation of deep brain stimulators for management of movement disorders, deep resection of tumors, and for resection of vascular lesions located in vital brain areas (language, memory, motor and sensory). One of the key requirements for using this technique is the need for patient co-operation. The anesthetist must have knowledge of specific problems that can occur during surgery and the need for continuous monitoring of the awake patient throughout surgery.

The two most important considerations for patients undergoing awake craniotomy are mental maturity and evaluation of the airway. Anxiety disorders, low pain tolerance, mental disorders, movement disorders, claustrophobia, obesity, and gastro-esophageal reflux are contraindications to awake craniotomy. Other factors to consider are tumor size and its effects on cardiovascular stability, risk of bleeding, and hemodynamic instability.[186]

Positioning of patients for awake craniotomy surgery has some important considerations. The anesthetist must have access to the patients face to evaluate her/his facial expressions and speech throughout the procedure. This is vital to success of the procedure. Both asleep-awake-asleep anesthesia and monitored anesthesia have been used for this procedure. Dexmedtomidine, propofol, and remifentanil are ideal for this purpose because their effects rapidly dissipate when they are discontinued, allowing the patient to “awaken” quickly for intraoperative neurocognitive assessment. Ensuring that the patient has sufficient analgesia to prevent pain, especially when pins are inserted into the skull, when the scalp is reflected, and when the dura mater is incised and manipulated is very important.

The sleep-awake-asleep technique involves administering a general anesthetic, commonly propofol and remifentanil. During the surgery, when electrocorticography (ECoG) is required, the propofol infusion is discontinued (usually 15 minutes before ECoG monitoring begins). This drug is discontinued to prevent it from interfering with interpretation of the ECoG signals. Remifentanyl 0.05mcg/kg/min is continued during the awake phase of anesthesia because it has little effect on the ECoG signals. Dexmedtomidine 0.2mcg/kg/h can also be used, since it has also no affects on ECoG.[187,188] Monitored conscious sedation is appropriate when the skull is being opened and closed. Bolus or continuous infusions of propofol or dexmedtomidine plus remifentanil or fentanyl are also used. High concentrations of oxygen must not be allowed to accumulate near the surgical field because a spark from the electrocautery could start a fire. The

number of complications occurring during awake craniotomy is low because patients undergoing this procedure are carefully selected and thoroughly prepared. However, airway problems, nausea, vomiting, cerebral edema, seizures, bleeding, agitation, air embolism, and hemodynamic instability have been reported in some patients.[189-191]

Interventional Neuroradiological Procedures

It is increasingly common for children with intracranial disease to require anesthesia for diagnostic or therapeutic neuroradiology procedures. Procedures such as angiography and embolization of vessels are common. Anesthesia is required for these procedures because the procedures are long, technically challenging, and uncomfortable for the patient, and often require manipulation of blood pressure and ventilation during the procedure. The images obtained are also of better quality in anesthetized, immobile patients. Any complications that occur during these procedures can be taken care of more quickly in anesthetized children.[192]

General anesthesia and tracheal intubation are required for these procedures because 10-to-15 second periods of apnea are required to obtain the best quality images. Enough IV fluid is given during these procedures to maintain normovolemia, adequate renal perfusion, and good urine output. Failure to do so may result in renal failure, which is caused by the large volumes of hypertonic contrast medium used. While less than four percent of patients undergoing cerebral angiography have complications, bleeding occasionally occurs. When bleeding does occur, it usually does so at the site where the radiologist obtained vascular access (femoral vessels). Adverse reactions to contrast medium occur, but do so in less than 1% of children undergoing neurointerventionl procedures. These reactions consist of nausea, rash, hemodynamic instability, bronchospasm, and cardiovascular collapse. They usually occur within one hour of administering the drug. Mild reactions are self-limited and are managed symptomatically. Severe reactions should be treated like anaphylactic shock. Patients at high risk for reactions to contrast material are those who had previous reactions to these drug, asthma, or atopic dermatitis. When a patient previously has any one of these problems, he/she should be premedicated with steroids and antihistamines before the procedure begins. The recommended dose for the administration of intravenous methylprednisolone is 1-2mg/kg whereas it is 1mg/kg of diphenhidramine**.**

Therapeutic interventions, such as embolization of arteriovenous malformations, arteriovenous fistulas (AVF), or aneurysms, and the administration of intra-arterial chemotherapy for tumors, usually require general anesthesia. The procedures are typically long and muscle relaxation and paralysis are needed to prevent patient movement that could prove to be catastrophic. The anesthetist must keep track of how much heparinized saline the radiologist gives during these procedures and compensate by reducing the amount of IV fluid the anesthetist gives. Deliberate hypotension is sometimes used to facilitate placement of micro particles or coils in high-flow lesions. Micro particles, such as ONYX, can cause severe bradycardia, especially in infants. The risk

of rupturing an intracranial vessel is 0.5%. When this occurs, the patient must be immediately transferred the patient to an operating room for surgery. After embolization procedures, patients should go to a critical care unit for strict monitoring and control of blood pressure to reduce the risk of postoperative bleeding.[2]

Conclusions

The anesthetist's main goal for patients with neurologic pathology is to assure that comprehensive care is provided. This requires knowledge of normal and abnormal brain anatomy, differences in physiology at the different stages of development and the pathophysiological consequences imposed by the medicosurgical condition. Knowing the effects of anesthetics on the brain physiology of pediatric patients significantly improves outcomes and contributes to a significant reduction in morbidity and mortality.

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Chapter 11:
ANESTHESIA FOR THORACIC SURGERY

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Introduction

Many infants require thoracic surgery to correct congenital anomalies. Older children require surgery to remove tumors and pulmonary abnormalities. This chapter provides information on the lesions for which surgery is required and on the anesthesia care required. It also provides information on pneumothorax and its treatment. The information contained in this chapter will help the anesthetist meet the challenge of delivering safe care for the baby or child undergoing thoracic surgery.

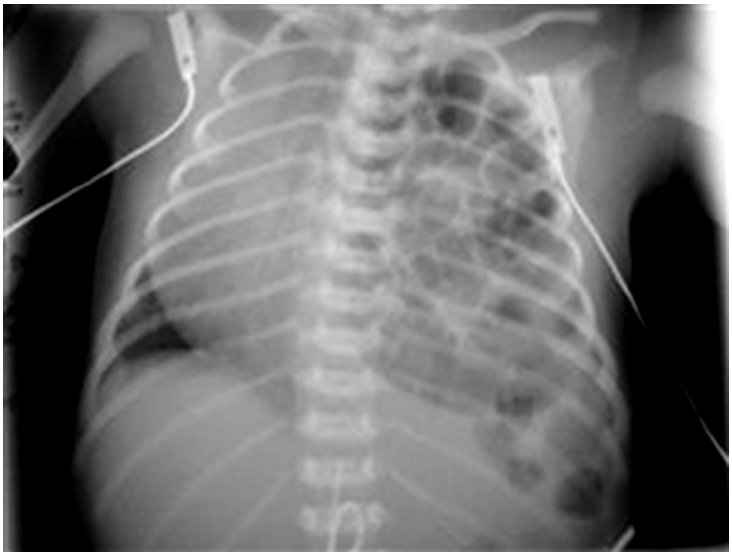
Thoracic Surgery for Neonates and Infants

Some neonates and infants require thoracic surgery to treat congenital lesions, such as diaphragmatic hernia, trachea-esophageal fistula, and several congenital abnormalities of the lung. The following section discusses these lesions and provides information concerning the anesthesia care required.

Congenital Diaphragmatic Hernia (CDH)

The diaphragm forms at about 10 weeks of gestation. If its formation is incomplete, the baby is born with a congenital *diaphragmatic hernia*. The hole in the incompletely developed diaphragm allows organs that normally reside in the abdomen to enter the thoracic cavity in utero and produce bilateral lung hypoplasia, pulmonary hypertension, and abnormal pulmonary vascular reactivity. Other congenital anomalies may also be present. The herniated abdominal contents, which may include the stomach, colon, kidney, and liver, often interfere with lung development. (**Figure 11-1**)

Figure 11-1: Diaphragmatic Hernia in a Neonate.



This radiograph shows a large amount of bowel and probably part of the liver in the left chest of this neonate who had a CDH. His heart is displaced into the right chest. Note: no visible bowel is seen in the abdomen. Courtesy of Han Minh Lee, MD[2]

The severity of the defect in lung development and subsequent lung hypoplasia are related to the age at which the abdominal organs enter the chest and on the amount of tissue herniated. The earlier the organs enter the chest and the more abdominal contents herniated, the worse the lung hypoplasia. Once they enter the chest, the abdominal organs compress the lung on the affected side and cause it to stop growing. As a result it is very hypoplastic. The abdominal contents also displace the mediastinum to the other side of the chest, which compresses that lung and causes that lung to also be somewhat hypoplastic. As a result these infants are frequently born with about two-thirds of a functioning right lung as their only means of oxygenation and CO_2 removal.[1,2] The abdominal mass not only hinders normal lung development, but it also reduces the total cross-sectional area of the pulmonary vascular bed and alters its reactivity, which frequently results in pulmonary hypertension. These abnormalities of the pulmonary circulation prevent the natural transition from intrauterine to extra-uterine circulation. Due to elevated right-sided intravascular pressures, venous blood is shunted right-to-left across the foramen ovale (PFO) and ductus arteriosus (PDA), causing severe hypoxemia. Neonates with a CDH usually exhibit signs and symptoms of respiratory distress and hypoxemia immediately after birth. The classic triad of CDH is cyanosis, shortness of breath, and *apparent* dextrocardia. The dextrocardia is apparent (and not actual) because the herniated abdominal

[2] This chapter is a modification of a chapter in Gregory GA, Andropoulos DB. Gregory's Pediatric Anesthesia., Wiley-Blackwell, Oxford UK, 2012

contents push the heart and mediastinum to the opposite side of the chest. When the bowel is removed from the chest, the heart returns to its normal position in the chest. Physical examination shows a scaphoid abdomen (a baby's abdomen normally protrudes), a bulging chest, decreased breath sounds on the affected side, distant or displaced heart sounds, and occasionally the presence of bowel sounds in the chest that contains the herniated bowel. A chest radiograph, if available, shows a bowel gas pattern in the chest, poor aeration of the affected chest, and shift of the mediastinum to the contralateral side (**Figure 11-1**). In the past, CDH was considered a neonatal surgical emergency. This is seldom the case today. Because many patients with CDH have severe hypoxemia, in the past these neonates were hyperventilated with 100% oxygen in the hope that the alkalosis and the high oxygen concentrations in the lung would produce pulmonary vasodilation and improve oxygenation. However, the mechanical ventilation required to produce respiratory alkalosis and the increased inspired oxygen required damaged the only functioning lung. The initial management of patients with CDH is now to delay surgical intervention until the acidosis and hypoxemia are corrected. Medical management focuses on stabilizing the cardiorespiratory system and improving pulmonary circulation by improving oxygenation, correcting metabolic acidosis, and reducing right-to-left shunting of blood. However, cardiorespiratory stabilization should not come at the expense of further damage to the lung (volutrauma or barotrauma) from overly aggressive ventilatory strategies.

It is best to avoid attempting to ventilate the lungs with a bag-and-mask before intubating the trachea at birth because doing so may fill the bowel and stomach with gas, which will further decrease lung compliance and increase the risk of volutrauma and barotrauma in the only functioning lung. Early tracheal intubation and decompression of the stomach via an oral gastric tube are important initial steps to prevent/treat distention of the bowel and stomach. Echocardiography, if available, can help document associated cardiac anomalies, pulmonary hypertension, pulmonary blood flow, right-to-left shunting of venous blood, and right heart dysfunction.

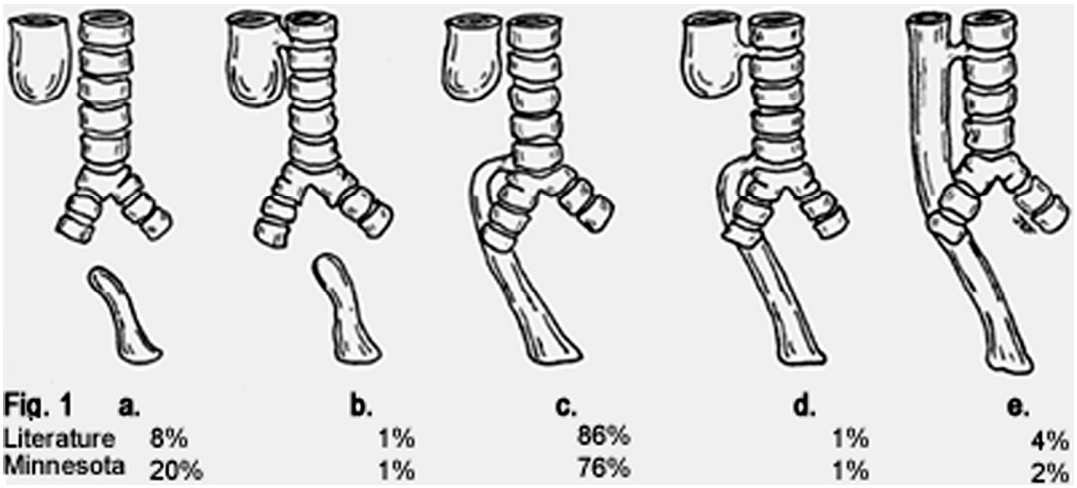
The anesthetist's goals during anesthesia are to provide sufficient analgesia and an adequate depth of anesthesia with drugs that have minimal effects on pulmonary vascular resistance and myocardial function. The ventilatory goals during surgery are the same as those before surgery, optimizing acid–base status and oxygenation, decreasing right-to-left shunting of blood, and avoiding changes in the pulmonary circulation by minimizing barotrauma or volutrauma. It is usually best to avoid making large changes in the mechanical ventilation established by the neonatologists preoperatively unless it is absolutely necessary to do so. They have spent a considerable amount of time finding the best ventilation pattern and inspired oxygen concentration for the patient. Making unnecessary changes in ventilation can be disastrous for the patient. If there is rapid deterioration in the patient's cardiorespiratory status during surgery, it should be suspected that he/she is having a pulmonary hypertensive crisis or a pneumothorax. A pulmonary hypertensive crisis can sometimes be treated acutely with hyperventilation, but

hyperventilation should only be used for this purpose when absolutely necessary. Meticulous attention should be paid to the newborn's temperature, as hypothermia may further increase pulmonary vascular resistance and decrease pulmonary blood flow. Nitrous oxide is usually avoided in these patients because they require higher inspired oxygen concentrations (FiO_2) than can be achieved during nitrous oxide administration. Furthermore, the nitrous oxide may diffuse into the herniated abdominal contents and worsen lung compression. If the neonate is hemodynamically stable and an anesthesia machine is available, low-concentrations of inhaled anesthetic may be administered. However, it is often better to use a high-dose narcotic technique (e.g., Fentanyl 100-200mcg/kg titrated to effect) with a muscle relaxant, because this combination of drugs usually causes less hemodynamic instability by reducing/preventing the release of catecholamine. Catecholamines raise pulmonary vascular resistance and initiate a pulmonary hypertensive crisis. If the patient is given high doses of narcotics and is paralyzed with muscle relaxants, he/she will certainly require postoperative mechanical ventilation and intensive care. Adequate postoperative pain relief is important because untreated pain also worsens hypoxemia and carbon dioxide retention by limiting movement of the chest and diaphragm.

Tracheoesophageal Fistula and Esophageal Atresia

Patients with a *Tracheoesophageal Fistula (TEF)* and *Esophageal Atresia* have an interruption of the esophagus and usually have an abnormal connection between the trachea and a portion of the esophagus. Other congenital anomalies may also be present. For instance, approximately 25% of them have congenital heart disease. Many have the *VATER* association, which includes *V,* vertebral defects; *A,* anal defects; *T,* tracheoesophageal fistula; *E*, esophageal atresia; and *R,* radial or renal anomalies or the *VACTERL* association, which includes *C,* cardiac, and *L,* limb anomalies in addition to those in the VATER association. The most common type of TEF (about 90%) is type C, where a fistula exists between the trachea and the lower esophageal segment (**Figure 11-**2). The upper esophageal segment ends in a blind pouch in the mediastinum. The opening between the trachea and the esophagus is usually posterior and is just above the bifurcation of the trachea. This connection may allow gastric fluid to enter the lung and cause aspiration pneumonia.

Figure 11-2: Tracheoesophageal Fistulae



This figure shows the different types of T-E fistulas. It also gives the incidence of each type of fistula in general and at the University of Minnesota. Type C is the most common form. http://www1.umn.edu/eatef/whatis.html

TEF should be suspected if there is maternal polyhydramnios (too much fluid in the amniotic sac) or a TEF is seen on prenatal ultrasound. Polyhydramnios occurs because the esophageal obstruction prevents the baby form swallowing amniotic fluid and the fluid normally produced by the lungs each day. Newborn infants with TEF have excessive salivation, drooling, cyanotic spells, and coughing and choking following feeding because fluid or milk enters the lungs through the fistula. Esophageal atresia can usually be confirmed in the delivery room because it is impossible to pass a suction catheter from the oropharynx into the stomach. If a plain radiograph of the chest and abdomen is available, the stomach is often distended with air that entered the stomach via the TEF. The lungs should be protected from aspiration of fluid or gastric contents through the fistula by avoiding feedings, positioning the infant upright to prevent reflux of gastric contents through the fistula into the lungs, and by intermittently or continuously suctioning secretions from the upper esophageal pouch. Other anomalies should be sought preoperatively, particularly cardiac anomalies. Complete repair of a TEF is often accomplished in one-stage; the fistula is ligated and the proximal and distal ends of the esophagus are then connected. However, the fistula ligation and esophageal repair should only be done at the same time if the bowel anastomosis can be accomplished without causing excessive tension on the suture line. If this happens, the suture line may break down. If the infant has other significant anomalies or if her/his ventilation is impaired by gastric distention due to air entering through the fistula, a gastrostomy is performed under local anesthesia to decompress the stomach and minimize aspiration of gastric contents. When this is done, definitive repair if the TEF is usually performed 24-to-72 hours later. The surgical approach for repair of a TEF is a right thoracotomy, unless the aortic arch is known to be right-sided.

The major anesthesia consideration during TEF repair is maintaining adequate ventilation of the lungs without forcing gas through the fistula into the stomach and bowel. This must be avoided. Spontaneous breathing of inhaled anesthetics for the induction of anesthesia allows reduces the risk of gastric distention and worsening hypoxemia and acidosis. The tracheal tube is placed following direct laryngoscopy. Correct placement of the tube is confirmed by auscultating both lung fields and by listening over the stomach. If breath sounds are loud over the stomach (there should always be some breath sounds heard there) the tube is proximal to the TEF and should be advanced slowly until the breath sounds over the stomach diminish or disappear. One way to place the tracheal tube in the correct position is to insert it into the trachea with the long part of the tube's bevel posterior. The tracheal tube is advanced into the right main-stem bronchus while someone listens with a stethoscope over the stomach. The tube is then slowly withdrawn until loud breath sounds are heard over the stomach. Then the tube is advanced into the trachea until the breath sounds over the stomach decrease or disappear, and the breath sounds of both lungs are heard equally. These patients should be allowed to breath spontaneously if possible, but the patient's cardiorespiratory status may require muscle relaxants and gentle positive pressure ventilation to assure adequate oxygenation and removal of carbon dioxide. It is important to assure that the fistula is still occluded by the TT after the patient is turned on her/his side for surgery. This is done by listening for breath sounds over the down lung and by hearing poor or no breath sounds over the stomach. Attaching a stethoscope to the chest wall over the down lung allows the anesthetist to detect changes in ventilation during surgery. If a gastrostomy was placed preoperatively to decompress the stomach, correct positioning of the TT can be confirmed by submerging the end of the gastrostomy tube 1-2cm into a container of water and looking for gas bubbles during ventilation. If bubbles are seen, the tip of the TT is proximal to the fistula. If no gas bubbles are observed, the TT is distal to the fistula. The gastrostomy tube can also be connected to a capnograph, if available. If the endotracheal tube is proximal to the fistula, there will be CO_2 in the gas aspirated from the gastrostomy; if the tip of the tracheal tube is distal to the fistula, no CO_2 is observed. Collapse and retraction of the lung during the thoracotomy will impair ventilation. Blood clots and/or secretions in the airway may occlude the tracheal tube, which will necessitate frequent suctioning of the tube during surgery. An arterial catheter, if available, should be inserted in newborns whose cardiorespiratory status is unstable and in those with associated anomalies, especially cardiac anomalies. After the fistula is ligated, an air leak will develop, potentially impairing ventilation; the air leak lasts until the esophageal anastomosis is completed. During surgery a catheter is usually passed into the blind upper pouch to identify the upper esophagus. Under direct vision the surgeon advances it into the distal esophagus during surgery and performs the esophageal anastomosis around this catheter. When the anastomosis is complete, the catheter is withdrawn until its tip is just above the suture line; the distance from the tip of the catheter to the lips is determined and a mark is made on the catheter; catheters longer than this should not be used to suction the esophagus postoperatively, otherwise the catheter may disrupt the suture line. If the distal portion of the esophagus is absent or too short

to be anastomosed to the proximal segment, the fistula is ligated, and the upper segment of the esophagus is sometimes externalized via an esophagostomy to drain saliva. The two segments of esophagus can be bridged with an interposed bowel segment or tube graft at about one year of age. The decision to extubate the trachea at the end of surgery requires a discussion between the surgeon and the anesthetist. They must balance the child's cardiorespiratory status, healing of the esophageal anastomosis, and the availability and need for postoperative mechanical ventilation and intensive care (ICU). Traditionally, the trachea was extubated when the patient had awakened from anesthesia, but this was found to be associated with an increased risk of intraventricular hemorrhage (IVH).[3] Tracheomalacia could sometimes occur at the fistula site and cause the trachea to collapse during inspiration. If this occurs it may be necessary to leave a tracheal tube in place and ventilate the child's lungs after surgery.

Congenital Cystic Lung Disease

Congenital cystic lung disease occurs in 1/10,00-1/25,000 live births and may be categorized as congenital lobar emphysema (CLE), bronchopulmonary sequestrations (BPS), congenital cystic adenomatous malformations (CCAM), and bronchogenic cysts (BC). Small lesions may be asymptomatic, but larger lesions may cause respiratory distress in the newborn. Initially asymptomatic lesions may become infected or cause a pneumothorax; a malignant tumor may develop later in life. Serial imaging has revealed, however, that many large lesions may actually decrease in size with time. While there is consensus that all symptomatic lesions should be resected, there is some debate whether asymptomatic lesions should be observed rather than resected. If asymptomatic CCAMs are followed conservatively, only 10% of them will require surgery later. It is therefore still recommended that a CCAM, intralobar BPS, or BC is resected between 3-6 months of age. Extralobar BPS can remain asymptomatic for life but can also be associated with problems. A period of observation has been advocated. Asymptomatic CLE may resolve spontaneously and should be observed.

When anesthetizing neonates with these congenital lung lesions, it is important to determine if the patient will tolerate positive pressure ventilation (PPV). PPV in lesions that have a bronchial connection to abnormal lung parenchyma can result in over-distension of the abnormal lobe and compression of normal lung tissue through a ball-valve effect. This can lead to compromised ventilation, mediastinal shift, compression of the great vessels, and decreased cardiac output. When there is doubt whether a bronchus connects to the thoracic lesion, spontaneous ventilation is indicated during induction and maintenance of anesthesia. Generally, CCAM and CLE have a bronchial connection. (**Table 11-1**)

Table 11-1: Whether a Bronchus Connects to the Lesion and Whether Positive Pressure Ventilation Can Be Done.

Lesion	Connects with Bronchus	Positive Pressure Ventilation OK?
Cystic Adenomatoid Malformation	Yes	No
Congenital Lobar Emphysema	Yes	No
Bronchopulmonary Sequestration	No	Yes
Bronchogenic Cyst	No	Yes

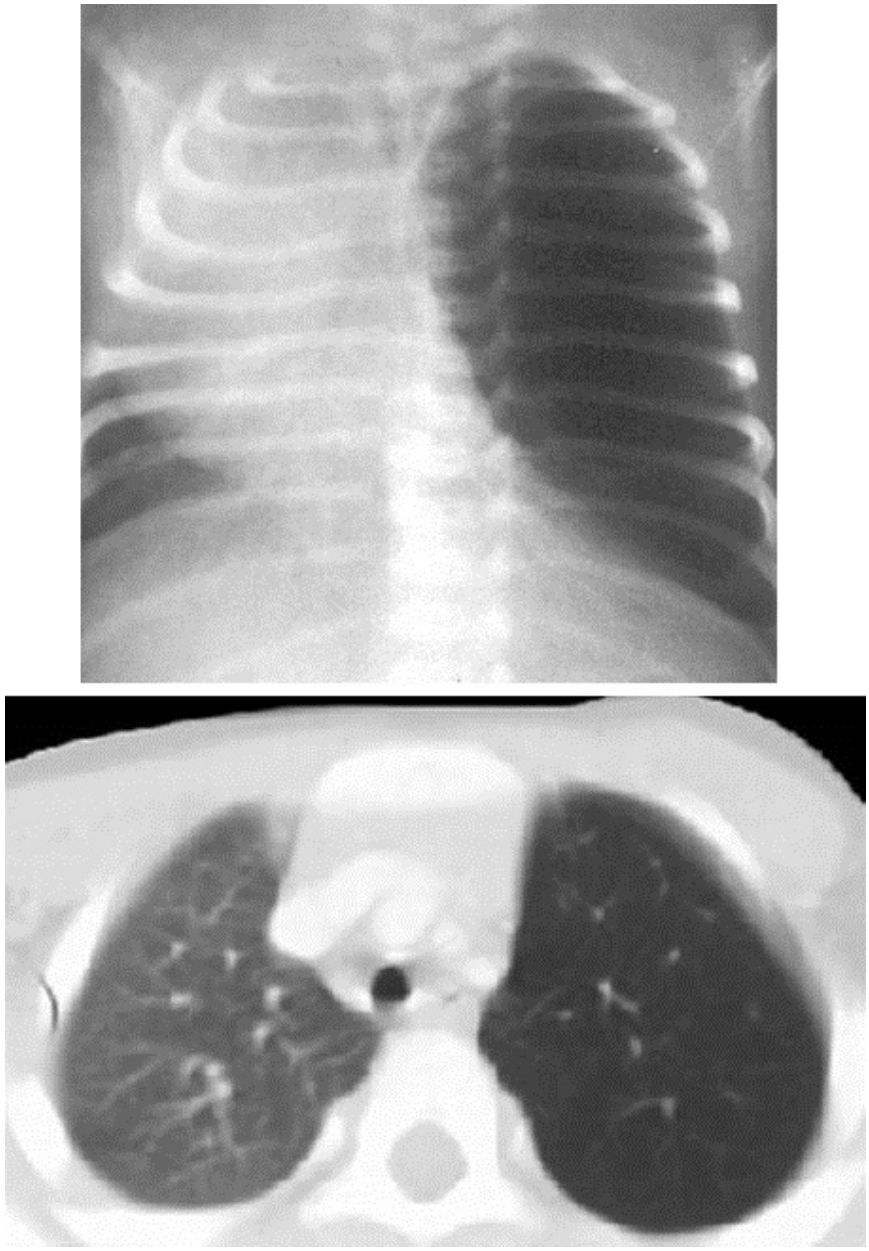
This table lists common thoracic lesions and whether they are connected to a bronchus. If they are connected, it is possible to over distend the affected lesion with positive pressure ventilation and cause further respiratory distress.

If OLV is used PPV and neuromuscular blockade can be safely used once the affected lung is isolated. For postoperative analgesia, intravenous opioids or, preferably, a thoracic epidural can be placed at the desired dermatome level. A catheter can also be threaded to the desired position from the caudal canal (**See Chapter 21**). Recently more congenital lung lesions have been resected using video assisted thoracic surgery (VATS). While this technique is challenging in infants and results in significantly longer operative time, it is safe and may reduce hospital stay.

Congenital Lobar Emphysema (CLE)

CLE is an abnormally emphysematous pulmonary lobe that communicates with the bronchial tree (**Figure 11-3**). It is most common in the left upper lobe, followed by the right middle lobe and the left lower lobe. It often enlarges before 28 weeks gestation because fetal lung fluid becomes trapped within the lesion, similar to postnatal air trapping. Between this time and birth, the lesion may regress and result in a normal looking lung at birth.

Figure 11-3: Congenital Lobar Emphysema



This radiograph and CT scan show CLE taking up most of the left lung.

Even though usually asymptomatic, it is important that patients with CLE are carefully evaluated at birth, since they are at risk for trapping air in the emphysematous lobe. Overinflation of the lung lesion may ultimately lead to "tension emphysema" and compression of the contralateral lung. At this stage it can be confused with a tension pneumothorax, and a chest tube may be inappropriately placed, leading to further respiratory distress. Large lesions may decrease cardiac output and eventually cause cardiac collapse. These patients need emergent thoracotomy and rapid lobe exteriorization.

The primary anesthetic concern for patients with CLE is avoiding overdistension of the effected lung tissue. PPV can cause rapid expansion of the lesion through a ball-valve effect, so an effort should be made to allow the patient to breath spontaneously. If PPV is necessary, low inflating pressures should be used. OLV is typically necessary for isolation of the affected area, and once initiated it is safe to switch to PPV. Nitrous oxide should be avoided throughout the case. At the end of the procedure one should return to two-lung ventilation to check for air leaks at the resection site. The trachea should be extubated early or, if the patient is to remain intubated, he/she should be allowed to breathe spontaneously to avoid reduce the risk of an air leak occurring.

Bronchopulmonary Sequestration (BPS)

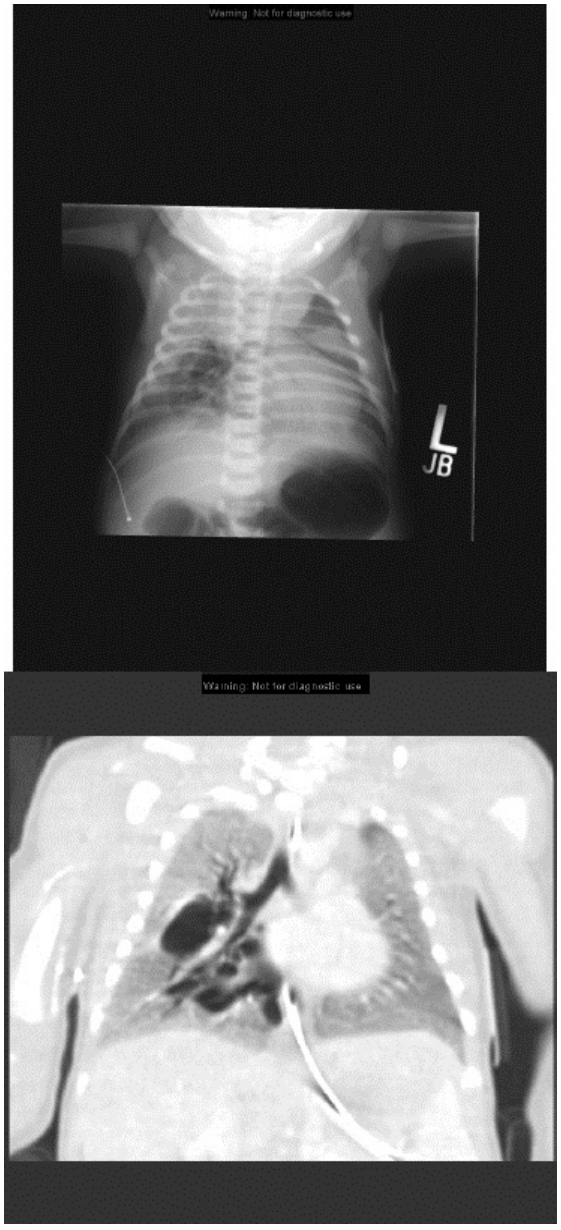
A BPS is a portion of nonfunctioning lung tissue without a bronchial connection. It typically has an anomalous blood supply. BPS is usually found in the lower lung lobes, with the majority of these lesions found intralobar (inside lobe pleura); the remainder of them is extralobar (with their own pleura). BPS can be confused with CCAM and some lesions are considered 'hybrid', having features of both BPS and CCAM.

BPS is often diagnosed in utero. At birth it is usually asymptomatic and presents later in life as pneumonia resistant to antibiotic therapy. Sometimes when a BPS is large, it can compress the lungs and cause respiratory distress. If it has a large blood supply, it can also lead to high output heart failure. There are few unique anesthesia concerns. OLV is helpful for surgical resection. Because there is no connection between a bronchopulmonary sequestration and the bronchial tree, it is safe to use positive pressure ventilation throughout anesthesia and surgery.

Congenital Cystic Adenomatous Malformation (CCAM)

A CCAM is a discrete intrapulmonary mass that may be either solid or cystic, and is typically characterized by increased adenomatous respiratory bronchioles. Cysts can be of various sizes, from 1mm to over 10cm and can be diagnosed by computerized tomography (CT). **Figure 11-4**. Although the lesion is nonfunctional, it communicates with the normal tracheobronchical tree and this can lead to air trapping during positive pressure ventilation. CCAMs are usually found only in one lobe but occur in all lobes with equal frequency. When the lesion does involve more than one lobe, a pneumonectomy may be required. Associated anomalies are uncommon. Most CCAMs are asymptomatic and are resected electively in the neonatal period. While there is a communication between the lesion and the tracheobronchical tree, CCAMs are usually solid or have small cysts, which causes them to act more like a solid lesion. It is safe to use positive pressure ventilation in these patients. Most lesions can be resected without lung isolation, but if necessary, OLV can be accomplished by intubating a mainstem bronchus.

Figure 11-4: Congenital Cystic Adenomatous Malformation



Radiograph and CT scan of a CCAM. Notice that the lesion occupies a significant portion of the right lower lung.

Bronchogenic Cysts (BCs)

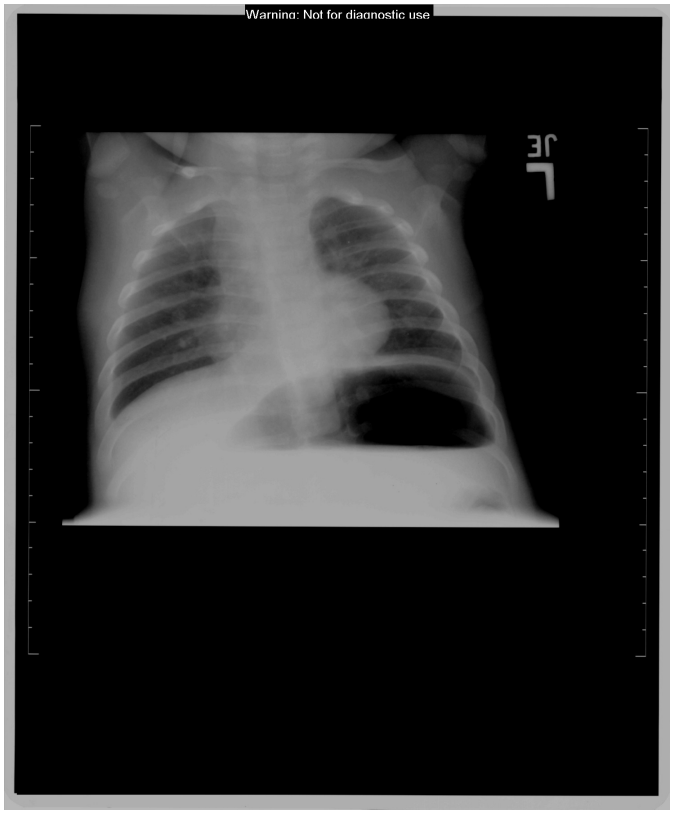
BCs are the result of abnormal budding or branching of the tracheobronchial tree. They are usually mediastinal, solitary, unilocular cysts filled with air, fluid, or mucous. They do not communicate with the bronchopulmonary tree and therefore, like bronchopulmonary sequestrations, pose few additional anesthetic concerns. Positive pressure ventilation can be used.

Thoracic Surgery for Older Children

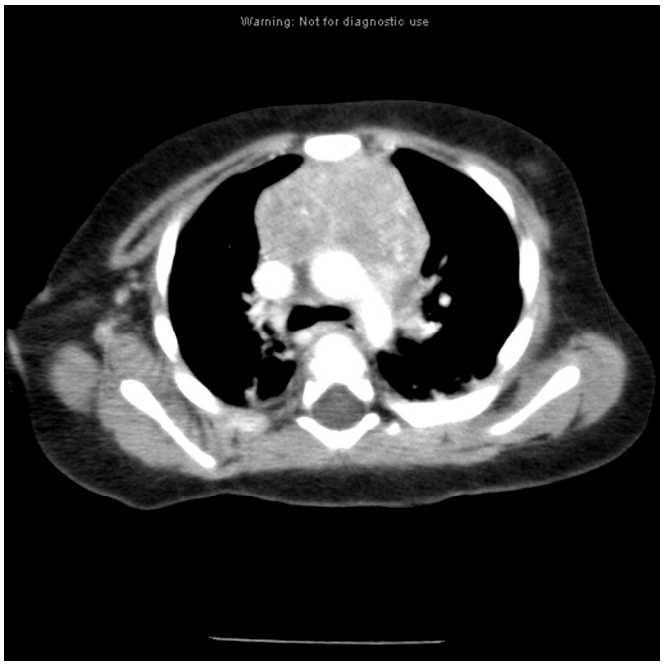
Mediastinal Tumors

The mediastinum is divided into anterior, middle, and posterior segments. Anterior mediastinal masses are usually neoplasms of the lung, mediastinum, or pleura, including lymphomas, lymphangiomas, teratomas, thymomas, and thymic cysts that usually occur after 4-5 years of age. Middle mediastinal masses are usually bronchogenic cysts, granulomas, and lymphomas and may or may not cause significant respiratory symptoms, depending on whether or not they compress the trachea, bronchi, and blood vessels. Posterior mediastinal masses are usually enteric cysts or tumors of neuroendocrine origin and less frequently cause respiratory complications. Signs and symptoms from mediastinal masses are related to compression of the airway, great vessels, and heart. These symptoms include dyspnea, orthopnea, chest pain, cough, wheezing, stridor, and swelling of the upper arms, face, and neck (*superior vena cava syndrome*). Orthopnea and superior vena cava syndrome are the symptoms associated with the highest risk of perioperative complications. The patient's history and physical examination should focus not only on signs and symptoms of airway, great vessel, and heart compression, but also on changes in the patient's symptoms when he/she changes body position and activity. Do the symptoms worsen or get better when the patient sits or lies in one position or another or exercises? A chest radiograph may show a widened mediastinum; a CT scan may demonstrate the mass size and the presence and degree of airway or great vessel compression (**See Figure 11-5**).

Figure 11-5: Anterior Mediastinal Mass.



Radiograph showing a widened mediastinum that is caused by the tumor.



CT scan showing an anterior mediastinal mass compressing the trachea and great vessels.

Establishing the correct diagnosis of the tumor type requires a tissue biopsy, and many oncologists require this before beginning chemotherapy, radiation therapy, or surgical intervention. The anesthetist should determine if the procedure is diagnostic or therapeutic because diagnostic tissue biopsies can often be obtained from a peripheral lymph node (if there is one) or from bone biopsy and can be obtained under local anesthesia or minimal sedation, making general anesthesia unnecessary. Ketamine is often used when obtaining these biopsies because it allows spontaneous ventilation and provides hemodynamic stability. Elective surgical procedures that require general anesthesia should be delayed until chemotherapy and/or radiation therapy have reduced the mass size. Therapeutic procedures, on the other hand, frequently necessitate using general anesthesia. Induction of anesthesia can be associated with immediate and severe airway obstruction and circulatory collapse. *It is important to remember that airway obstruction and circulatory collapse can occur even in children with anterior mediastinal masses who have no preoperative respiratory or cardiovascular compromise.* During the induction of anesthesia and during surgery, the child should be placed in the position that maximizes ventilation, minimizes airway obstruction, and reduces both hypotension and hypoxemia. Induction of anesthesia with inhalation drugs and spontaneous ventilation is common because it maintains normal transpulmonary pressure gradients and gas flow through the conducting airways. Continuous positive airway pressure (CPAP) is often applied to stent open the airways and prevent atelectasis from occurring. Tracheal intubation should be performed while the patient is deeply anesthetized and spontaneously breathing. Positive pressure ventilation is avoided whenever possible. Muscle relaxants are seldom used in these children because these drugs decrease chest wall tone and this increases the risk of airway compression. If obstruction occurs or worsens with the induction of anesthesia, changing the child's position to the lateral, recumbent, or prone position or intubating the trachea and advancing the TT past the obstruction may alleviate airway obstruction and make ventilation possible. This usually does not alleviate obstruction of the great vessels.

Practical Considerations for Anesthesia for Thoracic Surgery

The Lateral Decubitus Position

In the lateral decubitus position, ventilation is preferentially distributed to the dependent (down) lung of awake, spontaneously breathing older patients. The distribution of perfusion follows a similar pattern, with more blood flow occurring in the down lung. Thus, ventilation and perfusion are well matched. In infants, however, ventilation is preferentially distributed to the non-dependent (up) lung. Because the hydrostatic pressure gradient between the up lung and the down lung of infants is small, the difference in blood flow between the two lungs is small. Ventilation and perfusion are, therefore, not as well matched in infants as in older patients.

Several factors in the lateral decubitus position significantly affect ventilation/perfusion (V/Q) matching during thoracic surgery. General anesthesia, neuromuscular blockade, and mechanical ventilation decrease the functional residual capacity (FRC) of both the up and down lungs. The down lung becomes atelectatic because the weight of the mediastinum and abdominal contents compress it. Surgical retraction collapses the operative up lung. Finally, hypoxic pulmonary vasoconstriction (HPV) is inhibited by inhaled anesthetics and by vasodilating agents. In non-anesthetized patients, HPV improves V/Q matching by diverting blood away from poorly ventilated areas. The effects on HPV are similar in patients of all ages.

When adult, spontaneously breathing patients with unilateral lung disease are placed in the lateral decubitus position, they oxygenate better when their healthy lung is down and their diseased lung is up. This occurs in part because blood flow to the up, poorly ventilated, diseased lung is decreased, improving V/Q matching. Infants with unilateral lung disease, however, oxygenate better when the healthy lung is up. Since infants have a soft, easily compressible rib cage that will not support the down lung, the down lung collapses and the FRC is closer to the residual volume. This makes airway closure more likely in the down, compressed lung, even during normal tidal volume breathing. In the lateral decubitus position, infants also have less cephalic displacement of the down hemidiaphragm by their abdominal contents. Therefore the contraction force in the down hemidiaphragm is less than that of the up hemidiaphragm, limiting the ventilation efficiency of the down lung. Ventilation is distributed preferentially to an infant's up lung. The hydrostatic pressure gradient between the up and down lungs is smaller, so the preferential distribution of perfusion to the down lung is less than that of adults.

One-Lung Ventilation

Indications for One-Lung Lentilation (OLV) Include:

(1) Controlling the distribution of ventilation,

(2) Avoiding spillage of infected material or contamination of the other lung, and

(3) Providing a quiet operative field for the surgeons.

Many techniques are available to provide OLV in children. The surgeon may retract the operative lung mechanically, pack it with sponges, or insufflate CO_2 when the procedure is performed thoracoscopically. All of these maneuvers collapse the lung. Advancing a standard endotracheal tube (TT) into the non-operative bronchus is the simplest way to achieve OLV. When passed blindly the TT usually enters the right main-stem bronchus but can be selectively advanced into the left main stem bronchus by rotating the child's head to the right and then rotating the TT so the beveled portion of the tube faces right while the TT is advanced. This places the long, pointed portion of the bevel to the left. With selective endobronchial intubation, it is possible to quickly change from OLV to two-lung ventilation, since the TT can easily be pulled back into the trachea.

Using a standard TT may not be ideal, however. If a small, uncuffed TT is used, it may be difficult to completely seal the operative main-stem bronchus. With the tube in the opposite main-stem bronchus, the lung on which surgery is being performed cannot be suctioned, and hypoxemia may occur if the upper lobe bronchus becomes obstructed, particularly on the right.

Devices Used to Permit OLV:

Bronchial blockers have a small balloon on the end of a catheter that is inflated in the main-stem bronchus of the lung undergoing surgery. The other lung is then ventilated during surgery via a standard TT. In infants and children less than six years of age, *embolectomy catheters* are often used as bronchial blockers. The catheter can be inserted through or alongside a TT and advanced into the bronchus. The balloon is then inflated and the other lung is ventilated via the TT. Embolectomy catheters have a central stylet that can be bent to the left or right to guide placement of the catheter into the appropriate main stem bronchus. If the embolectomy catheter is accidently dislodged into the trachea during surgery, ventilation may be completely blocked. If this occurs, the balloon should be immediately deflated and both lungs ventilated.

The *Arndt Endobronchial* blocker is a bronchial blocker with an inflatable cuff and a central lumen through which a wire with a loop on its end is passed. The bronchial blocker is passed through a special adapter at the proximal (anesthesia circuit) end of the TT. The adapter contains: (1) a connection for the TT, (2) a standard anesthesia 15mm circuit adaptor, (3) a port that can be tightened around the bronchial blocker to prevent it from moving, and (4) a port for a flexible bronchoscope. (**Figure 11-6**).

Figure 11-6: Arndt Endobronchial Blocker

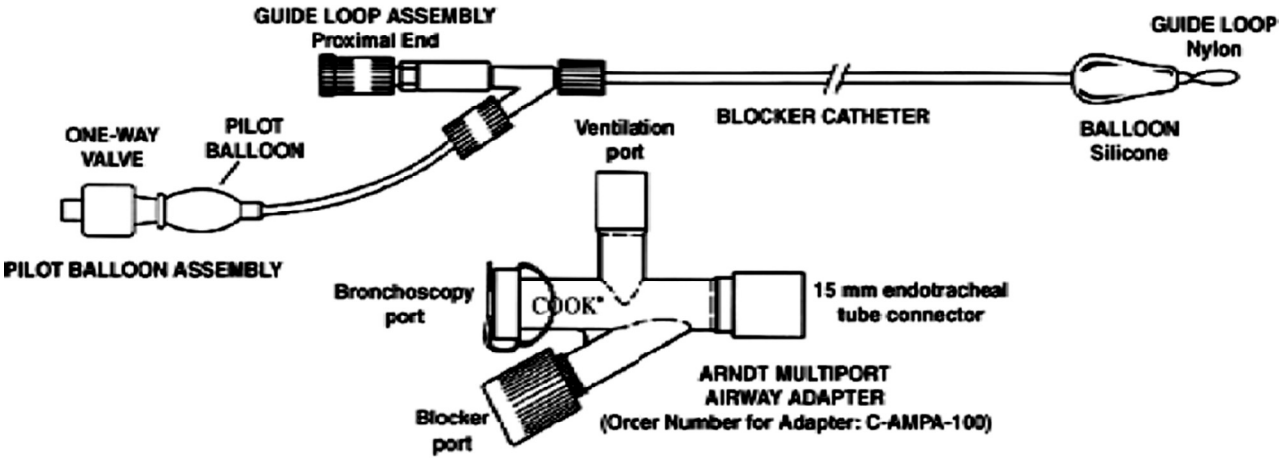
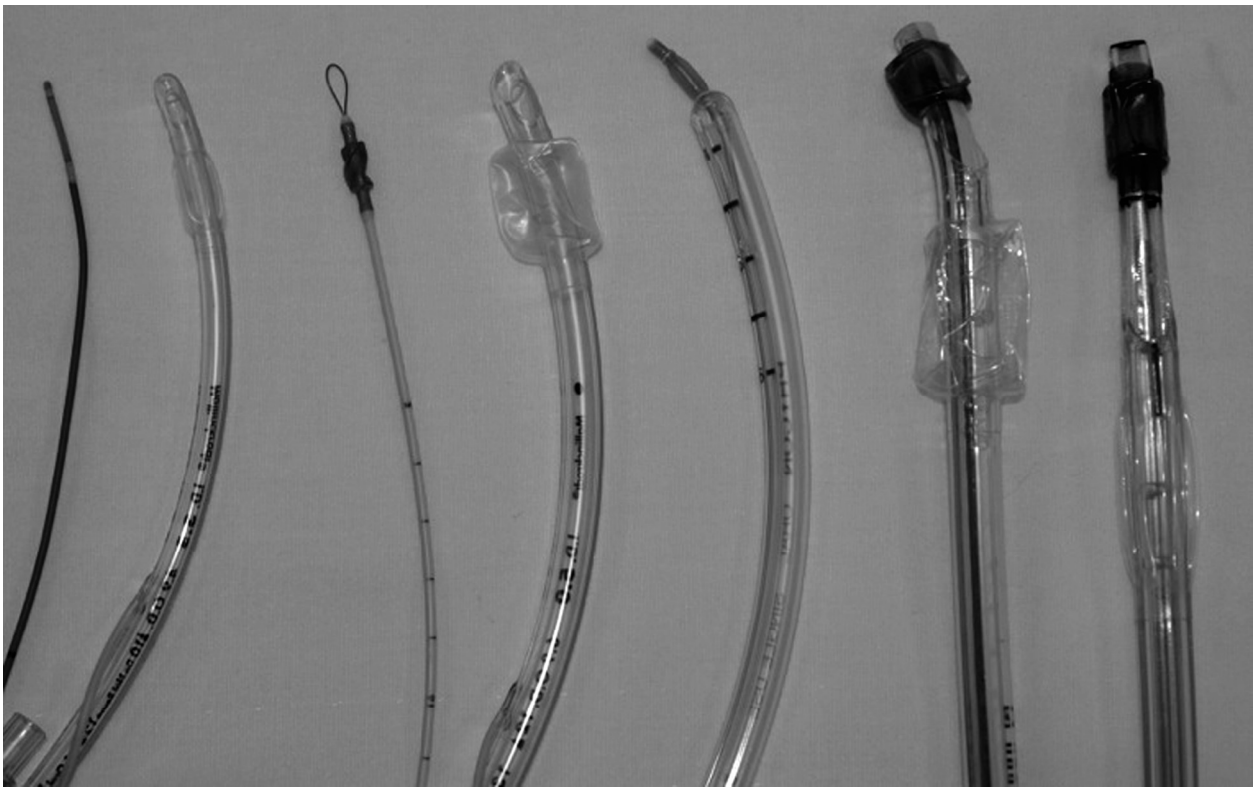


Diagram of Arndt endobronchial blocker (Cook®) and its special airway adapter. Its use requires a flexible bronchoscope.

The flexible bronchoscope and the blocker are passed under direct vision through the TT into the

operative main stem bronchus. The bronchoscope is then withdrawn into the trachea and the balloon inflated, again under direct visualization. Next, the guide wire is removed from the central channel. Once the wire has been removed, it cannot be replaced. The smallest endobronchial blocker available is 5-French, and the smallest TT that can be used with a 5 French blocker is one with a 5.0mm internal diameter.

Figure 11-7: Devices That Can Be Used to Allow One Lung Ventilation



Tubes used for one lung ventilation: From left to right: 1) Embolectomy catheter; 2) Standard 3.5mm TT; 3) Arndt endobronchial blocker; 4) Standard 5mm TT; 5) Univent tube; 6) Double-lumen tube - left; 7) Double-lumen tube – right.

The *Univent tube* is a single-lumen standard TT with a moveable bronchial blocker in the sidewall (**Figure 11-7**). The bronchial blocker contains a low-pressure-high-volume cuff and a central canal that can be used to suction the isolated lung if necessary. Univent tubes are inserted like standard TTs. The bronchial blocker is then advanced into the operative main stem bronchus. The cuff of the bronchial blocker portion of the tube is inflated to isolate the operative lung. The bronchial blocker can be deflated and withdrawn into the main channel of the tube at the end of the procedure to convert from OLV to two-lung ventilation. The smallest Univent tube available has a 3.5mm internal diameter and an external diameter of 8mm, making it comparable to a 6.0mm internal diameter uncuffed standard TT, far too large for small children but useful for someone who is 10 years of age or older.

Double-lumen tubes (DLTs) are the gold standard for OLV in adults because they allow rapid achievement of OLV, facilitate suctioning of both lungs, allow switching from OLV to two-lung ventilation easily and allow the application of CPAP and the administration of oxygen to the operative lung if necessary for treatment of hypoxemia. However, the smallest DLT available is a 26 French, which is appropriate for children 8-10 years of age. If a child needs postoperative mechanical ventilation, the DLT must be replaced with a standard TT. A Univent tube, on the other hand, can be left in place with the blocker withdrawn into the main channel of the tube.

OLV should be initiated as soon as possible after the bronchial blocker is in place so there will be sufficient time for the blocked lung to deflate and become atelectatic. Peak airway pressures should initially be confirmed with two-lung ventilation and then reconfirmed with OLV to be sure that excessive airway pressures are not being applied to the ventilated lung. Blocking ventilation to one lung allows the residual oxygen in that lung to be absorbed from unventilated alveoli and cause absorption atelectasis. This is made easier by administering 100 percent oxygen for at least five minutes before initiating OLV. Mismatching of ventilation/perfusion occurs if portions of the unventilated lung continue to be perfused. Fortunately, surgical manipulation, an open hemithorax, and hypoxic pulmonary vasoconstriction reduce blood flow to the unventilated lung and decrease this V/Q mismatch. The ventilation strategy chosen for one-lung ventilation should focus on preventing acute lung injury (ALI). Recommendations for protective lung ventilation include tidal volumes of 6ml/kg, limiting plateau airway pressure to <20cmH_20, and using 5-10cmH_20 of positive end-expiratory pressure (PEEP). The FiO_2 should be high enough to maintain the oxygen saturation (SpO_2) above 94%. Several techniques can be used to relieve hypoxemia should it occur during OLV. CPAP 10cmH_2O can be applied to the non-dependent (up) operative lung. One hundred percent oxygen is insufflated to achieve the desired CPAP level. This usually does not cause sufficient lung inflation to interfere with surgical conditions. The position of the lung isolation device should be reconfirmed by auscultation or by flexible bronchoscopy, if available. If persistent hypoxemia occurs, OLV should be converted to two-lung ventilation. Once the hypoxemia is relieved, OLV can again be re-initiated.

Pneumonectomy

Children with congenital abnormalities, neoplasms, trauma, or infection often require *pneumonectomy* or *partial pneumonectomy*. To decide if a patient will tolerate removing all or part of a lung, a good history and physical examination is very important. The history should focus on how well the patient tolerates walking, moving, and other exercise and whether he/she has evidence of hypoxemia while exercising or especially when at rest. The physical examination should look for signs of airway obstruction, such as wheezing, stridor, or cyanosis. The anesthetist should also look for evidence of a tracheal shift from its normal midline position, asymmetric chest movement, abnormal auscultatory findings, and signs of respiratory distress as evidence of airway obstruction. Children frequently tolerate loss of large amount of lung without symptoms.

However, if the patient develops significant respiratory symptoms after a pneumonectomy, it is an ominous sign. Infants with insufficient functioning lung mass to meet their needs often have nonspecific symptoms, such as poor feeding, irritability, choking, or changes in sleep habits. Insertion of an arterial cannula, if possible, should be done to monitor arterial blood gas and arterial blood pressure during and after a pneumonectomy. OLV is usually required for pneumonectomy. Preservation of lung capacity and function following a pneumonectomy decreases with increasing age. Consequently, babies do better than 20-year olds.[4]

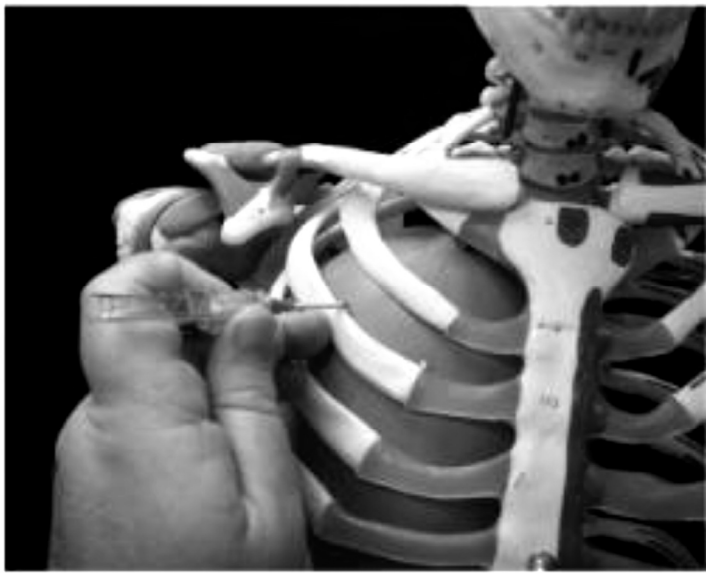
Post pneumonectomy syndrome is due to progressive hyperinflation of the remaining lung. This leads to increasing dyspnea, bronchomalacia, and recurrent lung infections. This syndrome is common after a right thoracotomy, due to counterclockwise rotation of mediastinal structures and compression of the left main stem bronchus or left lower bronchus between the aorta and spine posteriorly and pulmonary artery anteriorly. To correct this problem, mediastinal structures are sutured to the anterior chest wall to lift the vessels off of the bronchi.

Chest Trauma & Pneumothorax

Pulmonary contusion and rib fractures constitute 50% of pediatric chest trauma. Twenty percent of patients with chest trauma have a pneumothorax. If it impairs breathing and/or circulation, chest trauma can be life threatening. Life threatening injuries include tension pneumothorax, open pneumothorax, flail chest, and direct airway injuries.

A *tension pneumothorax* occurs when gas accumulates in the pleural space, is trapped there, and impairs both breathing and return of blood to the heart. The trachea is deviated and breath sounds are decreased on the affected side. The heart sounds are often shifted to the unaffected side. Hypotension and the need to use higher airway pressure to ventilate the lungs are often present. Relieving the high intrapleural pressure caused by the pneumothorax is the first priority. This can initially be accomplished by inserting a large-bore intravenous catheter into the pleura through the second intercostal space in the mid-clavicular line (**Figure 11-8**). This will immediately reduce the intrapeural pressure and improve ventilation and perfusion until a chest tube can be inserted. The cut-off finger of a glove can be attached to the catheter to act as a flap valve until a chest tube can be inserted. During inspiration the piece of glove is pulled over the end of the catheter. During expiration it is pushed away to allow the exit of gas.

Figure 11-8: Inserting a Needle to Relieve a Pneumothorax.



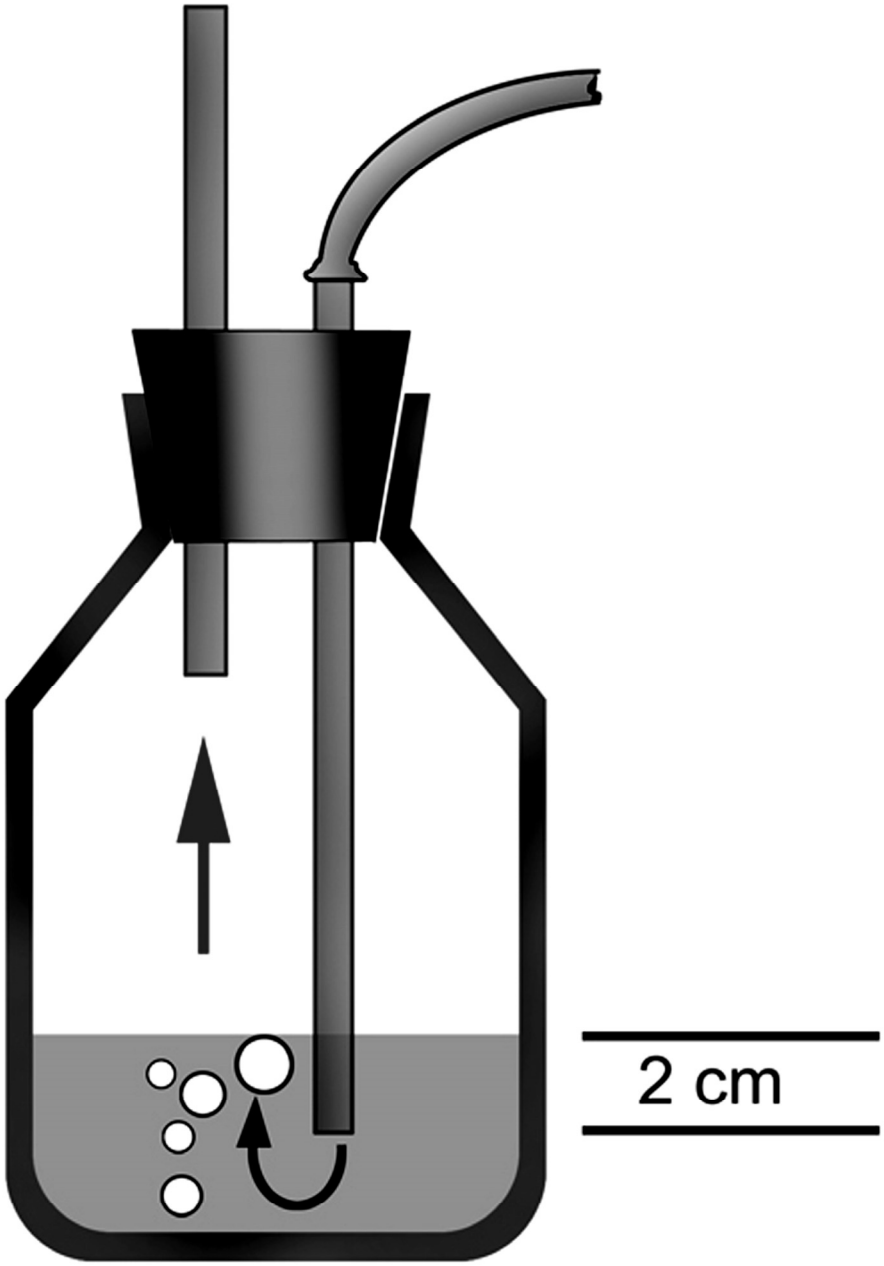
A large bore needle is being inserted over a rib (to avoid injuring the intercostal vessels) to relieve a pneumothorax. The hand is steadied against the rib cage during needle insertion.

An *open pneumothorax* occurs when a chest injury produces a hole in the chest and allows the pressures between the pleural space and atmosphere to equalize. The lung beneath the hole collapses; air moves through the chest wall defect (hole) during breathing. An occlusive dressing should be placed over the defect and a chest tube inserted. If a chest tube is not immediately available, a dressing that is closed on three sides may act as a flap valve to prevent more air from entering the chest.

A *flail chest* occurs when multiple ribs are fractured. During inspiration, the fractured area moves inward while the remaining chest moves outward. This interferes with ventilation.

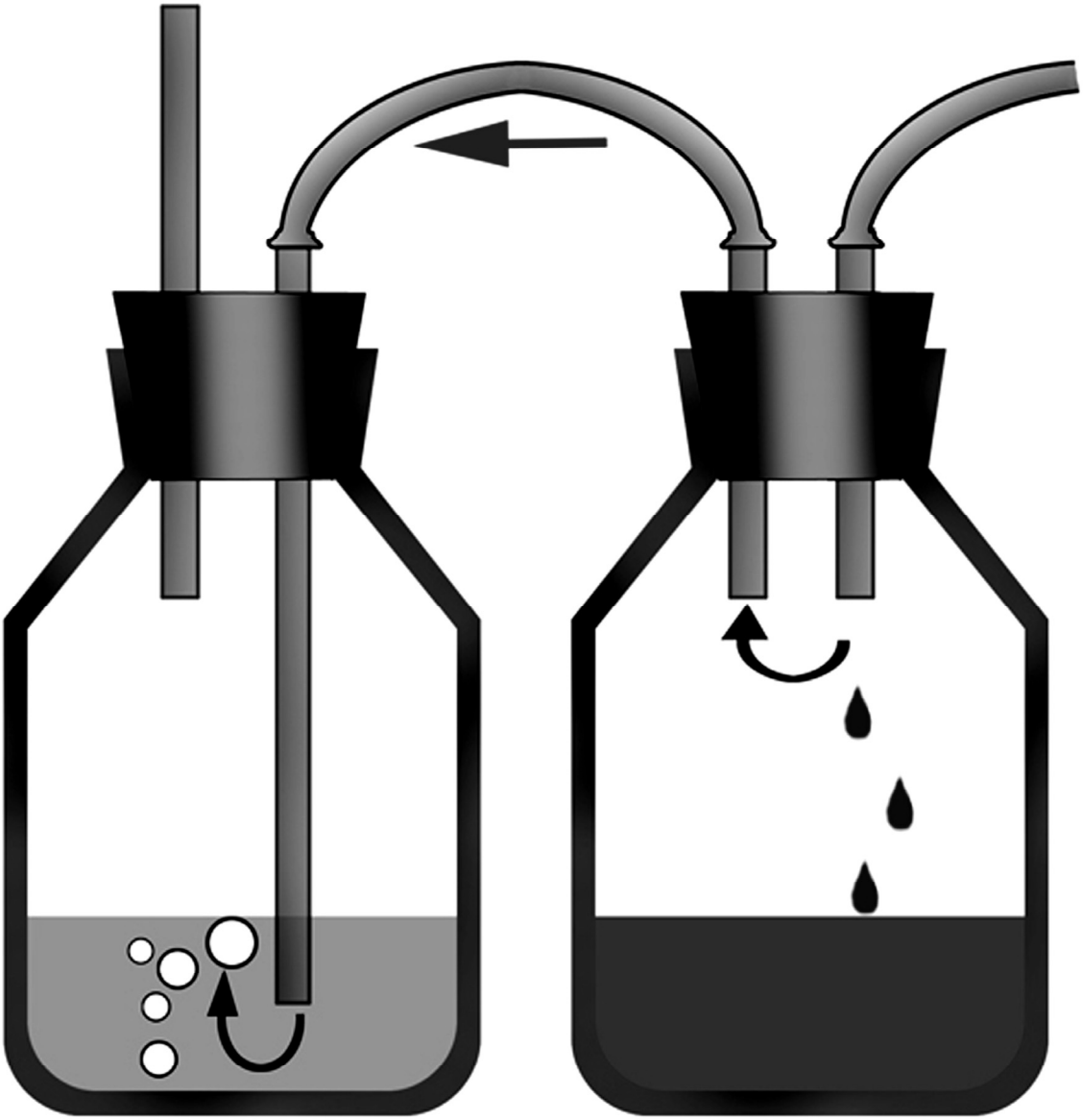
It is usually necessary and desirable to connect a chest tube to a suction bottle system to remove the drained gas or fluid/blood from the pleura and re-expand the collapsed lung. Below are three systems used for this purpose (**Figures 11-9 to 11**).

Figure 11-9: Single Bottle Under Water Seal



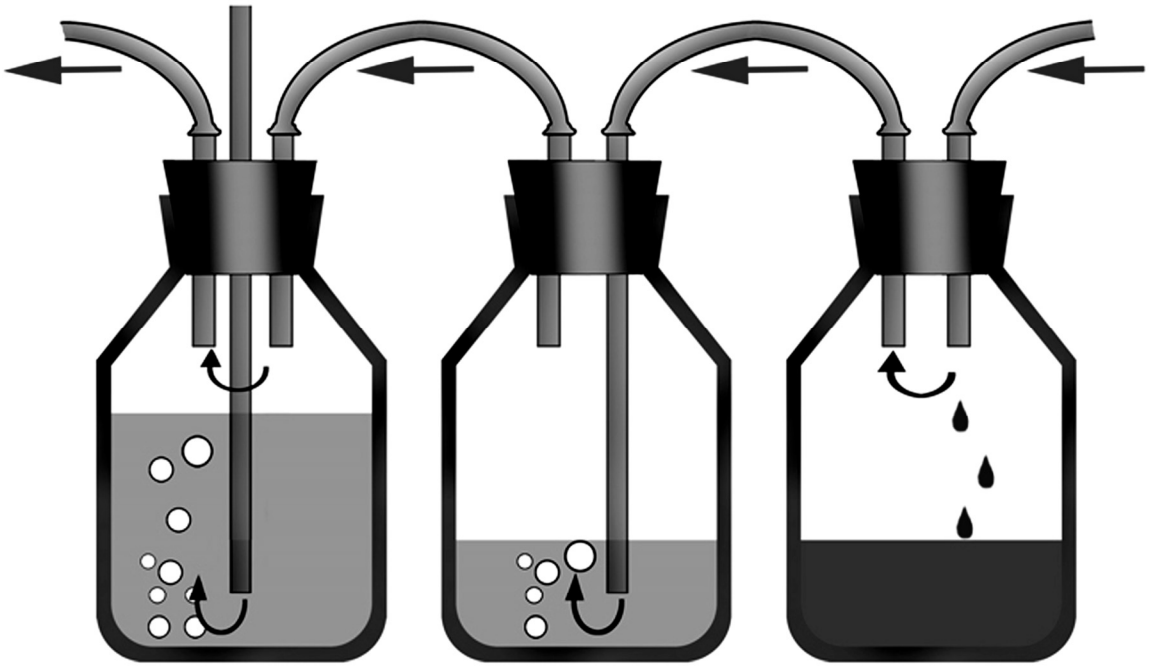
This is a schematic diagram of an underwater seal bottle containing sterile water. A chest tube is attached to the tube that is under water. The bottle is placed 100cm or more below the level of the patient to generate sufficient negative pressure to relieve the pneumothorax. A single bottle works well for an isolated pneumothorax when suction is not available. The collapsed lung may not re-expand as quickly as it would if suction is applied. Asking the patient to repeatedly perform the Valsalva maneuver (take in a deep breath and forcefully exhale) can aid in re-expanding the collapsed lung more quickly. *http/::emedicine.medscape.com:article:1503275-overview - a09*

Figure 11-10: Two-Bottle Suction System



A two-bottle drainage system is needed when fluid or blood must be drained from the chest. The bottle on the right is for fluid and/or blood collection. http/::emedicine.medscape.com:article:1503275-overview - a09

Figure 11-11: Three-Bottle Drainage System



If suction is required and available, a third bottle is added to the system. The maximum amount of suction produced is determined by how far the atmospheric vent is placed underwater in the suction regulation bottle. The bottle on the right is for collecting fluid, and blood. The center bottle is a water seal. When the patient inhales and produces a negative pressure, fluid is drawn into the under tube and air is prevented from passing back into the pleural cavity. Applying suction to the bottle on the left helps remove gas and fluid from the lung. If there are gas bubbles in the center and left bottle, there is still a gas leak from a hole in the lung. http//:emedicine.medscape.com:article:1503275-overview - a09

CONCLUSION

Providing safe anesthesia for patients who require thoracic surgery requires an understanding of the surgical lesion and the effects of these lesions on lung function, oxygenation, and the ability to remove CO_2. This chapter has reviewed the neonatal thoracic surgery conditions of congenital diaphragmatic hernia, tracheoesophageal fistula, and congenital cystic lung disease. It has also reviewed childhood mediastinal tumors. The pathophysiology of one-lung ventilation has been explained, as well as practical advice on techniques and equipment needed to safely deliver OLV. Lastly, management of traumatic pneumothorax, including setting up a chest drainage system, has been discussed.

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Chapter 12
Anesthesia for Patients with Congenital Heart Disease

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Introduction

Congenital heart disease (CHD) is the most common birth defect requiring surgical or other invasive intervention. It occurs in approximately eight per 1000 live births throughout the world. Because of this, anesthetists caring for children require a basic understanding of CHD anatomy and pathophysiology. At times, they may have to anesthetize these children for cardiac or non-cardiac surgery. In addition, cardiac repairs are increasingly performed in many parts of the world, especially the complete repair of two-ventricle cardiac lesions where a single surgery can dramatically improve the patient's quality of life and significantly increase her/his life expectancy. This chapter first presents a classification system for CHD and then discuss an approach to its diagnosis, including history and physical examination, laboratory examinations, and diagnostic imaging (focusing on echocardiography). Next, there is a discussion of the major cardiac lesions with pathophysiology, surgical approach, and anesthetic considerations presented. Following this discussion, there is a general discussion of cardiopulmonary bypass management. Finally, the subject of non-cardiac surgery in CHD is summarized.

Even if an anesthetist will not provide care for patients with CHD, an understanding of the principles discussed in this chapter will improve her/his care of other patients. Pediatric patients with normal hearts still encounter problems with poor cardiac function; fluid overload with cardiomegaly; low blood pressure from infection; hypovolemia; or bleeding; and pulmonary hypertension, especially if they are neonates. Some patients not known to have CHD will become hemodynamicaly unstable and subsequntly be discovered to have heart disease.

Classification of Congenital Heart Disease

A common classification of CHD divides these lesions into seven basic categories: 1) Left-to-right shunts, where an obstruction to right sided outflow causes blood to flow through an intracardiac or extracardiac communication from the left side to the right side of the heart; 2) Right-to-left shunts, where obstruction to right heart outflow causes blood to flow from the right side to the left side of the heart through an intracardiac or extracardiac communication; 3) Left sided

obstructive lesions, where flow out the left side of the heart (at any level) is obstructed; 4) Right sided obstructive lesions, where flow is obstructed through the right side of the heart, usually without cyanosis; 5) Regurgitant lesions, where a cardiac valve is insufficient; 6) Mixing lesions, where there are two cardiac ventricles but extracardiac malformations allow mixing of blood from both sides of the heart; and 7) Single ventricle lesions, where there is a single functional right or left ventricle and complete intracardiac mixing of blood. **Table 12-1** summarizes this classification and presents common examples. It is very important to understand that many patients have more than one type of lesion; for example aortic stenosis with a ventricular septal defect (VSD) is both a left-sided obstructive lesion and a left-to-right shunting lesion.

Table 12-1: Classification of Congenital Heart Disease

Category	Cyanosis	Pulmonary Blood Flow	Common Examples
Left –to-Right Shunts	No	Increased	VSD, ASD, PDA
Right-to-Left Shunts	Yes	Decreased	TOF, pulmonary atresia
Left-Sided Obstructive Lesions	No	Normal	CoA, aortic stenosis, mitral stenosis
Right-Sided Obstructive Lesions	No*	Normal	Pulmonic stenosis
Regurgitant Lesions	No	Normal	Ebstein's anomaly
Mixing Lesions	Yes	Variable	TGA, truncus arteriosus, TAPVR
Single Ventricle Lesions	Yes	Variable	Tricuspid atresia, HLHS

**Cyanosis may be present if accompanied by a septal defect. VSD, ventricular septal defect; ASD, atrial septal defect; PDA, patent ductus arteriosus; TOF, tetralogy of Fallot; CoA, coarctation of aorta; TGA, transposition of the great arteries; TAPVR, total anomalous pulmonary venous return; HLHS, hypoplastic left heart syndrome*

Table 12-2 presents the incidence of the most common congenital heart disease lesions in children from birth to 18 years of age in the United States.

Table 12-2: Incidence of Common Lesions in Children With CHD

Source: Circulation 2012;125:e97.

Lesion	Incidence (%)
Ventricular Septal Defect	20.1
Atrial Septal Defect	16.8
Valvar Pulmonic Stenosis	12.6
Patent Ductus Arteriosus	12.4
Tetralogy of Fallot	7.0
Coarctation of Aorta	6.8
Valvar Aortic Stenosis	5.5
Atrioventricular Septal Defects	3.9
Transposition of the Great Vessels	3.6

Diagnostic Evaluation of Congenital Heart Disease

The fundamentals of diagnostic evaluation of CHD include five basic components: history, physical examination, chest radiograph, electrocardiogram (ECG), and hemoglobin level (Hgb). From these five components, an accurate idea of the cardiac lesion can be discerned in nearly every case. In addition, significant insights can be gained into the pathophysiologic effects of the lesion(s) on individual patients. To these five basic components are added cardiac imaging modalities: echocardiography, cardiac catheterization, cardiac magnetic resonance imaging (MRI), and cardiac computed tomography (CT). Arterial oxygen saturation (SpO_2) completes the diagnostic picture.

History

The patient's history is crucially important for determining her/his baseline status, duration and severity of symptoms, and previous cardiac diagnostic and therapeutic interventions. For infants, tachypnea and feeding difficulties are very common features of left-to-right shunting lesions. Shunting leads to increased work of breathing and poor ability to breastfeed. Diaphoresis (profuse sweating) is also a common finding with congestive heart failure (CHF). Pallor, listlessness, and cyanosis may be present with more serious cardiac lesions. Poor growth is a common finding in patients with congestive heart failure (CHF). In older children, cyanosis is very common with lesions like tetralogy of Fallot (TOF). Patients with TOF often assume a squatting position to increase their systemic vascular resistance, decrease right-to-left blood flow (shunting), and to increase pulmonary blood flow, which relieves the cyanosis. Older children with CHD often have

dyspnea on exertion and cannot keep up with siblings or peers during play or exercise. Significant left ventricular or aortic obstruction may be accompanied by fainting spells; this is also seen with cardiac arrhythmias, such as supraventricular or ventricular tachycardia. Angina is observed in some older children with coronary artery difficulties or with significant aortic stenosis. Children who had cardiac surgery previously may have sternotomy or thoracotomy scars. If they do this should provoke questioning about the procedure and careful examination of previous medical records to understand what procedure was performed and what (if any) residual lesions remain. Cardiac catheterization procedures may include interventions, such as dilation of aortic and pulmonary valves or closure of a ventricular septal defect. The nature and the result of these interventions should be well understood if possible.

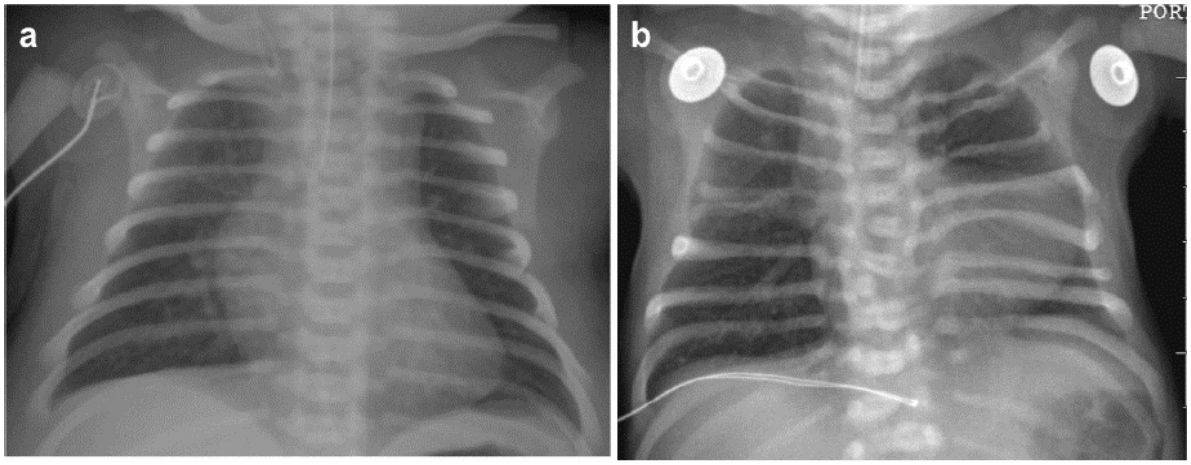
Physical Examination

Physical examination is best performed when the child is calm. One effective way to calm young children is to have her/him sit in the parent's lap; distracting them with toys is also effective. Vital signs are measured, including arterial blood pressure (BP) in four extremities, heart rate (HR), respiratory rate (RR), temperature, and the "5th vital sign of CHD": oxygen saturation (SpO_2) by pulse oximeter. Vital signs outside normal ranges are critically important: HR and RR are often elevated in CHF. The level of cyanosis, as determined by SpO_2, has major implications for anesthesia and surgery. For the physical examination, the first step is *observation* of the child: pallor, tachypnea, listlessness, failure of normal interaction with people, cyanosis, edema, jugular venous distension, and failure to thrive are important findings in patients with significant CHD. *Palpation* is also important: strength and quality of radial and femoral arteries pulses, perfusion (as judged by capillary refill time and temperature of the extremities), edema, hepatosplenomegaly, a cardiac thrill, and position of the cardiac apex all provide important information. *Auscultation* of the heart and lungs is next. Quality and loudness of first and second heart sounds (S1 and S2) are important—muffled heart sounds can indicate low cardiac output; a split S2 accompanies atrial septal defects (ASD), a loud S2 is heard with pulmonary hypertension, and a single S2 is heard with pulmonary atresia. Third and fourth heart sounds (S3 and S4) are heard with congestive heart failure and cardiomyopathy. Cardiac murmurs are important. Systolic murmurs are heard with restrictive flow, as with aortic or pulmonary stenosis, or a ventricular septal defect (VSD). Diastolic murmurs can be caused by mitral stenosis, and systolic-diastolic or continuous murmurs by patent ductus arteriosus (PDA). Gradation of murmurs from grade I-II/VI (soft-medium intensity murmur), grade III/VI (loud murmur), or grade IV-VI/VI (soft thrill, loud thrill, or audible thrill) gives important clues to the severity and location of the cardiac lesion. Auscultation of the lungs is also very important because it allows the examiner to detect rales (heard in congestive heart failure), wheezing (heard with the "cardiac asthma" that is associated with pulmonary venous obstruction due to left-sided obstructive lesions), or diminished breath sounds (lobar collapse from enlarged cardiac structures).

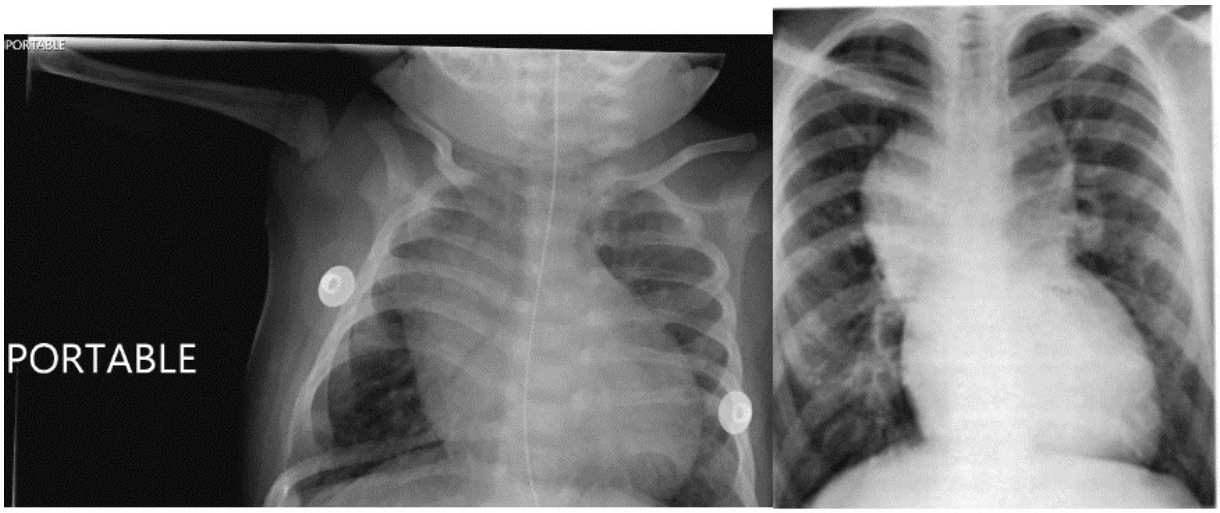
Chest Radiograph

The chest radiograph (CXR) yields information about both the cardiac lesion itself and the pathophysiology of the individual patient. An anterior-posterior (AP) CXR is often sufficient, but a lateral CXR can add important information. Heart size is an important consideration; a cardiothoracic ratio greater than 55% indicates cardiomegaly. Cardiomegaly is a sign of congestive heart failure, which may be the result of left-to-right shunting or cardiomyopathy. Cardiac silhouette is also important; a "boot shaped heart" is an indication that the main pulmonary artery (PA) is hypoplastic, which occurs in TOF. The narrow mediastinum seen with dextrotransposition of the great vessels (d-TGA) is often referred to as an "egg on a string". The "snowman" or "figure of eight" appearance of the mediastinum is caused by a distended vertical vein, which is often observed with obstructed supracardiac total anomalous pulmonary venous return (TAPVR). The appearance of the lung fields is crucial: "black" lung fields indicate diminished pulmonary blood flow; normal faint vascular markings indicate a normal pulmonary blood flow; and increased vascular markings and distended circular shadows on the CXR indicate small pulmonary arteries en face, which is indicative of increased pulmonary blood flow. The immediate postoperative CXR is important for assessing position of the tracheal tube, central lines, and evidence of intrathoracic bleeding or intrathoracic air. **Figure 12-1** displays examples of common CXR findings.

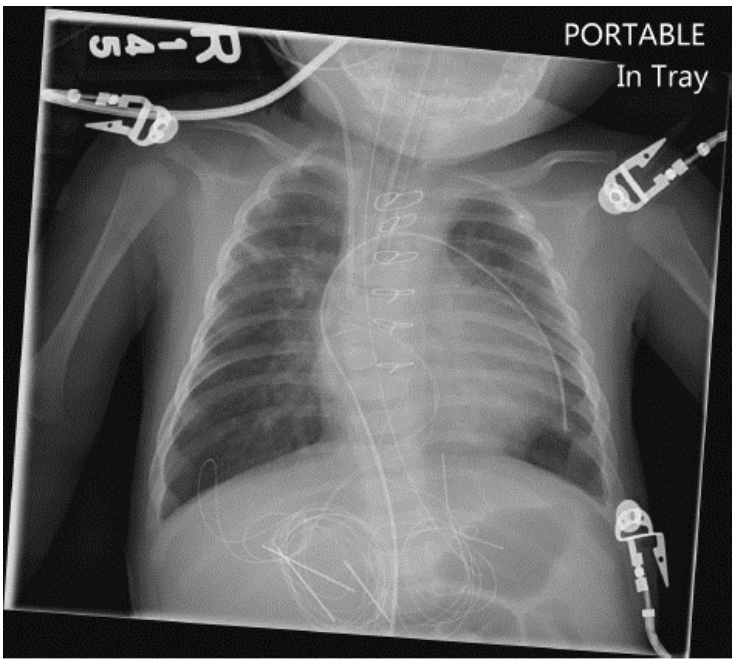
Figure 12-1: Chest Radiograph in Patients With Congenital Heart Disease



A. B.



C. D.



E.

A: *Transposition of the great arteries with ventricular septal defect: narrow mediastinum and increased pulmonary vascular markings;* ***B:*** *Tetralogy of Fallot: "boot-shaped" heart and normal pulmonary vascular markings;* ***C:*** *Cardiomyopathy: cardiomegaly with increased pulmonary vascular markings;* ***D:*** *Total anomalous pulmonary venous return: "Figure of 8" heart with increased pulmonary vascular markings;* ***E:*** *Postoperative CXR after ventricular septal defect repair; endotracheal tube is too low, and catheter (arrow) is in place.*

Hemoglobin Level

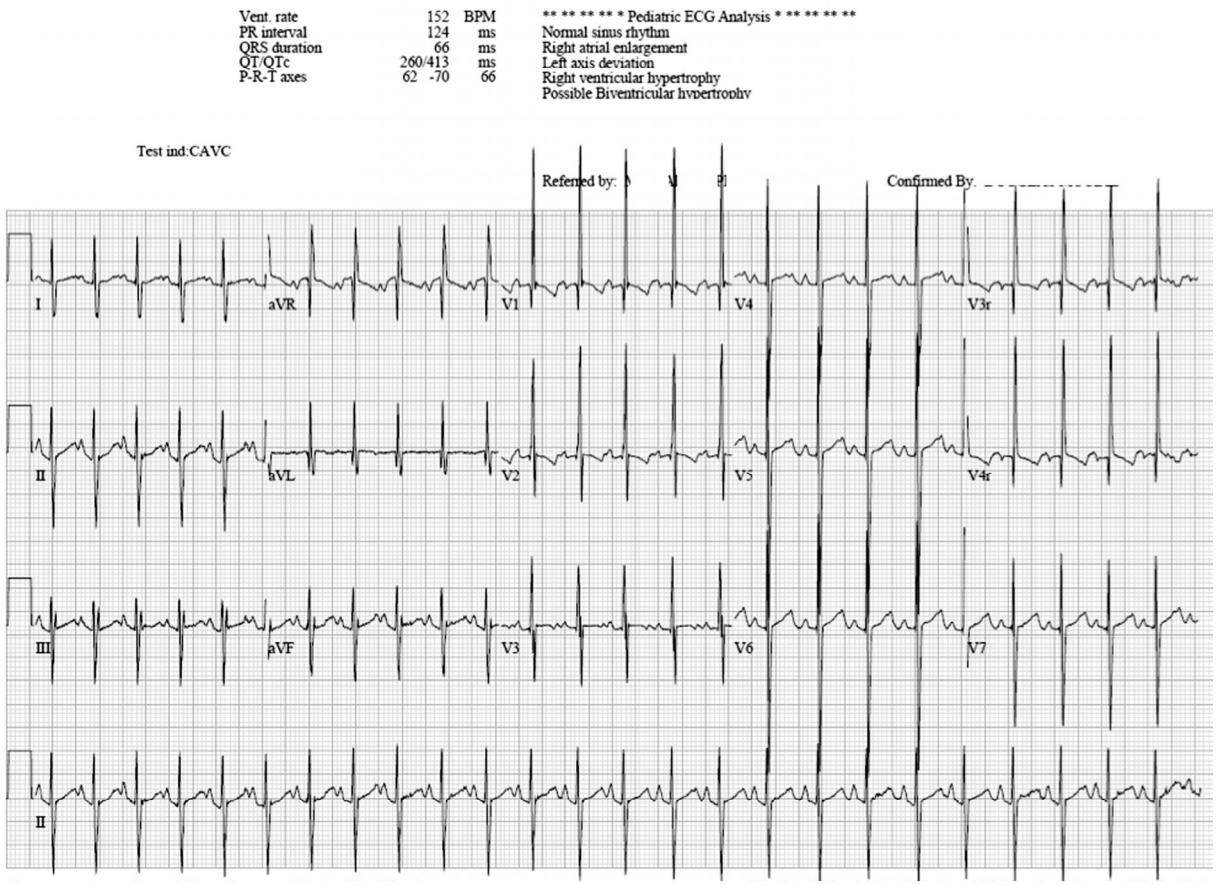
The baseline hemoglobin (Hgb) level is an important functional test of the degree of cyanosis in CHD. When blood oxygen tensions are lower than normal, the kidney secretes erythropoietin, which induces hematopoiesis and increases red cell mass. This increases the oxygen carrying capacity of blood to compensate for cyanosis (**See Chapter 1**). In general, the higher the Hgb

level, the lower the resting SpO_2; a normal infant undergoes a physiologic nadir in Hgb to about 10-11g/dl by 3-4 months of age. In cyanotic infants, the increase in Hgb levels is inversely proportionately to the patient's baseline SpO_2, sometimes reaching 15-16g/dl. Older children and teenagers with chronic, longstanding, severe cyanosis may have Hgb concentrations exceeding 20g/dl, which are often associated with signs and symptoms of hyperviscosity, including headache, visual changes, poor circulation in the extremities, clubbing of the finger and toe nails, and polycythemia-induced coagulopathy. Patients with mixing lesions and mild cyanosis may have near normal Hgb levels.

Electrocardiogram

A 12-lead ECG is an important source of preoperative information and for the diagnosis of CHD because it not only provides heart rate and rhythm, it also provides important information about chamber hypertrophy, intraventricular conduction delays, and myocardial strain (ST segment changes). The preoperative ECG also serves as a baseline for comparison if cardiac rhythm changes are suspected postoperatively. **Figure 12-2** is an example of the preoperative ECG in of patient with complete atrioventricular canal.

Figure 12-2: Electrocardiogram of a 4-Month Old Patient with Complete Atrioventricular Canal

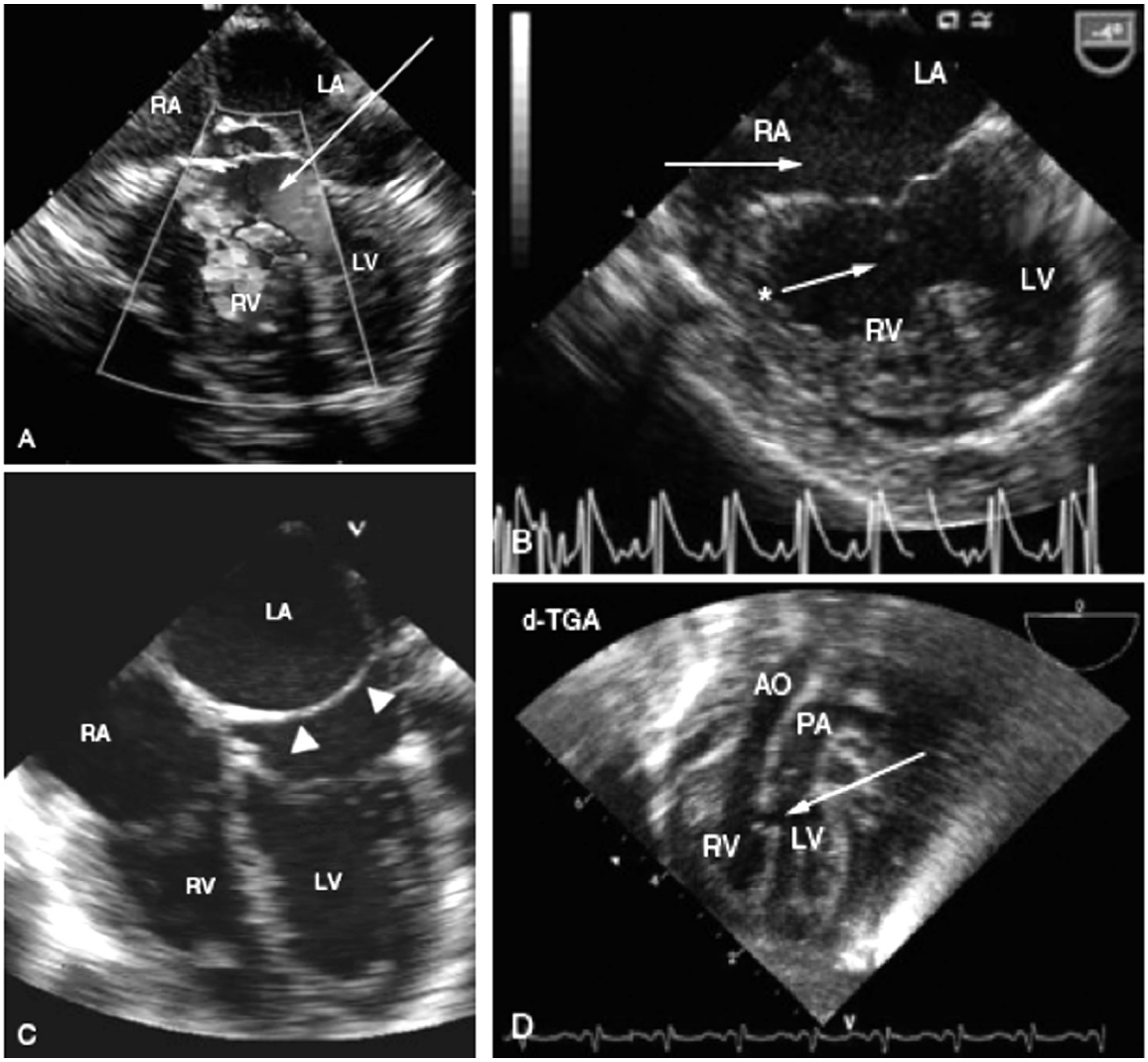


This is a complete, 15-lead ECG obtained in the Cardiology Clinic. Leads I, II, III, aVR, aVL, aVF get data from electrodes on the left and right arms, and left and right legs. Leads V1-V7 obtain data from leads on the chest, starting with V1 just to the left of the sternum near the costal margin. Leads V2-V7 follow a line just above the costal margin, progressively more lateral, until V7 lead is placed at the mid-axillary line. Leads V3r and V4r are placed to the right of the sternum. The long lead II tracing at the bottom of the printout is to diagnose arrhythmias. This ECG shows normal sinus rhythm (P-QRS-T), a rate of 150 beats/min, and the QRS voltages in all of the V leads indicate biventricular enlargement. The electrical axis of the R wave is -70 degrees, the "Northwest Axis" as one would see on a map; this finding is very characteristic of complete atrioventricular canal, or endocardial cushion defect.

Echocardiography

Transthoracic echocardiography is the mainstay of imaging diagnosis for congenital heart disease. It is portable, compact, non-invasive, relatively cost effective compared to other imaging methods, and highly accurate for imaging intracardiac anomalies and for cardiac function. These studies can be completed in a short period of time and can be repeated after an intervention. Two-dimensional imaging is the standard for anatomic diagnosis. This information is enhanced by color-flow Doppler interrogation to assess flow across valves, septa, and through shunts. Pulsed wave or continuous wave Doppler interrogation can be used to measure or calculate peak and mean velocities, as well as to calculate pressure gradients across intracardiac communications, valves, or outflow tracts. Ejection fraction and shortening fraction are accurate measures of ventricular function. If possible, the anesthetist should at least have access to the echocardiography reports. However, it is preferable to view the echo images in a presurgery planning conference with a radiologist and surgeon. Transesophageal echocardiography is used in the operating room pre- and postoperatively to assess anatomy, function, and residual defects after surgery. **Figure 12-3** shows examples of echocardiography for common lesions.

Figure 12-3: Echocardiography in Congenital Heart Disease



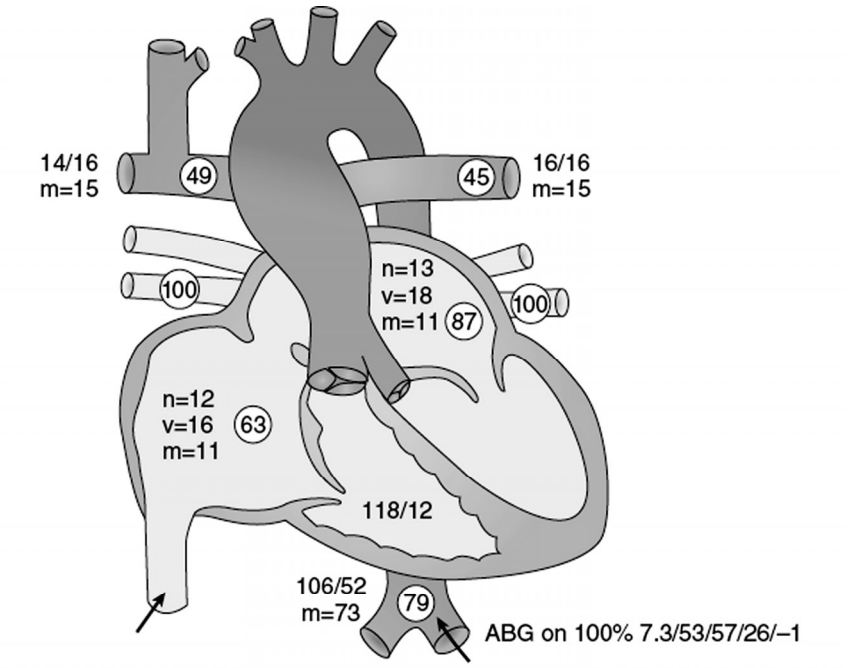
***A:** Ventricular septal defect (arrow) in 4-chamber view; **B:** Complete atrioventriular canal, with primum atrial septal defect (upper arrow) and ventricular septal defect (lower arrow); **C:** Cor triatriatum (double arrows)—a rare defect consisting of a membrane in the left atrium; **D:** dextrotransposition of the great vessels. Note the pulmonary artery (PA) arising from left ventricle (LV) and aorta (AO) from right ventricle (RV). Arrow is pointing to a ventricular septal defect. AO = aorta; PA = pulmonary artery; RV = right ventricle; LV = left ventricle; RA = right atrium; LA = left atriiumSource: Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 86.*

Cardiac Catheterization

Cardiac catheterization is capable of determining the anatomy and physiology of CHD. It is increasingly used for catheter-based interventions, i.e., balloon dilation of pulmonary or aortic valve stenosis, closure of PDA or ASD, or stenting of coarctation of the aorta. Although information from cardiac catheterization may be very useful in preoperative planning, in most settings the echocardiogram will be sufficient for this purpose. An example of information

obtained from a cardiac catheterization is shown on the diagram in **Figure 12-4**. On a single page, the anatomy, intracardiac and vascular pressures, oxygen saturations in each cardiac chamber, and pulmonary to systemic blood flow ratios are summarized. In addition, the previous surgical history is also listed, making this diagram extremely useful in planning an anesthetic for the cardiac surgery. Patients with simple cardiac lesions almost never undergo cardiac catheterization.

Figure 12-4: Cardiac Catheterization Diagram



Ht 48 cm Wt 4.3 kg BSA 0.22 m^2 Hgb 14.3 Hct – % LSVC –

Qp 1.47 L/min/m^2 (Fick) Qs 2.57 L/min/m^2 (Fick) Qp:Qs 0.6:1 PAR 2.72 U·m^2 Rp:RS –

VO_2 cons 160 ml/min/m^2 (assumed)

DIAGNOSIS:

1. Hypoplastic left heart syndrome (mitral and aortic stenosis)
2. Norwood procedure with a 3.5 mm Blalock-Taussig (BT) shunt
3. Right subclavian artery occlusion
4. Right ventricular dysfunction
5. Bidirectional Glenn
6. Poorly controlled atrial tachycardia

This patient has a very complicated CHD lesion; patients with simple cardiac lesions rarely, if ever, undergo cardiac catheterization. a = a wave pressure in mmHg; v = v wave pressure in mmHg; m = mean pressure in mmHg. ABG = arterial blood gas; numbers in white circles are measured oxygen saturations. Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 87.

Cardiac Magnetic Resonance Imaging (MRI) and Computed Tomography (CT)

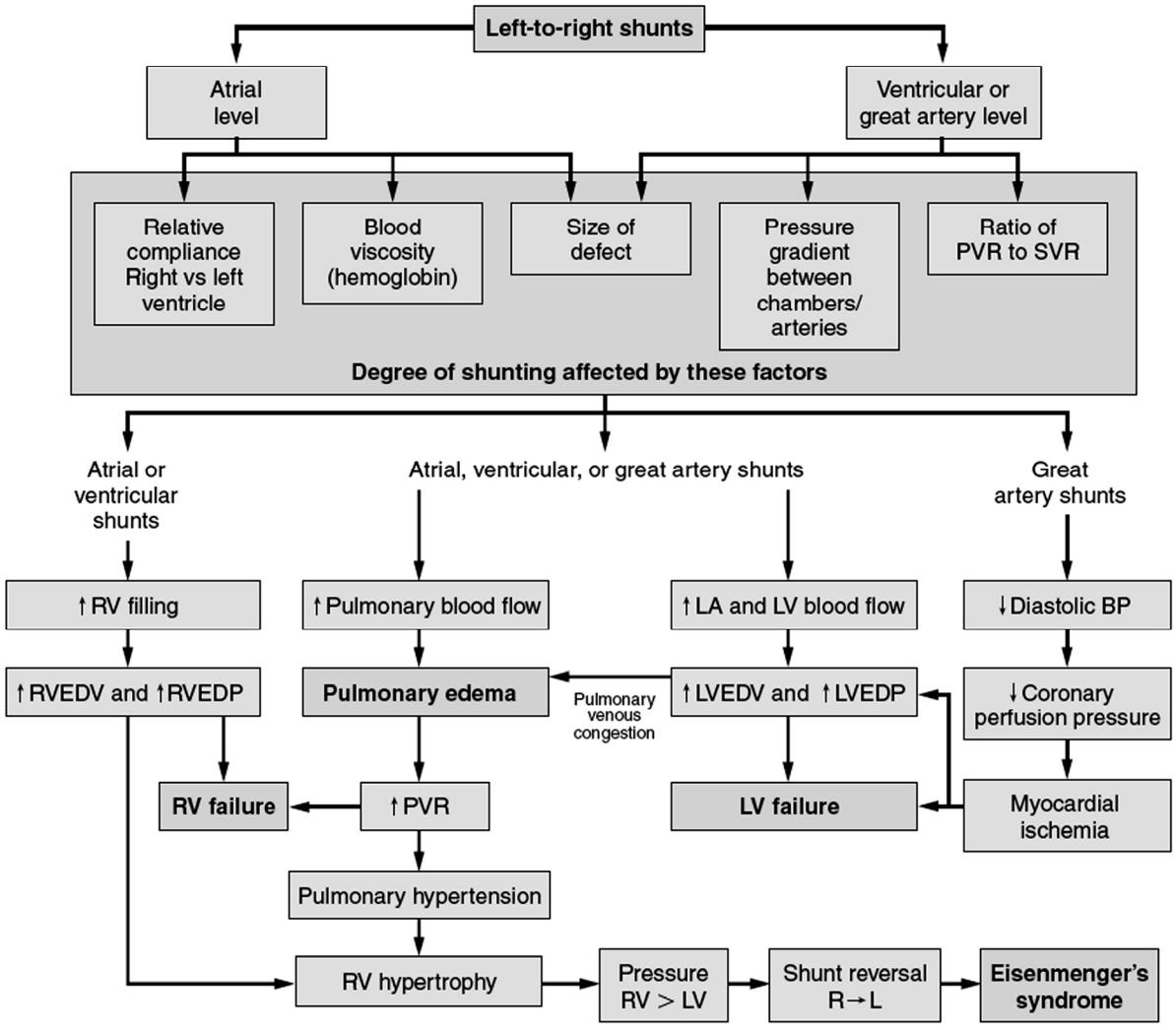
Cardiac MRI and CT scanning are non-invasive methods of providing accurate images of cardiac anatomy and are particularly suited for extracardiac structures, i.e., the aorta and its branches, pulmonary arteries and pulmonary veins. The equipment is complicated and expensive to maintain; therefore it may not be available in some programs. The advantage of MRI is that no ionizing radiation is involved; however at least 30-60 minutes are required for each study, and breath holding is sometimes required during the study. This means that infants and young children require sedation, or even tracheal intubation and general anesthesia for the study. CT scanning is a much shorter examination, requiring only 3-5 minutes to image cardiac structures; even young children require little or no sedation for the procedure. The major drawback to a CT scan is that radiation exposure can be significant; consequently, many cardiologists prefer MRI. The reader is referred to the bibiliography for more information about MRI and CT.

Left-to-Right Shunt Lesions

Left-to-right shunt lesions account for just over 50% of CHD and are the most common lesions requiring surgical correction in many cardiac surgical programs. The pathophysiology of these lesions is highly variable and depends on the size and location of the communication and the relative pressure and resistance on either side of the communication. The pulmonary-to-systemic blood flow ratio, or Qp:Qs, is highly variable and ranges from just over 1.0 with small shunts, to >5:1 with very large shunts. In the latter instance, pulmonary blood flow is five times systemic flow. A shunt is termed restrictive if the communication between cardiac chambers or great vessels is small enough to cause flow acceleration, designated by a peak pressure gradient of >10-20mm Hg, and a Doppler peak velocity of greater than 2-2.5m/sec. The smallest shunts tend to be ASDs because the pressure gradient between left and right atria is small, even if the ASD is large. Ventricular level shunts range from small VSDs of <2-3mm in diameter, to very large unrestrictive VSDs that allow large amounts of blood to flow from left-to-right. The largest shunts may occur at the level of the great arteries, with a large PDA or aortopoulmonary window producing continuous left-to-right shunting from the aorta directly into the pulmonary artery (PA). Besides the pressure gradient across the ventricular septum and the relative compliance of right and left ventricles, the resistance in the systemic and pulmonary circulations influences Qp:Qs. With relatively high systemic vascular resistance (SVR) and low pulmonary vascular resistance (PVR), the Qp:Qs can be >3:1. This degree of pulmonary over circulation often causes RV dilation, pulmonary edema, pulmonary venous congestion, and congestive heart failure. Maneuvers that further decrease PVR, i.e., FiO_2 of 1.0 and hyperventilation, can increase Qp:Qs even further and cause a steal phenomenon (blood is “stolen” from the aorta), decreases aortic pressure, and reduces coronary artery blood flow, which may cause myocardial ischemia. To prevent a “steal” is the rationale for lowering FiO_2, even to 0.21, and allowing some degree of hypercarbia, $PaCO_2$ 45-50mmHg, to increase the PVR and reduce the Qp:Qs.

A longstanding unrestrictive left-to-right shunt chronically exposes the pulmonary circulation to high flows and pressures, which results in pulmonary hypertensive changes, including thickening of the muscular medial layer of the pulmonary artery walls and extension of muscularization outward in to smaller pulmonary arterioles. Pulmonary pressure and resistance increase further; at some point they become fixed and unreactive, resulting in right-to-left shunting of blood during systole, and eventually during the entire cardiac cycle. This condition of fixed pulmonary hypertension and right-to-left shunting is termed Eisenmenger Syndrome, which prevents safe surgical closure of the VSD because doing so causes acute right heart failure. The pathophysiology of left-to-right shunts is diagrammed in **Figure 12-5**.

Figure 12-5: Pathophysiology of Left-to-Right Shunts



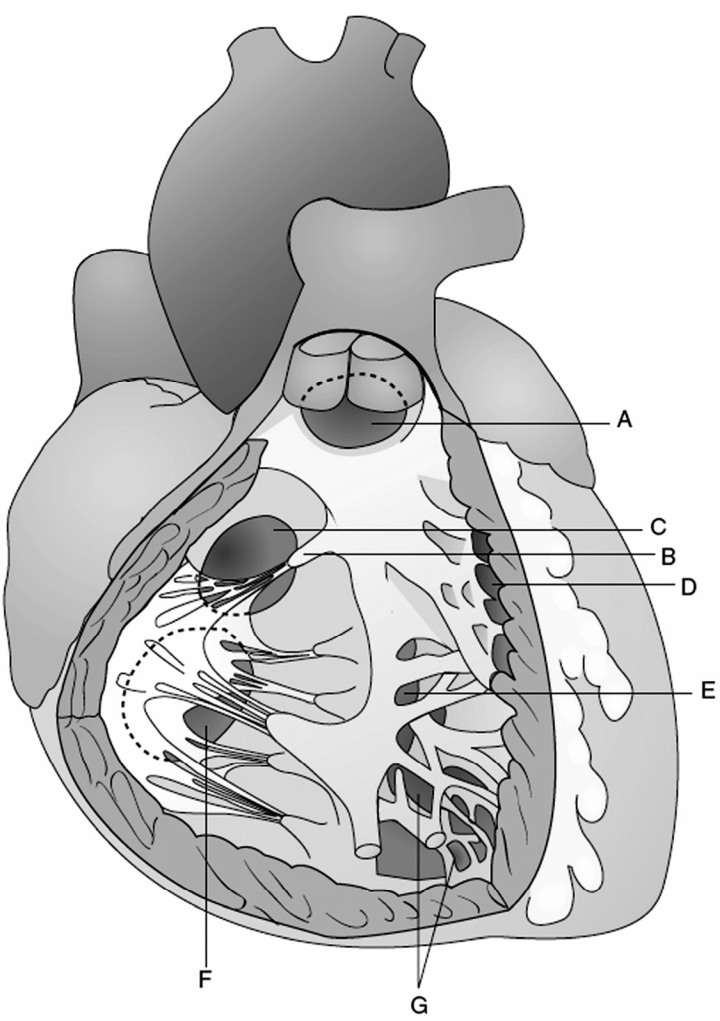
RVEDP= right ventricular end-distolic pressure; LVEDP = left ventricular end-diastolic pressure. Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 91.

Ventricular Septal Defects

VSDs are the most common form of CHD, occurring in about 20% of patients with CHD and of these perimembranous VSDs (those located in the perimembranous septum) are the most common type. Other types of VSDs include: supracristal or subarterial VSDs, which are located just beneath the pulmonary valve; inlet VSDs are located in the inlet septum just underneath the septal leaflet of the tricuspid valve; and muscular VSDs, which can be located anywhere in the muscular septum (**Figure 12-6**). Subarterial VSDs can cause aortic valve prolapse and aortic insufficiency from the Venturi effect created by blood flowing at high velocity and causing negative pressure that "sucks" the leaflet toward the VSD. Significant sized VSDs with Qp:Qs >3:1 produce significant right ventricular dilation and may cause pulmonary edema. The infant with a significant VSD has tachypnea, diaphoresis, poor feeding, pulmonary congestion, infections, and failure to thrive. Anticongestive therapy with diuretics, including furosemide, and afterload reduction with angiotensin converting enzyme (ACE) inhibitors is often required before surgery. Longstanding large VSDs result in fixed pulmonary hypertension and may produce Eisenmenger Syndrome. Findings on physical examination include tachycardia, displaced point of maximal impulse (PMI) downward and lateral at the left 5th or 6th interspace, a grade II-III/VI systolic murmur at the left sternal border, and if Qp:Qs >3:1, a diastolic murmur that is caused by increased blood flow across the tricuspid valve. The patient is acyanotic unless he/she has fixed pulmonary hypertension, in which case the degree of cyanosis parallels the right-to-left shunting of blood. Patients with larger VSDs have cardiomegaly and increased pulmonary vascular markings on CXR. ECG may be normal or reveal evidence of biventricular and/or atrial enlargement. Echocardiography is a very accurate means of diagnosing the location and number of VSDs, as well as their size and the flow velocity across them, which aids in determining if the defect is restrictive.

Large symptomatic VSDs are normally surgically repaired in infancy, which requires cardiopulmonary bypass, aortic cross clamping, and an autologous pericardial patch to close the VSD. Smaller VSDs are often repaired later in childhood. Transesophageal echocardiography (TEE) is very helpful during surgery to ensure that no residual defects remain. With subarterial defects, any aortic valve prolapse may require repair. With repair of the VSD, the ventricular dilation and pulmonary hypertensive changes usually regress over the weeks and months following surgery; the child should have a normal life expectancy.

Figure 12-6: Ventricular Septal Defect Anatomy



Types of VSD: ***A:*** *supracristal or subarterial;* ***C:*** *perimembranous;* ***F:*** *inlet or canal-type;* ***D,E,G:*** *muscular VSD.* ***B:*** *tricuspid valve cordae. Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 96.*

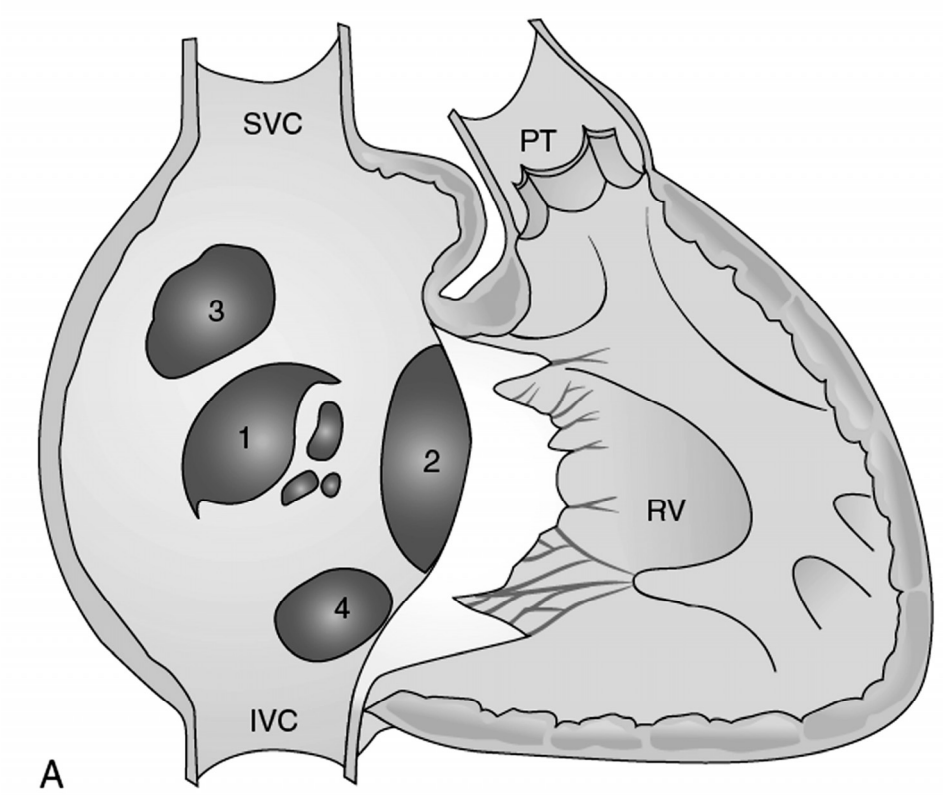
Atrial Septal Defects

Atrial septal defects (ASD) are the second most common CHD defect, comprising about 17% of lesions. The most common of these is a secundum ASD, located in the middle of the atrial septum secundum. The primum ASD, located in the septum primum, is often associated with a partial atrioventricular canal (see below). Other types of ASD include the inferior sinus venosus, and the superior sinus venosus ASDs (**Figure 12-7**). The superior sinus venous ASD is often associated with partial anomalous pulmonary venous return (PAPVR) where two right superior pulmonary veins return abnormally to the superior vena cava (SVC). Some patients have multiple small ASDs, and their patent foramen ovale (PFO) may be large enough to require closure. Because the degree of

left-to-right shunting with ASDs is relatively small, due to the low pressures and low-pressure gradient between the left and right atria, Qp:Qs usually remains at or below 2:1 and symptoms may develop late, sometimes not until the 3rd or 4th decade of life. Dyspnea with exertion is the most common presenting symptom. Pulmonary vascular disease rarely develops, but if it does, it only does so after the 5th decade of life. Physical examination may be normal, except for a soft grade I-II/VI systolic murmur at the left sternal border. The second heart sound is often split and fixed, representing delayed closure of the pulmonary valve. The delay caused by increased right ventricular stroke volume. CXR may reveal cardiomegaly and increased pulmonary vascular markings when ASDs are large. An ECG is often normal, but it can show atrial or ventricular enlargement. Echocardiography provides accurate diagnosis of the lesion and its location.

ASDs are surgically closed during childhood, usually by age 3-5 years of age if diagnosed early. Older children, teenagers, and adults often have their ASDs diagnosed and closed later in life. ASD closure may be done in the catheterization laboratory via a catheter-deployed “clamshell” device that is inserted through a large sheath in the femoral vein and guided into position by echocardiography. This procedure is only possible if the ASD is a secundum defect in the center of the atrial septum and if there is an adequate rim of tissue surrounding the ASD to which the device can be anchored without impinging on the aortic valve. All other ASDs are closed surgically with an autologous pericardial patch, which requires CPB and a short period of aortic cross clamping or induced ventricular fibrillation. Patients with sinus venosus ASD and PAPVR require an ASD patch that includes the pulmonary veins and directs blood flow from the veins under the patch to the left atrium. Intraoperative TEE is helpful for detecting residual surgical defects. Patients with an ASD usually recover very quickly and have no residual defects. Their life expectancy should be normal.

Figure 12-7: Atrial Septal Defect Anatomy



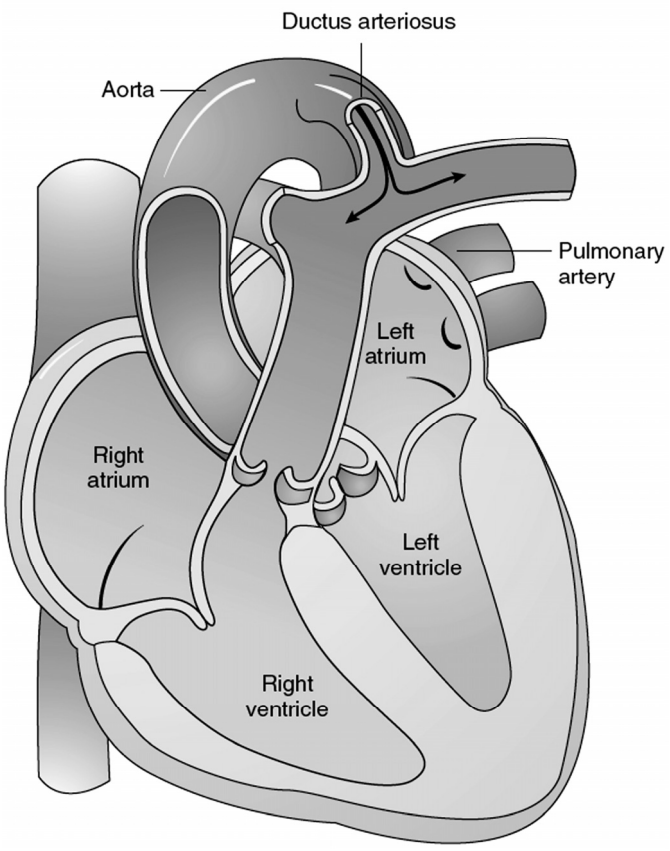
*ASD types: **1:** secundum; **2:** primum; **3:** superior sinus venosus; **B:** inferior sinus venosus. SVC = superior vena cava; IVC = inferior vena cava; RV = right ventricle; PT = pulmonary trunk. Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 95.*

Patent Ductus Arteriosus

Paternt ductus arteriosus (PDA) is another common left-to-right shunt lesion, observed in about 12% of patients with CHD (**Figure 12-8)**. The lesion may be isolated but is often part of a constellation of other cardiac lesions. Ductus arteriosus (DA) is part of normal fetal circulation. It functionally closes during the first 24-48 hours of life in most babies and is permanently closed by fibrosis during the first several weeks of life. If the PDA fails to close, it may persist throughout life. After birth a PDA may be essential for providing systemic blood flow for several CHD lesions, including hypoplastic left heart syndrome (HLHS) and moderate to severe coarctation of the aorta. PDA closure in patients with these lesions markedly reduces systemic perfusion, which, for patients with HLHS, is usually rapidly fatal. In patients with severe CoA, PDA closure produces subdiaphragmatic viscera and kidney ischemia, acidosis and shock, cardiac arrest, and death if not reversed. Patients with pulmonary atresia or severe pulmonic stenosis require a PDA with left-to-right shunting to provide enough pulmonary blood flow to sustain oxygenation and maintain organ perfusion and function. Prostaglandin E-1 (PGE-1) infusion 0.025-0.05mcg/kg/min

maintains a PDA in patients with these lesions until surgical palliation or surgical correction of the lesion can be accomplished. Isolated PDAs vary greatly in size and length. Because of this, the magnitude of left-to-right shunt is highly variable, ranging from Qp:Qs of just over 1:1 to >3:1. Symptoms vary from none to tachypnea, tachycardia, and CHF in some infants. Because there is continuous blood flow from aorta to PA, the PVR of large unrestrictive PDAs increases rapidly, causing early pulmonary vascular disease, fixed PVR, and right-to-left shunting. Adults with large untreated PDAs may present with huge, aneurysmal, calcified PDAs and Eisenmenger Syndrome, a situation generally considered untreatable. Physical examination may reveal signs of CHF, with tachypnea, tachycardia, and rales; the pulse pressure is often widened by the continuous diastolic runoff of blood into the PA, which lowers diastolic BP and causes bounding pulses. A long systolic, or continuous murmur is heard at the left sternal border. CXR often reveals cardiomegaly and increased vascular markings. Echocardiography is useful for determining size, length, and the velocity of flow in the PDA. Except for tachycardia, the ECG if often normal; in rare instances ST segment changes are seen on ECG, indicating myocardial ischemia.

Figure 12-8: Patent Ductus Arteriosus Anatomy



Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 92.

Closure of a PDA in premature infants is often attempted first with indomethacin or ibuprofen. If the PDA remains open, it is surgically closed through a left thoracotomy. This is often done at the bedside, not in the operating room. In premature infants, care must be taken during PDA ligation to monitor lower extremity pulses and perfusion by pulse oximeter to prevent the surgeons from inadvertently ligating the descending thoracic aorta, which may be smaller than the PDA. If the aorta is occluded, hypoxemia rapidly develops in the lower body. If the PDA is of appropriate size and length, patients weighing more than 5kg can have their PDA closed in the catheterization laboratory with occlusion devices placed through a sheath in the femoral artery. However, the most common approach is surgical ligation and division of the PDA via left thoracotomy. A PDA can also be closed thoracoscopically. A PDA or ligamentum arteriosum may be components of a vascular ring encircling the trachea or esophagus. At the end of surgery, the trachea of older infants can be extubated immediately; this is not usually the case for premature infants. Recovery from the procedure and anesthesia is rapid. Pulmonary hypertension may develop in older children or adults with a PDA. If so, they are at greater risk of morbidity or mortality from right heart failure following PDA closure.

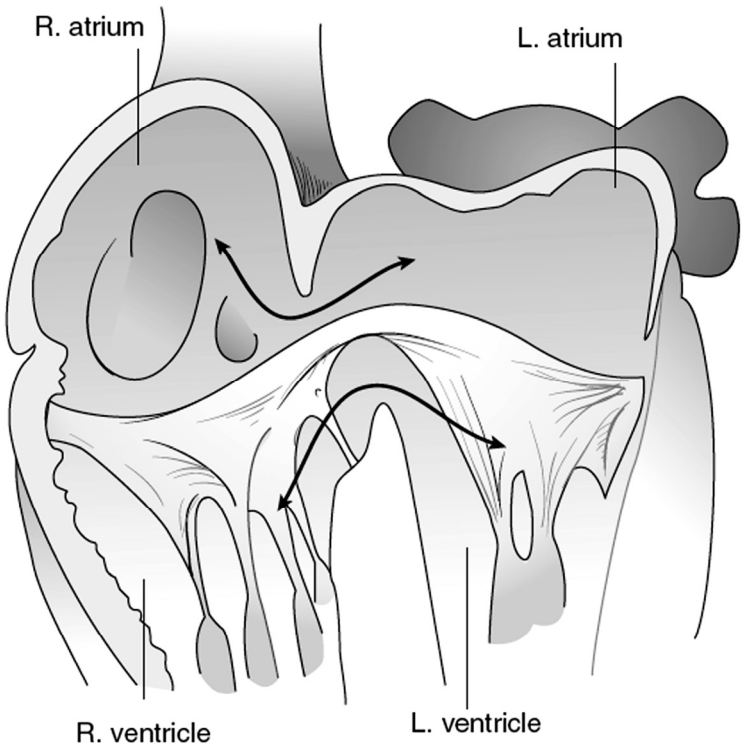
Atrioventricular Canal Defects

Atrioventricular canal (AVC) defects, also known as atrioventricular septal defects (AVSD), are relatively common left-to-right shunt lesions that occur in about 4% of patients with CHD. AVCs consist of a primum ASD, an inlet VSD, and abnormalities of the atrioventricular (AV) valves, ranging from a cleft in the mitral valve to a single common AV valve. A partial AVC consists of a primum ASD, cleft mitral valve, and no VSD or a VSD covered by tricuspid valve tissue that prevents left-to-right shunting of blood. An intermediate or transitional AVC has the findings of a partial AVC, but also has a small inlet VSD. A complete AVC (CAVC), also known as an endocardial cushion defect, has a primum ASD, large inlet VSD, and common AV valve (**Figure 12-9**). Complete AVC is commonly found in patients with Trisomy 21 (Down Syndrome) who often develop irreversible pulmonary hypertension during the first 1-2 years of life if the lesion is not repaired. Symptoms in patients with an AVC depend on the size of the left-to-right shunt. Partial canals tend to be minimally symptomatic, intermediate canals have a higher chance of being associated with heart failure, and CAVC is likely to present with symptoms of heart failure in infancy (tachypnea, poor feeding, poor growth), and frequent respiratory infections. Physical examination may reveal tachycardia and tachypnea with rales, normal S1, fixed split S2, and because the AV valves are often regurgitant, a grade III/VI harsh systolic murmur caused by flow across the VSD and by mitral and tricuspid valve regurgitation. An S3 is often present when the Qp:Qs exceeds 3:1. CXR reveals cardiomegaly and increased pulmonary vascular markings. ECG findings in CAVC are unique because the endocardial cushion defect changes the electrical vector of the heart, placing the R axis in the 0 to -90° quadrant, a “Northwest Axis”, which is essentially pathognomonic for CAVC. The ECG in **Figure 12-2** demonstrates this finding. Biventricular and atrial enlargement are also frequently observed in CAVC. Definitive diagnosis is by

echocardiography, which accurately images the ASD, VSD, and the AV valves and their regurgitation. Medical treatment is with oral diuretics, and possibly with ACE inhibitors for afterload reduction. Because of very large left-to-right shunting, the pulmonary circulation of patients with CVAC is exposed to high flows and pressures. If CAVC remains untreated, pulmonary hypertension develops and becomes fixed and non-reactive, which will prevent surgical repair due to the associated high mortality. As their disease worsens, right-to-left shunting of blood causes these patients to become cyanotic. Their CHF symptoms improve, and the CXR now shows a normal sized heart and oligemic lung fields. Growth often improves, giving a false sense of security that the patient has improved and can undergo surgery. Patients with Down Syndrome develop irreversible pulmonary hypertension earlier I life than patients with normal chromosomes; Down syndrome patients with CAVC often become inoperable if their lesion(s) are not repaired in the first 1-2 years of life.

Surgical correction of CAVC requires CPB and involves patch repair of the VSD (if present), patch closure of the ASD, and repair of the AV valves, which usually requires repair of mitral valve cleft and anchoring the AV valve to the patch. CAVC repair is technically challenging for the surgeon. Because of this, varying degrees of residual AV valve regurgitation may remain after surgery. Intraoperative TEE is used to assess the repair at the end of CPB. If abnormalities are found, it may be necessary to return the patient to CPB for further repair. Because of the frequent incidence of pulmonary hypertension, the tracheas of these patients are usually not extubated early. Sedation and mechanical ventilation are required until their pulmonary artery pressures (PAP) decrease. If available, inhaled nitric oxide (iNO) is sometimes used in this setting; however this therapy is a very expensive and may have limited effectiveness. CAVCs are ideally repaired at <6 months of age; transitional AVC is usually repaired at 1-2 years of age, and partial AVC is repaired at about 3-5 years of age. Residual mitral regurgitation or stenosis is a significant problem with AVC repair, and these patients require regular follow-up with a cardiologist because repeat surgeries may be necessary.

Figure 12-9: Complete Atrioventricular Canal Anatomy



Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 98.

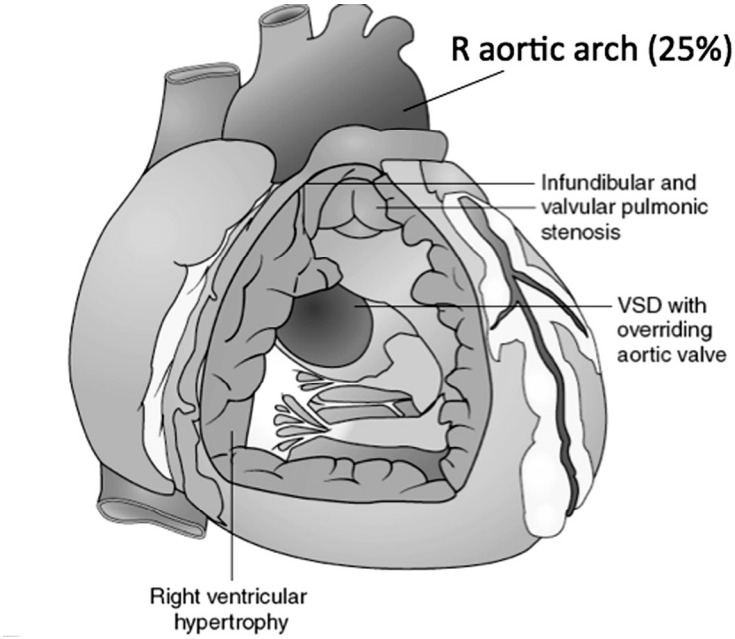
Right-to-Left Shunt Lesions

Tetralogy of Fallot

Tetrology of Fallot (TOF), the most common *cyanotic* CHD, occurs in about 7% of patients. The four components of TOF are: 1) Large subarterial VSD with over-riding aorta; 2) RV outflow tract obstruction (RVOTO) at the level of the sub-pulmonary valve infundibulum; 3) RV hypertrophy, and 4) Right-sided aortic arch (about 25% of patients) (**Figure 12-10**). Main and branch pulmonary arteries (PA) may be hypoplastic because reduced pulmonary blood flow reduces/prevents growth of pulmonary vessels. The symptoms of TOF are highly variable and depend on the degree of RVOTO. If RVOTO is mild, TOF can be acyanotic ("pink tet") and have symptoms similar to those with a large left-to-right shunt, e.g., VSD. With increasing RVOTO, dynamic obstruction to pulmonary blood flow occurs. This increases RV pressure, which causes right-to-left shunting through the VSD and cyanosis. Because RVOTO is a dynamic muscular obstruction, hypercyanotic spells ("Tet Spells") can occur when anything (e.g., crying, pain, hunger, thirst, etc) increases sympathetic stimulation. The spells may be so severe that the hypoxemia they produce causes the patient to loose consciousness. This lowers endogenous catecholamine secretion and ends the

TOF spell. Treatment of TOF spells outside of the operating room includes oxygen administration, IV volume infusion, and sedation. Morphine effectively treats TOF spells. Older children with unrepaired TOF and cyanotic spells often prevent or treat a spell by assuming a squatting position. Squating "kinks" the iliofemoral arterial system and increases SVR; this decreases the right-to-left shunting. This principle is used intraoperatively as well (see below). Physical examination reveals varying degrees of cyanosis; clubbing of fingers is a late sign. Cardiac examination reveals normal S1, diminished S2, and a harsh grade II-IV/VI long systolic murmur at the left sternal border. CXR often reveals a "boot-shaped heart", which is due to the hypoplastic main PA. The reduced pulmonary blood flow makes the lung fields of cyanotic patients oligemic. Patients with acyanotic TOF may have normal or even increased pulmonary vascular markings. ECG may reveal RV hypertrophy. The elevated Hgb level (polycythemia) of patients with TOF is proportional to the degree of cyanosis. The worse the cyanosis, the higher the Hgb. Older children with unrepaired lesions may be polycythemic and have Hgb concentrations that exceed 20g/dl. If they do, they can have headache, severe fatigue, and sluggish peripheral circulation. Definitive diagnosis of TOF is by echocardiography, which accurately displays all of its features. Medical therapy for TOF may include a β-adrenergic receptor blocker, such as propranolol, to decrease the force of RV contraction and RVOTO; these drugs often prevent "Tet spells". Severely cyanotic neonates may require infusion of PGE-1 to maintain the PDA until surgery can be performed.

Figure 12-10: Tetralogy of Fallot Anatomy

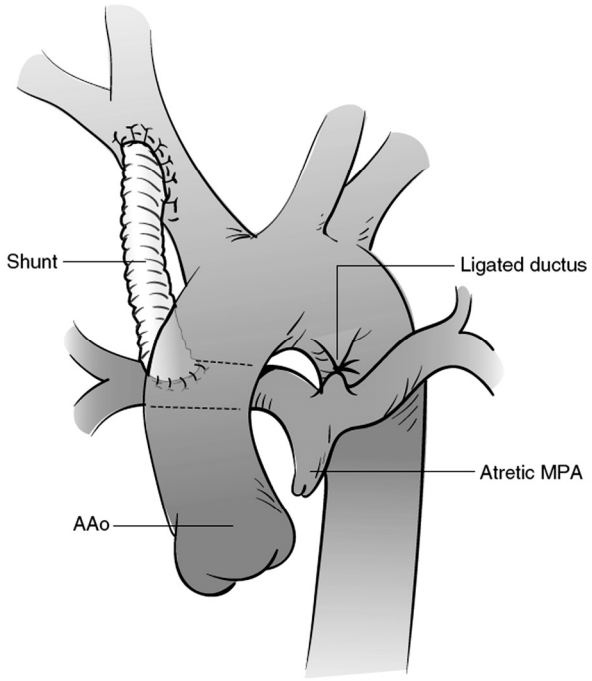


Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 111.

The age at which TOFs are repaired varies greatly. A severely cyanotic infant may require early surgery to create a systemic-to-pulmonary artery shunt (Blalock-Taussig, or BT shunt), which is a

3-5 mm Goretex® graft from the right subclavian or innominate artery to the pulmonary artery (**Figure 12-11**). This surgery is usually performed via right thoracotomy and normally does not required CPB. Placing the shunt allows the infant to survive and grow until he/she is ready for corrective surgery. Corrective surgery is performed at any age from neonate to adult and consists of VSD repair, RVOT muscle resection, and often includes placing a patch across the pulmonary valve annulus onto the proximal PA, which necessitates incising the pulmonary valve annulus. "Tet spells" may occur under anesthesia. If they do, treatment includes IV volume infusion to increase RV stroke volume; deepening anesthesia without lowering the arterial blood pressure, often with an opioid like fentanyl to decrease catecholamine release and reduce RVOTO; increasing SVR with a vasoconstrictor, such as phenylephrine, to produce more left-to-right shunting through the VSD and raise SpO_2. If the sternum is open, the surgeon can increase SVR by partially occluding the aorta. After surgery, varying degrees of either pulmonary insufficiency, or RVOTO may remain. TEE is important for immediate assessment of the repair. Direct RV pressure measurement is often used to determine if the RV pressure following CPB is <50% of systemic, as desired. Older children or teenagers with TOF often require further surgery to place a pulmonary valve. Immediate post-CPB problems include: 1) a non-compliant RV, that necessitates the use of high filling pressures; 2) treatment of arrhythmias, including junctional ectopic tachycardia (JET), which causes low cardiac output. Treatment of JET includes: 1) reducing catecholamine infusion (if being used), 2) cooling the patient, 3) placement of temporary atrial pacing via wires on the RA appendage, and 4) administering procainamide or amiodarone. JET usually resolves in 48-72 hours.

Figure 12-11: Systemic-to-Pulmonary Artery Shunt (BT Shunt)



This figure shows a shunt (graft) between the pulmonary artery and the subclavian artery. AAO = ascending aorta; MPA = main pulmonary artery. Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 112.

Pulmonary Atresia

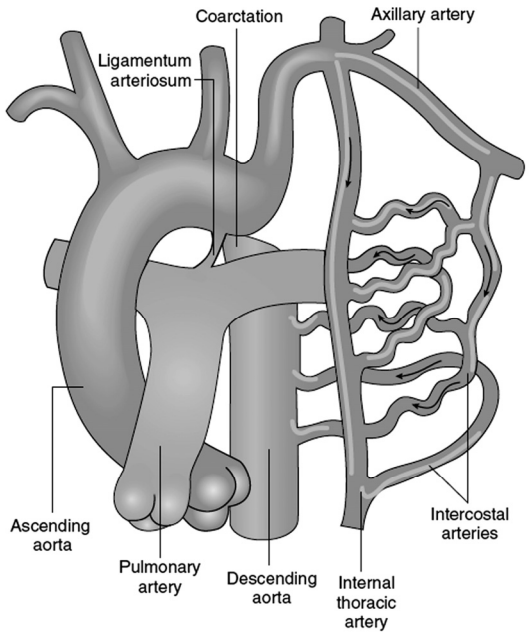
Pulmonary atresia is a complex lesion in which the anatomy varies from simple valvar PS with a large VSD and normal sized PAs, to a much more severe form that includes an intact ventricular septum and no true pulmonary arteries. Major aortopulmonary collateral arteries (MAPCAs) supply blood flow to the PAs. For a baby to survive, blood must flow from RA or RV (through a septal) defect to the LA or LV. During neonatal life the PDA supplies pulmonary blood flow. The severe forms of pulmonary atresia will not be discussed further. Pulmonary atresia with VSD and normal sized PAs is often considered a severe form of TOF and is treated in the same way as TOF. Neonates require a BT shunt. When older, the child will undergo complete repair, which consists of closure of the VSD and a transannular patch and RVOT resection, if the pulmonary valve can be used. If the pulmonary valve cannot be used, a conduit (which usually contains a valve) is placed from the RV to PA. Surgical and anesthetic considerations are similar to those for TOF.

Left-Sided Obstructive Lesions

Coarctation of the Aorta (CoA)

Coarctation of the aorta (CoA) is the most common left sided obstructive lesion, and is present in almost 7% of patients who have CHD. The aorta is usually narrowed just beyond the isthmus of the descending thoracic aorta, at the level of the ductus arteriosus. In cases of severe CoA, a PDA supplies blood to the lower body. PGE-1 is often required in neonates to keep their PDA open until they undergo definitive surgery. PDA closure may lead to severe LV strain, dilation, and dysfunction, or to cardiovascular collapse and death. Less severe CoA allows the PDA to close, which leads to hypertension of the upper body and upper extremities. LV function is usually preserved. This situation may be tolerated for many years. However, gradual LV hypertrophy develops; collateral arterial blood vessels grow from mammary and thoracic arteries, which are proximal to the CoA. These vessels anastomose with blood vessels distal to the CoA to provide lower body blood flow (**Figure 12-12**). These enlarged collateral arteries give rise to the characteristic rib notching seen on CXR. Patients may be asymptomatic; but as their upper body hypertension progresses, they may develop headaches. Untreated, adults develop early hypertensive arteriosclerosis. Some patients with severe hypertension have cerebrovascular accidents. Physical examination of a neonate with severe CoA and a closing PDA reveals poor perfusion and pulses in the lower body, delayed capillary refill, acidosis, and eventually cardiovascular collapse. Older infants and children have hypertension in the right arm, diminished femoral and pedal pulses, and a blood pressure gradient from right arm to legs that increases with more severe CoA and in the presence of inadequate collateral arterial circulation. Cardiac auscultation often reveals a soft grade I-II/VI systolic murmur at the left sternal border that radiates to the back. CXR may reveal some cardiomegaly if the patient has left ventricular hypertrophy or left ventricular dilation. Rib notching is observed in older children who develop a collateral blood supply. ECG often reveals LV hypertrophy and possibly a LV strain pattern with ST segment depression in the lateral leads. Definitive diagnosis is by echocardiography, which will delineate position and severity of CoA, whether a PDA is present, flow velocity through the CoA, and LV size, thickness, and function. Collateral arterial supply is not visualized well by echocardiography; CT, MRI, and cardiac catheterization are better tests if available. Medical therapy before surgery may include inotropic support and PGE-1 to keep the ductus arteriosus open in sick neonates who have LV dysfunction, or antihypertensive treatment with β-blocking agents or ACE inhibitors may be required.

Figure 12-12: Anatomy of Coarctation of Aorta with Collaterals in an Older Child

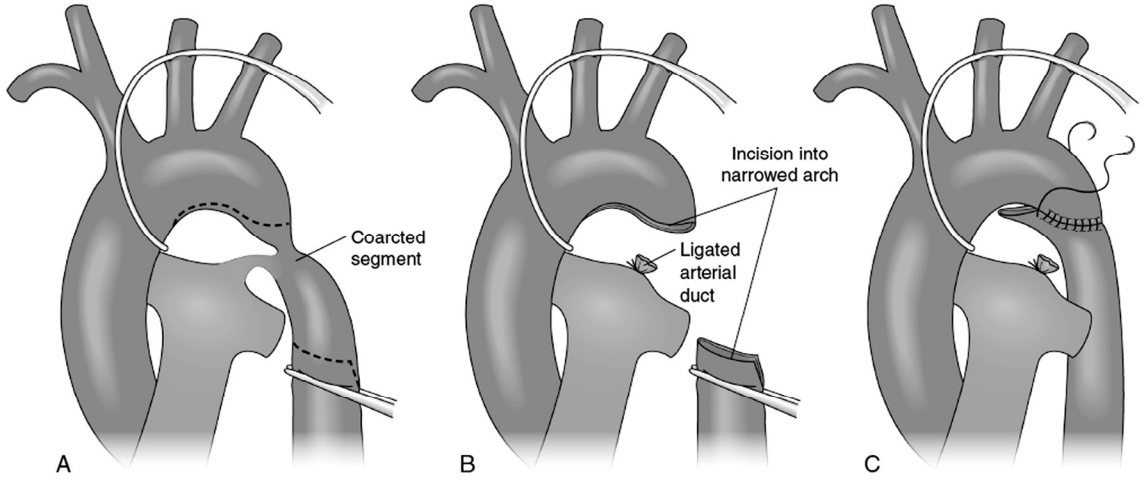


Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 105.

Surgical correction of CoA is performed soon after diagnosis is made and is normally done through a left thoracotomy without CPB. Arterial pressure monitoring in the *right arm* is necessary. In the modern era, the usual repair involves resecting the CoA tissue and remaining ductal tissue and anastomosing the proximal and distal ends of the aorta end-to-end (**Figure 12-13**). Previous approaches used subclavian flap angioplasty, but this is no longer done because the recurrence rate of CoA was too high. Because the aorta is cross clamped during the repair, there is usually a 15-30 minute period where the only blood flow to the lower body is via collateral circulation. Since the anatomy of collaterals is so variable, there is a small risk of the patient being paraplegic from spinal cord ischemia after CoA surgery. With modern techniques, the risk of this devastating complication is about 0.1%. Risk factors for paraplegia include older children without a well-developed collateral arterial supply and aortic cross clamp times in excess of 30 minutes. For these reasons, the anesthesia and surgery teams use spinal cord protective measures during the repair, including cooling the patient to about 34°C; maintaining a high-normal blood pressure during cross clamping to promote blood flow through collaterals; avoiding hyperventilation, which reduces spinal cord blood flow; and sometimes administering corticosteroids. Limiting aortic cross clamp time is very important. Immediately after surgery, the patient is assessed for her/his ability to move their lower extremities. Balloon angioplasty (via a sheath in the femoral artery) can also be used to treat CoA. Expandable stents can also be placed in the narrowed area, but surgery is normally the preferred treatment in infants and young children. Transcatheter

treatment is often used for recurrent CoA.

Figure 12-13: End-to-End Repair of Coarctation of the Aorta

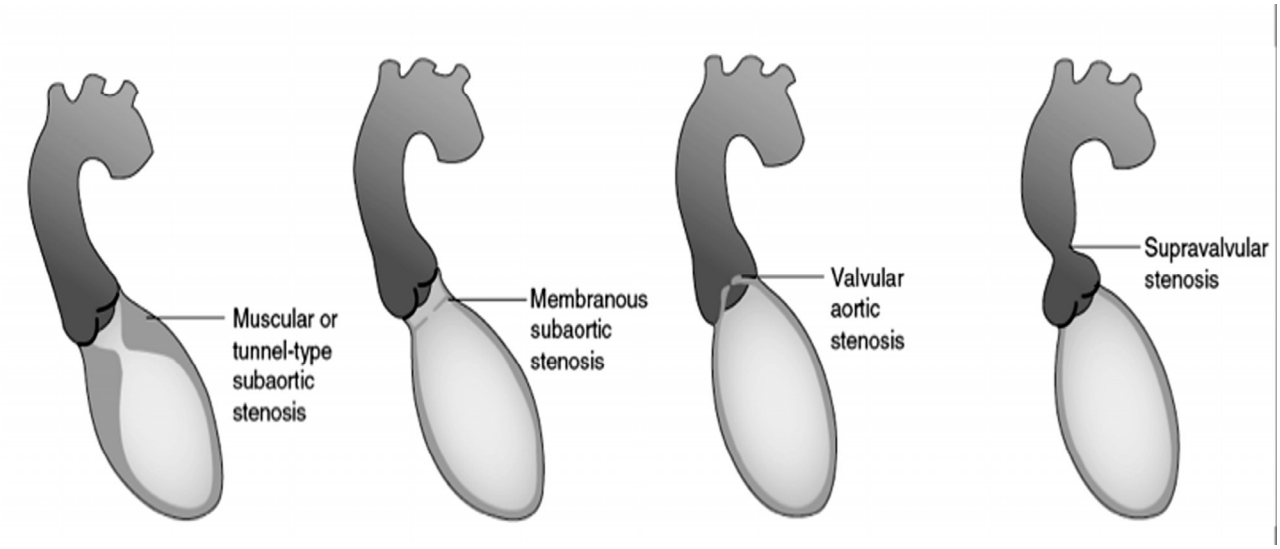


A: *Coartation of the aorta. The figure shows where the incision will be made (dotted lines). Note that the coarcted area is where the ductus arteriosus enters the aorta.* ***B:*** *Coarcted segment of aorta has been resected and the ductus arteriosus has been ligated.* ***C:*** *The descending aorta has been anastomosed to the arch of the aorta. Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 106.*

Aortic Stenosis

Aortic stenosis (AS) may occur at valvar, supravalvar, or subvalvar areas. There are many causes of AS, including congenital bicuspid aortic valve, Williams Syndrome (chromosome 7 elastin gene defect causing supravalvar AS with coronary artery involvement), and fibromuscular subaortic membrane (**Figure 12-14**). Signs and symptoms of AS range from severe obstruction in a neonate that requires PGE-1 infusion to maintain the PDA and prevent cardiovascular collapse until the AS can be corrected by surgery or in the catheterization laboratory, to completely asymptomatic patients who only have a systolic ejection murmur. After the neonatal period, patients with AS develop LV hypertrophy to maintain stroke volume. If the AS worsens, coronary ischemia may develop, and the patient may experience chest pains or syncope with exertion. This is especially the case for Williams Syndrome, where in addition to supravalvar narrowing at the sinuses of valsalva, the coronary artery ostia may be partially obstructed by abnormal tissue. Ventricular arrhythmias and sudden death may occur in untreated patients. LV dilation and failure are late signs of severe AS. Findings on physical examination include a harsh grade III-IV/VI long systolic murmur at the left sternal border. LV hypertrophy may displace the PMI downward and leftward. CXR reveals normal or increased heart size, and ECG usually indicates LV strain with ST segment depression in lateral leads. Echocardiography makes the definitive diagnosis and will accurately image the obstruction, the velocity of flow across the valve, the degree of LV hypertrophy, and the LV function.

Figure 12-14: Anatomy of Aortic Stenosis



Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 107.

Treatment of critical AS in neonates involves emergent balloon angioplasty in the catheterization laboratory. A balloon catheter is inserted through a sheath in the femoral or umbilical artery. PGE-1 and inotropic support must be maintained, and resuscitation drugs must be available because balloon dilation of the valve obstructs all flow to the coronary arteries for a brief time, which may cause ventricular tachycardia, fibrillation, and cardiac arrest. Older infants and children with AS undergo cardiac surgery with CPB. Many surgical treatments of AS are used, including aortic valvotomy or commissurotomy, aortic valve replacement with a mechanical valve, tissue valve, or autologous pulmonary valve (Ross procedure), pericardial patching with supravalvar AS, and resection of a fibromuscular membrane or LV outflow tract (LVOT) muscle for subaortic stenosis. The risk of coronary ischemia increases with the severity of AS. Patients with peak gradients in excess of 50-60mmHg are at high risk for coronary ischemia. Patients with Williams Syndrome are at even higher risk because of their coronary ostial obstruction. Hypovolemia, tachycardia, non-sinus rhythm, hyperdynamic LVOT contraction, severe depression of LV contractility, and hypotension all significantly increase the risk for cardiac arrest, because all of these conditions decrease stroke volume and coronary perfusion pressure or flow. Because the LV wall is thickened, the LV is at greater risk for inadequate perfusion from low arterial pressures. Low BP increases turbulent flow, which increases the resistance to blood flow across the obstruction. Prolonged fasting, hypovolemia, overdoses of inhaled or IV anesthetics that depress myocardial contractility and lower arterial blood pressure, tachycardia, excessive sympathetic stimulation that increases LVOT force of contraction, must all be avoided during anesthesia care for these patients. TEE is extremely useful after AS repair for detecting any remaining LVOT obstruction or aortic insufficiency (AI) due to the valve repair. Because the obstruction has been

relieved, hypertension is often a problem after CPB. Vasodilators, such as sodium nitroprusside, and β-blocking agents, such as esmolol, are often needed.

Mitral Stenosis

Mitral stenosis (MS) may be congenital, due to rheumatic heart disease, or residual after repair of AVC. Despite the fact that there is great anatomic variation of the mitral valve in MS, all patients have similar symptoms. As the stenosis worsens, obstruction to flow of blood into the LV causes LA enlargement, LA hypertension, and pulmonary venous congestion and hypertension. Pulmonary capillary engorgement develops, as do interstitial and alveolar edema. With increasing severity of MS, the patient develops pulmonary arterial hypertension over time. Initial symptoms of MS include tachypnea, frequent respiratory infections, and "cardiac asthma"—narrowing of small bronchioles by thickened pulmonary interstitium, and alveolar edema. Atrial arrhythmias, such as atrial flutter, can occur. Late signs include orthopnea and cachexia. Similar to AS, syncopal episodes may occur, due to inadequate systemic blood flow. Findings on physical examination include normal to slow HR and a "diastolic rumble" or a loud S3 caused by turbulent flow across the mitral valve. With significant MS, tachypnea, wheezing and rales are common. CXR reveals an enlarged LA and increased pulmonary vascular markings. ECG is consistent with LA enlargement, and once again echocardiography definitively shows the valve anatomy, the peak and mean flow velocity across the stenotic valve (from which the mean and peak gradient can be calculated), and the degree of LA enlargement. Pulmonary artery pressure can be estimated if the patient has tricuspid or pulmonary regurgitation.

Surgical treatment of MS involves CPB, aortic cross clamping, and comissurotomy or other repair of the mitral valve. Mitral valve replacement with a mechanical valve is possible, but most surgeons avoid this in growing children because it will be necessary to replace the valve as the heart grows and because long-term anticoagulation with warfarin is required. Warfarin therapy is very difficult to manage in children. The physiological considerations for anesthesia for MS surgery are similar to those for AS: HR is best kept normal to low to increase diastolic filling time across the obstruction. BP is kept normal to high to decrease the pressure gradient across the mitral valve, and hypovolemia is avoided so stroke volume can be maintained. Although pulmonary hypertension may be a problem, maneuvers to acutely lower pulmonary artery pressure (PAP) (FiO_2 of 1.0 and hyperventilation) may increase pulmonary blood flow but worsen pulmonary edema and pulmonary mechanics due to the fixed anatomic obstruction at the mitral valve. Non-sinus rhythm is poorly tolerated. TEE is extremely important for assessment of the quality of repair and cardiac function after CPB.

Right-Sided Obstructive Lesions

Pulmonic Stenosis

Pulmonic stenosis (PS) occurs at the level of the valve and at sub- or supravalvar areas in about 13% of patients with CHD. PS is normally well tolerated. Pressure overload causes compensatory RV hypertrophy. Only in the late stages of the disease, when the RV dilates and fails, does the patient have symptoms of right-sided heart failure, (hepatomegaly, peripheral edema, and ascites). Patients are often asymptomatic until relatively late in their course, when decreased RVOT flow leads to fatigue, dyspnea with exertion, and at times syncope. Physical examination of the patient reveals a harsh systolic murmur, often grade III-IV/VI, at the left upper sternal border. S2 may be soft and single. Since there is no VSD, the patient is often acyanotic. However, if there is right-to-left shunting through a PFO, neonates with severe PS may be cyanotic. A PGE-1 infusion is often needed to maintain the PDA until intervention on the pulmonary valve occurs. CXR may be normal or it may show a degree of right heart hypertrophy. ECG may be normal or show right ventricular hypertrophy. Echocardiography is the best diagnostic test and will precisely image the location and degree of obstruction, flow velocity across the valve, and the degree of RV hypertrophy. Isolated valvar PS is often treated in the catheterization laboratory by balloon dilation of the valve. The balloon is advanced through a sheath placed in a femoral vein. Neonates may require this procedure emergently; but in older infants or children, it is often performed electively. Valvuloplasty leaves varying degrees of pulmonary insufficiency (PI), which is well tolerated. Surgical approaches require CPB and involve incising the valve, with or without resection of infundibular muscle. A patch may be placed across the annulus, similar to TOF. Supravalvar PS is repaired with an autologous pericardial patch of the main PA. TEE is useful for both types of PS to determine the amount of PI and whether the PS has been relieved. Patients usually do very well after surgery. In later years they may require pulmonary valve replacement due to RV dilation and failure from longstanding PI.

Regurgitant Lesions

Aortic Regurgitation

Aortic regurgitation (AR) can be congenital or the result of balloon valvuloplasty for AS, endocarditis, or rheumatic heart disease. Aortic insufficiency (AI) usually presents in late infancy or childhood, not in the neonatal period. As the regurgitant fraction increases, LV diastolic volume and stroke volume increase, and the LV dilates over time, due to slowly developing AR. Acute AR (bacterial endocarditis) can quickly lead to LV failure. Diastolic backflow of blood into the LV decreases diastolic BP and coronary perfusion pressure, causing coronary ischemia. The ischemia leads to more LV dysfunction and ventricular arrhythmias. If not emergently addressed, acute AR may result in cardiac arrest and death. More slowly developing AR produces LV and LA enlargement, pulmonary venous congestion, tachypnea, “cardiac asthma”, dyspnea, and orthopnea. Physical examination reveals tachypnea and a long diastolic murmur at the left sternal border. Signs of low cardiac output, such as pallor, poor peripheral perfusion, and shock, may accompany acute AR. Angina may be present in patients with coronary insufficiency. Because the

diastolic runoff widens the pulse pressure; peripheral pulses are bounding, the so-called "water-hammer pulse". CXR often shows cardiomegaly with LV and LA enlargement and pulmonary venous congestion. ECG also shows evidence of LA and LV enlargement.

Emergency surgery may be required to correct acute AR. In patients with slowly progressing AR, non-emergent surgery can be required, usually outside the neonatal period. CPB and cross clamping of the aorta allows the surgeon to use one of a number of different techniques to repair the aortic valve. Aortic valve replacement may be required. In older children and teenagers, placement of a mechanical valve is usually well tolerated, which immediately improves the patient's condition. Infants and young children only undergo aortic valve replacement when their native valve cannot be repaired, or if a cryopreserved homograft, autograft (Ross procedure), or porcine-valved Dacron® conduit can be placed. If the aortic root is replaced, the coronary arteries must be re-implanted. Whenever possible, mechanical valves are put into smaller children. Hemodynamic goals for anesthesia in acute AR are maintenance of coronary perfusion pressures, which may require boluses of vasoconstrictor agents or continuous infusion of these drugs. Tachycardia is best avoided; myocardial contractility should be preserved with inotropes if necessary. For slowly progressive AR, faster HRs reduce the regurgitant fraction. Lower blood pressures reduce afterload and promote forward blood flow into the aorta. Hypovolemia is avoided. TEE is very useful for assessment of the repair and for determining the presence of residual defects. It is also helpful for evaluating LV function. Significant bleeding may be a problem post-CPB; adequate hemostatic blood products should be available.

Mitral Regurgitation

Mitral regurgitation (MR) can be congenital or from endocarditis, rheumatic heart disease, or AVC repair. As the stenosis worsens regurgitation of blood from LV into the LA produces LA enlargement, LA hypertension, pulmonary venous congestion and hypertension. The LA enlarges because a high percentage of each stroke volume is ejected backwards into the LA and increases atrial end-diastolic volume. If the mitral valve annulus enlarges, this will make it difficult for the valve leaflets to coapt and further worsens MR. Pulmonary capillary engorgement and interstitial and alveolar edema occur. Over time, severity of MR often increases, and this leads to pulmonary arterial hypertension. Initial symptoms of MR are similar to those with MS and include tachypnea, frequent respiratory infections, and "cardiac asthma". Atrial arrhythmias, such as atrial flutter, occur. Orthopnea and cachexia are late signs of MR. Physical examination often reveals tachycardia and tachypnea, plus a regurgitant jet-induced loud and long systolic murmur at the left sternal border. The PMI is displaced downward and to the left by the LV enlargement. CXR reveals cardiomegaly and enlargement of both LV and LA. ECG demonstrates LA and LV enlargement and may reveal atrial arrhythmias, such as atrial flutter. Echocardiography demonstrates mitral valve anatomy in detail, along with a qualitative estimate of the regurgitant fraction and the size and function of the LV and LA. During systole color-flow Doppler

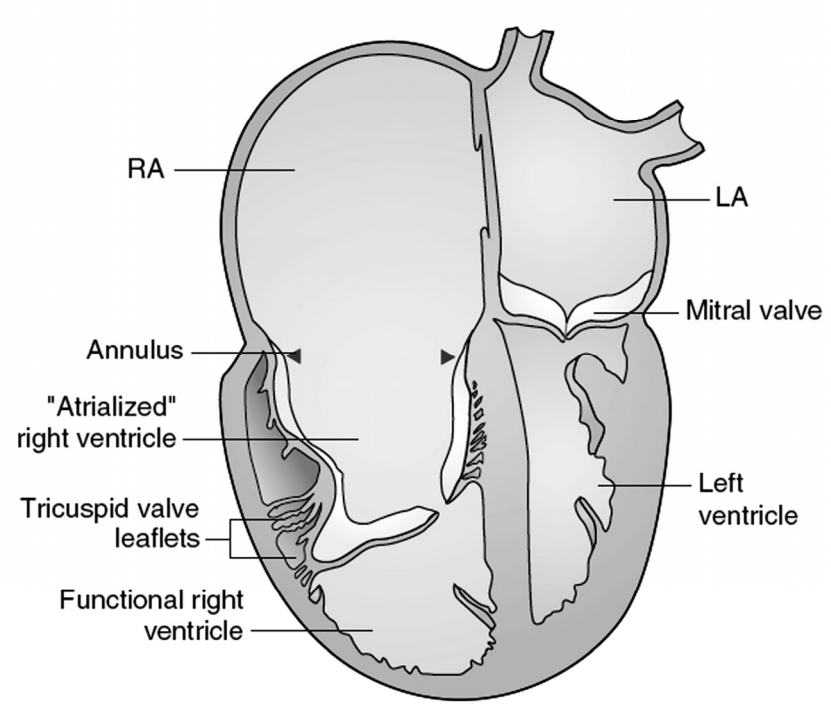
echocardiography reveals reversal of blood flow in one or more pulmonary veins in patients with severe MR.

Correction of MR requires repair of the valve and decreasing the size of the annulus. As with MS, mitral valve replacement is avoided in growing children whenever possible. Hemodynamic goals during anesthesia include maintaining a high-normal HR to decrease ejection time and reduce the amount of blood regurgitated into the atrium. Avoiding hypovolemia helps maintain adequate stroke volume. Reducing the arterial blood pressure decreases SVR and promotes forward blood flow. Finally, LV contractility should be maintained at normal levels. Following CPB, it is important to examine the heart for evidence of residual MR or MS and to help decide if further correction is needed.

Tricuspid Regurgitation

Tricuspid regurgitation is most often caused by Ebstein's anomaly, a congenital malformation of the tricuspid valve in which the valve annulus and leaflets are improperly placed into the body of the RV. The tricuspid valve itself is also malformed and has shortened chordae, resulting in varying degrees of TR (**Figure 12-15**). Neonates with severe TR caused by Ebstein's anomaly have poor forward blood flow into the RV and PA. For perfusion of the LA and LV, there must be an ASD or large PFO, which causes cyanosis. Because PA blood flow is low, a PDA is commonly required for maintenance of oxygenation. The electrical conduction system is also abnormal. In the neonatal period, severe Ebstein's anomalies are often fatal. Less severe degrees of TR can occur, and these patients may present in later childhood or even as adults. The TR causes RA enlargement and signs and symptoms of right heart failure, such as hepatomegaly, pleural effusions, and peripheral edema. The RV may also enlarge because the regurgitant fraction is high. Atrial dysrhythmias, such as atrial flutter and supraventricular tachycardia, are very common. Physical examination reveals the loud systolic murmur of TR at the right sternal border and RV impulse displacement; an enlarged liver, ascites, peripheral edema, and tachypnea may be evident. CXR reveals cardiomegaly; the pulmonary vascular markings are often normal or slightly increased; and there may be evidence of pleural effusion. ECG frequently demonstrates atrial premature contractions or atrial arrhythmias. Echocardiography accurately diagnoses malformation of the valve, the degree of regurgitation, presence of a PFO or ASD, and RV and LV size and function. Surgery entails a complicated repair of the tricuspid valve and plication of the arterialized portion of the RV. Anesthetic goals include: 1) promoting blood flow into the RV and PA by lowering PVR; 2) using high FiO_2; and 3) mild hyperventilation. Atrial arrhythmias must be diagnosed and aggressively treated; external or internal cardioversion may be required. After CPB, TEE of the heart is very important for assessing the repair. PVR must be kept low to optimize cardiac output.

Figure 12-15: Anatomy of Ebstein's Malformation



Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 110.

Mixing Lesions

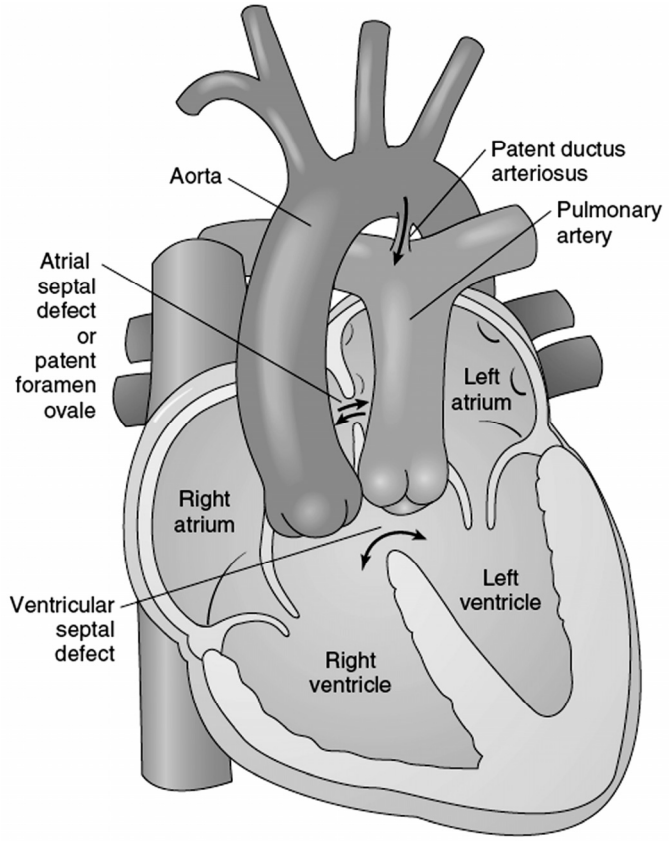
Mixing lesions are major malformations of the heart and great vessels that allow mixing of pulmonary and systemic blood. With these lesions, there are varying degrees of cyanosis. To oxygenate blood and provide adequate systemic oxygen delivery, both right-to-left and left-to-right shunting of blood must occur. Three of the most common and important mixing lesions will be discussed: dextro-transposition of the great arteries (d-TGA), total anomalous pulmonary venous return (TAPVR), and truncus arteriosus.

Transposition of the Great Arteries

d-TGA, also known as *transposition of the great vessels*, is a common form of CHD and occurs in about 3.6% of all CHD patients; it presents in the neonatal period. Early surgery is required for survival. The aorta arises abnormally from the RV and the PA from the LV. Approximately 20-25% of these patients also have a perimembranous VSD (**Figure 12-16**). Oxygenated pulmonary venous blood returns to the left side of the heart, i.e., to the LA and LV, where it is ejected into the PA. Unoxygenated systemic venous blood returns to the RA and RV and is ejected into the aorta. This parallel circulation (rather than the normal series circulation) prevents oxygenated blood from reaching the systemic circulation unless there is a communication at the atrial, ventricular, or PDA

level. These communications let oxygenated blood to enter the RA, RV, and aorta, which is necessary for survival. Without these communications, the patient is profoundly cyanotic and will only survive a few days if this situation is not corrected or paliated. These infants are profoundly cyanotic at birth, and 100% oxygen does not relieve the hypoxemia. Despite the sever hypoxemia, these patients shows little evidence of respiratory distress. This observation led to the "hyperoxia test", which differentiates cardiac from respiratory disease. The test consists of measuring SpO_2 or PaO_2 before and after the patient breaths a FiO2 of 1.0. Despite the high FiO_2, the SPO_2 of patients with *d-TGA* does not increase above 85% (PaO_2 below 45mmHg). Patients with pulmonary disease, on the other hand, often increase their oxygenation significantly. Physical examination reveals cyanosis without initial respiratory distress and a soft grade I-II/VI systolic murmur from a PDA or VSD, if present. CXR reveals a narrow mediastinum due to reversal of aorta and PA positions in the mediastinum ("Egg-on-a-String" appearance). The lung fields are black, due to reduced pulmonary blood flow. ECG is non-specific but often shows sinus tachycardia. The echocardiogram makes the definitive diagnosis by showing the PA and its branches arising from the LV and the aorta with its sinuses arising from the RV (**Figure 12-3D)**. Those patients who also have a VSD frequently have sufficient oxygenation to survive the neonatal period without further intervention. Because only 20-25% of patients with d-TGA have a VSD, the remainder requires PGE-1 to maintain the PDA, most of whom will require a balloon atrial septostomy at the bedside under echocardiographic guidance. To do this, a balloon-tipped catheter is introduced through the umbilical or a femoral vein into the LA. The balloon is inflated and vigorously yanked to tear a larger hole in the atrial septum to increase mixing of oxygenated and unoxygenated blood. This immediately increases the SpO_2, usually into the 80-90% range. After a septostomy, it is generally possible to discontinue PGE-1 and allow the patient to recover and feed before surgery.

Figure 12-16: Anatomy of Dextro-Transposition of the Great Arteries

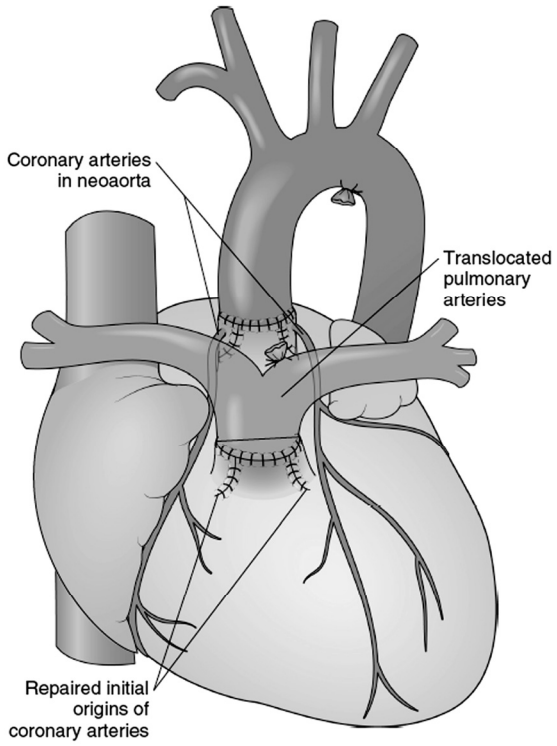


Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 116.

Surgery for d-TGA is now exclusively the arterial switch operation (ASO). During CPB and aortic cross clamping, the aorta and PA are transected, and their positions switched to restore normal anatomic relationships. In addition, the coronary arteries are translocated with a button of aortic tissue to their proper anatomic location on the aorta, which now arises from the LV. The VSD and ASD are also repaired (**Figure 12-17**). Coronary artery anatomy is variable, which presents challenges for the surgeon who must translocate these vessels without twisting, kinking, or obstruction them. The ASO is normally performed in the first several weeks of life before the LV becomes deconditioned, which occurs if there is no VSD and the surgery is delayed beyond 4-6 weeks of age. Deconditioning occurs because the pulmonary artery pressure and resistance decrease over time, making it easier for the left (pulmonary) ventricle to eject blood. When deconditioning occurs, the deconditioned LV will have difficulty pumping blood against the higher systemic pressures and resistances when the vessels are switched. Patients who have large VSDs maintain RV and LV pressures equal to systemic pressure, which allows surgery to be delayed until 2-3 months of age. Those with a VSD may develop excessive pulmonary blood flow and early pulmonary vascular disease as PVR decreases. Challenges for the anesthetist include treating low

post bypass cardiac output, which is often the result of coronary obstruction, a deconditioned LV, or inadequate myocardial protection during surgery. LA pressure is often monitored and maintained below 10mmHg to avoid excessive preload to the LV; excessive preloads may be poorly tolerated. Echocardiography, either epicardial or TEE, is an important tool for assessing myocardial function and diagnosing any residual issues from the surgery. Mortality after ASO is low and decreasing. The majority of patients require no further intervention for the remainder of their lives. However, some patients experience coronary artery problems, aortic insufficiency, or supravalvar pulmonic stenosis after surgery.

Figure 12-17: Anatomy of d-TGA After Arterial Switch Operation



Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 116.

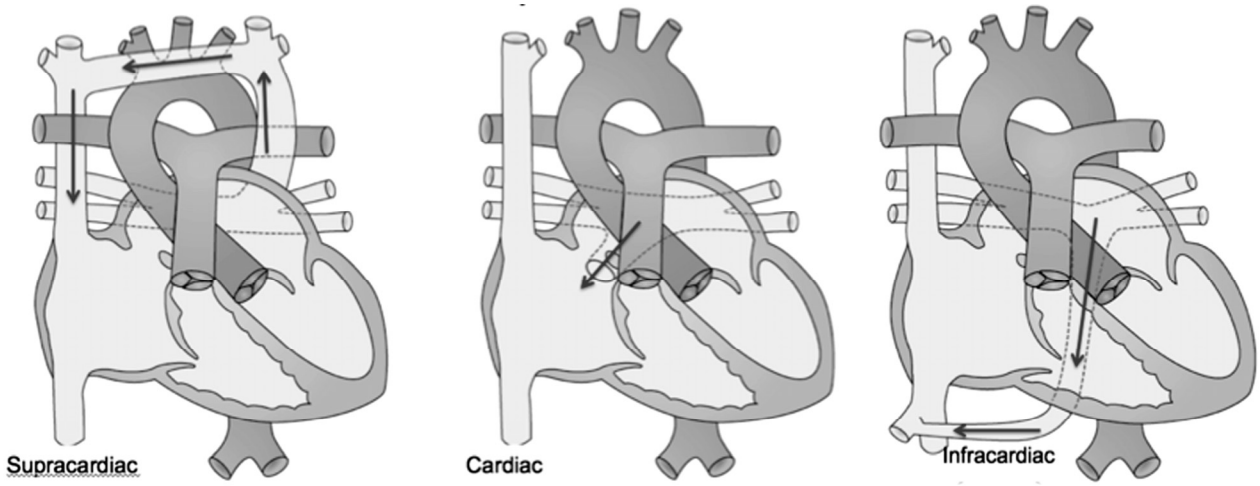
Total Anomalous Pulmonary Venous Return

Total anomalous pulmonary venous return (TAPVR) occurs when all four pulmonary veins connect abnormally to the right side of the heart. The three major types are: 1) Supracardiac, where confluence of the pulmonary veins connects via a vertical vein to the innominate vein; 2) Cardiac, where the pulmonary veins connect to the coronary sinus and drain to the RA; and 3) Infracardiac, where the pulmonary veins drain downward through an abnormal vertical vein and connect to the inferior vena cava or hepatic veins (**Figure 12-18**). There must be an atrial level communication (ASD or large PFO) for oxygenated blood to go into the left side of the heart to

maintain systemic cardiac output and systemic oxygen delivery.

The signs and symptoms of TAPVR at presentation depend on the degree of pulmonary vein obstruction and the size of the atrial level communication. Severe pulmonary vein obstruction, with minimal communication at the atrial level, leads to marked pulmonary venous engorgement, profound cyanosis, and death if not treated emergently by surgery. Lesser degrees of pulmonary venous obstruction occur in patients who have adequate atrial communication. These patients have mild to moderate cyanosis and variable amounts of respiratory distress during the neonatal period. In general, the risk of obstruction is high in patients with infracardiac TAPVR; supracardiac TAPVR has an intermediate risk; and cardiac TAPVR has the lowest risk of obstruction. CXR reveals a small heart and pulmonary venous engorgement. The CXR of patients with supracardiac TAPVR often shows a “figure of 8” or “Snowman” appearance due to the abnormal vertical vein on the left side of the mediastinum (**Figure 12-1D**).

Figure 13.18: Anatomy of Total Anomalous Pulmonary Venous Return



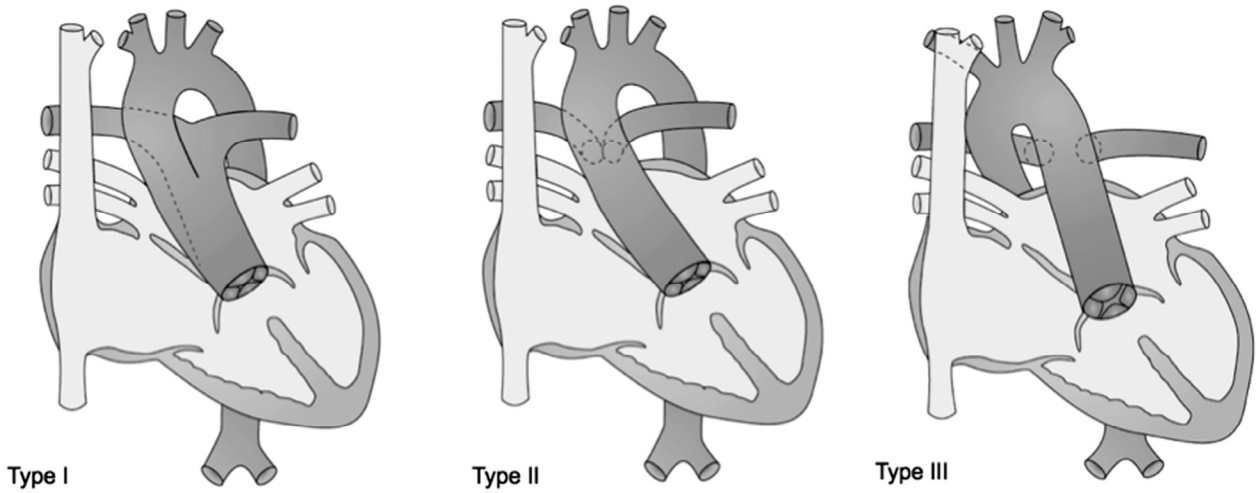
Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 103.

Urgent surgery may be required if supportive care (tracheal intubation, pulmonary ventilation, and inotropic support) does not effectively restore adequate oxygen delivery to tissues. CPB with deep hypothermic circulatory arrest (DHCA—see below) is necessary for this surgery. The abnormal vertical vein is ligated, divided, and the pulmonary venous confluence anastomosed to the back of the LA. The ASD is closed. Pulmonary hypertension is commonly encountered in the immediate postoperative period, which usually necessitates a period of mechanical ventilation, sedation, and pulmonary vasodilator therapy with sildenafil, inhaled nitric oxide (iNO), or nebulized prostacyclin, if available. Long-term outcome is usually good if the patient survives the neonatal period and surgery. A few patients have recurrent pulmonary vein obstruction and require further treatment.

Truncus Arteriosus

Truncus arteriosus consists of a single arterial trunk that arises from the left and right ventricles and gives rise to both the aorta and the PA (**Figure 12-19**). A large subarterial VSD is always present, and there is a single truncal valve that opens into the truncus. The truncal valve may be abnormal and may have anywhere from two to six leaflets plus stenosis, regurgitation, or both. The three major anatomic subtypes of truncus arteriosus are: Type I: the truncus arteriosus gives rise to the aorta and a short main PA that branches into left and right PA; Type II: there is no main PA, but the left and right PAs arise from the back of the truncus arteriosus; and Type III: there is no main PA, but the left and right PAs arise from the sides of the truncus arteriosus. Truncus arteriosus results in unique pathophysiology - the systemic, pulmonary, and coronary circulations exist in parallel and have profound interactions that affect cardiac and pulmonary function. At birth, pulmonary vascular resistance is elevated and Qp:Qs is near 1:1, resulting in mild cyanosis from mixing of oxygenated and unoxygenated blood without excessive pulmonary blood flow; aorta and coronary flow are relatively well preserved. As PVR decreases over the first days and weeks of life, Qp:Qs increases to 2-4:1, resulting in pulmonary over-circulation, pulmonary edema, and signs of CHF. The decrease in PVR allows a “steal” of blood flow away from the aorta and coronary arteries toward the pulmonary circulation. Diastolic BP is low, which frequently causes coronary insufficiency, myocardial ischemia, low cardiac output, ventricular arrhythmias, and death, if not treated. Physical examination demonstrates tachypnea, tachycardia, poor peripheral perfusion; cardiac examination reveals a single S2 and a grade II-IV/VI systolic murmur from the VSD or AS. There is often a S3 heart sound from AR or increased diastolic flow into the LV. CXR reveals cardiomegaly with increased pulmonary vascular markings. ECG may reveal sinus tachycardia, LV enlargement, and LV strain with ST segment changes in the lateral leads. Echocardiography produces a very accurate picture of the anatomy of the truncus arteriosus and pulmonary arteries, as well as the anatomy, flow, regurgitation, or stenosis of the truncal valve. The size and configuration of the VSD and the size and function of the ventricles are also imaged. If TA goes untreated and the patient survives, her/his CHF will be followed within weeks to months by pulmonary vascular disease, due to exposing the pulmonary circulation to high flows and pressures. A fixed shunt usually develops within the first year of life and makes the condition inoperable. Right-to-left shunting results in increasing cyanosis.

Figure 12-19: Anatomy of Truncus Arteriosus



See text for description of the different types of truncus arteriosus. Source: Andropoulos and Gottlieb; Congenital Heart Disease, Anesthesia and Uncommon Diseases, 6th Ed., Fleisher L., (ed.) 2012, p. 101.

Correction of truncus arteriosus occurs in the neonatal period using CPB and aorta cross clamping. The repair consists of closing the VSD with an autologous pericardial patch, separating the PA's from the truncus arteriosus, and inserting a RV-to-PA conduit that contains a valve. Problems with the truncal valve are addressed, and the ASD may be closed. This is a complex operation and major issues for the anesthetist post-CPB include bleeding, pulmonary hypertension, and myocardial dysfunction. If the patient survives the operation and the neonatal period, long-term outcomes are generally good, but subsequent cardiac surgery will be required as the patient grows to replace the RV-to-PA conduit. The truncal valve may also require further corrective procedures.

Single Ventricle Lesions

The most common *single ventricle lesions* occur with tricuspid atresia, which causes hypoplasia of the right heart, and hypoplastic left heart syndrome. Surgical treatment of single ventricle lesions is done in the neonatal period to provide stable pulmonary or systemic blood flow. Then, at age 3-6 months a superior cavopulmonary connection is created (bidirectional Glenn operation). Finally, at 2-4 years of age, a total cavopulmonary connection (Fontan operation) is performed. In general patients with single ventricles require multiple surgical and catheterization procedures and intensive use of resources. They experience much higher mortality and complication rates than patients with two-ventricle. The reader is referred to the Bibliography for extensive information about anesthesia for patients with single ventricles.

Table 12-3 summarizes the desired hemodynamic goals for the major classes of cardiac lesions, based on the pathophysiological considerations discussed above.

Table 12-3: Desired Hemodynamic Goals for Major Cardiac Lesions

Lesion	HR	Contractility	Preload	SVR	PVR	FiO2	PaCO2
Left sided obstruction: AS	↓	↓*	↑↑¶	↑↑	---	---	---
Left sided regurgitation: aortic/mitral	↑	↑	↑	↓↓	---	---	---
Left-to-right shunts: large VSD in infants	↓	↑	↓↓	↓↓	↑	↓	↑
Right-to-left shunts: TOF	↓↓	↓↓	↑↑	↑↑	---	↑↑	---

*HR, heart rate, SVR, systemic vascular resistance; PVR, pulmonary vascular resistance; AS, aortic stenosis; VSD, ventricular septal defect; TOF, tetralogy of Fallot. Changes from baseline: ↓, mild decrease; ↓↓, moderate decrease; ↑, mild increase; ↑↑, moderate increase; ---, no change. *Do not decrease if depressed at baseline; do not increase with depressed baseline contractility with high preload ↓*

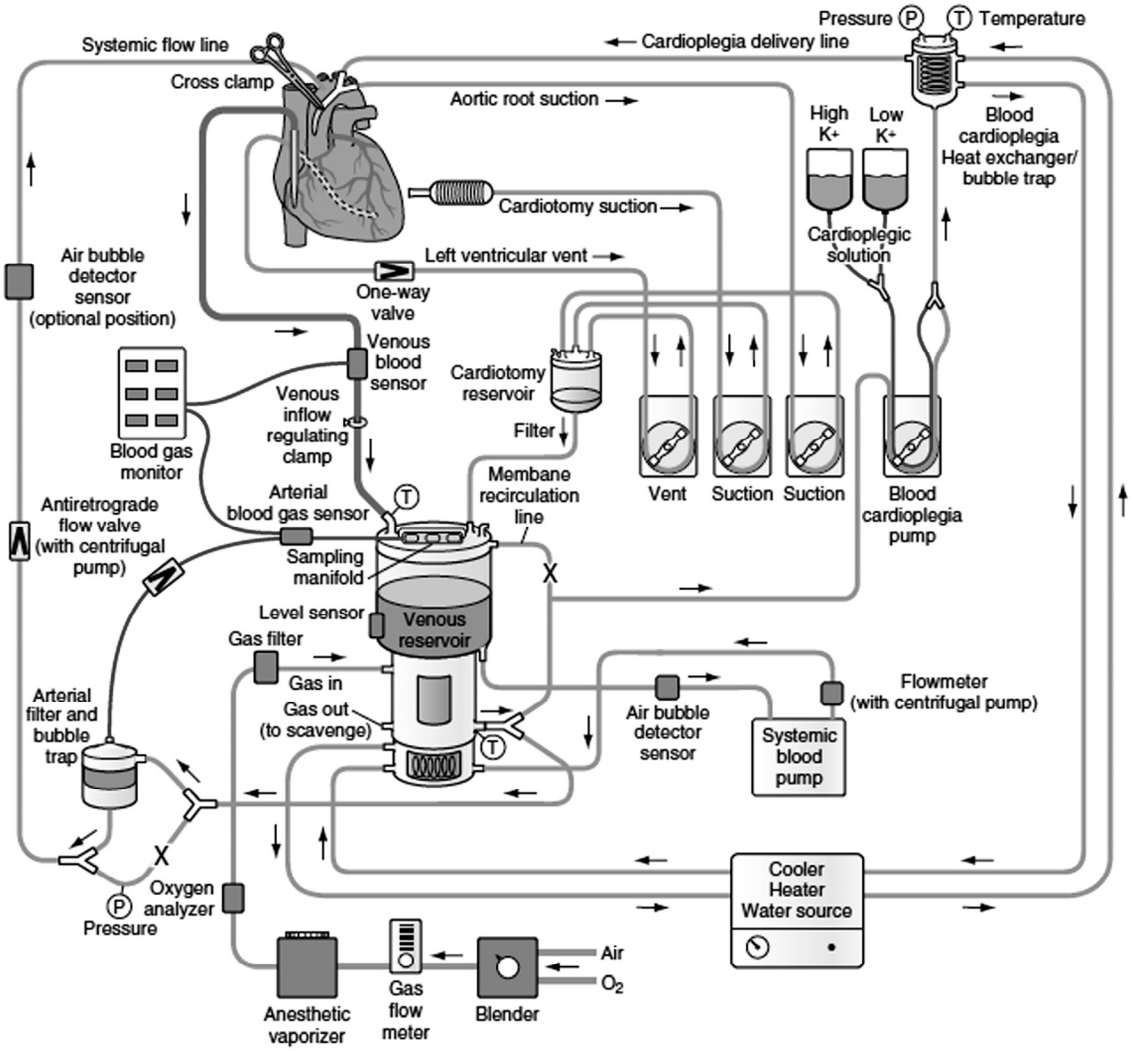
Cardiopulmonary Bypass

Basic Aspects

The components of a CPB circuit are displayed in **Figure 12-20**. The main components are a bypass machine with roller pumps for aortic perfusion, cardiotomy suction, and cardioplegia, an oxygenator-heat exchanger, a venous reservoir, tubing for return of venous blood and for aortic perfusion, and cannulae for aortic perfusion and drainage of venous blood. Monitors of flow, pressure, and blood oxygen saturation are incorporated into the circuit. In addition, monitors are incorporated into the venous reservoir and arterial perfusion side of the circuit to detect entrainment of air. An arterial filter is also incorporated to remove thrombi and trap air. An oxygen-air blender and an anesthetic vaporizer are incorporated into the sweep gas line to adjust the FiO_2 and add volatile anesthetic. The basic components of pediatric circuits are similar to adult CPB circuits, however, because of the small and variable sizes of pediatric patients, several different sizes of venous reservoirs and tubing are necessary for CHD surgery. Because the blood volume of infants and children is only 75-90ml/kg and the minimum volume of a CPB circuit is 250-350ml, smaller infants and children usually require priming of the CPB circuit with blood [packed red blood cells (PRBC) with or with or without fresh frozen plasma (FFP), or whole blood] to prevent extreme dilution of hematocrit and coagulation factors. Configurations of these

systems vary greatly, but in general the patient must be larger than 10-15 kg before it is possible to prime the circuit without blood (bloodless prime). The composition of fluid used to prime the system in small infants is often blood plus some isotonic balanced salt crystalloid solution, plus heparin, usually 2000-3000 units. The pH may be adjusted to normal with sodium bicarbonate. Larger patients have a crystalloid prime with heparin added, and the pH is adjusted to normal. Modern manufacturing has significantly reduced costs of the CPB circuits, and pre-packaged circuits with appropriate sized tubing, oxygenator, and venous reservoirs are available for infants and children at low cost.

Figure 12-20: Basic Components of a Cardiopulmonary Bypass Circuit



Source: Gertler R, Andropoulos DB. Chapter 17: "Cardiopulmonary Bypass and Management." In, Cote CL, Lerman J. (eds.) A Practice of Anesthesia for Infants and Children, 5th ed, Elsevier, Philadelphia PA, 2012, pp. 386-407.

Phases of CPB

The *Pre-Bypass Period* involves induction of anesthesia, tracheal intubation, insertion of vascular access and monitoring lines (including peripheral IVs, arterial catheter, and central venous catheter, if used). A TEE probe, if used, is inserted and the cardiac anatomy confirmed. Antibiotics and corticosteroids (if used) are administered, and antifibrinolytic agents started (tranexamic acid or aminocaproic acid), if used. Sternotomy is accomplished; a subtotal thymectomy is normally done to increase access to the heart and increase space in the mediastinum; the pericardium is incised; and the heart and great vessels are dissected and prepared for cannulation.

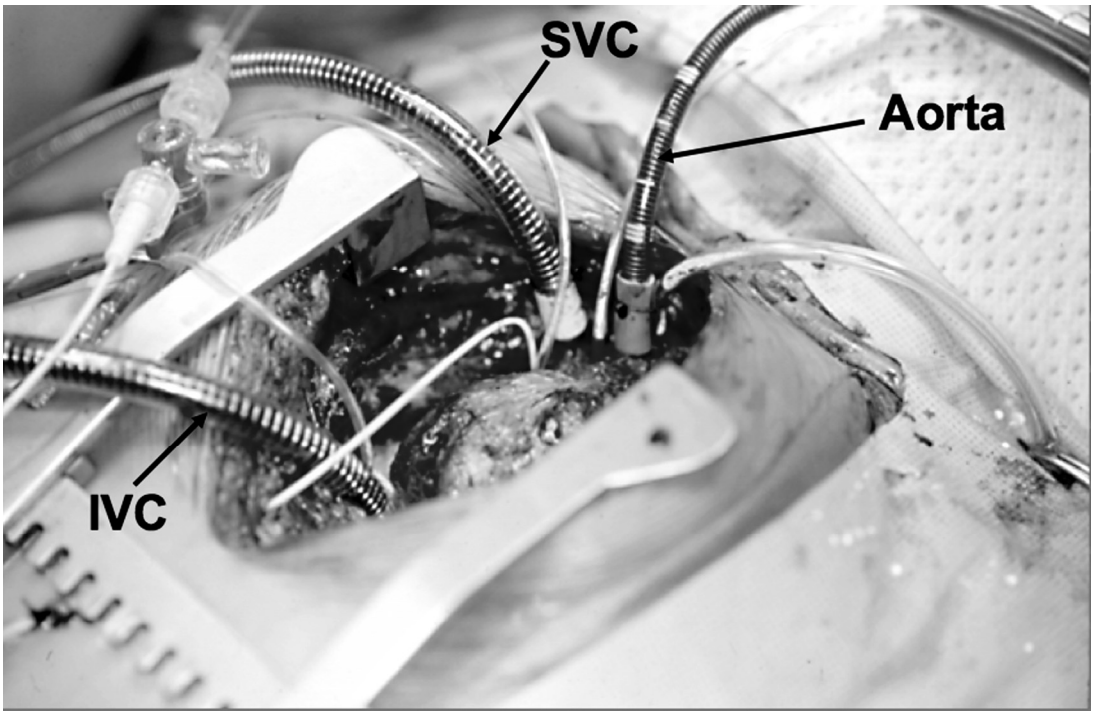
The *Cannulation Period* starts with the surgeon placing purse string sutures in the root of the aorta to prepare for aortic cannulation. At this point heparin, 300-400 units per kg of body weight, is given to ensure profound anticoagulation during bypass. Heparin must be given through a secure catheter, preferably a central line that has good blood return; alternatively the surgeon can administer the heparin directly into the heart. The activated clotting time (ACT) is commonly used to measure heparin effect; the baseline ACT is normally 120-150 seconds. The target ACT during bypass is 480 seconds. There must be adequate anticoagulation before beginning bypass to prevent clotting of the CPB circuit, which, if it occurs, is a fatal and preventable mistake. The surgeon will then place purse string sutures for insertion of the venous cannulae; most often this is for both superior vena cava (SVC) and inferior vena cava (IVC) cannulae to allow intracardiac access. Then, the aortic cannula is placed, the arterial perfusion line is connected and de-aired, and the correct position of the aortic cannula confirmed by measuring the pressure and by infusing a small amount of volume from the venous reservoir. Next, the IVC and SVC cannulae are placed and connected to the venous return tubing. Cannulation in small infants is often a time of significant hemodynamic instability; some surgeons initiate CPB only after the SVC cannula is placed. **Figure 12-21** shows cannulation in an infant for cardiac surgery.

The *CPB Initiation Period* begins when surgeon, perfusionist, and anesthetist agree that adequate anticoagulation has been achieved, cannulae and tubing are connected correctly, and the pump is ready to assume full flow support. CPB is initiated at low flows; the perfusionist assesses venous drainage by checking venous reservoir volume and the surgeon checks for emptying of the heart. CPB flow is increased over the first minute or so to full flow, normally 100-150ml/kg/min for patients <10kg, and 2.4-2.8l/min/m^2 for larger patients. Mean arterial pressure (MAP) is assessed, and target MAP ranges are achieved (30-50mmHg for neonates and infants, 40-50mmHg for older children, 50-60mmHg for teenagers and adults). Additional IV anesthetics or opioids are administered into the CPB circuit and volatile anesthetic is added to the sweep gas if indicated. The sweep gas FiO_2 is adjusted to avoid hyperoxia or hypoxia. Normally the FiO_2 is about 0.25-0.30. Blood gases and venous oxygen saturations are determined soon after initiating CPB to ensure adequate delivery of oxygen. Mechanical ventilation is stopped as soon as full CPB is

achieved. The anesthetist assesses the patient's face, scalp, and fontanelle for signs of superior vena cava (SVC) obstruction, which include bulging fontanelle (in babies), plethora, and cyanosis of the scalp and face.

The *CPB Cooling Period* starts when the team is satisfied that CPB has been successfully instituted. Cooling is initiated via the heat exchanger of the bypass machine. Hypothermia is frequently used to protect the myocardium, brain, and other organs because hypothermia greatly reduces oxygen consumption and cellular metabolic requirements. Mild hypothermia is 30-34°C and is used for many simple repairs, such as ASD and VSD. Moderate hypothermia is 22-30°C and is used for moderately complex repairs, such as TOF, ASO or CAVC. Deep hypothermia is 17-22°C and is used for complex surgery, such as TAPVR or truncus arteriosus repair that may require DHCA (see below).

Figure 12-21: Aorto-Bicaval Cannulation for Bypass in an Infant



Aorta = aortic cannula; SVC = superior vena cava cannula; IVC = inferior vena cava cannula.

The *Aortic Cross Clamping Period* begins after cooling when a clamp is placed on the aorta above the coronary arteries. Cardioplegia solution is infused into the aortic root through a separate small cannula to create diastolic arrest of the heart. This is accomplished by infusing a 20meq/l potassium (K+) solution into the coronary arteries to cause electrical silence. The ECG should be isoelectric within no more than 1-2 minutes after cross clamping occurs. Ice is often packed around the heart to maintain low myocardial temperatures. Cardioplegia can be re-infused at 20-minute intervals to maintain a quiet ECG; recurrence of ECG should be communicated to the

surgeon and additional cardioplegia given as needed. Aortic cross clamping is required for any surgery within the heart, such as ASD, VSD, TOF, ASO repairs. Aortic cross clamp time is also known as “ischemic time” because the myocardium is not perfused; prolonged periods of cross clamping cause myocardial ischemia, dysfunction, and possibly cardiomyocyte death. “Long” cross clamp times vary by institution and surgeon, but generally if cross clamping lasts longer than 90-120 minutes, the risk for myocardial dysfunction increases.

Deep Hypothermic Circulatory Arrest is utilized for some complex neonatal cardiac repairs, such as TAPVR or truncus arteriosus, to reduce blood return to the field and enable the surgeon to visualize and repair tiny structures accurately. At deep hypothermic temperatures, the CPB flow is turned off and all blood is drained from the heart. This leaves a quiet, bloodless field for the critical part of the repair. Safe periods of DHCA are generally thought to be up to 30-40 minutes; longer periods of DHCA are associated with brain injury and problems with long-term neurodevelopmental.

The *CPB Warming Period* begins when the major portion of the intracardiac repair is complete. The aorta is unclamped after the surgeon has performed de-airing maneuvers to reduce/remove air that entered the cardiac chambers while they were open to atmosphere during cross clamping. The heart should start beating spontaneously when the coronary arteries are again perfused with oxygenated blood and the high-K+ cardioplegia solution is washed from the myocardium. The patient is warmed to a nasopharyngeal temperature of about 36°C. The remainder of the cardiac repair is completed during warming. The surgeon places catheters in the RA or LA for pressure monitoring and vascular access; temporary cardiac pacing wires are placed on the atrium and ventricle, if needed. The desired hematocrit is achieved by either hemofiltration, adding blood to the venous reservoir, or both. Cardiac rhythm is assessed. If the rhythm is not normal sinus at the desired rate, temporary cardiac pacing is begun. If needed, inotropic, vasoconstrictor, and vasodilator drug infusions are started during rewarming once the ECG has returned to a near normal configuration. The lungs are inflated gently, the endotracheal tube is suctioned, and the lungs are inspected for bilateral inflation; any atelectatic areas of lung are expanded with several vital capacity breaths, additional suctioning, and possibly nebulized bronchodilators. Desired FiO_2 and ventilator settings are instituted. Calcium chloride is often added to the CPB circuit at this point to achieve normal ionized calcium levels, particularly in neonates.

The *Separation from CPB Period* begins with weaning of CPB flow and gradual impedance of venous return by the perfusionist. During this time the surgeon assesses cardiac function and filling and directs the perfusionist to further reduce flow and venous return until the CPB pump is turned off. Immediately after the patient is separated from CPB, blood pressure, SpO_2, filling pressures (central venous pressure, LA and RA pressures) are assessed, as well as myocardial contractility and ECG rhythm. A TEE examination of the heart is performed to assess the results of

the surgery and determine if there are residual defects, e.g., a residual VSD, that necessitates return to CPB for repair. Cardiac function is assessed. An arterial blood gas is checked to assess oxygenation, ventilation (CO_2), and acid-base status. The most common reasons for being unable to separate from CPB with good cardiac function and output are myocardial ischemia (due to prolonged cross clamp time) or residual cardiac defect, e.g., RVOT obstruction after TOF repair. In addition, air bubbles occasionally lodge in the right coronary artery during weaning, which results in RV dysfunction and ST segment elevation. This usually resolves over a short time with increasing the arterial blood pressure and flow through the coronary arteries. The most common inotropic agents used around the world are milrinone 0.25-0.75mcg/kg/min; epinephrine 0.02-0.1mcg/kg/min, and dopamine 3-10mcg/kg/min. Many patients undergoing simple corrective two ventricle repairs, i.e. VSD and ASD, require no inotropic agents after CPB. Once the team is satisfied with the patient's hemodynamic status, TEE results, and blood gas, the surgeon requests that protamine be administered to reverse the heparin effects, usually in a dose of 1.0-1.3 mg of protamine per mg of heparin (100 units heparin = 1 mg) given for the original dose. The ACT is checked 3-5 minutes after the protamine is given to ensure that the heparin effect is fully reversed. After protamine, the ACT should be within about 10% of baseline.

The *Post-CPB Period* extends from the administration of protamine until admission to intensive care unit (ICU) and handoff of care to the ICU team. In many children, bleeding is a primary concern following CPB. The most common cause is bleeding is from suture lines or from small vessels. During surgery the surgeon must meticulously search for and occlude any source of bleeding with an electrocautery, additional sutures, or other hemostatic drugs or techniques that are applied topically to the bleeding area. CPB itself causes problems with coagulation in several ways, the most important being dilution by the CPB prime of coagulation proteins and platelets in <8-10kg patients. Platelets also bind to components of the CPB circuit and tubing, are degranulated, and become inactive. Finally, fibrinolysis occurs by activation of the coagulation cascade, by contact of blood with foreign surfaces, and by the inflammatory response to bypass. In small infants, administration of platelets is the first line of therapy and will resolve most post-CPB bleeding, especially since platelets are suspended in plasma that contains some coagulation proteins. A platelet dose of 1 random donor unit per 5kg patient weight increases the platelet count by about 50,000 per microliter (**See Chapter 4**). Inadequate fibrinogen is the next most common deficiency in infants, and cryoprecipitate 15-20ml is effective in restoring normal levels of fibrinogen in most infants. Finally, coagulation proteins can be deficient; FFP is often utilized after CPB to correct these deficits. If available, the use of fresh whole blood is far preferable to component therapy for bleeding. Once bleeding is resolved, mediastinal drainage tubes are placed, and the sternum is closed. Many older patients weighing more than 20-30kg have simple cardiac surgery without blood transfusion. If cell salvage is available, residual blood in the CPB circuit at the end of bypass can be washed, filtered, and returned to the patient. However, this blood has few, if any, clotting proteins. After sternal closure, the patient is transported fully

monitored to the ICU and a full report and handoff given to the ICU team. Many patients undergoing simple cardiac repairs have their tracheas extubated in the operating room or shortly after arriving in the ICU.

Anesthetic Approach to Surgery for Congenital Heart Disease

Pre-Surgical Planning

Ideally a multidisciplinary conference with cardiologists, surgeons, anesthetists, and intensivists is held to present and discuss all cardiac surgery patients. The history and physical examination findings, CXR and laboratory results, and echocardiography and other images (such as cardiac catheterization, MRI, and CT) are presented. A surgical plan is proposed and discussed, and suitability of the patient for surgery is assessed. The group decides upon a final surgical plan. This plan may be altered if the surgeon's findings at operation differ from the presurgical conference data. The anesthetist plays an important role in this conference and should contribute to the discussion of perioperative risk and planning, particularly where non-cardiac medical problems are concerned.

Selection of Anesthetic Techniques and Agents

The preferred approach for designing an anesthetic for cardiac surgery is to devise a set of hemodynamic and ventilatory goals for each patient that are based on the patient's cardiac anatomy and on the pathophysiology of their cardiac lesion. Only when all of the information has been evaluated can an intelligent anesthetic plan by devised. The information in **Table 12-3** summarizes goals for the major categories of CHD.

Next, anesthetic drug selection is considered. The drugs chosen should have hemodynamic and myocardial effects that are most likely to allow the anesthetist to achieve the desired hemodynamic goals. For example, a patient with severe AS presenting for aortic valve replacement should not receive a large dose of propofol for induction of anesthesia because this drug will lower blood pressure, increase the AS gradient, worsen coronary perfusion, and possibly lead to cardiovascular collapse. All volatile anesthetic agents depress myocardial contractility to some extent if their concentrations are high enough, but there are significant differences among the available agents. For example, halothane depresses myocardial contractility more than isoflurane, sevoflurane, and desflurane in pediatric patients, most particularly in infant <6 months of age. If at all possible, halothane is avoided in infants with CHD or in other patients with CHD who have decreased myocardial function. If it must be used, the inspired halothane concentrations should be limited to <1 MAC. Sevoflurane should be used for inhaled induction of anesthesia in these patients when possible because it better preserves myocardial contractility. Isoflurane has even less effect on myocardial contractility and is a good choice of volatile anesthetic for maintenance of anesthesia for most CHD surgery. It is a poor choice for induction

of anesthesia because it causes laryngospasm and bronchospasm. Generally, opioids such as fentanyl have minimal effect on myocardial function and are well tolerated at a variety of doses. Ketamine is a particularly useful drug and may be used for IV or intramuscular induction of anesthesia; its vagolytic and sympathomimetic effect preserves cardiac output in most cases. **Table 12-4** summarizes the major hemodynamic effects of common anesthetic agents.

Table 12-4: Anesthetic Agents and Cardiovascular Effects

Agent	Myocardial Contractility	Heart Rate	Arterial Blood Pressure	Cardiac Output	Pulmonary Vascular Resistance
Fentanyl	---	↓↓	---	↓	↓↓
Midazolam	---	↓	↓	↓	↓
Ketamine	--- or ↓	↑	↑	↑	---
Propofol*	↓	↓	↓↓	?	---
Etomidate¶	---	---	---	---	---
Dexmedetomidine	---	↓↓	↓	↓	↓
Volatile agents	↓	--- or ↑	↓	--- or ↓	↓

*Changes from baseline: ↓ = mild decrease; ↓↓ = moderate decrease; ↑ = mild increase; ↑↑ = moderate increase; --- = no change. *Avoid propofol in preload/afterload dependent patients: L side obstruction, dilated cardiomyopathy, decreased LV function; ¶etomidate preferred in these patients*

Vascular Access, Monitoring, and Equipment for Cardiac Surgery

Secure peripheral IV access is essential for CHD surgery. In infants and children the saphenous vein is a reliable relatively large vessel into which at least a 22g catheter can be inserted in neonates and infants. Dorsal hand, wrist, or antecubital veins are large and thick-walled enough to accept large IV catheters. Arterial access is required for all CPB surgery and for most non-CPB CHD surgery, such as repair of CoA. This catheter provides crucial information on beat-to-beat arterial blood pressure and for measuring blood gases and pH. The radial artery is preferred in most cases. However, the femoral artery is large and well collateralized and can be used when the radial artery cannot be accessed. Percutaneous central venous access is utilized for many CHD surgeries, with the right internal jugular vein or femoral veins being the preferred site for placing a double-lumen catheter. Many simple surgeries, such as ASD and VSD, do not require central venous access; two large peripheral IVs suffice for many of these cases. Finally, the cardiac surgeon can place a transthoracic atrial catheter through the chest wall during the warming period on CPB that can be used for several days after surgery. The approach to vascular access varies widely according to the institutional setting, surgeon and anesthetist's preference and experience, and resources available. **Chapter 2** presents a complete discussion of vascular access

and monitoring.

Monitoring for CHD surgery also includes at least a 3-lead ECG that accurately displays cardiac rhythm and allows rapid diagnosis and treatment of arrhythmias, which are very common during CHD procedures. An oscillometric blood pressure cuff is an important monitor for determining systolic, diastolic, and mean arterial pressures at least every 5 minutes before arterial access is obtained and for monitoring these pressures should the arterial catheter fail during surgery. Pulse oximetry is crucial, not only for routine monitoring of oxygenation, but also to detect the frequent changes in desaturation that occurs with surgical interventions. The plethysmograph function of the pulse oximeter is also important for monitoring peripheral perfusion. While vasoconstriction, low temperature, and low cardiac output will affect this signal, complete loss of the pulse oximeter signal must be addressed immediately. Temperature monitoring is crucial, especially during hypothermic CPB. Two sites: nasopharyngeal, which reflects brain temperature; and rectal, which reflects core organ temperature, are preferred. If it is only possible to measure one temperature, nasopharyngeal is preferred because of the high blood flow to the brain in infants and need to protect this vital organ. Use of a capnograph that work appropriately with an infant's small tidal volumes is very important, both for standard monitoring of ventilation and for detection of the variable end-tidal to arterial CO_2 gaps commonly seen in cyanotic infants. A large gap (end-tidal CO_2 that is much lower that the arterial CO_2) signifies low pulmonary blood flow, low cardiac output, or both. Anesthetic gas monitoring is also desirable. The anesthesia machine ventilator must be capable of delivering small tidal volumes in the pressure control mode.

Early Tracheal Extubation After Congenital Heart Surgery

VSD, ASD, and PDA comprise 50% of congenital heart disease. The tracheas of most of these patients can be extubated at the end of surgery. The benefits of early tracheal extubation are also becoming more widely accepted for lesions such as TOF, AS, CoA, and other two-ventricle corrective surgeries. In general, if the patient did not require mechanical ventilation preoperatively, the surgery was uncomplicated, and there are no residual anatomic defects, there is no significant ongoing bleeding, no significant inotropic agent requirement or dysthymia requiring pacing or drug therapy, and there is no significant pulmonary hypertension, the patient is a candidate for early tracheal extubation, either in the operating room or within the first 2-4 hours after admission to ICU. Limiting the doses of long acting fixed agents, i.e., fentanyl or morphine, and full reversal of neuromuscular blockade are important preparations for tracheal extubation. The anesthetist must also plan for the patient's analgesic needs following tracheal extubation. Whenever possible, anesthetists must be available in the immediate postoperative period to assist with early airway management of extubated patients.

Anesthesia for Cardiac Catheterization

Cardiac catheterization, as noted above, can be used diagnostically to delineate anatomy and physiology, including Qp:Qs, intracardiac pressures and oxygen saturations, cardiac function, and the degree of pulmonary hypertension and response to pulmonary vasodilators. However, cardiac catheterization is used far less often for diagnosis in the modern era because echocardiography suffices for surgical planning for most patients. Either the cardiologist, or anesthetist provides sedation during diagnostic catheterization. IV ketamine, propofol, dexemedetomidine, opioids, and midazolam, can all be used to sedate these patients. In addition, the anesthetist can provide general anesthesia by endotracheal tube or laryngeal mask airway, if needed. Whatever the sedation or anesthetic technique used, the goals for diagnostic catheterization are the same: provide a steady baseline state from which valid hemodynamic and oxygen saturation data can be obtained. This normally means providing the lightest level of anesthesia possible, a FiO_2 of 0.21, and a blood pressure, HR, and ventilation (end-tidal CO_2) that are as close to the patient's awake baseline values as possible. Because the most stimulating and painful part of cardiac catheterization is gaining vascular access, usually via the femoral vessels, a brief period of deeper anesthesia plus local anesthetic infiltration of the groin area are sufficient. Once the vessels are catheterized, the anesthetic level is lightened to achieve the hemodynamic and ventilatory goals.

Cardiac catheterization is increasingly used for interventional procedures, such as aortic valve balloon angioplasty in neonates who have critical AS, for ASD and PDA closure, and for dilation and stenting a CoA. For these procedures, a cardiologist may sedate some patients, but in many institutions an anesthetist provides this care. Anesthetic management of patients undergoing interventional cardiac catheterization often requires a general anesthetic and tracheal intubation to provide a stable airway and allow neuromuscular blockade to immobilize the patient during critical phases of the intervention. Diagnostic information is often available before these interventions, and the same principles noted above apply, i.e., providing baseline conditions for hemodynamics and ventilation during the procedure. Before an intervention, e.g., dilation of AS, the anesthetist normally increases the FiO_2 to 1.0 and prepares to resuscitate these high-risk patients if necessary. Drugs needed for resuscitation and a cardiac defibrillator must be immediately available. Thorough pre-procedure discussion and planning with the cardiologist for her/his requirements for the procedure and for untoward events is important. Blood transfusion is frequently needed, and this blood should be immediately available for high-risk procedures. Although the trachea of most patients can be extubated at the end of the procedure, and the patient can be returned to a cardiac ward after recovery, some patients will require ICU care, which must be planned for in advance.

Anesthesia for Non-Cardiac Surgery

Because CHD is so frequent and because survival of CHD patients after surgical or catheter interventions is improving all over the world, patients with CHD will increasingly present for non-cardiac surgery. The types of surgery are generally not different from those required by patients without CHD, circumcision, hernia, myringotomy and tubes, tonsillectomy and adenoidectomy, appendectomy, and incision and drainage of abscesses. The approach to pre-anesthetic evaluation and planning is similar to that for cardiac surgery. A history of previous cardiac surgery and of residual lesions and symptoms is crucially important. The anesthetist must review the latest imaging study, usually echocardiography, the CXR, the last ECG, and the resting SpO_2 as part of their search for evidence of residual cardiac lesions. A number of studies over the past decade have provided a much more accurate picture of which CHD patients are at highest risk of cardiac arrest and death when undergoing anesthesia for non-cardiac surgery. The highest risk group appears to be those with pulmonary hypertension, particularly when the PA pressure is at or above systemic levels. Patients with significant LVOT obstruction are also at very high risk and have the greatest risk of death following cardiac arrest. Infants with a single functional ventricle are another group at significant risk for cardiovascular collapse under anesthesia. Finally, patients with cardiomyopathy and decreased myocardial function are at very high risk for less than desired outcomes. Even patients with CHD without PA hypertension are at higher risk for cardiac arrest under anesthesia than patients without cardiac disease. Included in this group are infants with unrepaired large VSDs. Another risk factor for cardiac arrest during non-cardiac surgery is the patient with unrepaired or palliated CHD. These high-risk groups are summarized in **Table 12-5.**

Table 12-5: Highest Risk Patients for Cardiac Arrest with Anesthesia for Non-Cardiac Surgery

High Risk Group	Example	Reasons
Left sided obstruction lesions	Aortic stenosis	Anesthetics lower blood pressure resulting in ↑ stenosis and ↓ coronary perfusion
Pulmonary hypertension	Large untreated VSD in older child	Cathecholamine release from light anesthesia, ↑$PaCO_2$ and ↓PaO_2 from ventilation problems will ? PVR and right to left shunt
Ventricular dysfunction	Dilated cardiomyopathy	Anesthetics lower blood pressure and preload, and decrease myocardial contractility leading to ↓ stroke volume
Single ventricle infants	Hypoplastic left heart syndrome	↑FiO_2 and↓$PaCO_2$ with intubation will ↑Qp:Qs; anesthetics decrease myocardial contractility

VSD = ventricular septal defect; PVR = pulmonary vascular resistance; Qp:Qs = pulmonary-to-systemic blood flow ratio

Infective endocarditis (IE) prophylaxis is an important consideration for patients with CHD who are undergoing non-cardiac surgery. In 2007 the American Heart Association revised its official guidelines, which are very similar to other authorities' guidelines around the world. To require IE prophylaxis, a patient must have both a cardiac condition and a surgical procedure indication that increases risk for IE. For example, a patient with a repaired VSD >6 months ago who has no residual defect does not need IE prophylaxis for any procedure. A cyanotic patient with an unrepaired TOF would require IE prophylaxis. A surgical procedure with no or very low risk of seeding the blood with bacteria does not require IE prophylaxis, even if the patient has a cardiac condition that might warrant it. For example, simple cystoscopy or endoscopy does not require IE prophylaxis. Dental procedures require IE prophylaxis. If IE prophylaxis is indicated, choice of antibiotics depends on the procedure. A single dose of IV antibiotics 30-60 minutes before the procedure is sufficient. **Table 12-6 A, B, and C** displays the current recommendations for IE prophylaxis. Antibiotic drugs are recommended for dental and respiratory tract procedures. For other cases, such as infected skin and musculoskeletal tissues, the antibiotic already being given for the infection is sufficient (nafcillin or methicillin, for example, for methicillin-sensitive *Staphylococcus aureus* infections). For gastrointestinal or genitourinary surgery, an antibiotic is used for prophylaxis against likely bacterial species that would be released into the blood and infect the heart when mucosa are incised. For example, ampicillin, with or without gentamycin, is effective for many urinary tract and gastrointestinal procedures. Clindamycin is often used for gastrointestinal procedures. Discuss the choice of antibiotics with the surgeon. In general, use the same antibiotic that would be used as surgical prophylaxis for patients without heart disease.

Table 13-6A: Cardiac Conditions Associated with Highest Risk for Infective Endocarditis

Prosthetic cardiac valve or material	
Previous infective endocarditis	
Congenital Heart Disease*	
	Unrepaired CHD, including palliative shunts
	Repaired CHD with prosthetic material; first 6 months after procedure
	Repaired CHD with residual defects
Cardiac transplant recipients with valvulopathy	

*CHD, congenital heart disease. * Except for those conditions listed, antibiotic prophylaxis is no longer recommended for any other form of CHD. Modified and reproduced with permission from Circulation. 2007;116:1736-54.*

Table 13-6B: Surgical Procedure Indications for Infective Endocarditis Prophylaxis*

Dental procedures involving manipulation of gingival mucosa or periapical region of teeth
Respiratory tract procedures: tonsillectomy/adenoidectomy, bronchoscopy, thoracic surgery
Infected skin, skin structures, or musculoskeletal tissues
Gastrointestinal or genitourinary procedures where mucosa are incised or biopsied (surgery or endoscopy); not needed for simple endoscopy or cystoscopy without biopsy

** Except for those conditions listed, in Table 13-6A, antibiotic prophylaxis is no longer recommended for any other form of CHD. Modified and reproduced with permission from Circulation. 2007;116:1736-54.*

Table 13-6C Antibiotic Regimens for Endocarditis Prophylaxis for Dental or Respiratory Tract Procedures

Setting	Antibiotic	Dose: Single Dose 30-60 Minutes Before Surgery
No penicillin allergy	ampicillin	50 mg/kg IV
Mild penicillin allergy	cefazolin or ceftriaxone*	50 mg/kg/IV
Severe penicillin allergy	clindamycin	20 mg/kg IV

**Do not use cephalosporins with a patient history of anaphylaxis, angioedema, or urticaria with penicillin or ampicillin. Give test dose of cephalosporins with history of mild penicillin allergy: 5 mg/kg IV, wait 5 minutes to assess for any reaction, then give remainder of dose slowly. IV = intravenous. Modified and reproduced with permission from Circulation. 2007;116:1736-54.*

Conclusions

This chapter has presented an overview of anesthesia for CHD, focusing on two-ventricle lesions that undergo complete corrective surgery. Because these lesions comprise >50% of patients with CHD, patients with these lesions are increasingly undergoing surgery world wide and surviving. Following surgical correction, most of these patients can lead a normal life and have normal life expectancy. Patients with a single ventricle and those with other very complex lesions comprise a smaller proportion of patients with CHD, require a large commitment of resources, and their outcomes in terms of mortality, morbidity, quality of life, and duration of life in general are not as good. The reader is referred to the Bibliography for a more extensive discussion of both the simpler and the more complex CHD patients. Anesthesia for CHD is a complicated field that is constantly changing, but because CHD surgery is increasingly practiced in many settings all over the world, it is hoped that this introduction will be useful to anesthetists who are participating in the care of these patients.

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Chapter 13
ANESTHESIA FOR ABDOMINAL SURGERY

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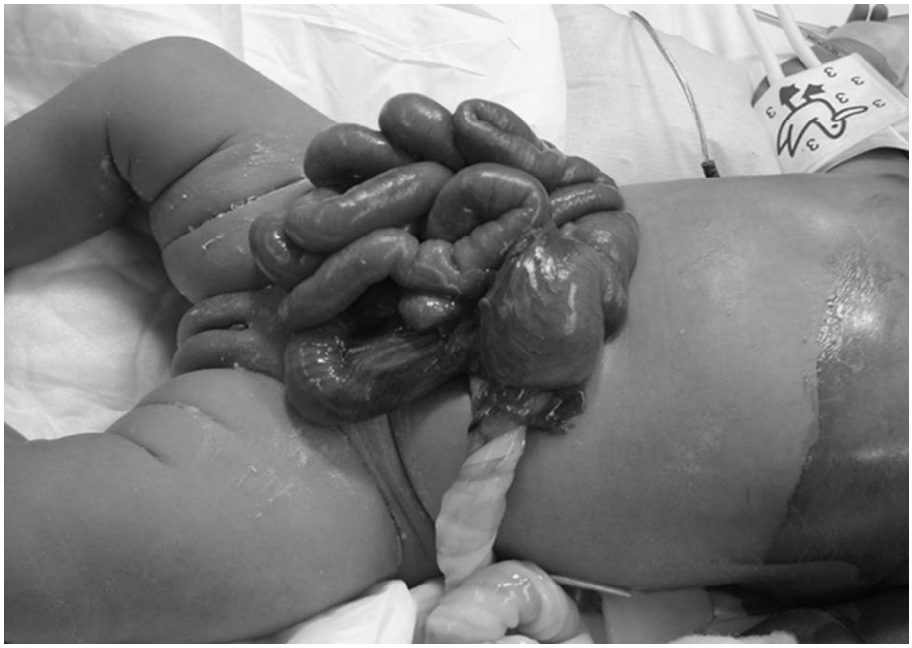
Introduction

Congenital anomalies of the abdomen are fairly common in neonates, infants, and children and often occur, along with other congenital anomalies, as part of a syndrome. Consequently, when an abdominal lesion is detected, the anesthetist must search for other anomalies, especially those related to the heart, as these anomalies are likely to complicate the anesthetic and the patient's perioperative course. This chapter discusses common abdominal lesions that require surgery and anesthesia.

Gastroschisis

Gastroschisis occurs in about 1:3,000 live births and is characterized by a defect in the abdominal wall that is usually located to the right of the umbilicus. It is characterized by a hole in the abdominal wall and exposed loops of bowel. **Figure 13-1**.

Figure 13-1: A Large Gastroschisis, Exposed Bowel, and Absent Abdominal Wall Covering.



Etiology

The exact etiology of gastroschisis is unclear, but it is believed to be due to disruption of the omphalomesenteric artery or the right umbilical vein. It has also been suggested that it might be the result of failed development of the abdominal covering after the intestines return to the abdominal cavity at about 10 weeks of fetal life.

Presentation

Gastroschisis usually occurs in isolation. Other congenital anomalies are seldom present. Affected babies are often born prematurely or have a low birth weight. Because there are no covering membranes, multiple loops of bowel are exposed to the environment and are prone to injury. Chronic exposure to amniotic fluid in utero causes fibrin strands that result in scaring, fibrosis, and multiple bowel adhesions. Attempts to take down these adhesions after birth result in marked bleeding from the bowel surface. With time the adhesions will spontaneously disappear and the bowel will float free in the peritoneum. Sometimes the bowel is strangulated and ischemic, which gives it a very edematous, blue, or dusky appearance.

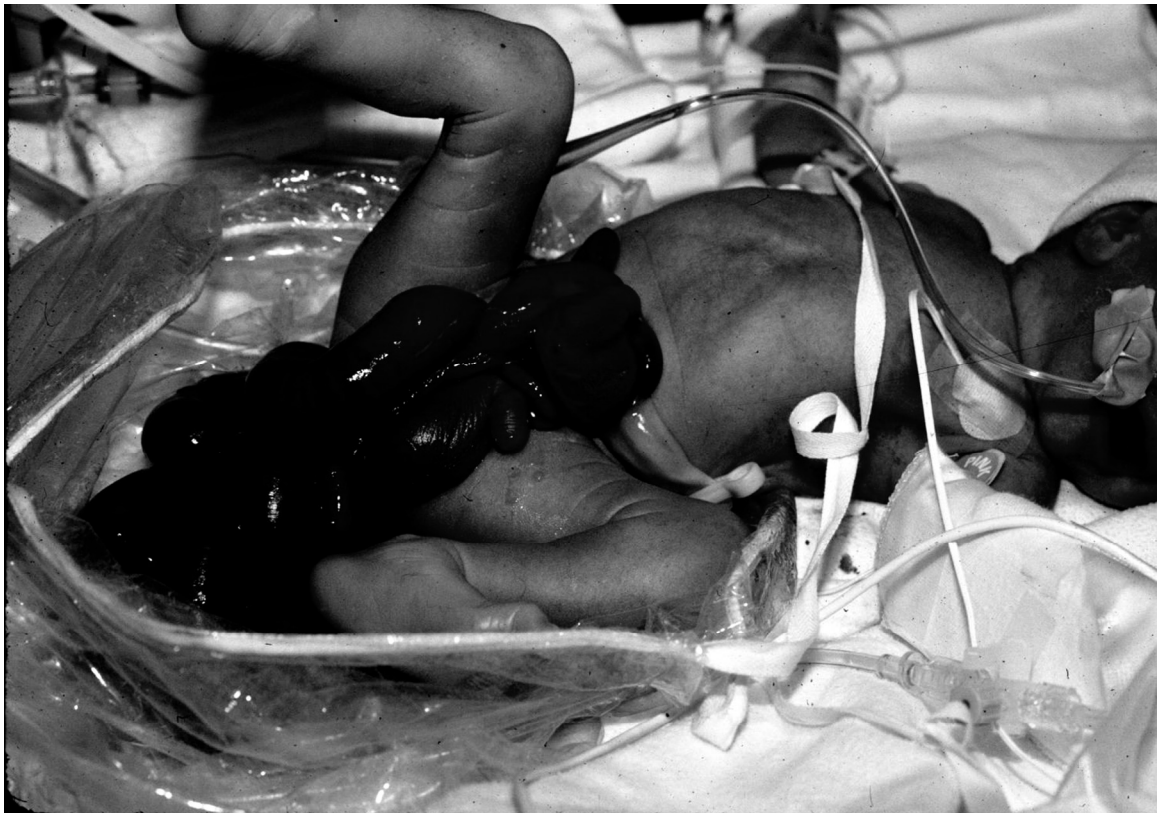
Pathophysiology

Depending when the baby presents for care after her/his symptoms begin, the baby may be dehydrated, i.e., may have sunken fontanels, sunken orbits, dry mucous membranes, and loss of skin turgor on physical examination (**See Chapter 1**). Infection may be present when the child is first seen. Depending on severity of the infection, the infant may be both septic and hypovolemic. Hypovolemia is usually the result of loosing large amounts of fluids from the exposed surface of the bowel and failure to take in sufficient fluids to compensate for these losses. Signs of hypovolemia include a very fast and thready pulse, cold extremities, and a mottled skin appearance.

Surgical Management

Before surgery occurs, it is important to prevent kinking of the bowel's blood supply. Supporting the bowel with sterile towels to keep it in the midline and prevent it from falling to one side or the other does this. At times it may be necessary to place the child on her/his side to protect the bowel. The bowel's blood supply must also be protected during transport of the patient to the hospital. To reduce the massive heat and water loss that occurs form the exposed bowel, it should be covered with a sterile plastic drape. Placing the entire child in a sterile plastic bag that covers her/his body from the feet to the mid chest will easily accomplish this. The plastic bag also allows collection of fluid lost from the bowel surface, which helps the anesthetist and surgeon determine fluid losses and requirements for fluid replacement.

Figure 13-2: Baby in a Plastic Bag for Warmth

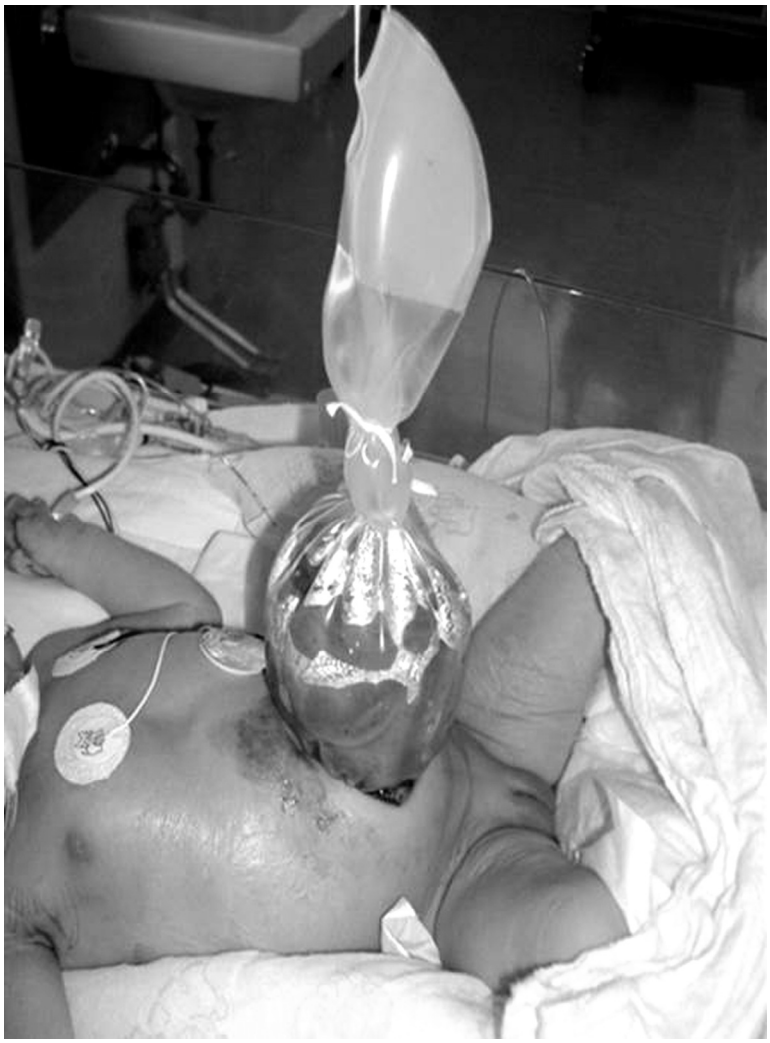


The baby shown here was transported for two hours by ambulance in the plastic bag in which she is lying. The bag was tied loosely around the baby's upper chest to enclose the entire lower body of the baby within the bag. Her temperature on arrival hospital was 36.8°C. There was 80ml of fluid in the bag that was lost from the exposed bowel. Courtesy of Joseph A. Kitterman, MD

If the infant presents to the treating hospital shortly after birth and the amount of extruded bowel is small, the abdomen can be closed immediately. However, if a large amount of bowel is exposed, and/or there is a delay in presentation to the hospital, it may not be possible to immediately close the abdomen. Because a significant amount of the baby's bowel was outside the abdomen during fetal life, babies with gastroschisis may have an abdominal cavity that is small and will not accommodate the entire bowel. In this case, the exposed bowel is enclosed in a silo (**Figure 13-2**). A silo is a plastic bag that is sewn to the abdominal wall and contains the entire extruded bowel. Each day the silo is squeezed gently and tied to make the bag smaller, thereby gently "forcing" the bowel into the abdomen. The bowel is slowly manipulated back into the abdomen by this technique over a few days, which allows the abdomen to stretch and accommodate the intestines. This process also allows the bowel edema to regress, making the bowel smaller and more likely to fit within the abdominal cavity. Once the extruded bowel has been reduced into the abdomen, the abdominal wall can be closed surgically. This gentle reduction of bowel is performed at the bedside. The amount of bowel reduced each day is

tailored to the infant's clinical response (effects on breathing and perfusion of the lower extremities) thereby reducing the infant's need for mechanical ventilation.

Figure 13-2: Baby With a Gastroschisis Who's Bowel Is Suspended in a Silo



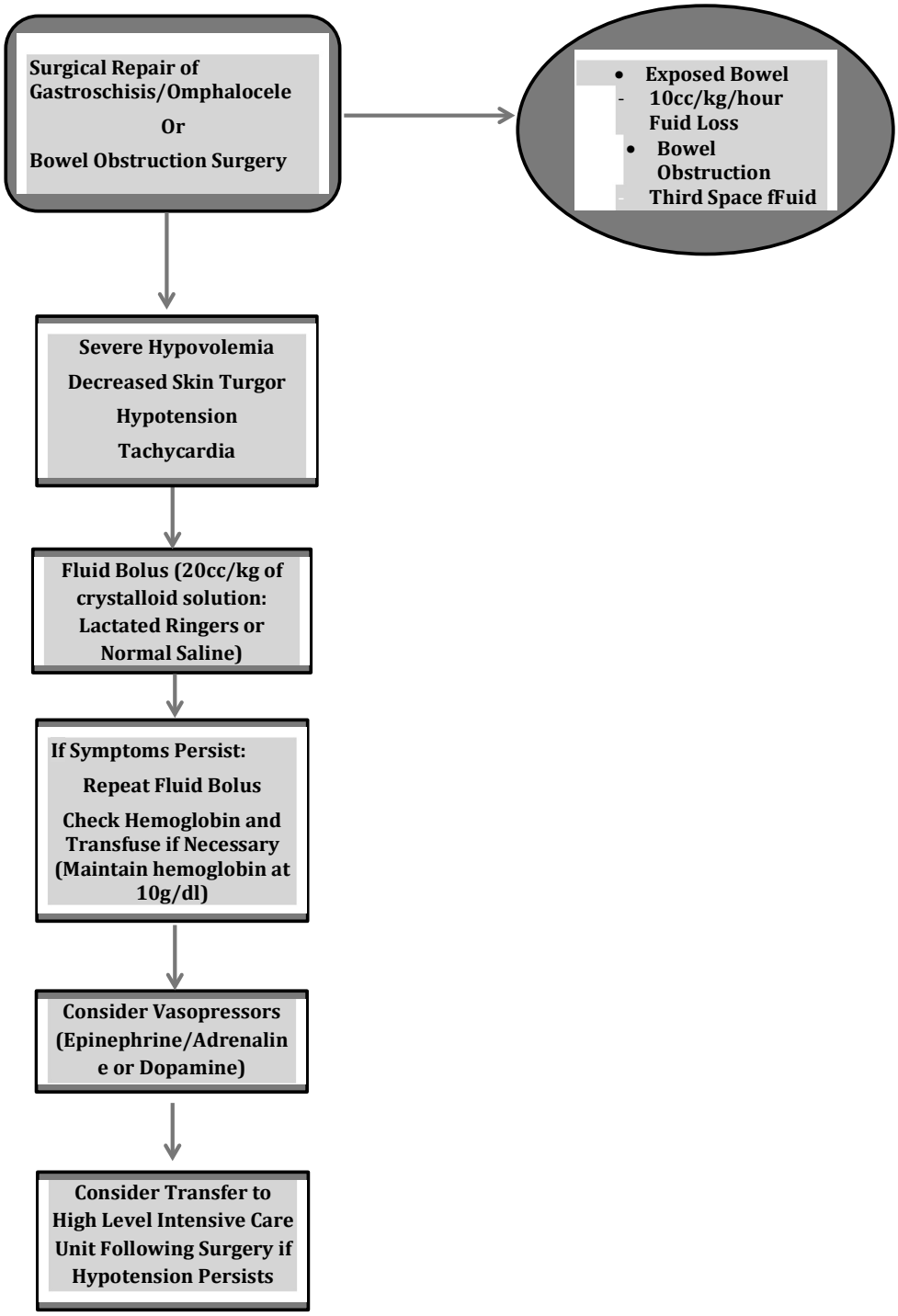
The sterile plastic bag is sewn to the abdominal wall and tied superiorly just above the bowel. Each day the tie is moved a little more towards the abdomen to slightly increase pressure on the bowel. The bag is tied to the top of the incubator to protect the blood supply and to aid in removal of edema fluid.

Anesthetic Management

General tracheal anesthesia with routine monitoring of arterial blood pressure, oxygen saturation, electrocardiogram, end-tidal carbon dioxide, and body temperature is required during surgical closure of the abdomen. Fluid resuscitation is very important in the peri-operative management of these patients, as a lot of fluid is lost from the exposed bowel. If the baby presents to hospital many hours after birth, he/she may be severely hypovolemic. Since this is a surgical emergency, efforts to resuscitate the baby with intravenous fluids should occur simultaneously with

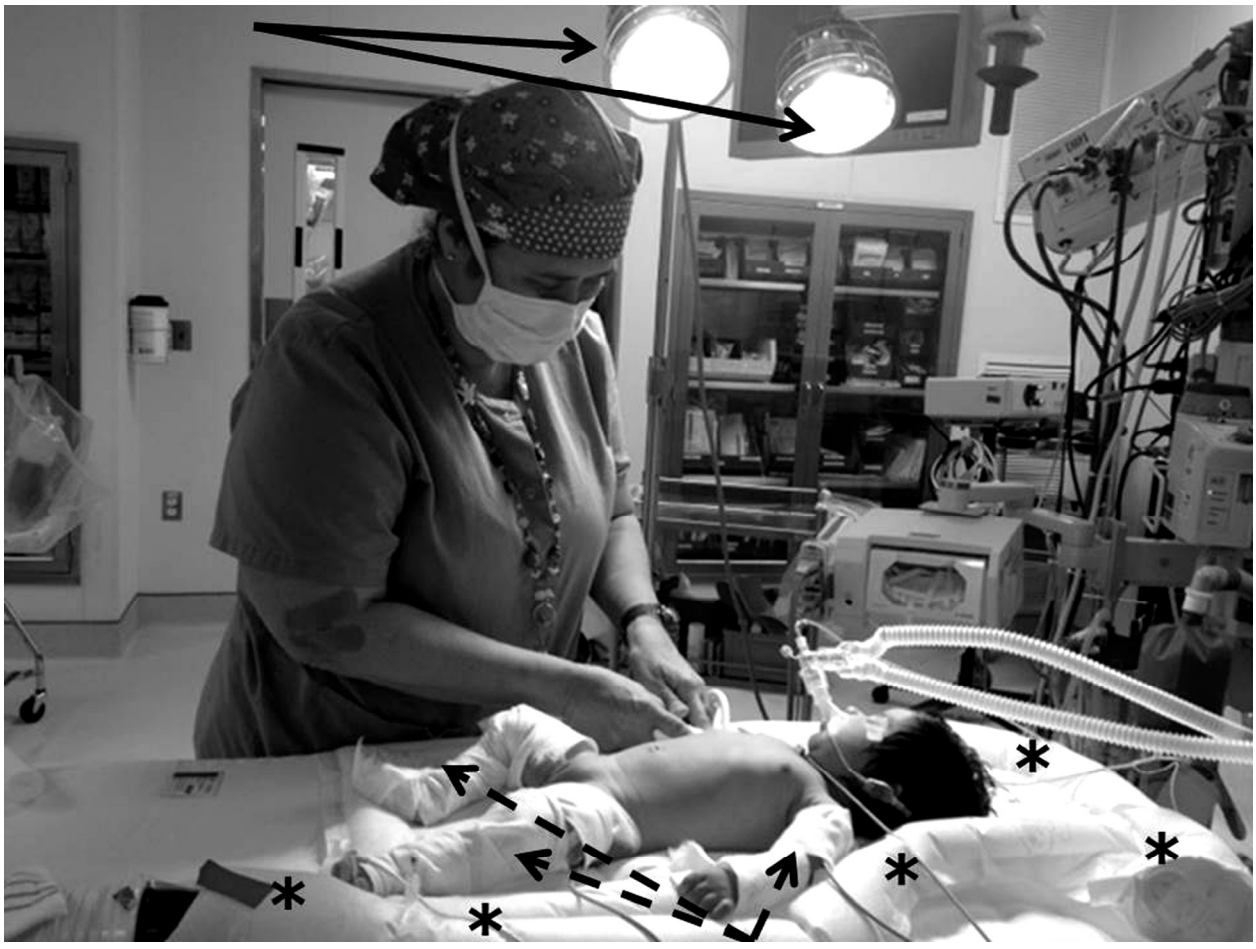
preparation for surgery. It is often necessary to continue fluid resuscitation during surgery because the exposed bowel is a continuous source of fluid loss **(Figure 13-3).**

Figure 13-3: Flow Chart Depicting Assessment of Volume Status and Management of Hypovolemia During Abdominal Surgery



Intraoperative hypothermia can easily occur with exposure of the bowel to the cool environment and with irrigating the exposed bowel with fluids (especially cool fluids), during surgery. The operating room should be kept very warm to decrease the incidence of hypothermia. A warm operating room is the most efficient method of preventing t hypothermia during surgery. Warming and humidification the inspired anesthesia gases also reduces heat loss (approximately 25% of heat loss occurs via the lungs during mechanical ventilation). In addition, the baby's exposed extremities can be wrapped with the same cotton material (sheet wadding) that is normally placed on extremities before applying a cast. The baby's head should be covered with a cap or with plastic, and intravenous fluids should be administered via a fluid warmer if possible. In situations where inflatable warming blankets are available, placing one of these blankets under the baby during surgery is a very effective way to maintain the body temperature.

Figure 13-4: Cotton Wrapped Around The Extremities of a Baby to Maintain Body Temperature



This figure shows several methods of maintaining a baby's body temperature in the operating room. The solid arrows-show an overhead heat lamp, which must be maintained at least three feet away from the baby's skin to prevent skin burns. The asterix show the forced-air warming blanket, and the dashed arrows show the baby's extremities wrapped in insulating material (sheet wadding).

Overhead radiant heat lamps may also be utilized to keep the baby warm during preparation for surgery, but the lamps should be placed no closer than three feet from the baby's skin to prevent skin burns. The lamp should be turned off if the skin is prepared with iodine containing solutions, because the combination of heat and iodine increases the chance of burns. **Figure 13-4** depicts several methods used to keep infants warm in the operating room. A humidifier, if available, should be inserted in the breathing circuit, as this is a very effective way of reducing heat loss and maintaining body temperature.

The use of a muscle relaxant during surgery facilitates abdominal closure. However, this sometimes makes it easier to replace the bowel in the abdomen and allows too much pressure to be applied to the abdominal contents. One good way to determine if the intra-abdominal pressure increases excessively when the surgeons attempt to place the bowel into the abdomen is to monitor the oxygen saturation and waveform of a pulse oximeter previously placed on a lower extremity. If the pulse waveform and the oxygen saturation (SaO_2) are normal, perfusion to the lower extremity is adequate and the intra-abdominal pressure is not excessive. However, if the oxygen saturation decreases and/or the pulse oximeter displays an abnormal waveform, this is a sign that placing the abdominal contents into the abdominal cavity is producing excessive intra-abdominal pressure. The surgeon should be immediately notified of this so he/she can consider placing the bowel in a silo and closing the abdomen later. The intra-abdominal pressure typically will continue to increase when the abdomen is closed because fluid will leak into the abdominal cavity from the surface of the injured bowel. Therefore, consideration should be given to placing the bowel in a silo if there is any sign that primary closure of the abdome will be difficult.

Returning the bowel to the abdominal cavity may decrease or prevent movement of the diaphragm, decrease tidal volume, and increase the end-tidal carbon dioxide concentration ($EtCO_2$). Therefore, monitoring of the end-tidal $EtCO_2$ is another way to determine if the intra-abdominal pressure is too high during surgical repair of a gastroschisis. If the $EtCO_2$ increases by more than 10mmHg during abdominal closure, the abdominal pressure is significantly elevated and consideration should be given to placing the bowel in a silo. If during mechanical ventilation the peak inspiratory pressure (PIP) increases to greater than $25cmH_2O$ when the surgeon attempts to place the bowel in the abdomen, consideration should be given to delaying surgical closure of the gastroschisis and placing the bowel in a silo.

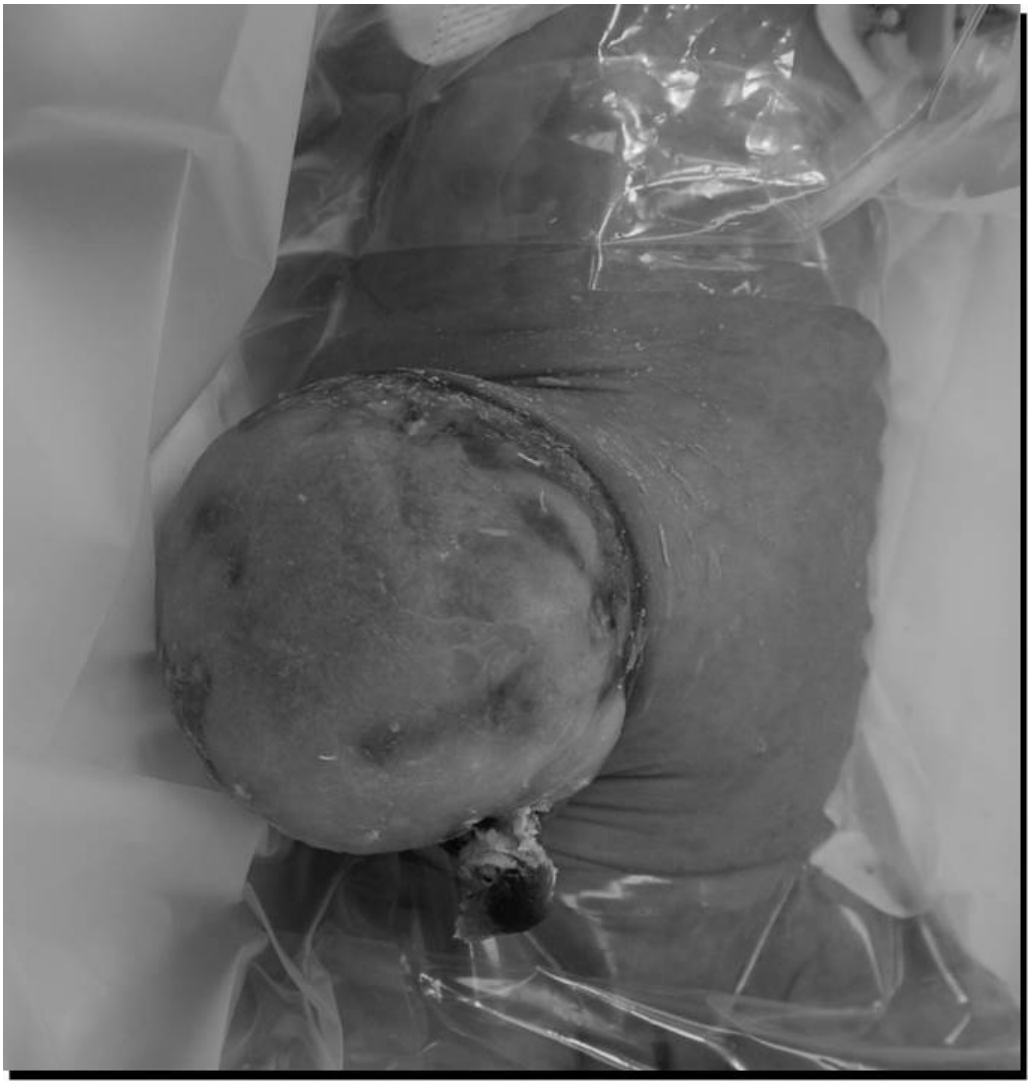
Due to the fairly significant increase in intra-abdominal pressure that occurs once the bowel is placed into the abdomen, there may be impaired movement of the diaphragm and impaired breathing. As a consequence, the majority of babies require mechanical ventilation after closures of a large gastroschisis. Tracheal extubation usually occurs a few days after surgery, when the intra-abdominal pressure has decreased. For environments that do not have the ability to maintain babies on mechanical ventilation with tracheal intubation, placement of a silo and

gradual reduction of the gastroschisis into the abdominal cavity is recommended before attempting to close the abdomen.

Omphalocele

Omphaloceles are characterized by herniation of the abdominal contents through the umbilical ring. In contrast to gastroschisis, the bowel of patients with an omphalocele is covered and protected by amnion. The size of an omphalocele varies. It can be very small (almost the size of an umbilical hernia) or very large (one that contains not only bowel but also other organs, e.g., the liver and spleen) **Figure 13-5**.

Figure 13-5: A Baby With an Omphalocele



This figure shows a large omphalocele with bowel and liver in the sac. Note the small size of the abdomen, which cannot accommodate the sac contents.

Presentation

Omphalocele is a midline lesion, and as such is associated with several other midline anomalies. Approximately 60% of affected infants have associated cardiac lesions; therefore the anesthetist must evaluate babies who have an omphalocele for cyanosis, low oxygen saturation, a cardiac murmur, or for abnormal pulses. A detailed history and physical examination plus an echocardiogram, when available, must be obtained to determine the severity of any accompanying cardiac disease (**See Chapter 12**).

Pathophysiology

Since omphaloceles are covered and not totally exposed, the threat of dehydration or sepsis is less, although still present.

Surgical management

Surgical management of an omphalocele is similar to that for gastroschisis. Large omphaloceles are often painted with an escharotic (scar producing) agent, such as silver sulfadiazine (silvadine) or betadine, wrapped, and gradually reduced in size. The contents are gradually eased into the abdomen over a period of time. Delayed abdominal closure is performed when the sac contents can be easily placed into the abdomen without compromising ventilation or bowel blood flow.

Anesthetic management

Intraoperative anesthetic management of omphalocele and gastroschisis is similar. However, the intra-operative fluid requirements for omphalocele surgery are fewer than those for gastroschisis, as the covering membrane prevents the bowel from being completely exposed, which minimizes fluid loss. Other anesthetic considerations include keeping the baby warm and monitoring oxygenation levels and the oxygenation saturation waveform patterns during closure of the abdomen to assure that the intra-abdominal pressures are not excessive.

Bowel Obstruction

Bowel obstruction is a common problem in infants and children. Many times it is due to a congenital anomaly; at other times it is acquired. The following section of this chapter provides an overview of this problem.

Pyloric stenosis

Pyloric stenosis is a common cause of gastrointestinal obstruction and occurs in 2-4:1,000 live births. Patients usually present with this malady between 2-8 weeks of age. Pyloric stenosis is the result of hypertrophy (thickening) of the stomach outlet (pylorus); it is more common in males

than in females. The cause of pyloric stenosis is unknown, but it occurs with higher incidence in bottle-feed infants and in twins.

Presentation

Projectile vomiting of non-bilious gastric contents is the usual first sign of pyloric stenosis. The vomiting increases with time and gradually becomes more forceful (projectile) in nature. Vomiting classically occurs after feeding but may occur at any time. The patient usually appears to be hungry and sucks vigorously, despite the vomiting. The repeated vomiting may lead to moderate to severe dehydration with loss of skin turgor and a depressed anterior fontanel. (**See Chapter 1**)

Diagnosis

A history of frequent vomiting in a child between 2-8 weeks of age is very suggestive of pyloric stenosis. Physical examination of the abdomen usually reveals hyper-peristalsis as well as an olive-sized mass in the right upper epigastric region, which is best palpated when the child is not crying or just after the child has eaten. The diagnosis can be confirmed by a barium swallow study, which reveals accumulation of barium just proximal to the gastric outlet and very little, if any, barium passing through the point of obstruction. Diagnosis of pyloric stenosis can also be made by ultrasound.

Pathophysiology

Gastric fluid is comprised of hydrogen, chloride, and potassium ions. Repeated vomiting of large amounts of gastric fluid depletes these ions and results in metabolic alkalosis and low serum concentrations of chloride and potassium. The kidneys compensate for the loss of hydrogen ion by exchanging potassium for hydrogen and sodium. This further decreases the serum potassium concentration and leads to an alkaline urine with a pH of >5. As vomiting progresses, the sodium and potassium stores become depleted, and the urine becomes acidotic (paradoxical aciduria). If this scenario persists, severe dehydration occurs, and the child has prerenal azotemia and metabolic acidosis.

Anesthetic Management

Surgical repair of pyloric stenosis is not an emergency. Repair should only be undertaken after the volume depletion and electrolyte abnormalities have been corrected. Pyloric stenosis is a medical emergency not a surgical emergency. Ideal fluid replacement should include either normal saline (NS) or 5% Dextrose in ½ NS with dextrose at a rate of 1.5 times maintenance fluid requirement. Four milli-equivalents (mEq) of potassium should be added to each 100ml of IV fluid and administered until the potassium concentrations have been repleted. Chloride, potassium, and bicarbonate concentrations should all be within normal limits before the child is taken for surgery.

Giving large volumes of 5% Dextrose in ½ NS may lead to hyponatremia (**See Chapter 3**).

Before anesthesia, the patient's stomach should be suctioned with a 10 or 14 French suction catheter to remove fluid and barium from the stomach. A suction catheter of this size is preferable because it is easier to remove residual gastric contents and/or barium from the stomach through them. Tilting the operating table into the head up and head down position and then turning the patients to their right and left sides allows more gastric contents to be removed. A vagal response may occur while suctioning the awake baby's stomach. Administering 20mcg/kg of atropine just prior to suctioning the stomach helps stop the suction catheter from causing bradycardia. The suction catheter should be inserted through the mouth into the stomach and suctioning continued until minimal aspirate is obtained. If there is blood tinged aspirate, stop the suctioning. If the child has a small nasogastric tube in place preoperatively, it should be removed and replaced with a larger sized catheter. While suctioning removes most of the gastric fluid, it does not remove all of it because suction catheters fail to reach all parts of the stomach. Consequently, a rapid sequence induction is still appropriate, even if gastric suctioning has already taken place. After gastric suctioning, the baby should be pre-oxygenated for 2-3 minutes before a rapid sequence induction is undertaken; cricoid pressure should is applied during induction of anesthesia to reduce the likelihood of regurgitation and aspiration of gastric contents. Cricoid pressure should not be excessive, or it may actually collapse and obstruct the soft tracheas of young children. If this occurs, release the pressure slightly. Oxygen desaturation may occur during induction of anesthesia if tracheal intubation is not accomplished on the first attempt. A styletted tracheal tube improves the likelihood of accomplishing tracheal intubation on the first attempt and is therefore a common practice with rapid sequence intubation.

During surgical repair of pyloric stenosis, large amounts of fluid are not required, especially if the surgery is done by a laparascopic approach. Ventilation is usually controlled during surgery, but it is important not to over ventilate the patient's lungs, as this will worsen existing alkalosis of patients who also have metabolic alkalosis. If the preoperative level of carbon dioxide (CO_2) is elevated and the pH is normal, the patient has compensated for the metabolic alkalosis. The anesthetist must only provide sufficient ventilation to maintain the preoperative CO_2 level, otherwise severe alkalosis and decreased cerebral blood flow will occur. The patient should be awake and responding to her/his surroundings at the end of surgery before removing the tracheal tube. This reduces the likelihood of aspirating gastric contents.

Pain Management

While correction of serum metabolic derangements often occurs before surgery, correction of electrolyte abnormalities in the cerebral spinal fluid takes longer. These abnormalities (especially an elevated bicarbonate level) can delay emergence from anesthesia. Thus, pain medication, particularly opioids, should be administered judiciously, if at all, during surgery. Acetaminophen

suppositories (40-45mg/kg) may be administered either during surgery, or the anesthetist can wait until after tracheal extubation. Alternatively the surgeon can infiltrate the operative site with local anesthetic towards the end of surgery. The combination of infiltrated local anesthetic plus rectal or postoperative oral acetaminophen elixir usually provides adequate pain control.

Other causes of bowel obstruction include malrotation of the bowel, volvulus of the bowel (twisting of the bowel around a focal point), intussusception (characterized by involution of a segment of bowel into another, the presence of Ladd's bands across part of the bowel, or atretic segments of bowel). Atresia of the bowel (abrupt ending of the bowel lumen) can be found in the duodenum, jejunum, or colon. Duodenal atresia often occurs in association with other congenital anomalies. It is often part of the syndrome whose acronym is VACTERL, which stand vertebral, ano-rectal, cardiac, tracheal-esophageal defects, radial bone (or renal) and limb anomalies. Duodenal atresia is a common finding in children with Down syndrome.

Bowel Obstruction Due to Atresia, Malrotation or Volvulus

Presentation

Continuous vomiting by patients with bowel obstructions causes moderate to severe dehydration, depending on the on the amount of time between the onset of the patient's symptoms and when the patient's presents to hospital. Signs of dehydration are discussed earlier in this chapter and also in **Chapter 1**. Severely distended bowel, increased abdominal girth, and visible loops of bowel below the abdominal wall are commonly detected on physical examination. An abdominal X-ray confirms the diagnosis when it reveals dilated loops of bowel and/or air fluid levels. The presence of free air in the abdominal cavity (bowel perforation) is a surgical emergency; The mortality rate with perforation is high. Bowel sounds may be hyperactive or decreased, depending on whether the child is in an early or late stage of the disease.

Pre-Operative Preparation

Laboratory values, including hemoglobin level and serum electrolytes, should be obtained, as these data help guide the patient's perioperative fluid and electrolyte management. Depending on the hemoglobin concentration, intra-operative blood transfusion may be required. An elevated serum sodium concentration (hypernatremia) is an indication of the severity of the dehydration is and is helpful in directing perioperative fluid resuscitation.

Surgical Management

In children, exploration of the abdomen and relief of a bowel obstruction is done under general anesthesia. Because these children have a full stomach, they are prone to aspirate their gastric contents during the induction of anesthesia. Appropriate precautions (rapid sequence induction of anesthesia with cricoid pressure) should be taken to prevent this from occurring.

Anesthetic Management

Bowel obstruction due to malrotation, volvulus, or bowel atresia requires urgent surgery to prevent the bowel from becoming ischemic and/or perforating, especially if the bowel's blood supply is compromised. Sometimes the bowel has perforated by the time the patient presents for care, further increasing the urgency of surgical repair. Use of routine anesthesia monitors, including electrocardiogram (ECG), arterial blood pressure, end-tidal carbon dioxide (when available), and core temperature are imperative. Fluid resuscitation is very important, both before and after induction of anesthesia. Therefore, adequate intravenous access is required for surgery. This usually means inserting at least two intravenous catheters. While 24 gauge intravenous catheters are easier to place, 22 or 20-gauge catheters permit more rapid infusion of fluids, blood, or albumin during surgery. A large bore catheter is especially important for resuscitation.

Adequate pre-oxygenation is mandatory because oxygen desaturation occurs commonly once anesthetic drugs are given. This occurs because the distended bowel compresses the diaphragm, causing atelectasis and inadequate lung expansion with inspiration. The stomach should be adequately suctioned before anesthesia is induced, not only to reduce the likelihood of aspirating gastric contents but also to reduce the size of the stomach and decrease compression of the diaphragm. If a nasogastric catheter is not present, one should be inserted and connected to suction to remove as much of the stomach contents and gas as possible before inducing anesthesia. Despite adequate preoperative gastric suctioning, regurgitation may still occur during induction of anesthesia. Therefore, the rapid sequence induction technique should be used when intubating the trachea. The intravascular volume must be corrected prior to inducing anesthesia to avoid causing severe hypotension. Rapid sequence induction typically involves administering of sedative-hypnotic drug, such as intravenous thiopental (4-6mg/kg), propofol (2mg/kg), ketamine (2mg/kg), or etomidate (2mg/kg), and a rapid acting muscle relaxant, such as succinylcholine (2mg/kg) or rocuronium (1mg/kg) while applying cricoid pressure to prevent regurgitation of gastric contents. On occasion it may not be possible to intubate the trachea on the first attempt. In such situations, gentle mask ventilation (enough to see the chest move with each manual ventilation) may be performed while continuing to apply cricoid pressure. This helps prevent or treat the rapid decrease in oxygen saturation, which is made worse by atelectasis that is induced by the distended abdomen. Ketamine and etomidate are particularly helpful in patients who have bowel obstruction and accompanying sepsis or moderate dehydration, as these drugs decrease arterial blood pressure less than thiopenthal or propofol, especially propofol, which causes significant decreases in systemic vascular resistance. It is important to note that neither ketamine nor etomidate prevents hypotension if the patient is severely hypovolemic. Thus, the importance of normalizing the intravascular volume as much as possible before the induction of anesthesia cannot be overemphasized. In the presence of perforated bowel, however, surgery may have to proceed while fluid resuscitation is ongoing. During this time vasopressors [adrenaline (epinephrine), dopamine] may be needed to support blood pressure and cardiac output.

Maintenance anesthesia is usually accomplished with halothane, sevoflurane or isoflurane and with the administration of intermittent doses of muscle relaxants. If possible, the muscle relaxant used should increase the patient's heart rate, as this helps sustain cardiac output and arterial blood pressure in young children. Muscle relaxation, which is very helpful during abdominal surgery, may be achieved by giving intermittent boluses of rocuronium or other short/intermediate acting muscle relaxants. Succinylcholine is usually avoided (except in a dire emergency) because it predisposes patients who have the genetic predisposition this disease to develop malignant hyperthermia (MH). This drug should be used very cautiously, especially in patients with undiagnosed muscle weakness or in those with a family history of members who developed malignant hyperthermia or febrile episodes that eventually led to death or the need for intensive care after surgery. The administration of succinylcholine to 3-8 year old males should be avoided as much as possible, as male children of this age can have undiagnosed muscle disease and develop severe hyperkalemia, cardiac arrest, and die after normal doses of succinylcholine.

After inducing anesthesia, catheterizing the urinary bladder allows the anesthetist to monitor urine output during surgery. This is a helpful guide for evaluating fluid status. If the urine output is at least 0.5-1ml/kg/hr during surgery, kidney perfusion is adequate. Lesser amounts of urine production suggest that resuscitation of intravascular volume is incomplete. Additional fluid boluses of 10ml/kg or more of Ringer's lactate or normal saline are indicated. These boluses of fluid should be dextrose free, as large volumes of dextrose containing solutions can cause hyperglycemia, particularly in septic patients. Maintenance fluids (*not volume replacement fluids*) should, however, contain 5% dextrose to provide sufficient glucose to meet the patient's needs, particularly during the anesthetic management of children less than one month of age or children with inadequate nutrition. If possible, the serum glucose concentration should be measured during surgery to be certain the serum glucose concentrations are not too high or low. During anesthesia serum glucose concentrations usually increase for at least three reasons: 1) decreased utilization of glucose; 2) catecholamine induced glucose production from glycogen; 3) interference by catecholamines with the uptake of glucose at the cellular level. All of these tend to cause hyperglycemia in both sick and in anesthetized patients. The only way to know the patient's glucose concentration is to measure it. If a cardiac arrest occurs when the glucose level is >200mg/dl, 50% fewer patients can be resuscitated and of those resuscitated, 50% will have central nervous system (CNS) injury. Similar injury can occur with hypoglycemia.

Patients with bowel obstruction vomit, which results in the loss of large amounts of gastrointestinal fluid. Their intravascular volume also decreases because fluid is sequestered into the injured bowel. Fluid resuscitation should start with 10cc/kg. of dextrose free balanced salt solution, such as lactated Ringer's solution and normal saline. A sufficient volume of crystalloid solution must also be infused continuously during surgery (10cc/kg/hr. or more) to replace the fluid losses caused by handling the bowel and exposing it to the environment during surgery. At times, much more fluid is required. The amount of fluid needed must be based on frequent

evaluation of the patient's arterial blood pressure, heart rate, tissue perfusion, and urine output (**See Chapter 3**). Colloid solutions, such as 5% albumin, can also be utilized for volume expansion, but they are more expensive than crystalloid solutions and have not been shown to be any more effective at restoring intravascular volume than normal saline. Maintenance fluids must also be given in addition to the replacement fluid. Maintenance fluids should contain 5% Dextrose. The usual administration rate is 4ml/kg/hr. for the first 10kg of body weight; 2ml/kg/hr. for the 2nd 10kg of body weight; and 1 ml/kg/hr. for the every kilogram of weight over 20kg (**See Chapter 3**). A balanced salt solution with 5% dextrose should be used for this purpose, not D5 ½ NS or ¼ NS. These latter two solutions can lead to severe hyponatremia. Maintenance fluids are usually administered continuously through one IV and replacement fluids for ongoing losses through a separate IV whenever possible.

Pain Management

Pain relief is usually accomplished by giving intermittent intravenous doses of pain medication, such as ketamine (1-2mg/kg), pethidine, (0.5-2mg/kg) fentanyl (0.5-1.0mcg/kg) or morphine (0.5-1.0mg/kg). Pethadine must be administered cautiously to young children because one of its metabolites causes significant respiratory depression and lasts a long time. In addition, excretion and metabolism of pethidine is reduced in neonates compared to adults, so its effects may last longer. Many children with bowel obstruction are premature and are at risk for post-operative apnea. Administration of opioids to these infants should be done with caution. Close postoperative monitoring for apnea and hypoxemia is therefore mandatory in these patients. The use of neuraxial blockade, such as epidural anesthesia, with infusion of local anesthetics, can also be employed for pain management (**See Chapter 21**). However, the sympathetic blockade that occurs when local anesthetics are given for these blocks may further accentuate intraoperative hypotension, especially if the child is hypovolemic. Neuraxial blockade is not an option for septic children, as it may further aggravate the infection and predispose the child to getting a central nervous system infection. For very sick patients, infusions of vasoactive medications, such as epinephrine (adrenaline) or dopamine, may be necessary during surgery to maintain adequate hemodynamics (blood pressure and heart rate).

Conclusion

Anesthesia for patients needing abdominal surgery requires knowledge of the lesions and of how they affect the patient's physiology. This knowledge must include evaluation of these patients for volume status and for the presence of other congenital anomalies. Cardiac anomalies are the most common accompanying lesions and may complicate both the anesthesia and the postoperative course. Despite all of the real and potential complications, survival of these patients is good if they receive appropriate preoperative, intraoperative, and postoperative care.

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Chapter 14
ANESTHESIA FOR GENITOURINARY SURGERY

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Pediatric patients appear for surgery with a wide range of urologic conditions. For most of these children, the surgery is elective, but emergent procedures do arise (i.e. testicular torsion, relief of acute urinary obstruction). Because they are usually otherwise healthy, these patients do not require extensive medical workup. However, if the patient has other congenital anomalies besides their urologic problems or they have a syndrome, preoperative assessment must include evaluation of the associated anomalies (i.e. airway, neurologic, pulmonary, cardiac, hepatorenal, hematologic, musculoskeletal, etc.) to determine if these anomalies will impact the anesthetic technique to be utilized.

Acute or chronic renal insufficiency is part of many urologic conditions. In general, *acute renal insufficiency* impairs the kidney's ability to remove or retain water, electrolytes, and products of metabolism. It is classified according to the anatomic location of the insult: pre-renal (impaired renal perfusion), renal or Intrinsic (structural damage to the renal parenchyma), or post-renal (obstruction of the lower urinary outflow tract). Intravascular and/or extravascular volume and electrolyte changes may be present and cause depletion to overload of either or both of these compartments. When renal insufficiency is suspected, laboratory tests are obtained to determine which (if any) electrolyte is abnormal. While all electrolyte concentrations can be abnormal, hyperkalemia is of particular concern because it may be life threatening. Renal insufficiency is often accompanied by acidosis. Consequently, evaluation of the patient's acid/base status prior to surgery is important.

Patients with *chronic urologic conditions* have pathophysiologic issues similar to those with acute renal failure (i.e., fluid and electrolyte abnormalities, acidosis); however, other organs are more likely to be injured in patients with chronic renal failure. Where available, some patients with chronic renal failure will be on renal replacement therapy (dialysis). Many of these patients are hypovolemic immediately after being dialyzed. Consequently, it is important to determine the patient volume status prior to surgery (**See Chapter 1**). For elective procedures, it is better for the patient if he/she is dialyzed the day before surgery. This minimizes the likelihood of her/him being hypo or hypervolemic at the time of surgery. The latter may cause hypertension and pulmonary edema. Comparing the patient's "dry weight" (weight when patients are not fluid overloaded) to her/his current weight will help the anesthetist determine the patient's volume status. Additional

issues for patients on renal replacement therapy include anemia and coagulopathy. Thus, the patient's hemoglobin level and coagulation status should be determined, when possible, prior to undertaking invasive surgical interventions. Based on the results of these tests and the likelihood of surgical hemorrhage, it may be appropriate to have suitable blood products immediately available. Systemic hypertension is also a common finding in patients with chronic renal failure, and it may be long-standing. Because the changes are chronic, it is desirable to maintain systemic blood pressure within 20% of the patient's *baseline* blood pressure, even if elevated. The target blood pressure during anesthesia is relative to the patient's elevated baseline pressure, not normotensive values for an ideal patient population. The goal is to assure sufficient blood pressure to maintain end organ perfusion (especially the brain). This is best done by maintaining the arterial pressure within the patient's normal range.

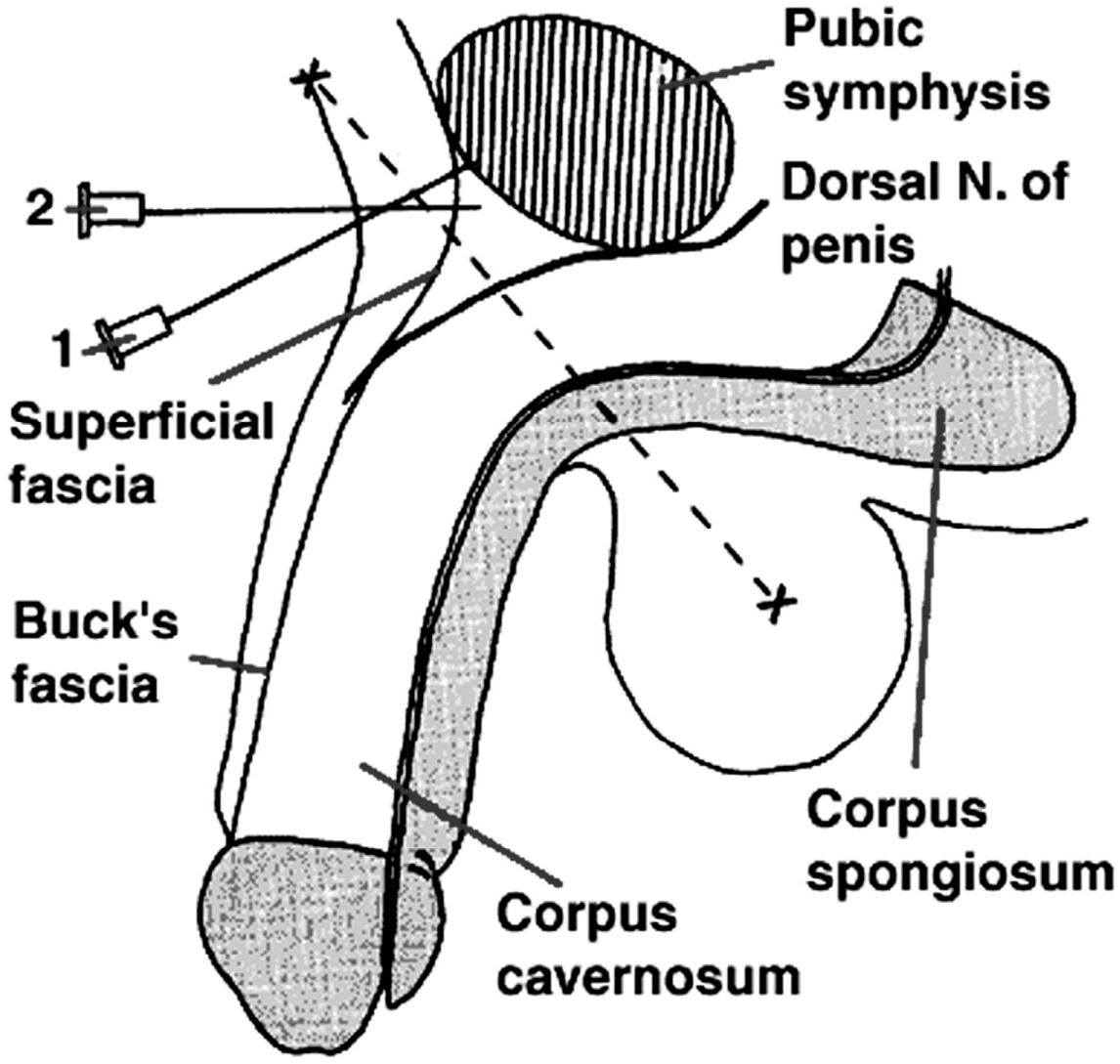
Specific Procedures

Circumcision

For centuries *circumcision* has been performed for religious or cultural reasons and for medical indications. Consequently, it is one of the most widely performed surgical procedures worldwide. Cited medical benefits of circumcision include reduction in the number of urinary tract infections (UTIs), some protection against sexually transmitted diseases,[1,2] a reduction in the incidence of penile cancers,[3] and possibly improved hygiene. [4,5] It is highly desirable to reduce the number of UTIs in infants with a congenital uropathy, including those with high-grade vesicoureteral reflux (VUR) and posterior urethral valves. The potential benefits of circumcision must be weighed against potential complications, including bleeding, infection, meatal stenosis, and complications of anesthesia.

In the United States, circumcision is usually performed by the obstetrician or pediatrician on the first or second day of life using a Gomco® clamp or Plastibell®. Little or no anesthesia is provided. However, given recent increased awareness that newborns do in fact experience pain, many of these procedures are being performed using penile nerve blocks (**Figure 14-1**).

Figure 14-1: Penile Anatomy And Block



Penile nerve blocks are done after sterilizing the skin below the symphysis pubis. The needle is inserted at a 30°angle in the midline at the level of the inferior border of the symphysis pubis and advanced posteriorly. As the needle passes through superficial and Bucks fasciae, the anesthetist should feel two distinct "pops". If the inferior border of the symphysis pubis is contacted with the needle, withdrawn it slightly and advance it slightly more inferiorly. When the needle is beyond the second "pop", apply gentle suction to the syringe to determine that the needle is not in a vein or artery. Then inject 0.25ml/kg of 0.25% bupivacaine. Large volumes of drug may cause undue pressure on the nerves and vascular supply of the penis and injure them. ***e-safe-anaesthesia.org****,* with permission.

Patients whose circumcisions were done without anesthesia respond more to painful stimuli later in life[6]**;** therefore, some type of anesthesia should be provided for circumcision from infancy through adulthood. Spinal anesthesia has been used for circumcision, but most of them are performed under general anesthesia. Modalities used to manage post-operative pain include penile nerve blocks (**See Above**) or caudal epidural blocks; they are often supplemented with IV

analgesics. When it is not possible to use a regional technique, IV narcotics combined with non-steroidal analgesia may be used. Although many analgesic modalities have been employed, no one technique has been proven superior to the others. A recent Cochrane review of the data found no differences between caudal or penile blocks and parenteral analgesics alone. But the reviewers noted that good evidence is lacking, secondary to small trials and poor methodology.[7]

Hypospadias - Chordee Repair

Hypospadias is an abnormal opening of the urethra or meatus on the penis. This opening is usually on the ventral surface of the penis, but can occur on the glans, the shaft of the penis, or the scrotum or perineum. Hypospadias occurs in 0.3-to-0.7 percent in live male births[8] and is more common in Caucasians[9]. The diagnosis is usually made during routine newborn physical examination. If he also has cryptorchidism (undescended testis), the patient may have a disorder of sexual development. A pelvic ultrasound will determine the presence of internal gonads and the possible presence of a uterus. These findings are complimented by karyotyping.

Surgical correction of hypospadias is often done in one stage; however, a series of stages, each lasting a few hours, may be required for more complicated corrections. The current recommendation of the American Academy of Pediatrics is to perform this surgery before 18 months of age, after which gender identity is defined.[10,11] In general, the surgery is carried out between 3 and 18 months of age. Earlier interventions (3-6 months) may be associated with improved wound healing because these children produce fewer pro-inflammatory cytokines.[12] If a second procedure is needed; it is performed after adequate wound healing has taken place.

Some anesthetists prefer general endotracheal anesthesia (GETA) rather than spinal or caudal anesthesia for these procedures, due to the young age of these patients and the length of the procedure. Because neuromuscular blockade is not necessary to facilitate surgical exposure, general anesthesia can be provided through a laryngeal mask airway (LMA). Excellent post-operative analgesia can be achieved with a caudal epidural neuraxial block (**See below**). Many anesthetists use 1ml/kg of 0.2% ropivacaine or 0.25% bupivacaine for these blocks. Some will add 1µg/kg of preservative-free clonidine to supplement the block or extend its duration (**See Chapter 21**). Placing a caudal block prior to surgical incision allows the block to be used as part of a balanced anesthetic technique. This offers a number of benefits, including need for less volatile anesthetic (which facilitates more rapid emergence from anesthesia) and for providing preemptive analgesia. Given the length of these procedures, it may be beneficial, in the author's experience, to administer an additional one-half of the original local anesthetic dose into the caudal space at the end of surgery. For example, if 10ml of 0.2% ropivicaine were injected at the beginning of the procedure, 5ml of 0.2% ropivicaine would be injected at the end. This reduced dose of ropivicaine at the end of surgery prolongs postoperative analgesia and minimizes local anesthetic toxicity.

Wilm's Tumor

Nephroblastoma, also known as *Wilms Tumor*, is the second most common solid organ tumor of childhood, after neuroblastoma. However, it is the most common primary renal malignancy of childhood. It occurs in approximately eight children per one million births and commonly presents as a unilateral lesion at 3-4 years of age. It presents at a slightly younger age when the tumor is bilateral. There is slight females predominance, with a 0.92:1 male: female ratio. The risk of developing a Wilms tumor is higher in people of African decent than in Caucasians. Those of Asian decent have the lowest incidence. Although most Wilms tumors are due to sporadic mutations, a familial relationship is present in roughly 1.5% of cases. Nearly 10% of all cases of Wilms tumor are associated with a genetic syndrome. These include WAGR (30-50% of cases), Denys-Drash (approximately 90% of cases), and Beckwith-Wiedemann (greater than 90% of cases).[13] Mental retardation (WAGR), nephrotic syndrome (due to mesangial sclerosis and progressive renal disease, DDS), macrosomia, macroglossia, and hypoglycemia (BWS) are characteristic of these patients that are important to anesthetists.

Wilms tumors most often present as painless abdominal masses. However, anorexia, vomiting, malaise, hypertension, microscopic hematuria, and rarely an acquired von Willebrand disease type 1 (**See Chapter 4**) are sometimes presenting symptoms. The diagnostic work-up starts with an abdominal ultrasound (when available), followed by a Computerized Tomography scan (CT) or more commonly a Magnetic Resonance Imaging (MRI) scan when available. If these scans are not available, the diagnosis is usually made during exploratory laparotomy. A tissue biopsy is **NOT** obtained because this increases the risk of tumor spillage and metastesis.

In the United States, most patients undergo surgical resection of the tumor before receiving chemotherapy. Surgeons attempt to remove the tumor en bloc without rupturing the tumor capsule.[14] To do this, a trans-peritoneal approach through a large transverse subcostal incision is used, which causes significant postoperative pain with breathing. Massive hemorrhage is possible during resection of the tumor because the tumor can extend into the renal vessels, inferior vena cava (IVC), and occasionally into the right atrium.

Chemotherapy is given preoperatively if the child has a solitary or horseshoe kidney, if the tumor is bilateral, extends into the inferior vena cava (IVC) or right atrium (RA), is considered unresectable due to its massive size, or there is respiratory distress from pulmonary metastasis. Whether external beam radiation therapy is added to the patient's treatment depends on the cancer stage at initial diagnosis.[15]

Some chemotherapy agent(s) used to treat these tumors will be of concern to the anesthetist. These include vincristine, doxorubicin, and dactinomycin. External beam radiation therapy may also be of concern. Known side effects of these agents include neurotoxicity (vincristine), cardiotoxicity, myocarditis, and pericarditis (doxorubicin), hepatotoxicity (dactinomycin), and

pulmonary fibrosis with scarring of the treated area (external beam radiation).[16] The anesthetist must search carefully for signs or symptoms of these side effects. Failure to recognize them could be fatal for the patient.

Children with Wilms tumors are frequently otherwise healthy. Resection of their tumor is likely to be the first in a series of medical interventions they will experience. Therefore, premedicating them with an anxiolytic drug with amnestic properties (e.g., midazolam) may be very beneficial. General endotracheal anesthesia is preferred for maintenance of anesthesia, due to the large abdominal incision and because neuromuscular blocking agents are needed to facilitate surgical exposure. Adequate large bore intravenous (IV) access is required, given the risk of hemorrhage from tumor and/or vascular rupture. Type specific blood products should be immediately available before surgery begins. At least one large bore IV should be placed when possible in an upper extremity, since it may be necessary to cross-clamp the IVC to control hemorrhage. Intra-operative anesthetic challenges include: less than adequate pulmonary ventilation, due to increased intra-abdominal pressure; large fluid shifts between compartments (**See Chapter 3**) that occur with volume resuscitation and an open abdomen; massive hemorrhage; an evolving coagulopathy (**See Chapter 4**); major vascular injury and/or temporary IVC cross-clamping; thermoregulation; and pulmonary emboli in patients who have intravascular extension of their tumor. Post-operative analgesia can be provided with parenteral opioids, but epidural analgesia is generally favored (**See Chapter 21 and below**). Given the large transverse subcostal incision, epidural analgesia facilitates more normal pulmonary function, early ambulation, and the return of normal gastrointestinal function.

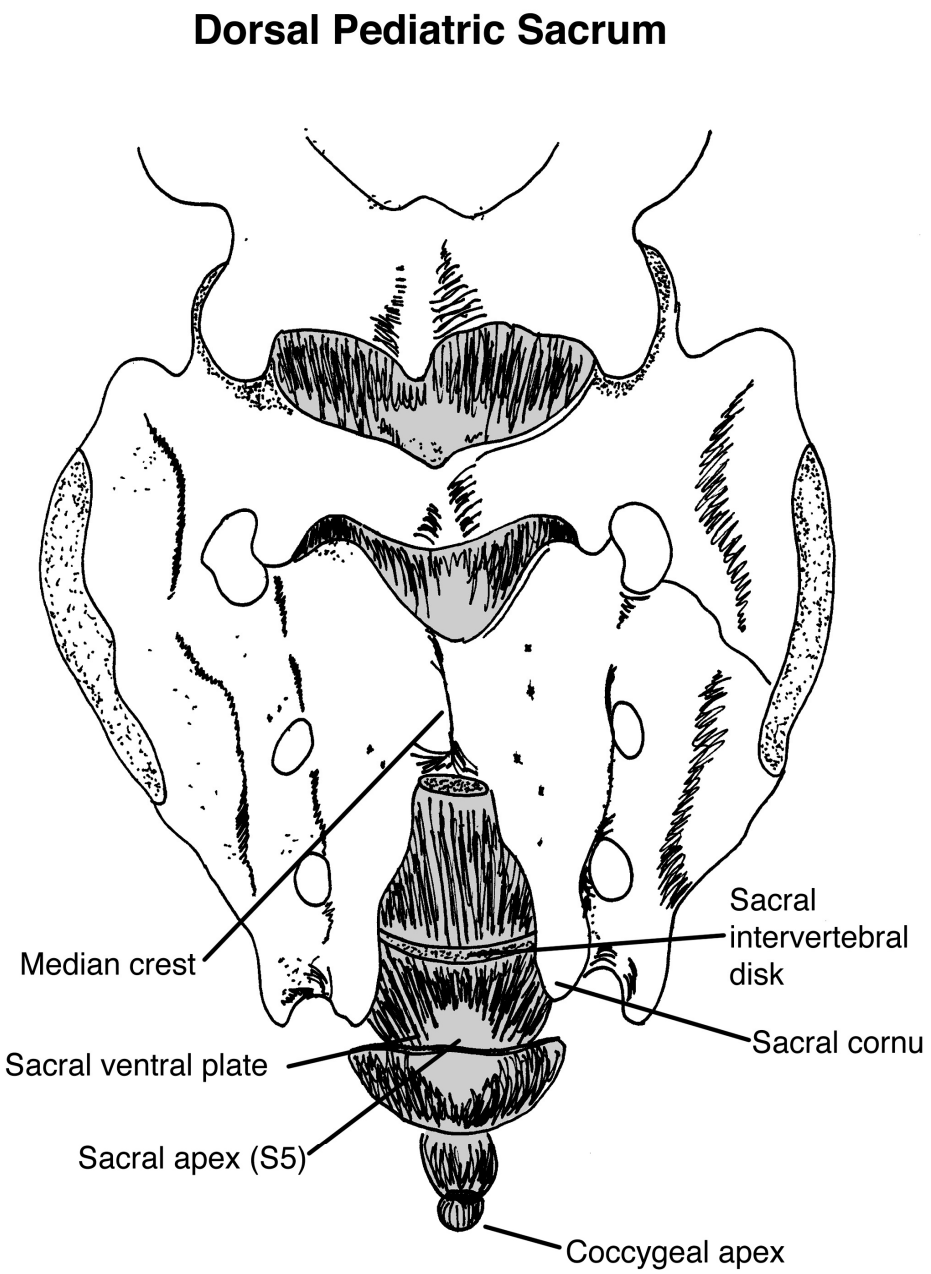
Neuraxial Anesthetic/Analgesic Techniques

Caudal Epidural Block

A caudal block is frequently utilized to provide analgesia for infants and children undergoing surgical intervention for urologic conditions. It is usually used as an adjunct to general anesthesia. Neuraxial blocks allow the use of less inhaled anesthetic during the procedure. In addition, it reduces or eliminates the need for intravenous opioid administration in the perioperative period, which facilitates rapid emergence from anesthesia and reduces/avoids opioid side effects, including nausea, vomiting, ileus, and pruritus.

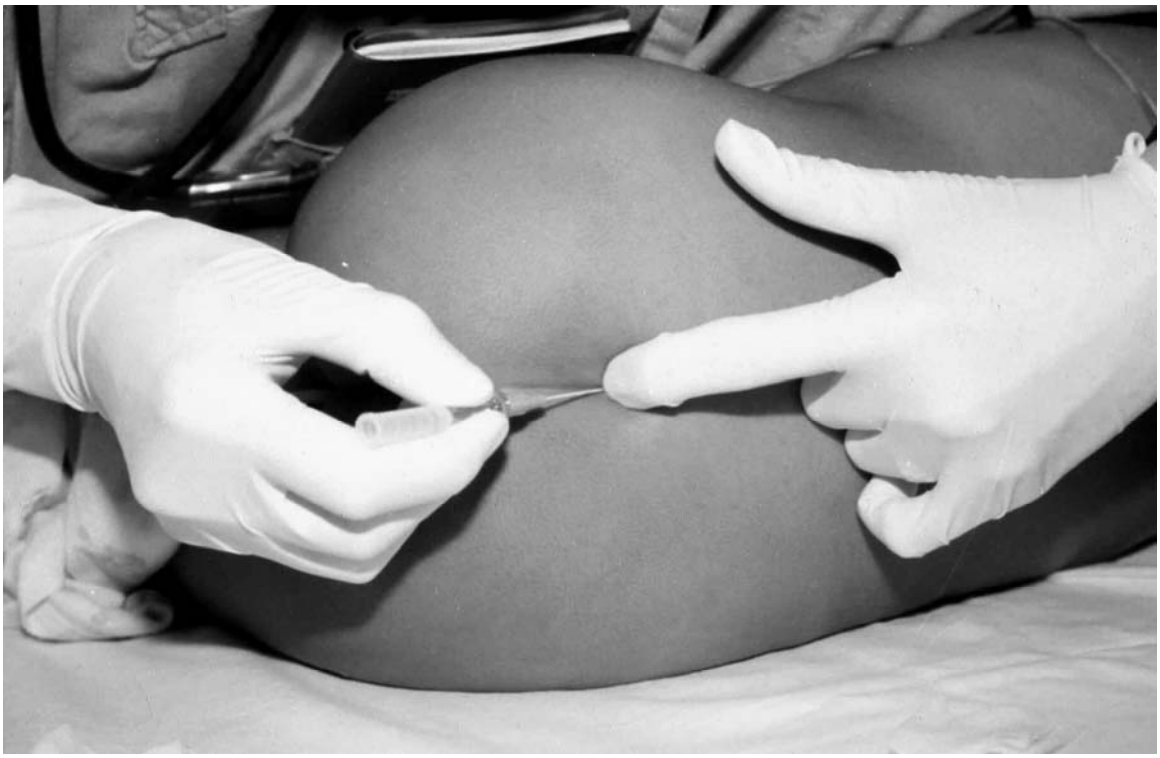
A caudal block is performed with the infant or child in the lateral decubitus position; both thighs and knees are flexed. The anatomic landmark is the sacral hiatus; a “recess” located between the 5th sacral cornua (**Figure 14-2, 14-3**). These cornua are palpated as two bony prominences that are about 0.5 cm apart and are located cephalad to the intergluteal fold (crease of the buttocks).

Figure 14-2: Sacral Anatomy of Young Children



This figure shows the bony anatomy of the sacrum. A caudal epidural is done by inserting a needle or catheter in the midline at the inferior level of the sacral cornua. See text.

Figure 14-3: Caudal Block in a Child



*Caudal block being performed in a child. A 22 gauge intravenous catheter is inserted into the caudal space after the back is sterilized. Once the needle tip has entered the space, the catheter is advanced off of the needle and the needle is withdrawn. This reduces the risk of entering the subarachnoid space. The catheter can be connected to sterile tubing for repeated injection of local anesthetic during and after surgery (**See below**). Photograph courtesy of Maurice Zwass, MD*

If it is difficult to identify the cornua, the coccyx is palpated and a finger is slid cephalad to the "recess". A 22-gauge short beveled needle or angiocatheter is inserted into the sacral hiatus (recess between the cornua) at a 45° angle. A "pop" is felt as the needle pierces the sacrococcygeal ligament. The "pop" is more easily felt with a short-beveled needle. With the needle in the caudal epidural space, the needle angle is decreased from 45°-to-0° (parallel to the long axis of the spinal canal). (IV catheters can be advanced to their hub.) The needle is then advanced *only* a few millimeters because the dural sac extends lower in infants (L2 or L3) than in adults (L1) (**See Chapter 10**); the dural sac can easily be entered if a needle is advanced too far cephalad. The needle or catheter now rests in the caudal canal, which is continuous with the epidural space in patients less than 5-6 years of age. The needle or catheter is aspirated to confirm the absence of blood and/or cerebrospinal fluid (CSF). Local anesthetic and any adjuncts being used are injected in increments with aspiration tests preceding each incremental injection.

The optimal dose and volume of drug to give in the caudal space has been studied extensively. Takasaki and colleagues[17] published an often-cited formula that determines the volume of drug needed to achieve the desired level (by dermatome) of blockade.

Volume of local anesthetic (ml) = 0.05ml/kg/dermatome to be blocked

Another technique described by Armitage involves giving: 0.5ml/kg for lumbosacral level blocks, 1ml/kg for thoracolumbar level blocks, and 1.25ml/kg for mid thoracic level blocks.[18] Bupivacaine 0.25% (maximum volume 20ml) is the drug most often used. Volume of drug injected affects the level of blockade; drug concentration affects density of the block. It is important not to exceed 3mg/kg of either ropivacaine or bupivacaine to avoid causing local anesthetic toxicity. Lower drug doses are recommended in infants less than six months of age because they have less alpha-1-glycoprotein. This increases the plasma concentrations of free local anesthetic and increases the likelihood of exceeding toxic drug levels.

Adjuvant medications are often injected along with the local anesthetic to extend or supplement the blockade. These include fentanyl 1-2µg/kg, preservative free morphine 30-70µg/kg, epinephrine (1:200,000), and/or preservative free clonidine 0.5-1µg/kg. The lower dose of local anesthetic is selected when the desired level of analgesia is below the umbilicus; the higher dose is selected when analgesia is required above the umbilicus.

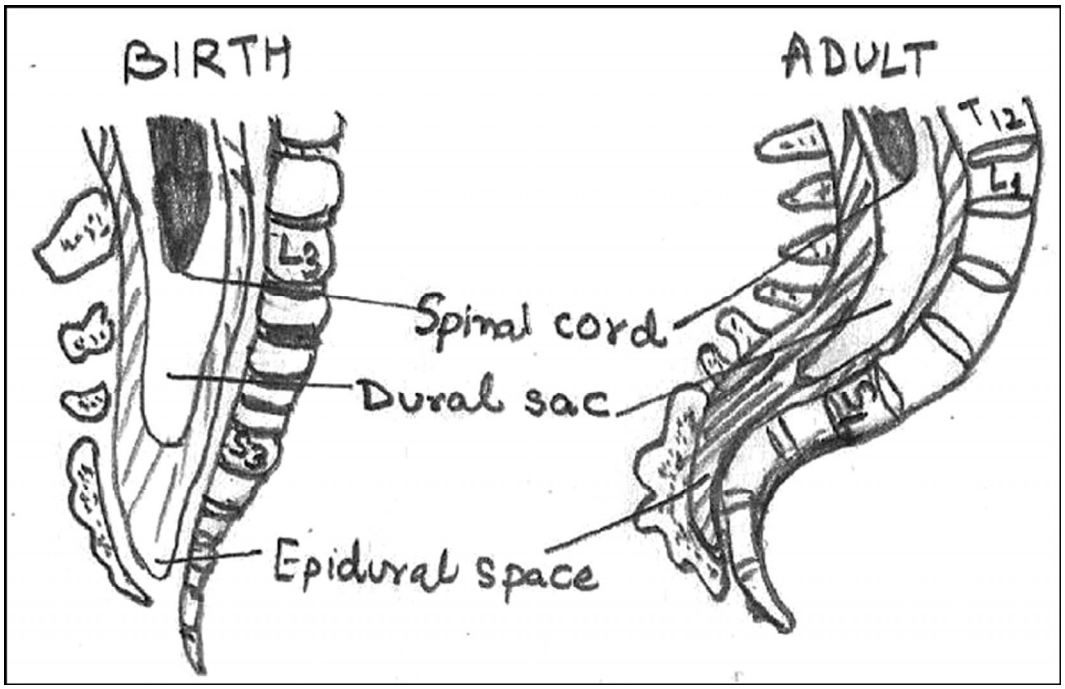
When the surgical procedure last longer than 3-4 hours, the block can be "re-dosed" at the end of the procedure. To allow repeated dosing of local anesthetic during and after surgery, an IV catheter can be inserted, left in place, connected to sterile tubing, and secured in-situ with a clear adhesive dressing. At the author's institution, it is common practice to re-dose the caudal with one half the initial volume of drug approximately 2.5 - 3 hours after giving the initial dose of drug (see above). If a medication infusion pump designed for neuraxial use is available, continuous drug infusion, 0.2-0.4mg/kg/hr. of a 0.1% - 0.2% solution of ropivicaine or bupivacaine can be used. The drug dose is reduced by 30% for <6-month-old infants[19].

Spinal Block

Spinal (intrathecal) anesthesia has been used in children for nearly a century (**See Chapter 21**) and is often the only anesthetic needed for many urologic procedures. Spinal anesthesia is particularly useful for repairing inguinal hernias in premature neonates because it reduces post anesthetic apnea in these patients.[20,21] While this is true, it is still advisable to observe these patients overnight in a monitored in-patient unit regardless of the anesthetic technique used. Some of them develop postoperative apnea no matter what anesthetic is used. Another advantage of spinal anesthesia is that it that it can be used for infants who have drunk clear liquids up to two hours before surgery. To calm babies during spinal anesthesia, a pacifier dipped in a 50 percent dextrose solution (D50) is often given to them to suck on during the procedure. The dense neuraxial blockade achieved with spinal anesthesia also appears to reduce the infant's level of consciousness; they often fall asleep after a spinal anesthetic is placed, possibly due to decreased sensory input reaching the reticular activating system.[22]

There are anatomic and physiologic differences in the spinal cords of infants, older children, and adults. In infants, the spinal cord (conus medullaris) ends at L3; it reaches L1, the adult level, after one year of age. Therefore, for subarachnoid block the safest needle insertion site in infants is the L4-L5 (or even L5-S1) interspace (**Figure 14-4**). In proportion to body weight, both the CSF volume and rate of CSF turnover are much greater in infants than in adults. Dilution of local anesthetics into this large volume of CSF explains why larger intrathecal doses of local anesthetic (mg/kg) are required to produce the desired block. The increased rate of drug turnover explains the shorter duration of subarachnoid block in infants.

Figure 14-4: Differences in Spinal Cord Between Infants and Adults



The spinal cord of neonates ends at L3, while that of adults ends at L1. The infant's dural sac ends at S3 and the adult's at S1. Thus, it is easier to enter the infant's dural sac while performing caudal anesthesia and produce a total spinal anesthetic. http://www.joacp.org. *With permission*

As noted above, intrathecal dosing of local anesthetics is different for infants and adults, but the drugs used are the same. The agents most commonly used for spinal blockade in infants are bupivacaine and tetracaine. The bupivacaine dose is 0.5 -1mg/kg as an isobaric (0.5% bupivacaine) or a hyperbaric (0.75% bupivacaine in 8.25% dextrose) solution.[23,24,25] The tetracaine dose is also 0.5 – 1mg/kg as a hyperbaric (0.5% tetracaine in 5% dextrose) solution.[26,27] Adding epinephrine to local anesthetics extends the block duration approximately 30%.[28] A technique for adding epinephrine to the local anesthetic is to fill a tuberculin syringe with epinephrine (1:1000) and then expel its contents; the residual amount of epinephrine in the syringe is sufficient to prolong the block. The higher local anesthetic dose (1mg/kg) produces motor blockade to mid-thoracic dermatome levels and may prolong the block to 1.5 hours.[29,30,31] Lower local anesthetic doses

(0.5 – 0.6mg/kg) are more appropriate for short duration lower extremity procedures.

Standard cardio-pulmonary patient monitoring (**See Chapter 2**) should always be used when placing a spinal block. Having adequate intravenous access during this time is also important. The block can be done with the child in either the sitting or lateral position. However, the CSF hydrostatic pressure in the lower back is higher in the sitting position, which increases flow of CSF through small spinal needles. Significant neck flexion is avoided to prevent causing airway obstruction, a common problem in young children. Infiltration of the skin with local anesthetic (1% lidocaine) is generally done with a 30G needle at the intended needle insertion site (midline at the L4-L5 or L5-S1 interspace). A 22G spinal needle with a stylette, is used to avoid the small risk of carrying tissue into the CSF and causing an epidermoid tumor. "Pencil point" spinal needles are available in pediatric sizes. Two tactile "pops" should be experienced, one at the ligamentum flavum and the other at the dura mater. Following the 2nd "pop", the stylette is removed; CSF flow through the needle confirms intrathecal placement of the needle. Smaller gauge needles are available; however, free flow of CSF through them is impeded. Local anesthetic is injected slowly to avoid causing increased cephalad displacement of the drug and a "high spinal". If an electro-cautery pad is needed for surgery, it can be placed on the patient's thigh.

Epidural Blockade

When innervation of the surgical incision site is from higher dermatomes (e.g., a Wilm's tumor incision), it is advantageous to place an indwelling epidural catheter at that specific dermatome level. This allows the anesthetist to provide extended post-operative analgesia, especially when a continuous infusion of local anesthetic (often with an opioid adjunct) is used. Inserting the epidural catheter at the higher dermatome has at least two advantages: 1) less likelihood of contaminating the insertion site with stool or urine; 2), smaller volumes of local anesthetic are needed to produce anesthesia/analgesia than are needed when the drugs are given by the caudal route. The safety of placing both lumbar and thoracic epidural catheters by experienced anesthetists has been demonstrated.[32,33,34] The technique for placing an epidural catheter for infants is similar to that for adults, but there are a few differences. In infants and children the ligamentum flavum is thinner, making it more difficult to recognize "engagement" of the epidural needle. Also, the angle of needle entry is less acute (more perpendicular) in children. The distance from the skin to the epidural space is estimated to be 1mm/kg of body weight for children between 6-months and 10-years of age.[35] Venous air embolism has occurred during placement of an epidural block in children, probably by injecting air into an epidural vein. Therefore, it is recommended that saline (not air) be employed when using with the loss of resistance technique to identify the epidural space.[36,37] Epidural catheters should be placed using equipment specific in size and caliber for infants and children. A 5cm 18G Tuohy needle, with either a 20G or 21G epidural catheter, can be used without difficulty. This shorter Tuohy needle offers better needle control during catheter placement. Epidural neuraxial blockade may be used intraoperatively with

the same advantages noted with caudal epidural block (**See above**). To initiate blockade, it is recommended that 0.3-0.5ml/kg of 0.1-0.25% bupivacaine[37] (or 0.1-0.2% ropivicaine) be incrementally injected after the aspiration test is found to be negative and that this be done before each increment of drug is injected. A continuous infusion of local anesthetic 0.2-0.4mg/kg/hr. is then initiated. This dose of drug is *decreased* by 30% for infants <6 months of age.

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Chapter 15
ANESTHESIA FOR ORTHOPEDIC SURGERY

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Care of patients undergoing orthopedic procedures requires attention to many anatomical and physiologic principles that may not be immediately apparent if one only focuses only on the proposed surgical procedure. Most patients undergoing these procedures are healthy, but they can have many other problems, depending on their age, acuity of disease, and whether their orthopedic problems are the result of congenital anomalies, syndromes or trauma. It is the responsibility of the anesthetist and other healthcare providers to provide safe care, including prevention of future injury, and a smooth, comfortable recovery from anesthesia and surgery.

In this chapter general principles, specific orthopedic procedures, and treatment options for particular orthopedic problems are discussed. As for many things in medicine, surgical and anesthetic management often includes very expensive surgical components, anesthetic agents, monitors, and appropriate specialists. Many of these vastly exceed the capabilities of the healthcare systems of some countries. Therefore, this chapter focuses on the more common, straightforward components needed to provide safe care for patients with orthopedic problems.

General principles

Patient Populations

Children of all ages, from infants with congenitally dislocated hips to young adults who are suffering from massive trauma, require orthopedic procedures. Routine preoperative assessment, including the patient's medical history and physical examination, is essential. Attention to the underlying disorders and surgical problems are important for avoiding anesthesia complications. The more we know about the patient, the more likely it is that we can prevent or reduce these complications. The mortality risk following orthopedic procedures is not as great as it is for patients with cardiac disease or those undergoing thoracic or abdominal surgery[1.] Similarly, surgery on the extremities generally has little direct impact on pulmonary function. Yet an anesthetist who is familiar with a patient's baseline cardiac and pulmonary status will be better able to anticipate potential risks of anesthetic techniques and agents, of blood loss, and of surgical manipulation on that individual patient.

Each patient should undergo a though examination before anesthesia is induced. In addition to airway, cardiac, and respiratory assessment, body habitus, including, weight, extremities, presence of limb contractures, bedsores, and other wounds must be sought and noted to determine the potential effects of anesthesia on them. Some lesions will affect positioning of the patient for surgery, and these should be known and planned for. The anesthetist should determine if the skin of a potential nerve block sites is intact, infected, or inflamed. The presence of any one of these conditions may prevent the anesthetist from using this particular technique. Extremes of weight for age increase the risk for positioning related injury. For patients with multiple trauma injuries, the extent of their various injuries and how they will affect the outcome of surgery and recovery must be evaluated. This includes the integrity of the spinal cord and the potential for further injury that may be caused by moving/positioning the patient for bag-and-mask ventilation, tracheal intubation, and surgery. If cerebral trauma or severe trauma to other organs has occurred and requires non-surgical treatment, it may be safer for the patient to delay most orthopedic procedures until the patient's other problems have been successfully treated. Visual inspection of an injured extremity may reveal massive swelling that should alert the anesthetist to the possibility of internal bleeding and the occurrence of hemodynamic instability when the surgeon incises the extremity. The presence of a femur fracture in an infant or toddler may be evidence of child abuse and should cause the anesthetist to search for other injuries. He/she should look for bruises or injuries suggestive of abuse and initiate social assistance and patient protection.

Pediatric Assessment

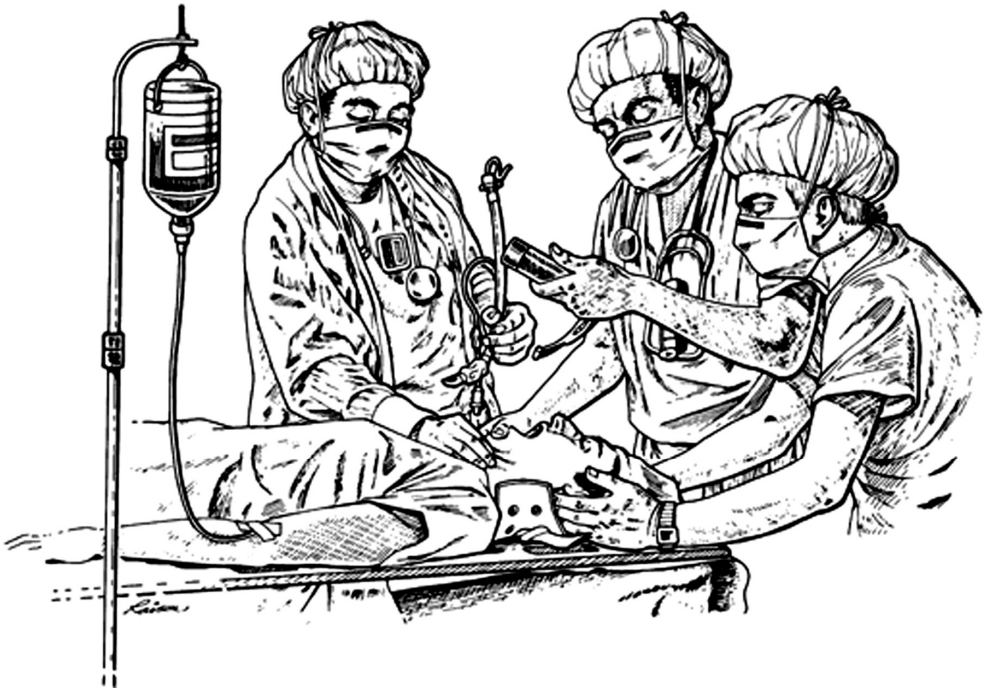
Neuromuscular Disease

Children with cerebral palsy often present for a variety of orthopedic procedures. It is important for the anesthetist to recognize the wide spectrum of neurologic impairment found in this patient population and to individualize the patient's care, based on her/his specific limitations. While the patient may have severely impaired motor function, her/his cognitive function may be normal. Some children have an expressive communication disability, but this does not mean they cannot understand what is being said or done. Airway reflexes, such as the gag reflex, may be minimal, which puts the child at risk for aspiration of secretions and gastric contents and for subsequent pulmonary compromise. Other anesthetic considerations for these patients include contracture-related positioning restrictions, difficult vascular access in contracted extremities, and chronic pain from muscle spasms. Seizure medications, if being taken, should be given the morning of surgery. The intravenous (IV) pharmacologic equivalent of their anti-seizure medication should be given if they cannot take their drugs orally.

Airway

Even if the anesthetist elects to employ regional anesthesia for the surgery, he/she must have a plan for emergently supporting the airway if this becomes necessary. Failure of blocks, local anesthetic toxicity, and other unexpected circumstances may require conversion from regional to general anesthesia and/or tracheal intubation. Trauma, especially when it is due to falls, diving, or motor vehicle accidents, may be associated with instability of the cervical spine. Inline stabilization of the neck is used during manipulation of the airway to reduce or prevent further injury to the spinal cord. Key components to this maneuver include 1) stabilization of the neck with head and neck traction by one assistant, 2) cricoid pressure by a second assistant to aide in visualization of the glottis during laryngoscopy (and possibly prevent aspiration), 3) retention of the back portion of the cervical collar to help in neck stabilization (**Figure 15-1**).

Figure 15-1: Stabilization of the Neck During Tracheal Intubation



The figure shows one assistant applying cephlad in-line traction to stabilize the neck; cricoid pressure is being applied by another assistant. The anesthetist is inserting a tracheal tube. http://web.squ.edu.om/med-Lib/MED_CD/E_CDs/anesthesia/site/content/v04/040133r00.htm

Degenerative changes, such as arthritis, scoliosis, or some congenital diseases (e.g., Klippel Feil syndrome) that involve the cervical spine may make tracheal intubation difficult. Patients with Down syndrome may have instability of cervical spines one and two. Instability of their cervical spine places such patients at potential risk for spinal cord injury if the anesthetist extends the head during mask ventilation or tracheal intubation. If the patient is able to move her/his head in

all directions before surgery without central nervous system symptoms, there should be no difficulty extending the head to intubate the trachea. Congenital anomalies of the upper spine should be detected preoperatively so that their impact on successful direct laryngoscopy can be anticipated. If advanced airway equipment is not available, such as a video-laryngoscope or a bronchoscope, laryngeal mask airways (LMA) often provide an effective airway in these urgent settings.

During positioning for surgery and during the surgical procedure the anesthetist is responsibility for protecting the patient's airway. Positioning of patients with orthopedic problems may involve dramatic suspension of the patient on fracture tables, rotation of her/his body, or turning the patient onto her/his abdomen. The anesthetist is responsible for coordinating the care team's efforts when positioning the patient and for maintaining the secured airway during the move. Fracture reduction may require the surgeon to apply sufficient tension to the affected extremity to move the patient, which may dislodge the LMA or tracheal tube.

For all patients, but especially in those who are to undergo a regional block as a component of the anesthetic, it is vital to establish baseline nerve function. Postoperative nerve injury may be secondary to a pre-existing nerve injury, intraoperative positioning of the patient, stretch or disruption of the nerve by the surgeon, or rarely by the block itself.

Pain Management

Pain is a significant component of many orthopedic surgical problems and must be evaluated preoperatively. The pain may arise from the surgical site itself or from other trauma sites. Baseline assessment of pain should determine: 1) character and location of the pain; 2) intensity of the pain; 3) things that make the pain worse; and 4) things that make it better (e.g., medications, splints, casts, immobilization of the extremity). Care must be taken to minimize pain at these sites when positioning the patient for surgery. The surgical approach for correction of the lesion is often limited by inability to place the patient in the desired position for surgery because of pain and/or other physical restrictions (contractures, etc.). This inability to position the patient appropriately may make it more difficult for the surgeon to perform the surgery.

As discussed in chapters 20 and 21, perioperative analgesic requirements for patients with chronic pain are often much greater than for patients with acute or little pain. Medications that are being given to treat chronic pain preoperatively should be continued or replaced with drugs that provide a similar degree of analgesia. Additional analgesics, either higher doses of the chronic medications or agents in different pharmacologic classes, are also required to cover the surgically induced pain. The use of several drugs to treat perioperative pain can minimize the side effects of each drug. Nerve blocks or infiltration of local anesthetics into the surgical field often provide excellent postoperative pain relief, but it must be remembered that the effects of these blocks and infiltration wear off in a finite amount of time, depending on the drug used. This

means that the patient will have pain, often severe, when the block is no longer effective. The anesthetist must plan for this must as part of planning for the patient's postoperative care. If the patient is going home following surgery, he/she should be told when to start oral analgesics, usually before the block wears off. To avoid toxic blood levels of local anesthetics, it is best to not exceed the stated safe total dose of drug (**See Chapters 20 and 21**). Other useful classes of analgesics include opioids, non-steroidal anti-inflammatory agents (NSAIDs), ketamine, alpha-2 agonists (clonidine), and benzodiazepines. While there is some evidence of delayed bone healing in animals that were given NSAIDs, well-designed studies in humans show this is not the case.[2] It is best for the anesthetist to determine if the surgeon has concerns about whether NSAIDs can cause delayed bone healing before giving them. Some orthopedic injuries and surgeries are complicated by neuropathic pain. Neuroleptic drugs, such as gabapentin, may help minimize this otherwise difficult type of pain. The anesthetist can play a very important role in the patient's recovery from surgery and anesthesia by working with the surgeon, nurses, family and other caregivers to ensuring that the patient is comfortable postoperatively. Patients who have little pain are more likely to participate in early mobilization and physical therapy, which facilitate recovery from surgery and prevent the complications associated with bed rest.

Regional Anesthesia

Many orthopedic procedures can be performed under regional anesthesia, including subarachnoid block (SAB) for treatment of lower extremity fractures in teenagers. Benefits of blocks include avoiding the risks of general anesthesia, improved analgesia, early ambulation, and earlier discharge from hospital.[3] Technical and other considerations for these blocks are discussed elsewhere (*http://www.nysora.com/).* Whether an epidural block or SAB is used depends on the surgical procedure, equipment and medication available, and anesthetist's skills. Physiologic changes, such as those accompanying the rapid sympathetic block following spinal anesthesia, may be profound. Being prepared to treat these changes is important. The quality of motor block and surgeon's satisfaction with operating conditions can be adjusted by the choice of local anesthetic agent (**See Chapter 21**). How long the block lasts can be controlled more easily with epidural anesthesia. Administration of preservative-free opioids or alpha-1 antagonists (e.g., clonidine) into the epidural or subarachnoid space may extend the analgesia well into the postoperative period. If opioids are used, however, the patient's condition must be carefully monitored after surgery to detect any potentially life-threatening side effects of these drugs, including apnea and cardiac arrest. Patients given subarachnoid or epidural opioids should be monitored overnight in an intensive care unit (ICU).

There are several well-established contraindications to regional anesthesia, including patient refusal of this type of anesthesia, infection at the site where the block is to be performed, and/or coagulopathy. The latter is important during performance of the block and when it is time to remove a catheter that was used for continuous infusion of local anesthetic to treat postoperative

pain. A recent publication by the American Society of Regional Anesthesia addresses these concerns. http://www.asra.com/publications-practice-advisories.php Because the incidence of temporary phrenic nerve blockade is high with intrascalene block, this block is contraindicated for patients with limited pulmonary reserve.

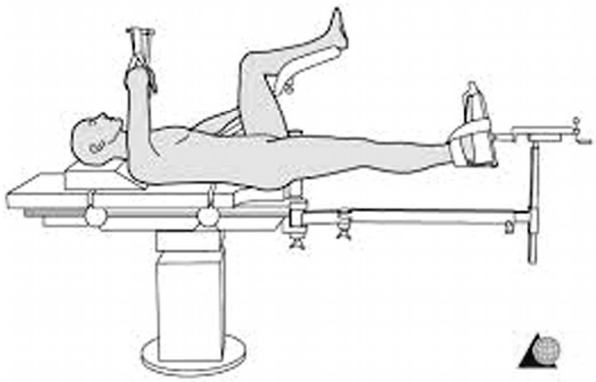
Muscle Relaxation

Neuromuscular blocking agents are used for some orthopedic procedures, often because surgeons request them to make reduction of a fracture easier. Alternatives to giving muscle relaxants, such as increased depth of anesthesia, are good options for patients who are at risk of developing complications from the use of muscle relaxants. For instance, succinylcholine must be avoided in patients who have spinal cord injury or muscular dystrophy because administration of succinylcholine to these patients can cause life-threating hyperkalemia. Patients with congenital disorders, such as spina bifida and cerebral palsy, are not at risk for developing hyperkalemia from administration of muscle relaxants. Residual neuromuscular blockade due to the use of muscle relaxants can cause respiratory compromise, hypoxemia, and hypercarbia postoperatively in any patient. This is particularly true for patients with muscular dystrophies. If the anesthetist has any concern that the patient has residual neuromuscular blockade, it is far safer to continue postoperative support of ventilation than to remove the tracheal tube and have the patient hypoventilate. Regional anesthesia or ketamine administration reduces both postoperative opioid requirements and the respiratory depression caused by these drugs.

Positioning of the Patient for Surgery

Proper patient positioning is very important for the success of orthopedic surgery because it optimizes the surgeon's view of and access to the surgical field, affects the amount of tissue perfusion and blood loss, affects the ability to obtain appropriate X-rays, and affect the ability to provide adequate patient comfort (**Figure 15-2**). It is the responsibility of each team member (surgeon, anesthetist, nurses) to check all pressure points during positioning of the patient and to do so intermittently throughout surgery. Excessive tension or pressure on extremities, eyes, ears, and genitals may lead to nerve damage or to pressure necrosis of tissues. When patients are placed in the prone position, it is especially important to prevent application of pressure to the eyes because this can lead to blindness. Exerting excessive pressure on the chest wall or abdomen (especially the abdomen) can impair ventilation. Conservation of body heat is important during the perioperative period, especially when he/she is uncovered during positioning and preparing the patient for surgery. If possible, the room temperature should be elevated during patient positioning, preparing the skin with antibacterial agents, and when performing anesthesia procedures; this will reduce heat loss and hypothermia. So will covering as much of the patient's body as possible with a plastic sheet.

Figure 15-2: Patient Positioning on a Fracture Table



This figure shows some of the positions possible on a fracture table. The arm can be suspended overhead to prevent brachial plexus injury. The legs can be positioned for surgery. Positioning on fracture table places patient at risk for injury at surgical and nonsurgical sites. www2.aofoundation.org

Radiation Exposure

Orthopedic surgeons frequently use fluoroscopy or X-rays during surgery to guide their work. While doing so is very important, it can expose both the patient and healthcare workers to radiation. Proper shielding of healthcare providers and patients with leaded devices (aprons) does this well. Radiation exposure dramatically decreases the farther a person is from the source of the radiation. If the anesthetist is fifteen feet from the X-ray machine, her/his exposure to radiation is minimal.

Blood loss

For some procedures, such as tendon transfers, closed fracture reduction, and hip stabilization, there is minimal blood loss. Other surgeries, such a spine surgery, trauma, open extremity surgeries, have a greater potential for substantial blood loss. The anesthetist can help reduce blood loss and the need for blood product administration. Recommendations have been developed for both elective and emergent surgery for patients on anticoagulants preoperatively.[4,5] http://www.uptodate.com/contents/management-of-anticoagulation-before-and-after-elective-surgery

It is important to obtain a preoperative hematocrit in patients who are expected to lose a lot of blood during surgery and in those with complex diseases, trauma, or who are at the extremes of age. Although anemia may is associated with perioperative mortality, blood transfusion also carries a risk (**See Chapter 4**).[6,7] If there is time, preoperative treatment of the anemia with iron or erythropoietin (when available) may be warranted. It is important to pay attention to

indicators of the intravascular volume status in hospitalized trauma patients and patients who are critically ill. Femur fractures or injuries that involve damage to vascular structures (e.g., disruption of blood vessels) may be associated with large unrecognized blood loss. Spinal procedures performed in the prone position are associated with lower venous pressures in the surgical site, which decreases blood loss. Hemodilution, controlled hypotension, and administration of anti-fibrinolytic agents may minimize blood loss during spinal procedures.

Tourniquets

Use of a tourniquet during extremity surgery reduces blood loss and improves the surgeon's ability to see the surgical site. The recommended inflation pressure for an upper extremity tourniquet is 50mmHg above systolic pressure; the recommended inflation pressure for the lower extremity is 100mmHg above the patient's systolic pressure. A tourniquet that is inflated for more than 120 minutes frequently causes significant discomfort in awake patients. Release of the tourniquet for several minutes reduces this discomfort. Appropriate selection of analgesics or sedatives for treatment of tourniquet pain must take into consideration its transient nature. Giving large doses of narcotics for this purpose is inappropriate. Prolonged periods of tourniquet inflation are not recommended because this may lead to permanent ischemic injury to nerves, muscle, and skin. After two hours of cuff inflation, transient metabolic changes including lactic acidosis, myoglobin release, and hyperkalemia may occur as a result of tissue ischemia. If the tourniquet must be used for longer, it should be deflated for 10-15 minutes and then re-inflated. Regional blocks, including nerve blockade of the region beneath the tourniquet, may reduce tourniquet-induced pain but not the nerve injury.

Infection Prevention

The anesthetist can take crucial steps during anesthesia to decrease the risk of infection. This is particularly important for patients with orthopedic injuries because infections can be devastating in these patients and may require weeks of antibiotic therapy to treat the infection. Open fractures should get priority on the surgical schedule to reduce/prevent infections from occurring. Recognition and management of preoperative infections, such as urinary tract infections and ear infections, reduce surgical site infections.[8,9] Administration of antibiotics within one hour before the surgical incision also reduces the number of perioperative infections. The antibiotics are re-dosed every 4-6 hours during long cases. The basic principles of hand washing, wound care, and sterile technique are critical for preventing infections.

Trauma

Patients with multiple trauma and orthopedic injuries present several additional challenges to the anesthetist. These patients frequently have full-stomachs, which put them at risk for vomiting and aspiration of their gastric contents. Trauma and the narcotics used to treat pain delay gastric

emptying. Spinal cord injury should always be considered as possibly present in trauma patients. As stated above, further spinal cord injury must be prevented during airway management and positioning of the patient for surgery. Surgical or medical management of traumatic injury to the brain, thorax, or abdomen may take precedent over repair of the patient's orthopedic injuries. Open repair of pelvic fractures is often accompanied by large blood loss. Consequently, blood products should be available before the start of surgery (**See Chapter 4**). Packing the pelvis with sponges and external fixation of the bone to prevent further injury may be the safer initial management of this injury. Discussion between the surgeon, anesthetist, and other caregivers is essential to decide on the best management of the patient with multiple-trauma.

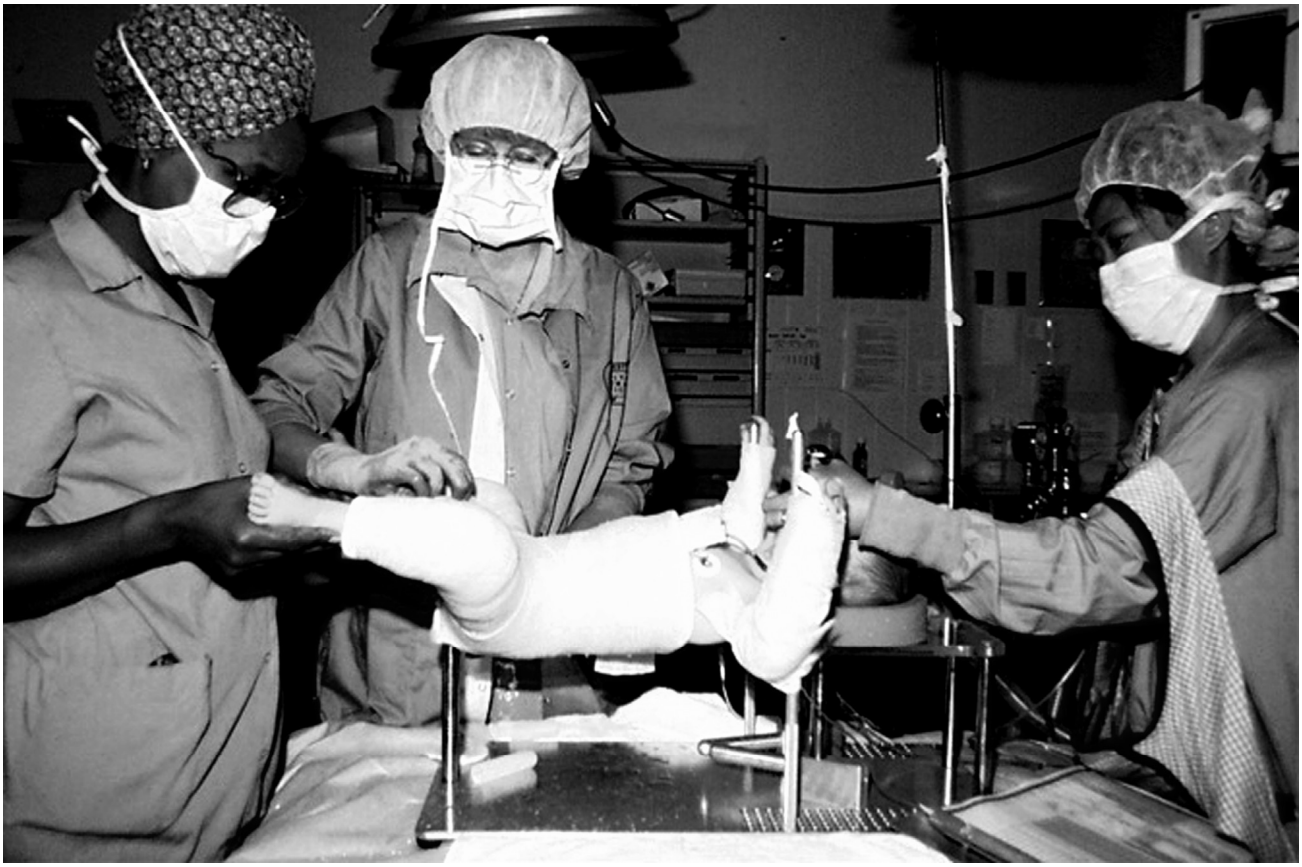
Venous Thrombosis

Lower extremity injury, surgery, and bed rest place patients at risk for venous thrombosis and pulmonary embolus. The morbidity and mortality from pulmonary embolus is very high. Excellent pain control and early ambulation lessen the patient's risk of developing venous thrombosis. However, the benefits of giving anticoagulants to prevent or treat venous thrombosis must be weighed against the risks of causing increased surgical bleeding.

Casts

Splinting or casting extremities is a frequent component of orthopedic surgery. For most children, these procedures are performed while the patient is anesthetized. Young children who are awake may not tolerate the noise cause by a cast saw; some patients are concerned that the saw will injure them. The eyes of the patient and anesthetist and all sterile surgical equipment should be protected from particulates and dust arising from cutting the cast. Some children are not happy when they awaken and find that they are cannot move because of a cast. They should be warned before surgery that they will be in a cast and unable to move normally. If the child cries a lot and is in distress after surgery, it is important to ensure that this is not the result of residual surgical pain, the pain from a tight cast, or the exothermic reaction caused by some cast materials. Young patients, especially infants, are frequently placed in lower body or spica casts as part of the treatment of congenitally dislocated hips. It may be necessary to repeatedly turn the patient while the cast is being applied, frequently while he/she is being balanced on a small cast table (**Figure 15-3**). The anesthetist is often the only one watching the patient during this time. Therefore, it is up to her/him to: 1) secure the airway and maintain adequate ventilation; 2) prevent stretch of the upper extremity and brachial plexus; 3) ensure that the cast does not restrict excursion of the chest and abdomen and interfere with breathing; 4) keep the patient warm while he/she is uncovered during application of the cast.

Figure 15-3: Application of a Spica Cast



The figure shows a patient on a Spica cast table. Notice that he is elevated above the surgical table and somewhat precariously suspended on the cast table. The anesthetist is protecting the airway to prevent accidental tracheal extubation. Photograph Courtesy of Mohamed Diab, MD.

Compartment Syndrome

Perfusion of an extremity may be impaired if bleeding or swelling occurs and increases the pressure within confined facial-muscle areas. Loss of distal pulses and, at times a decrease in oxygen saturation (SaO_2), may herald the onset of a compartment syndrome. The ischemia that results from the increased pressure in the compartment is usually very painful. One clew that this is happening is the need for excessive amounts of analgesics, requirements that far exceed those usually anticipated for the type of surgery that was done. It has been suggested that regional anesthesia or patient controlled analgesia can delay the recognition of compartment syndrome, but this usually is not the case. If there is the potential for developing a compartment syndrome following surgery, lower concentrations of local anesthetics can be used for the block. Prompt recognition of a compartment syndrome, relief of the increased pressure, and restoration of perfusion are mandatory if massive metabolic changes, muscle breakdown, and permanent nerve injury are to be prevented.

Surgical Sites

Spine

Surgical procedures to correct curvature of the spine (scoliosis) are the most complex of orthopedic operations. The curvature is often the result of a neuromuscular disorder or other degenerative disease. The cause of scoliosis in an otherwise healthy patient is often unknown (idiopathic). Readers are referred to other publications for in depth discussions of the anesthetic management of patients for posterior spinal fusion.[10]

http://bja.oxfordjournals.org/content/99/1/86.full

Specific concerns for spinals surgery include the patient's cardiopulmonary status, blood conservation, prevention of spinal cord injury, and pain management. The extent of pulmonary compromise correlates with the degree of spinal curvature and rib cage distortion. Patients with severe curvature, especially those with neuromuscular disorders, may have minimal pulmonary reserve and need prolonged ventilator support post-repair. Blood loss tends to be greater in patients with neuromuscular disorders. Blood conservation techniques, previously mentioned, are particularly important during spinal surgery for patients with neuromuscular disorders. These include proper positioning of the patient and control of her/his arterial blood pressure. Spinal cord function can be compromised if the surgeon put tension on the spinal cord while trying to reduce the curvature and when he/she is placing the hardware to keep the spine in the desired position. Devastating paralysis can result from excessive tension on the spinal cord. Maintenance of systemic blood pressure within each patient's normal range is important at this point of the operation. Keeping the patient warm is also necessary, because hypothermia can reduce blood flow to the spinal cord and add to any injury from other causes. Postoperative vision loss is a rare but devastating complication of surgery performed with the patient in the prone position. Risk of this occurring is increased by prolonged surgical times, large volume fluid administration, and having the head in a dependent position relative to the heart. Pain management should employ the multimodal approach (**See Chapter 20**), including infiltration of local anesthetics into the surgical site. The patient may have had chronic pain as part of her/his presenting surgical scenario and required a significant amount of drugs to treat the pain. This may make providing adequate analgesia for these patients difficult following surgery.

Shoulder Surgery

Anesthetic concerns for shoulder surgery include: 1) the patient's baseline neurologic status; 2) assuring airway and anesthesia circuit security during surgery; and 3) monitoring the patient's hemodynamic status with the patient in the sitting position. Intrascalene blocks (**See Chapter 21**) provide effective intraoperative and postoperative pain relief. However, the surgery is done in close proximity to the brachial plexus and sometimes patients awaken with a brachial plexus

injury. When this occurs, it is necessary to differentiate whether the injury is due to an uncommon transient or long-term injury to the plexus that was caused by the nerve block, or whether it is due to the surgery itself. The long-term outcomes may be very different. During this surgery, care must be taken to assure security of the airway and anesthesia circuit. The operating table is often rotated away from the anesthetist, making it difficult for her/him to see the circuit and tracheal tube connections. When the surgeon places traction on the shoulder, the connections between the anesthetic circuit and the tracheal tube may inadvertently separate. When this occurs, hypoxemia may occur in apneic patients. Because the surgical site is above the level of the heart, there is an increased risk that a venous air embolus (VAE) may occur. Although echocardiography and precordial Doppler monitoring are the most sensitive means of detecting VAE, changes detected with more commonly used monitors may give evidence of this serious clinical situation. These include sudden decrease in $ETCO_2$, decreased systemic arterial blood pressure, new machinery type cardiac murmur, and rhythm disturbances or ischemic changes.

Upper Extremity Surgery

Most major surgical procedures on the upper extremity are performed under general anesthesia, but regional anesthesia, supplemented by sedation or general anesthesia, is also used. The block selected for upper extremity surgery depends on the surgical site, the need for a tourniquet during surgery **See Chapter 21**), the patient's disease, and the anesthetist's experience. (**Table 15-1**) For brief wrist or hand surgery a Bier block with infiltration of local anesthetic is an option.

http://www.nysora.com/techniques/3071-bier-block.html.

Table 15-1: Blocks for Upper Extremity Surgery

Block Type	Area Covered by Block	Potential Complication
Intrascalene	Shoulder	Injection of drug into the phrenic nerve, epidural space, vertebral artery, or into the subarachnoid space – spinal anesthetic
Supraclavicular	Upper and lower arm	Pneumothorax, phrenic nerve block
Infraclavicular	Elbow, hand	Intravascular injection
Axillary	Distal arm to elbow	Hematoma, infection
Bier Block	Hand	Cardiovascular compromise if tourniquet is released too rapidly

Hip Surgery

Orthopedic surgery for treatment of hip fractures is one of the most common surgical procedures done in many countries. The patients are frequently elderly and have multiple medical problems that must be considered and understood by the anesthetist before surgery. If not contraindicated by the patient's coagulation status, regional anesthesia is often the anesthetic of choice because it eliminates the need for general anesthesia and the risk of general anesthesia-related cognitive impairment following surgery. Subarachnoid, epidural, or lumbar plexus blocks are suitable for hip surgery. These blocks can also be used for repair of congenital or developmental abnormalities of the hip, depending on the patient's age. Some surgeons want controlled hypotension during hip surgery to minimize blood loss. But it is usually best to maintain the blood pressure within 20% of the patient's baseline pressure to minimize ischemic injury to the heart, brain and other organs. *Fat emboli* following trauma to long bones or from the surgery its self may be deposited in the pulmonary capillary bed and cause an inflammatory reaction. Pulmonary embolism is usually diagnosed when the patient has difficulty breathing and problems with oxygenating (low SaO_2), changes in her/his mental status when awake, and a petechial skin rash. These symptoms are often become apparent 12-to-72 hours after the injury (when the patient is going to surgery) or after surgery. The anesthetist should suspect this diagnosis when the patient develops unexpected respiratory problems in the perioperative setting. Respiratory support and bone stabilization are necessary to reduce/prevent fat embolization. *Deep vein thrombosis* or *pulmonary embolism* are significant causes of morbidity and mortality for patients undergoing major hip and lower extremity surgery. Risk factors include advanced age, cancer, previous venous thromboembolism (VTE), and prolonged immobility. Prophylactic anticoagulant therapy is recommended for the patient at risk for VTE. Effective postoperative analgesia often allows these patients to be mobilized early, which decreases the occurrence of thrombosis and embolism.

Lower Extremity

Previously mentioned anesthetic considerations for orthopedic surgeries apply to procedures performed on the lower extremities as well. Careful patient assessment, proper patient positioning, blood conservation, employment of regional anesthetic techniques, pain management, and VTE prevention are included. Central neuroaxial, femoral, sciatic, lumbar plexus, or more peripheral blocks can be beneficial. Regional anesthesia is thought to reduce the development of phantom limb pain when amputating all or part of an extremity. Pain due to muscle spasm after lower extremity surgery can be substantial, and benzodiazepines can be used to reduce this discomfort.

Summary

Anesthetists are presented with numerous challenges in patients undergoing orthopedic surgery. This patient population and the extent of the surgery vary significantly. A coordinated, dedicated

team effort by the anesthetist, surgeon, and nurses is required. The surgical procedure is just one step to recovery from the initial problem. Many other problems that occur are under the control of the anesthetist, and proper planning for the anesthetic and its delivery can affect recovery from both anesthesia and surgery. Attention must be paid to underlying disorders, infection control, pain management, and respiratory care. Nerve blocks for upper/lower extremity provide excellent postoperative pain relief and shorten hospital stays.

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Chapter 16
ANESTHESIA FOR PLASTIC SURGERY

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Anesthesia for cleft lip and palate surgery is common and very rewarding because of the life changing effects these operations have on both patients and their families. The cosmetic benefit of repairing a cleft lip leads to greater acceptance by the local culture, and the improved speech that can occur with a cleft palate repair makes patients and families uniformly grateful for the care provided. This chapter discusses the etiology of cleft lip and palate, the different types of deformities seen, associated co-morbidities, and perioperative considerations for providing safe anesthesia to this group of patients.

Cleft lip and palate dysmorphology is really a collection of deformities that can occur in isolation or together with one another. Moreover, they can occur with other congenital defects that may have a significant affect upon the anesthetic care the patient requires. The exact cause of cleft lip and palate is unknown, though it is probably multi-factorial. There is a genetic predilection to clefting within ethnic groups (**Table 16-1**).

Table 16-1: Incidence of Clefts by Race or Country

Race/Country of Origin	Incidence/1000 Births
Native Americans	3.74
Japanese	0.82 - 3.36
Chinese	1.45 - 4.04
Caucasians	1.43 - 1.86
Latin Americans	1.04
Africans	0.18 - 1.67

Certain genetic syndromes are associated with a cleft lip and/or a cleft palate, but the genetics of clefts that occur when they are not part of a syndrome are poorly understood. While some genes are known to be associated with clefts, further discussion of this subject is beyond the scope of this chapter. Whatever the genetic associations, clefts tend to occur in families.

There also is some association between clefts and a variety of other factors, including maternal hypoxemia during pregnancy, maternal alcohol abuse, use of antihypertensive drugs during

pregnancy, maternal tobacco smoking, and maternal nutritional deficiencies, such as folic acid deficiency. Whatever the cause, a defect develops during the 12th week of pregnancy that results in a partial or complete unilateral or bilateral cleft lip plus or minus a cleft palate.

Why is it important to repair a cleft lip or cleft palate? Some practitioners consider a cleft lip a cosmetic defect. While it is true that a cleft lip alone, i.e., without any other defects, has no functional implications, it is also true that children born with a cleft lip are often shunned by society and frequently teased by other children, making it difficult for them to attend school and obtain an education. It may be impossible for them to gain employment because of the social stigma associated with a cleft lip. Repairing a cleft lip during infancy makes it much more likely that the child will go to school, gain an education, and become employed.

Cleft palate repair, on the other hand, does have functional implications because a cleft palate is an open passage between the nasal and oral cavities. This opening allows food or liquid to come out their nose when eating or drinking. This is often very distressing to both the child and the family. However, the most important and common effect of a cleft palate is its detrimental effect on speech development. During speaking, it is necessary to place the tongue against the palate to allow a person to make many language sounds. Absence of an intact palate makes it impossible to develop those sounds correctly and results in nasal sounding speech. This connection between the oral and nasal cavities allows *velopharyngeal inadequacy*, that is, air freely leaks through the nose during speaking. In addition to the nasal sounding voice, a cleft palate can cause numerous other errors in speech development.

If the palate is repaired before two years of age, the child has a much better chance of developing normal speech. Even though an isolated cleft palate cannot be seen, its repair and the subsequent development of more normal speech have enormous benefit to the patient, their family, and to society as a whole. The patient who has her/his cleft palate repaired is much more likely to become a contributing member to society. In fact, repair of either of these defects makes it more likely that a child will become a contributing member of society as an adult, thereby improving a statistic called the Disability Adjusted Life Years (DALY) of the country in which the child lives.

Unfortunately, just repairing a cleft lip or palate is not all that is required; it is only the beginning. Though it will not be discussed in detail in this chapter, it is essential that these patients receive additional comprehensive services when possible, including speech therapy and dental care, to achieve maximal benefit from the operation. The remainder of this chapter discusses the types of clefts and the perioperative considerations for taking care of patients with a cleft lip, cleft palate, or both.

Cleft Types

There are two primary types of clefts: cleft lip and cleft palate. These deformities can be categorized in several ways, including whether a cleft lip or cleft palate occurs alone or whether they occur together. Clefts can be complete (through all tissues) or incomplete (not through the entire structure). Incomplete lesions of the lip may consist of a simple notch in the vermillion border of the upper lip; a partial cleft palate may consist of only a soft palate notch. In contrast, a complete defect is one where the upper lip extends cleft clear through the vermillion border of the upper lip and may extend into the nose. A cleft lip occurs because the maxillary process and medial nasal process fail to fuse during development. The palate may be cleft from the soft palate all the way forward through the hard palate. The resulting defect may consist of anything from a cleft soft palate, to a hole in the hard palate, to a completely split palate.

Both complete and incomplete clefts can be present on only one side (unilateral), or on both sides of the oral cavity (bilateral). A unilateral cleft lip is far more common than a bilateral cleft lip. The ratio of unilateral/bilateral lesions varies, depending on the ethnic group. Finally, clefts can involve the nose or can be complex in other ways. The following outline categorizes the different types of clefts. Remember that the geographical location in the world and the particular ethnic group affects the incidence of these different cleft types (**Table 17-2**).

TABLE 17.2: Types of Clefts

Cleft Lip

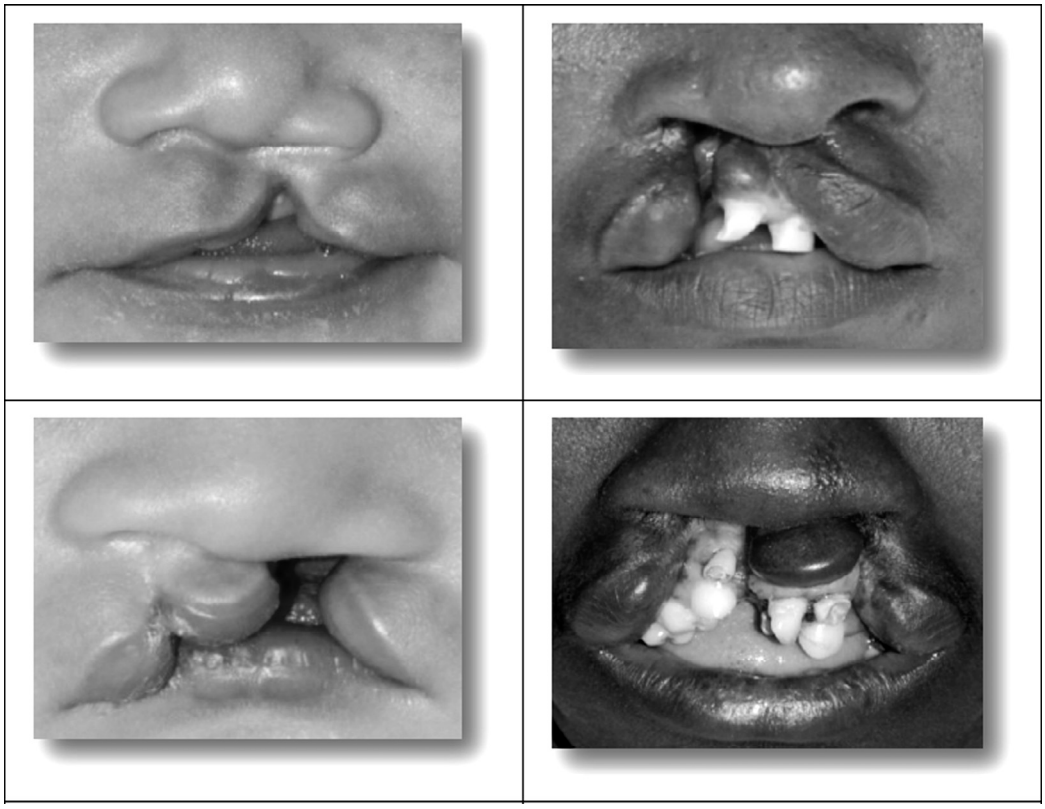
- Unilateral
  - Incomplete
  - Complete
- Bilateral complete

Cleft palate (plus or minus a cleft lip)

- Unilateral
  - Incomplete
  - Complete
- Bilateral
  - Incomplete
  - Complete

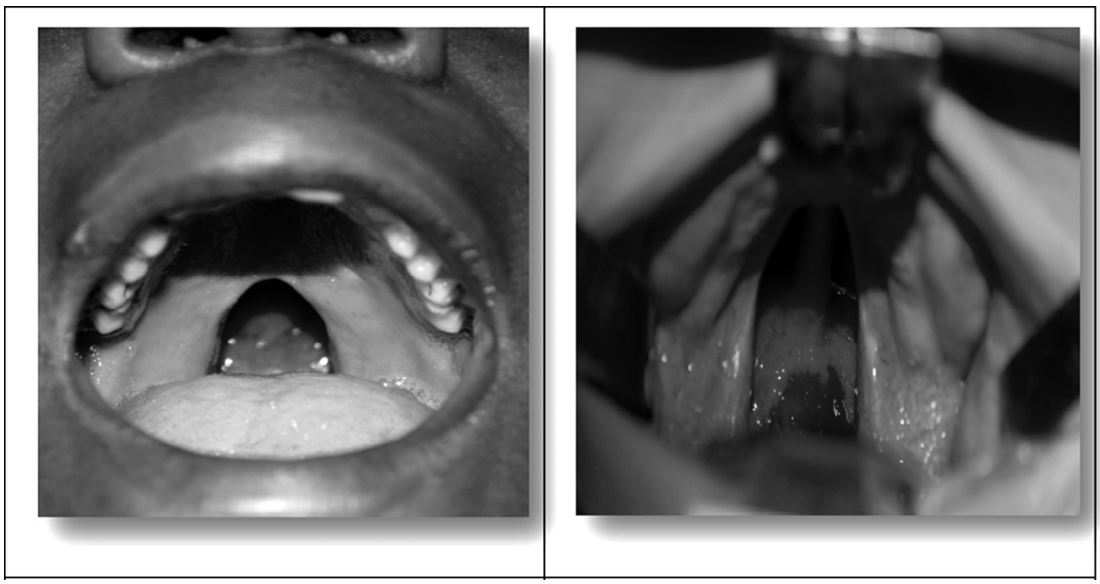
Cleft nose or other complex cleft pattern

Figure 17-1: Types of Cleft Lips



Upper left – Left lateral cleft lip; Upper right – Complete cleft lip; bottom left – Asymmetrical partial cleft lip; Bottom right – Bilateral cleft lip

Figure 17.2: Types of Cleft Palates



Veau 1 – Cleft palate – Soft palate

Veau 2 – Cleft palate - Complete

Whether these deformities occur separately or together, understanding differences between a cleft lip and a cleft palate procedure is essential to providing safe and effective anesthesia for these patients. Anesthesia for cleft palate surgery has significantly more perioperative considerations, intraoperative issues, and complications than anesthesia for a cleft lip repair. These differences will be discussed in detail in the following sections.

Preoperative Considerations For Patients with Clefts

The ideal time to repair a cleft lip is three months of age; the ideal time to repair a cleft palate is about one year of age. Unfortunately, when clefts get repaired often depends on the availability of a surgeon who is qualified to perform the necessary procedure(s), surgeon's and a anesthetist's who are comfortable with caring for infants, the availability of funds to pay for the surgery, having someone with whom the family can stay in the town where the operation will be performed, a satisfactory primary care giver (usually a parent), the adequate qualified nursing personnel on the ward, the availability of an "on call" physician, and the availability of a surgeon and anesthesia provider to handle complications after hours. Comprehensive care planning takes into consideration the availability of dental care and speech, and occupational therapy if required. These factors are especially important considerations for cleft palate repair because, if they are not available, surgery to correct these clefts is often delayed beyond the ideal age. Not performing palate surgery at the correct time makes it more difficult for the patient to develop normal speech, even with speech therapy when it is available.

To safely provide anesthesia for cleft lip and palate repair, the anesthetist must be comfortable providing anesthesia for infants and children. Because the surgeon and anesthetist must "*share the airway*", especially for a cleft palate repair, the anesthetist must feel particularly comfortable managing the airway and intubating the tracheas of infants and young children during an emergency and under difficult circumstances.

A small percentage of patients who have clefts have a syndrome, and the cleft is part of that syndrome. Depending on the syndrome and its manifestations (phenotype), there may be other significant preoperative considerations that make providing anesthesia challenging. For example, patients with *Treacher-Collins* or *Pierre Robin syndrome* often have micrognathia (small jaw) in addition to their cleft, which makes tracheal intubation difficult. Closing the cleft palate of patients who also have micronathia frequently causes significant postoperative airway obstruction because the patient's tongue (which is normal size) is now contained in a much smaller space after the cleft palate is closed. Patients with *Stickler syndrome* can have other craniofacial abnormalities besides a cleft. Other syndromes that have clefts as part of the syndrome usually have fewer perioperative considerations. Patients who have a cleft plus additional dysmorphic features must undergo very careful preoperative evaluation to determine if there are other abnormalities that can lead to anesthesia complications. It may not be clear

from an external physical examination whether there are other manifestations of the syndrome that can complicate the anesthesia and surgery or that can cause postoperative airway obstruction. Undiagnosed congenital heart disease can be particularly problematic. www.ncbi.nlm.nic.gov/omim is a website where free information about syndromes can be obtained.

In addition to the above considerations, patients with clefts may have difficulty eating and have malnutrition and/or anemia. Malnutrition may reduce the plasma protein concentrations, which can reduce the amount of protein available to bind drugs in plasma (*plasma protein binding*). This increases the amount of free drug available, making more free drug available to tissues, which increases the drug's effects and the probably occurrence of complications. In addition, malnourished patients may have poor postoperative wound healing and wound break down. Anemia decreases the hemoglobin reserve and oxygen carrying capacity of the blood. Depending on the amount of intraoperative blood loss, patients undergoing cleft palate repair may have insufficient oxygen available to supply the needs of vital tissues and assure wound healing. Therefore, blood must be available for transfusion if necessary.

Particular attention must be paid to a history of airway problems and to examination of the airway. How difficult it will be to intubate the trachea will depend on the malformation and the age at which the patient is undergoing repair. Older patients often have rotated and malpositioned teeth that interfere with placing the laryngoscope blade in the mouth. A bilateral cleft lip, with a prominent prolabium that sticks out, can get in the way of the laryngoscope, making visualization of the glottis difficult. The laryngoscope blade can also ride up into a large complete cleft palate, also making it hard to visualize the glottis. Repeated attempts at visualizing the glottis increase the probability of causing airway trauma. Cleft lip surgery that is only done outside of the oral cavity has fewer airway considerations for both tracheal intubation (bilateral lip excepted) and tracheal extubation. Good airway management skills are required for patients undergoing cleft palate surgery because it is more likely they will have airway obstruction and laryngospasm after tracheal extubation, due to postoperative bleeding, increased airway secretions, and airway edema.

The amount of blood lost and the possible need for red blood cell or whole blood transfusion are significantly greater during and after cleft palate than for cleft lip operations. Because significant blood loss rarely occurs during cleft lip surgery, it is acceptable to perform this surgery in slightly anemic patients (Hemoglobin 8-9g/dl) if he/she is otherwise healthy. Surgeons frequently infiltrate the tissues with local anesthetic containing epinephrine, which further decreases bleeding. In contrast to cleft lip repair, cleft palate repair can be associated with significant bleeding, especially if the patient is more than 10 years of age at the time of surgery or if the malformation the palate is large and the surgery is difficult. As a general rule, patients living below 5,000 feet altitude require preoperative hemoglobin concentrations greater than 10g/dl in

order to proceed with the operation. When the preoperative hemoglobin concentration is above this level, it is uncommon for patients with a cleft palate to require a blood transfusion. However, if significant postoperative bleeding occurs, and the patient must return to the operating room for control of bleeding, the need for transfusion increases. Though the need for a blood transfusion is unlikely, any hospital that performs cleft palate operations must have the ability to quickly provide blood for transfusion.

Preoperative planning for surgery must ensure that age appropriate anesthesia and surgery equipment are available. This includes devices for airway management, such as correct sized laryngoscope blades, oral airways, and masks. Correct size oral RAE tracheal tubes should be used whenever possible to minimize the possibility of tracheal tube kinking and airway obstruction during surgery. These tubes also move the tracheal tube and connectors away from the surgeon's field. When selecting the correct size RAE tube, both the diameter and location of the bend of the tube must be considered. RAE tubes are manufactured to fit children of average size for a given age, and the bend in the tube is at a specific point in the length of the tube. The distance from the bend to the distal end of the tube might be too short or too long if a patient is larger or smaller than average. This "mismatch" may make it hard to ensure that the tip of the tracheal tube is positioned correctly in the mid-trachea. If RAE tubes are unavailable, a standard tracheal tube may be used and secured in the midline of the lower jaw. However, use of these tubes significantly increases the risk of the tracheal tube kinking as the tube warms to body temperature. Appropriate size suction catheters are required for suctioning through the tracheal tube and for suctioning the oral cavity before and after tracheal extubation. Using a Yankauer suction tip can increase bleeding if the suction tip injures fragile tissues that have just undergone surgery. Other age appropriate supplies required are buretrols that can be placed in line with the intravenous tubing to reduce the possibility of accidently administering too much fluid to infants. Number 22 and 24 gauge intravenous catheters and 1ml and 3ml syringes are also very useful.

Preoperative patient preparation includes a discussion with the patient (when possible) and the family about expectations surrounding the surgery, risks of anesthesia, and the possibility of needing a blood transfusion if the patient must return to surgery for bleeding after repair of a cleft palate. Informed consent should be obtained from the patient (when possible) and/or family for both the anesthesia and surgery. Whether patients should be admitted to the hospital the day of or the night before surgery depends on local custom. In either case, it is important to ensure that he/she has followed the NPO guidelines, especially for solid food (**See Chapter 1**). The patient should drink as much clear liquids (liquid through which one can read a newspaper – water, juice without pulp) as they desire up to two hours before the induction of anesthesia. Larger volumes of fluid are cleared from the stomach more rapidly than small volumes of fluid. Avoiding dehydration, especially in a warm climate, will facilitate IV placement and decrease the likelihood of developing hypotension during the induction of anesthesia. If halothane will be used as the primary anesthetic, it is especially important that the patient be well hydrated with clear

fluids within the proper time frame to prevent hypotension on induction of anesthesia. For an older child who is particularly anxious, a preoperative anxiolytic drug, such as midazolam, 0.5mg/kg diluted in one-to-two ounces (30-60ml) of soda, may be administered. The maximum dose of midazolam is 20mg.

Intraoperative Considerations

Following preoperative evaluation and obtaining consent for anesthesia and surgery, the patient can be prepared for the operating room. It may be helpful, depending on local custom and logistics, to have the parent present during the induction of anesthesia. A parent, if properly prepared for what to expect, can have a calming influence on the child as he/she goes off to sleep. It is important to explain to the parent that the child may struggle, the eyes may roll upwards, her/his breathing may be irregular, and the child will have involuntary movements but that this is common and expected and that there is no pain associated with an inhalation induction of anesthesia.

Unless it is anticipated that the airway will be difficult or that there is a specific reason that an IV will be needed prior to the induction of anesthesia, an inhalation induction of anesthesia, with sevoflurane if available, is usually preferred for pediatric patients. Halothane *should* only be used for an inhalation induction of anesthesia if the practitioner is very familiar with its use. However, one must be particularly careful about using halothane in small infants if they have been NPO and are dehydrated because halothane has significant cardiac depressive effects and can cause significant bradycardia and decreased cardiac output. The toxic to therapeutic index of halothane is quite narrow, necessitating extreme caution during its use.

After anesthesia is induced, monitors can be applied and an IV can be placed (**See Chapter 2**). A precordial stethoscope is applied to the chest for monitoring breath sounds and for listening to heart tones. This device is an especially valuable for monitoring ventilation if end-tidal carbon dioxide monitoring is unavailable. If the trachea is to be intubated without giving muscle relaxant, it is especially important to make certain that the patient is adequately anesthetized. If the patient has been anesthetized with sevoflurane, look for conjugate pupils (looking straight forward) that have returned to normal size (no longer dilated), apnea, and normal blood pressure to indicate that the patient is sufficiently anesthetized for tracheal intubation to be accomplished without causing laryngospasm or hypoxemia. When using halothane, a decrease in heart rate, along with the above indicators, are indicative of adequate anesthesia.

Visualization of the glottis and placement of a tracheal tube may take longer in patients with altered anatomy of their mouths, which can increase the likelihood of laryngospasm in patients who have not been given muscle relaxants. When difficulty is encountered or the “depth” of anesthesia is uncertain, the anesthetist must decide whether or not to administer a muscle relaxant to aid with tracheal intubation. While giving a muscle relaxant (other than

succinylcholine) precludes the possibility of the patient rapidly returning to spontaneous ventilation, administration of a muscle relaxant usually makes tracheal intubation easier and safer. The use of succinylcholine routinely for tracheal intubation in infants and small children is controversial and carries some risk of causing arrhythmias, especially bradycardia. This can be a significant problem if the patient is anesthetized with halothane. Some practitioners pre-treat all children with atropine before administering succinylcholine. There is also the theoretical concern that using succinylcholine in patients with an undiagnosed myopathy may cause hyperkalemia and death.

There are several ways to tape a RAE tube in place; surgeons usually request that the tube be secured in the midline and be taped to the lower lip and chin. Remember that the patient will be fully covered with surgical drapes, that the anesthesia provider will have limited access to the tracheal tube and airway, and that the table and patient's head will be turned 90 degrees; thus, it is important to make sure that all tracheal tube connections are tight and that there is no kinking of the tube when the surgeon expands the Dingman oral retractor. If there is kinking, the pressure needed to ventilate the lungs will increase and the breath sounds and end-tidal CO_2 will decrease. End tidal carbon dioxide monitoring, if available, is particularly valuable for ensuring that ventilation is occurring when there is limited access to the head, airway, and tracheal tube. If the retractor occludes the tracheal tube, the end-tidal CO_2 will go to zero. Use of a precordial stethoscope is very helpful as described above, because the breath sounds will diminish or be absent.

Blood and airway secretions are common during cleft palate surgery. Using a cuffed tracheal tube (when available) decreases the possibility that the patient will swallow blood and have it get into the airway. Often there is no need to place air in the cuff. If the cuff is to be inflated, only sufficient air should be injected into the cuff to eliminate the leak. Using a cuffed tube does not always eliminate aspiration of blood during surgery.

The surgeon will place a throat pack in to the oropharynx to further prevent blood and secretions from ending up in the airway or the stomach. It is important to make certain that when a throat pack is inserted, the surgeon leaves part of the pack outside the mouth so everyone in the operating room can see it and know that the pack is in place. A sign should be placed on the OR table or wall in plain view of everyone stating that the throat pack is in place. Everyone in the OR *must* be responsible for assuring that the throat pack is out at the end of surgery. There are multiple case reports of airway obstruction after removal of the tracheal tube because the throat pack was not removed at the end of the case. When this occurs, **IT IS OFTEN LIFE THREATENING.** If the end of the throat pack is not protruding from the mouth, it may be forgotten until the trachea is extubated, and the patient's lungs cannot be ventilated and he/she is hypoxemic or dies.

During the maintenance phase of anesthesia, it is usually best for patients to breath spontaneously when possible. If a muscle relaxant is used for tracheal intubation, it is best that the drug not be re-dosed so spontaneous ventilation can resume quickly. Usually a combination of inhaled anesthetic and an analgesic are all that is required for maintenance of anesthesia. Analgesic considerations for cleft lip and palate repair depend on what is available locally and whether or not the surgeon or anesthesia provider performed a nerve block. (See the next section for details.)

Towards the end of surgery, the surgeon often places a heavy suture (2-O silk) in the tongue and tapes the ends of the suture to the cheeks. This makes it possible for the anesthetist or nurses to relieve airway obstruction by pulling the tongue forward. Near the end of surgery, it is also necessary to decide which method of tracheal extubation will be used: awake or anesthetized. No matter which method is chosen, it is necessary to have both the equipment and drugs available for immediate tracheal intubation or tracheostomy should one or the other be required. This is especially important for patients undergoing cleft palate repair, since their risk of airway obstruction is greater following tracheal extubation.

The tracheal tubes of most patients undergoing cleft lip surgery are usually removed when they are awake. Those who have undergone cleft palate surgery are extubated when the patient is awake or when they are still anesthetized. There are pluses and minuses for each form of extubation. Extubating the trachea when the patient is awake significantly decreases the likelihood that he/she will develop laryngospasm after tracheal extubation. This is because the patient has already gone through stage two of emergence. Having the patient awake before leaving the operating room also improves airway muscle tone, ensures a positive gag reflex, and reduces the likelihood that the tongue will cause airway obstruction. For these reasons, extubating the trachea awake is preferred when anesthetists have little experience providing anesthesia for repair of cleft lips and for cleft palates.

Tracheal extubation may occur while the patient is still anesthetized if the surgeon is worried that the patient will cough during the wakeup and start bleeding from raw surfaces of the palate. By extubating the trachea of patients while he/she is still anesthetized, the possibility of coughing on the tracheal tube is prevented and the likelihood of bleeding is reduced. However, the above-mentioned advantages for awake tracheal extubation are absent when the tracheal tube is removed from anesthetized patients. Awake extubation may place the patient at increased risk for airway obstruction and laryngospasm if the anesthetist misjudges the patient's level of anesthesia and extubates the trachea during "stage two". Only anesthetists skilled in the airway management of young children, who can quickly "rescue" a patient who develops airway obstruction or laryngospasm, should extubate the trachea of an anesthetized patient. If deep tracheal extubation is planned, a tongue stitch must be in place so the anesthetist can pull on the suture and move the tongue forward to relieve the airway obstruction. It is also helpful to place

the anesthetized child on her/his side following tracheal extubation; this allows the tongue to fall forward, not backward, and minimizes airway obstruction.

It is also important to pay careful attention to the method by which the airway is suctioned during emergence from anesthesia. If the anesthesia provider fails to take meticulous care during airway suctioning during wakeup, he/she can damage or disrupt the surgeon's repair and cause bleeding or dehiscence of the repair. Thus, when suctioning the airway, it is important to use a suction catheter rather than a Yankauer suction tip and only suction outside the teeth next to the inside of the cheeks or in the midline away from the repair. When suctioning in the midline, care must be taken to avoid disrupting the suture lines, especially following a cleft lip repair. To avoid injury to a palate repair, it is safer to open the mouth and suction in the midline just over the tongue. After suctioning, verify that the palate is appropriately dry and not bleeding. If more than a trace amount of blood is suctioned continuously from the palate, it is preferable to have the surgeon take another look to determine if there is a bleeding site that can be seen and cauterized, even if this means re-anesthetizing the patient. The safest place to deal with a patient whose palate is bleeding is the operating room, not the recovery room or on the ward. It is important to make sure the palate is dry before the trachea is extubated and before leaving the operating room.

Analgesia Considerations

Opioids

The most appropriate opioid to use during surgery is one that is long acting and causes little respiratory depression, especially if the plan is to have the patient breath spontaneously throughout surgery. At present morphine is our best option. Giving a total dose of 0.1mg/kg every four hours, divided into three or four doses, usually keeps the patient breathing spontaneously. Fentanyl is a poor choice because it only provides 30-40 minutes of pain relief, and in many countries is more expensive. Hydromorphone, a drug that has five times the potency of morphine, is also an acceptable choice. The usual dose of Hydromorphone is 0.015-0.02mg/kg of drug given in divided doses over 2-to-6 hours. The specific dose of drug chosen should depend upon whether or not the patient has had or will get local anesthetic, either by the surgeon or the anesthetist, and whether the patient is also being given non-opioid analgesics. Local anesthesia decreases the need for intravenous opioids. A long acting local anesthetic, such as bupivacaine, when given as infra-orbital nerve block or sphenopalatine block (**See Chapter 21**) for cleft lip or palate repairs respectively, significantly decreases or eliminates the need for opioid analgesics.

Non-opioid agents

Ketamine is a good adjuvant to use with opioids because it provides good analgesia without causing significant respiratory depression. However, using ketamine for cleft surgery tends to increase airway secretions in patients who already have significant amounts of secretions.

Nonetheless, Ketamine use significantly decreases opioid requirements. The dose of ketamine is 0.5-1.0mg/kg IV or IM as an adjuvant to morphine.

Acetaminophen can be given orally preoperatively or rectally intra-operatively. The problem with rectal administration of the drug is that very large doses of drug are required, 40mg/kg or more. Half of this dose (20 mg/kg) can be repeated every six hours after surgery. The PO syrup of Acetaminophen can be given preoperatively in a dose of 15mg/kg. Acetaminophen 15mg/kg can also be given intravenously. When Acetaminophen is given intravenously, it must be administered over 15 minutes to avoid causing excessive blood concentrations of the drug.

The use of non-steroidal analgesics, such as oral ibuprofen or intravenous ketorolac is controversial, especially for cleft palate surgery. Some surgeons believe that they increase postoperative bleeding, although the evidence for this belief is poor. Many surgeons accept the intraoperative use of these drugs. They and others accept their use postoperatively if good hemostasis was obtained and the surgical field was very dry at the end of surgery. Communicate with the surgeon and get her/his opinion before administering these drugs.

Local anesthesia can be a particular benefit for patients after cleft surgery. For cleft lips, either local infiltration of the wound or bilateral infra-orbital nerve blocks can be done (**See Chapter 21**). To have the maximum effect, bilateral infra-orbital nerve blocks are required for cleft lip repairs. These blocks are easy to do and are associated with few complications. Infiltration of local anesthetic into the surgical site or a sphenopalatine nerve block provides good postoperative pain relief after cleft palate surgery. A sphenopalatine block is more difficult to do and should not be attempted unless the anesthetist has been formally trained to perform this block. These blocks are best done at the beginning of the case while the landmarks are clear. This minimizes opioid requirements during surgery, though performing them at the end of surgery provides a longer duration of analgesia.

Post Anesthesia Considerations

There are three main considerations for patients following cleft surgery: ensuring airway patency, being vigilant for postoperative palate bleeding, and controlling pain. The three are related. Palate bleeding may affect airway patency, and continuous crying and screaming due to pain may cause palatal bleeding. If airway compromise requires insertion of an oral or nasal airway or re-intubation of the trachea, the surgical repair can be disrupted. One of the best ways to calm a child after surgery is to have the mother sit at the bedside and hold the child. She can help monitor for bleeding, calm the child, and encourage the child to take oral fluids. She can help prevent the child from touching her/his cleft lip repair. If local custom allows parental visitation in the Post Anesthesia Recovery Room (PACU), it is strongly recommended that this be done.

Following cleft palate surgery, children should be closely observed in the PACU for the presence

of bleeding. If there is significant bleeding, a sponge is placed against the roof of the mouth and light pressure is applied to control the bleeding. By applying gentile pressure, mild bleeding usually stops. Applying too much pressure may disrupt the surgical repair and increase bleeding. However, if there is more than just a small amount of bleeding, or if the bleeding does not stop after applying pressure for 10 minutes, immediate re-exploration of the surgical site in the operating room should be seriously considered. If there is any doubt about the seriousness of the bleeding, it is much safer and better for the patient to explore the wound early rather than wait until later. Delaying exploration of the wound of a bleeding patient until later often results in returning to the operating room in the middle of the night, which is less safe than doing so during the day. It may require persistence and convincing on the part of the anesthetist to get the surgeon to agree that the wound should be explored; thus, it is important that the surgeon is made aware of the bleeding as soon as the amount of bleeding becomes a concern. If bleeding is significant, especially if a return to the OR is being considered, it is imperative that the patient's hemoglobin be determined. Patients can have significant bleeding that can go undetected if the blood is swallowed rather leave the mouth. A type and cross match of packed red blood cells or whole blood should be done if there is any possibility that a blood transfusion will be required. Remember that a "normal" or elevated hemoglobin concentration (higher than before surgery) may be present in patients after significant bleeding, if fluid replacement for the bleeding was inadequate.

When patients are awake, responding to voice and commands, able to maintain a normal oxygen saturation (SpO_2) in room air, and have little pain, they are ready for transfer from the PACU to the ward. Patients who have undergone cleft lip repair can usually have their IV removed in the PACU because they usually take oral fluids readily and are less likely to have postoperative complications. However, the IV of patients who have undergone cleft palate repair should remain in place for the first 24 hours after surgery, because they often take fluids by mouth poorly and because there is a risk of postoperative bleeding. Both of these scenarios require the presence of an IV to provide proper therapy.

Pain control is a significant issue, especially following cleft palate repair, and has been addressed above. Specific medications that should be administered on the ward will depend upon local availability of drugs and sufficient nursing care. Use caution when administering opioids postoperatively, especially to infants, on the ward.

Special Considerations

Surgery to create a *pharyngeal flap* is a type of cleft palate associated repair that deserves special consideration. The goal of this surgery is to decrease the hyper-nasality of the patient's speech by restoring velopharyngeal competence or by functionally separating the nasal passages and the oral cavity. However, creating a pharyngeal flap can sometimes cause significant airway

compromise after surgery. This occurs because passage of air through the nasopharynx is often severely restricted by the presence of airway (flap) edema and by a significant decrease in the size of the oropharynx. Following tracheal extubation, the patient frequently unable to breath through her/his nose. This difficulty with breathing can be made worse if opioids given for pain reduce the respiratory rate. Opioids decrease respiratory drive and ventilation, which may lead to hypoxemia and necessitate rapid re-intubation of the trachea; this may be very difficult. Sometimes inserting a nasal airway will provide an adequate airway while the trachea is being re-intubated. At times it is necessary to take down the flap to assure an adequate airway. When pharyngeal flap operations are done, it is essential that all equipment and drugs needed for ~~a~~ rapid re-intubation of the trachea be immediately available prior to extubation. These patients usually require observation in an intensive care unit for the first 24 hours after surgery.

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Cleft lip repair under local anesthesia (video): http://www.youtube.com/watch?

Chapter 17
ANESTHESIA FOR EAR, NOSE AND THROAT (ENT) SURGERY

Olutoyin Olutoye M.D., M.Sc., FAAP

Introduction

Ear, Nose and Throat (ENT) surgeries are the most common surgeries performed in children, and very often require the surgeon and anesthetist to share the same workspace. Hence communication between them is essential. Tracheal tubes are more likely to become dislodged from the trachea during these procedures than during most other procedures because the surgeon must frequently move the patient's head to accomplish the surgery. This chapter discusses common ENT surgeries and the special precautions required for some procedures.

Middle Ear Procedures

Myringotomy & Insertion of Pressure Equalization Tubes

Chronic otitis media (OM) is characterized by fevers and ear pain (plus or minus ear discharge) and is common in young children. It often occurs in association with upper respiratory tract infections of viral or bacterial origins. In many instances OM is treated with and responds to oral antibiotics, but repetitive infections usually require surgery, which entails creation of a hole in the eardrum (myringotomy) to relieve pressure and to drain middle ear secretions. A small pressure equalizing metal or plastic tube is commonly inserted to keep the hole open and prevent fluid accumulation. These tubes remain in place for up to six months and fall out spontaneously; occasionally they have to be surgically removed if they become impacted. Surgery for placement of these tubes is very short but requires the child to remain very still; hence anesthesia is usually required.

Anesthesia for insertion of pressure equalizing or myringotomy tubes usually includes inhalation induction of anesthesia with halothane or sevoflurane with or without nitrous oxide (if available) and oxygen. N_2O may distend the eardrum and make it easier for the surgeon. Spontaneous ventilation is maintained throughout surgery and is delivered through a facemask. An oral airway prevents airway obstruction and movement with breathing. If available, surgeons use a microscope or magnifying glasses to perform myringotomies. These magnifying devices improve

the surgeon's ability to see the operative field and improves tube placement. During insertion, the patient must lie perfectly still because any movement is magnified by the microscope and makes it harder to perform the surgery. The procedure usually lasts 10-15 minutes, even when tubes are placed in both ears. Children with Trisomy 21 (Down syndrome) have very narrow ear canals, which makes it more challenging for the surgeon and prolongs the surgery and anesthesia. Since most of these surgeries are very short, some practitioners do not feel it necessary to place and IV for surgery. If this is done, the anesthetist must know beforehand where there is an adequate vein into which he/she can quickly place an IV if a problem arises. Pain is usually minimal after these procedures, and only mild analgesics are typically required (oral acetaminophen 10-15mg/kg, intra-operative rectal acetaminophen 40-45mg/kg, intranasal fentanyl 2mcg/kg or intramuscular morphine 0.1mg/kg up to 2mg total).

Tympanoplasty

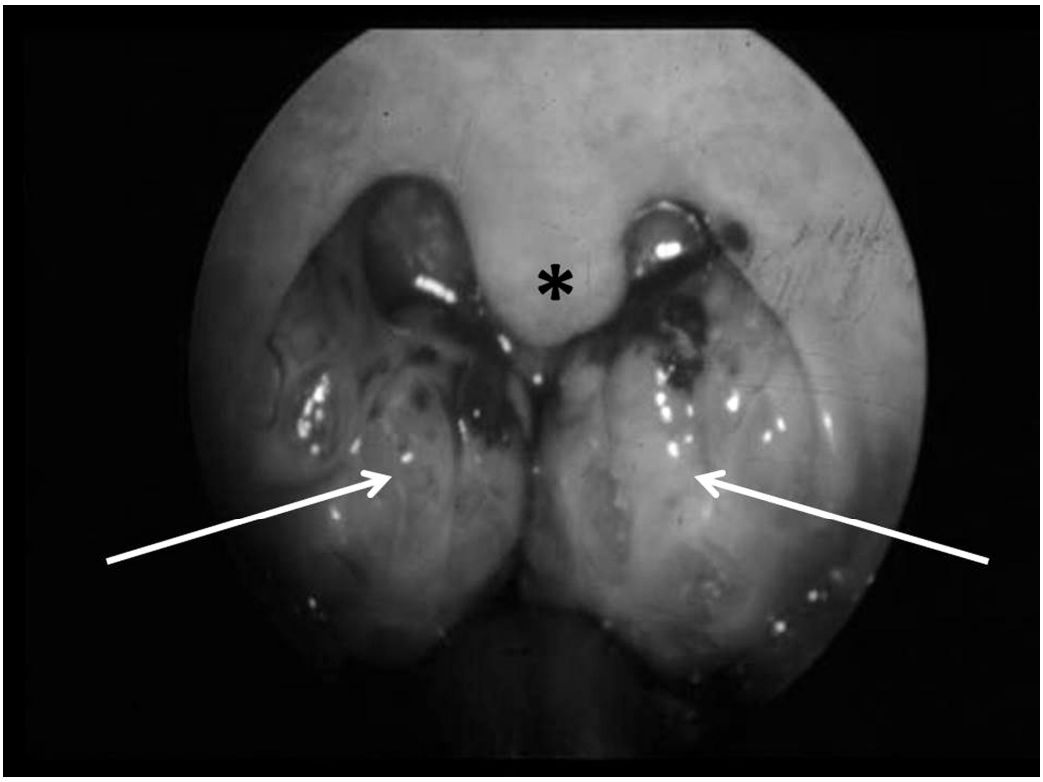
Severe ear infections may progress to chronic middle ear infections, especially the mastoid bone (mastoiditis). Persistent repeated ear infection may cause a large hole in the eardrum that cannot be easily closed with a patch. In this case a fat graft may be required to close the holes. These holes, together with skin growth into a persistent hole in the ear drum produces a condition known as cholesteatoma. Repair of cholesteatomas requires a posterior auricular approach rather than the transauricular approach for surgical repair of the tympanic membrane (tympanoplasty) and lasts much longer than myringotomy and/or placement of ear tubes. It also requires rotation of the operating room bed 180 degrees away from the anesthetist and requires tracheal intubation. Inhalation induction of anesthesia is performed with halothane or sevoflurane in oxygen and nitrous oxide, if the latter is available. However, continued use of nitrous oxide during surgery is *not* recommended because N_2O easily expands any air in the middle ear. Discontinuing the N_2O allows the gas in the middle ear to be rapidly absorbed, which may change the configuration of the eardrum and displace the graft from its normal position. Therefore, nitrous oxide is contraindicated for maintenance of anesthesia in these patients. Nitrous oxide can also aggravate the postoperative nausea and vomiting that commonly accompany middle ear surgery.

After induction of anesthesia, intravenous access is obtained and a sedative hypnotic agent, e.g., propofol 2-3mg/kg, or ketamine 2mg/kg, is administered to facilitate tracheal intubation. The use of muscle relaxants is avoided because facial nerve monitoring is required for this surgery to help the surgeon from inadvertently cutting the facial nerve. Intra-operative opioids or additional ketamine may be required for pain control during and after surgery. Because postoperative nausea and vomiting are common following middle ear procedures, intravenous decadron 0.15-0.5mg/kg and ondansetron 0.15mg/kg should be administered at the beginning and near the end of surgery respectively when possible.

Tonsillectomy With or Without Adenoidectomy

Tonsillectomy and adenoidectomy (T&A) are two of the most common surgical procedures performed in children. Approximately 530,000 tonsillectomies are performed annually in the United States in children below 15 years of age. The indication for tonsillectomy without adenoidectomy is frequent tonsillitis, while the indication for both tonsillectomy and adenoidectomy is usually airway obstruction from enlarged tonsils and adenoids (adenotonsillar hypertrophy) **Figure 17-1**. Adenotonsillectomy is a frequent cause of obstructive sleep apnea (OSA).

Figure 17-1: Hypertrophied Tonsils



*This photography shows massively hypertrophied tonsils. The arrows are pointing at the hypertrophied tonsils, which leaves very little space between them for breathing. It may be difficult to insert an oral airway in patients with large tonsils; doing so may cause bleeding. Oral airways should be inserted gently. *- Uvula. Courtesy of Carla Giannoni, MD*

Pathophysiologic Consequences of Adenotonsillar Hypertrophy:

Sleep Apnea

Severe airway obstruction from adenotonsillar hypertrophy leads to disordered sleep patterns and *obstructive sleep apnea (OSA*). The latter is characterized by heavy snoring and pauses in breathing during sleep that last >20sec and in lack of air movement during the obstructive

periods. Gasping for air while asleep, mouth breathing, and daytime somnolence also occur. Halitosis (bad mouth odor) is also present when patients with adenotonsillar hypertrophy who also have an intra-oral infection. Because affected children typically have disturbed restless sleep, they are tired when they awaken in the morning and may remain so throughout the day. This lack of sleep occasionally results in poor school performance. Persistently impaired breathing chronically elevates blood CO_2 levels, which constrict the pulmonary artery and can cause pulmonary hypertension.

Pulmonary Hypertension

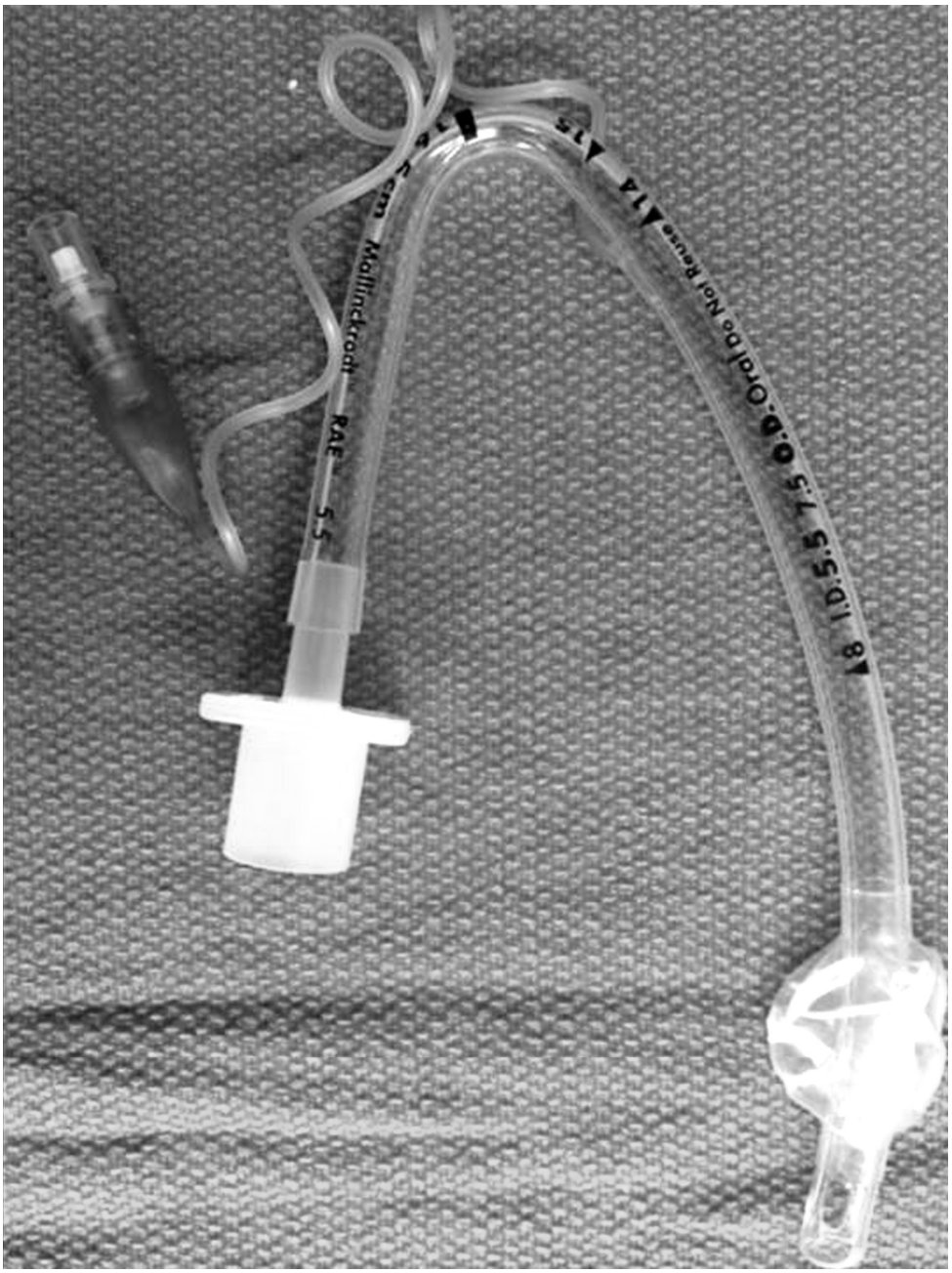
Pulmonary hypertension elevates the $PaCO_2$ levels and leads to worse hypoxemia during sleep, both of which constrict the pulmonary arteries. The increased intra-arterial pressure induces new muscle formation in the arteries, and this causes the pressures in the pulmonary arteries to be continuously elevated. Pulmonary hypertension is suspected during physical examination when the second heart sound of patients who have adenotonsillar hypertrophy is loud and the systolic murmur of tricuspid regurgitation is present. Other signs include neck vein distention and a prominent right ventricular heave, i.e., a lifting of the chest just to the right of the sternum, which can be both seen and felt. The liver is often enlarged (i.e., >2cm below the right costal margin) and its edge is rounded and not sharp, due to liver congestion. The chest X-ray of children with pulmonary hypertension shows evidence of cardiomegaly, right ventricular hypertrophy, and prominent enlargement of the right atrium. The electrocardiogram also shows evidence of right atrial enlargement (increased amplitude of the p wave). If the pulmonary hypertension is left untreated, cor pulmonale and right heart failure occur. The signs and symptoms of right heart failure include liver congestion and enlargement (hepatomegaly). It may also be pulsatile on palpation. In addition, there may be jugular vein distension. In severe cases, edema of the lower extremities is also present.

Where available, patients with severe OSA and pulmonary hypertension can be treated with a continuous or bi-level positive airway pressure-breathing device for a few days before surgery, particularly during nighttime sleep. This improves breathing, reduces decrease carbon dioxide levels, and decreases the amount of pulmonary hypertension. Prompt surgical management of adenotonsillar hypertrophy and treatment of obstructive sleep apnea-induced pulmonary hypertension usually prevents progression of the pathophysiology of pulmonary hypertension.

Surgical Management

To gain access to the adenoids and/or tonsils, the surgeon inserts an oral mouth gag that holds the mouth open. It is helpful if a special preformed tracheal tube can be used to facilitate mouth gag insertion, make it easier for the surgeon to see the tonsils, palate and upper lip, and minimize tracheal tube compression or obstruction by the mouth gag **Figure 17-2**.

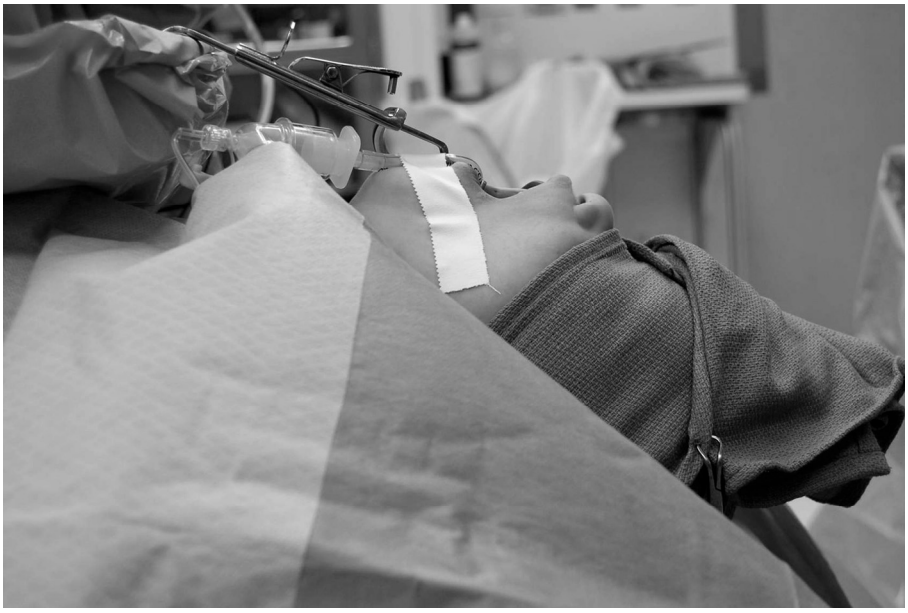
Figure 17-2: Oral RAE Tracheal Tube



*This figure shows an oral RAE tube for use during oral surgery. It is preformed so that the tube connector is over the chest when the tube is in place. This gets the anesthesia circuit out of the surgeon's way so he/she can see. The cuff is inflated to reduce aspiration of blood and to decrease the leak of anesthetic gases and oxygen into the mouth. The latter could cause an airway fire (**See Chapter 8**).*

Following tracheal intubation, the outer portion of this tube is taped in the midline onto the child's chin **Figure 17-3**.

Figure 17-3: Oral RAE Tube and Mouth Gag in Place for Surgery.



This picture shows a mouth gag present in the patient's mouth, which is overlying, but not compressing, an oral RAE tracheal tube. The mouth is held open by the gag.

The surgeon should open the mouth gag carefully to ensure that doing so does not compress or obstruct the tracheal tube. If the tracheal tube is compressed by the mouth-gag, there will be difficulty positive pressure ventilating the patient's lungs; much higher ventilation pressures will be required. Spontaneously breathing patients show signs of obstructed breathing, including indrawing of the chest and simultaneous outward movement of the abdomen (rocking respirations) plus intercostal, suprasternal and infrasternal retractions. In severe cases, air movement ceases completely. This requires immediate release and repositioning of the mouth gag.

Anesthetic Management

Anesthesia induction is commonly accomplished by inhalation of either halothane or sevoflurane. If the child has an IV in place before surgery, and intravenous anesthesia induction with thiopental 4-6mg/kg, propofol 2-3mg/kg, or ketamine 1-2mg/kg can be used. Again, if the child does not have an IV, the anesthetist should determine where he/she will rapidly place and IV in an emergency. Tracheal intubation can be achieved during deep inhalation anesthesia (without muscle relaxants) with sevoflurane or halothane, or it can be done following administration of an intravenous induction agent, with or without a short acting muscle relaxant. Depending on availability, short to intermediate acting muscle relaxants, including intravenous succinylcholine 1-2mg/kg, rocuronium 0.6mg/kg, atracurium 0.5mg/kg or cis-atracurium 0.2mg/kg can be administered along with the sedative-hypnotic agents to facilitate tracheal intubation. Muscle

relaxants should only be given if the anesthetist is fairly certain he/she can maintain an adequate airway and easily ventilate the patient's lungs when he/she becomes unconscious. This is not always easy to determine in patients with enlarged tonsils and adenoids. In the United States, succinylcholine is only used to emergently place a tracheal tube in patients who are considered to have a full stomach. This restriction is the result of reports of hyperkalemia and cardiac arrest during induction of anesthesia and succinylcholine administration, particularly in young males who have not yet started walking. These patients may have an undetected/undiagnosed form of muscular dystrophy that predisposes them to muscle break down and severe hyperkalemia with succinylcholine-induced muscle contractions. Since succinylcholine is the only muscle relaxant available in some countries, it should be used with great caution in this patient population.

Patients with severe obstructive sleep apnea are very sensitive to opioids. Therefore, the recommendation is to not give opioids until the trachea has been extubated. The dose of opioid must be titrated to effect, i.e., give enough opioid to prevent pain but not enough to cause apnea. This can be done by administering small amounts of opioid at a time and waiting 3- 5 minutes to determine their effect before giving more drug. Another option in patients undergoing adenotonsillectomy includes giving intra-operative intravenous ketamine 1-2mg/kg for pain management. Ketamine has the benefit of not depressing respiratory efforts. However, it may cause hallucinations, which are disadvantageous for older children. Hallucinations usually do not occur with these low doses of ketamine. Use of intravenous diclofenac 0.3mg/kg can also be considered. Administration of intraoperative dexamethasone 0.15 to 0.5mg/kg, reduces edema at the surgery site, decreases postoperative pain, and reduces the incidence of nausea and vomiting after surgery.

During surgery, the inspired oxygen concentration (FiO_2) should be below 30% to reduce the risk of an airway fire (**See Chapter 8**). The use of cuffed endotracheal tubes decreases the potential for an airway fire by reducing the amount of oxygen-containing gas in the pharynx. The three components needed for an airway fire to occur include an oxygen-rich environment (FiO_2 ≥0.30% and/or nitrous oxide), an ignition source (electrosurgical devices or laser), and fuel (endotracheal tubes, surgical drapes, gauze, alcohol containing solution and masks). Most airway fires are occur when an electrocautery burns through an endotracheal tube that has >30 percent oxygen flowing through it.

If an airway fire occurs, the flaming endotracheal tube should be immediately removed from the airway, the oxygen source completely turned off, and the airway flooded with saline or water. After the fire has been extinguished, the extracted endotracheal tube should be inspected to verify that it is intact and no fragments are left in the airway. Ventilation should be managed via a facemask. Bronchoscopy is done to detect the extent of mucosal damage and to remove burned, coagulated tissue. A tracheal tube is then inserted to assure and airway.

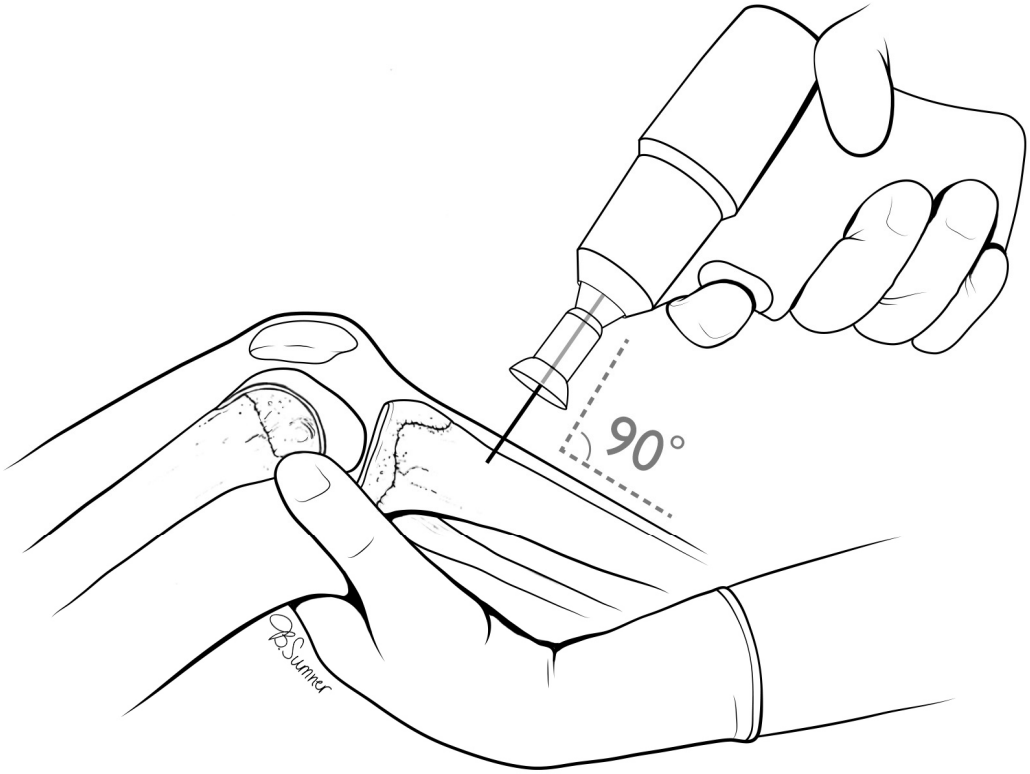
Post-Operative Management

Young children, particularly those under three years of age, should be monitored overnight in an intensive care unit (ICU) when possible to provide a higher level of supervision. Obstructive symptoms, if they exist, may worsen after surgery due as the operative area swells. Close monitoring during this early post-operative period will allow prompt detection of respiratory depression if it develops in response to the giving pain medication after surgery.

Post Tonsillectomy Bleed

Post tonsillectomy bleeding may occur immediately after surgery while the child is still in the recovery room, in which case the child should be taken quickly back to the operating room for re-exploration and cauterization of the bleeding source. Bleeding may also occur 7-10 days after surgery when the wound scab or eschar peels off. When bleeding occurs, the child initially swallows the blood. By the time he/she starts coughing up or regurgitating blood, the stomach typically contains significant amounts of blood and the hemoglobin concentration has decreased. By the time the child arrives at the hospital, he/she may be dehydrated from not drinking and hypovolemic from bleeding, making it very challenging to secure intravenous access. History obtained from the parents and child may indicate that vomiting of frank red blood (recent) or dark brown old blood. However, this does not always indicate either the duration or severity of the bleeding. A good history and physical examination are required to determine the severity of bleeding (**See Chapter 1**). Dry, parched lips and decreased skin turgor (persistent tenting of skin when it is lifted up) are signs of significant bleeding and severe volume loss. Emergency surgery is needed to stop the bleeding. When no available site exists for percutaneous intravenous access, the anesthetist should consider using the intraosseous route to institute fluid resuscitation and/or blood transfusion as indicated. To obtain intraosseous access a specially made needle or a large bore intravenous catheter, (such as a 16g catheter) is inserted into the tibial tuberosity, which is located approximately 1cm below the head of the tibia. **Figure 17-4** shows one technique for intraosseous needle placement.

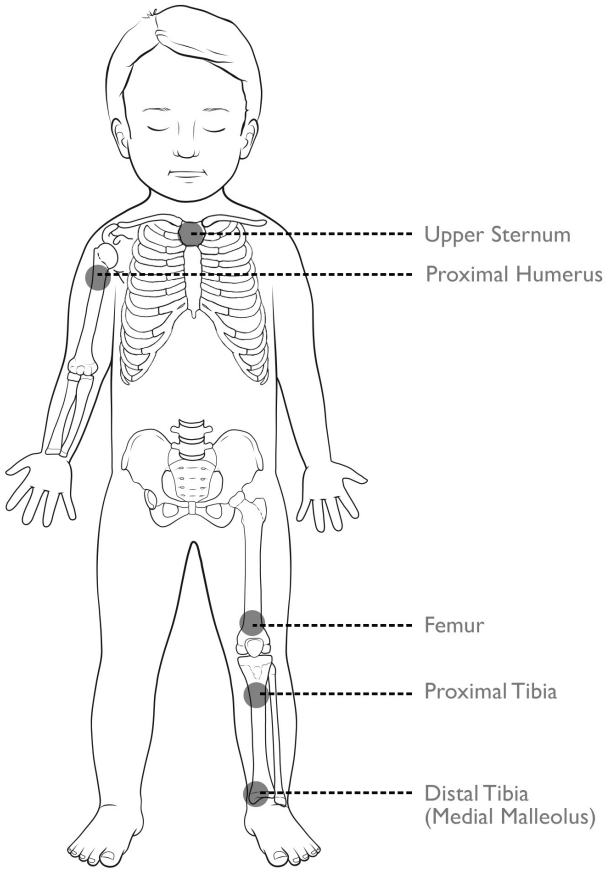
Figure 17-4: Technique for Insertion of an Intraosseous Needle



This figure shows insertion of an intra-osseous needle using an electric drill. Note the needle is being inserted at 90° to the long axis of the bone.

Care must be taken to avoid placing the needle in the growth plate, which could prevent that extremity from growing. Firm pressure is applied to the needle until definite loss of resistance is felt. Easy aspiration of bone marrow verifies correct placement of the needle in the marrow cavity. Alternative locations for intraosseous needle insertion include: head of the humerus and the upper 1/3 of the sternum. **Figure 17-5** shows these sites.

Figure 17-5: Sites for Intraosseous Line Placement



Anesthetic Management of Post Tonsillectomy Bleeding

A hemoglobin value obtained after the bleeding occurred should be evaluated to determine the need for peri-operative blood transfusion. Regardless of when the patient last TE, he/she has a stomach full of blood and must be managed by rapid sequence induction of anesthesia and rapid tracheal intubation. Once adequate intravenous access has been obtained, the intravascular volume is expanded with boluses of normal saline 20ml/kg until the arterial blood pressure is normal and perfusion has improved. This may require as much a 60ml/kg of normal saline. Then the patient can breath 100% oxygen for five minutes before giving intravenous propofol 2-3mg/kg or ketamine 1-2mg/kg and intravenous succinylcholine 2mg/kg or rocuronium 1mg/kg to facilitate rapid sequence induction of anesthesia and tracheal intubation. Propofol should be used with caution in these potentially volume-depleted patients because propofol decreases peripheral vascular resistance and may cause profound hypotension. Ketamine may be a better choice as an induction agent because it causes the release of catecholamines and increases peripheral vascular

resistance of patients with relatively normal intravascular volumes. However, volume depleted patients will become hypotensive when the drug is injected.

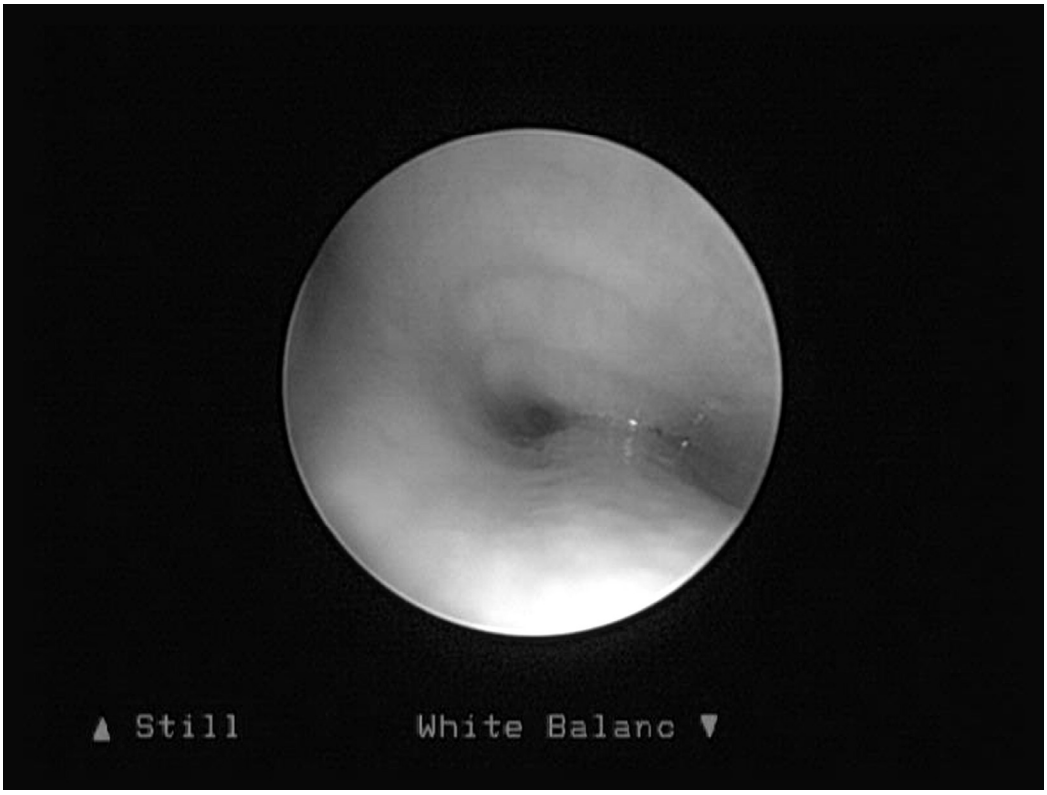
Two separate large bore Yankauer type suction tips should be available for removing blood from the oropharynx during tracheal intubation. Bleeding may be aggravated by direct laryngoscopy and obscure visibility of the vocal cords during tracheal intubation. If there is a lot of blood in the larynx, it may be difficult to view the glottis and vocal cords. If bubbles are identified coming through the blood during expiration, this often serves as a guide to the location of the vocal cords. Correct placement of the tracheal tube should be confirmed by noting bilateral chest rise with inspiration, seeing condensation inside the tracheal tube with positive pressure ventilation and the presence of an end-tidal carbon dioxide waveform on a capnograph, if available.

The surgical procedure to control bleeding usually only lasts a few minutes and involves identification/localization and cauterization of the bleeding site. Because the duration of surgery is short and the patient has a full stomach, succinylcholine is the drug most commonly used for tracheal intubation of patients with a full stomach. Rocuronium 1mg/kg can also be used, but it may not be possible to adequately reverse the neuromuscular block at the end of the short procedure. Likewise, pain medication should be administered judiciously, if at all, as surgery is short and narcotics may cause respiratory depression and make it difficult to extubate the trachea. It is usually better to wait until after the trachea has been extubated before giving pain medications. All volatile anesthetic agents can be utilized for maintenance of anesthesia. However, the drug should be titrated to an end-tidal anesthetic concentration that will allow rapid awakening from anesthesia and tracheal extubation. The tracheal tube should only be removed when the patient is awake.

Tracheomalacia

Tracheomalacia is due to underdevelopment of the tracheal rings or widening of the membranous portion of the trachea and is observed in extremely premature babies; particularly those who have had a tracheal tube in place for weeks. As a result, tracheomalacia usually occurs in tertiary care centers. The classic presentation of tracheomalacia is stridulous breathing or wheezing, especially during expiration because the intrathoracic airways collapse during exhalation because the extra tracheal pressure is more positive. Other symptoms include recurrent breath holding or apnea spells or a chronic cough in older children. This condition also occurs when an extrinsic vascular ring compresses the trachea (**Figure 17-6**).

Figure 17-6: Endoscopic View of Distal Tracheal Lumen Compression by Innominate Artery.



This figure shows tracheal compression by an extra thoracic ring. Photograph courtesy of Deidre Larrier, MD

Diagnosis and Treatment: Diagnosis and differentiation of these two conditions (tracheomalacia and vascular ring) involves rigid or fiberoptic bronchoscopy. Both require general anesthesia, commonly a combination of intravenous and inhalation anesthesia. Management of tracheomalacia and tracheal rings is initially medical and application of continuous positive airway pressure and bi-level positive airway pressure to prevent tracheal collapse during expiration. If symptoms persist, surgical approaches are employed to improve airway patency, such as airway stenting with tracheostomy and aortopexy. Aortopexy is performed in specialized institutions via video assisted thoracoscopic surgery and is described in more specialized textbooks.

Anesthetic Management

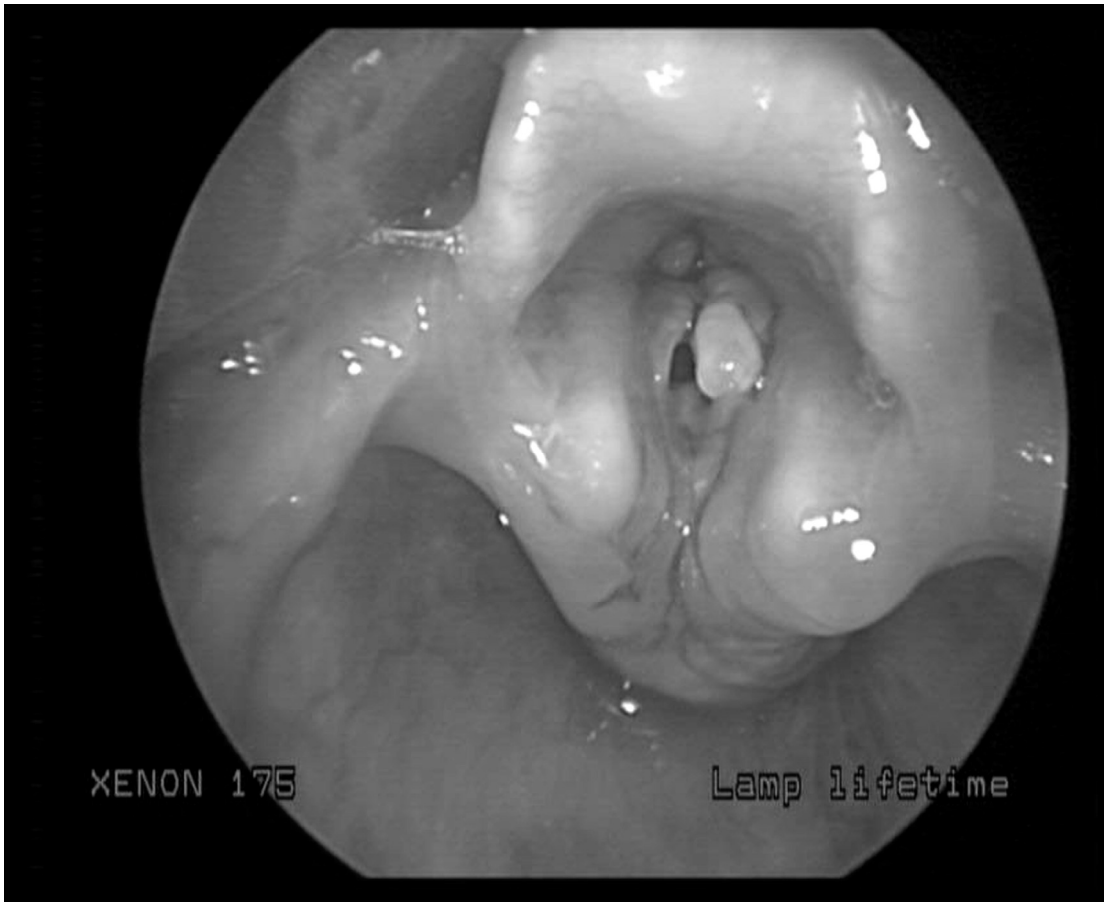
Diagnostic bronchoscopy: Inhalation induction of anesthesia is performed and intravenous access is secured once the monitors have been applied. The anesthetic goal during this surgery is to provide a sufficiently deep plane of anesthesia that prevents coughing during bronchoscopy while maintaining spontaneous ventilation. Spontaneous ventilation is needed so the surgeons can watch tracheal movement during inspiration and exhalation to determine if it collapes during exhalation. Intravenous access allows the anesthetist to administer intermittent boluses of

propofol or ketamine or to continuously infuse these drugs. Chest rise must be continuously verified during surgery, especially as it is difficult to ascertain end-tidal carbon dioxide ($EtCO_2$) concentrations due to lack of complete airway seal during surgery.

Tracheostomy: Tracheostomy is typically performed when it is difficult to wean a child from mechanical ventilation or when a baby has severe tracheomalacia or laryngomalacia. Inhalation induction of anesthesia is done via the indwelling tracheal tube. Giving muscle relaxants prevents patient movement while the tracheostomy tube is being inserted. Once the surgeon has opened the trachea, he/she will direct the anesthetist to slowly withdraw the tracheal tube to a position just above the hole in the trachea without removing it from the trachea. This allows reinsertion of the tracheal tube beyond the hole in the trachea if necessary. Once the tracheotomy tube is in place, the tracheal tube can be removed from the mouth. The anesthesia circuit is then connected to the tracheostomy, and the anesthesiologist manually ventilates the patient's lungs to ensure bilateral chest rise and the presence of carbon dioxide in the expired gases. Once correct placement of the tracheostomy tube is confirmed, it is secured around the neck by tracheal ties. Surgeons ensure that the ties are not too tight by confirming that he/she can easily get two fingers between the ties and the neck. If the ties are too tight, the jugular vein may be obstructed.

Airway granulomas or Subglottic narrowing: Both of these conditions can occur when a child's trachea has been intubated for long periods of time. The tube causes mucosal edema, which narrows the airway and causes stridulous breathing. Prolonged tracheal intubation may also cause formation of granulation tissue within the airway. Airway narrowing and stridor characterize both conditions. Airway granulomas **Figure 17-7**, often present with more severe symptoms of airway obstruction, particularly if the granuloma is large enough to obstruct the airway and is pedunculated. This may cause a ball-valve effect during breathing.

Figure 17-7: Airway Granuloma Resulting from Tracheal Intubation

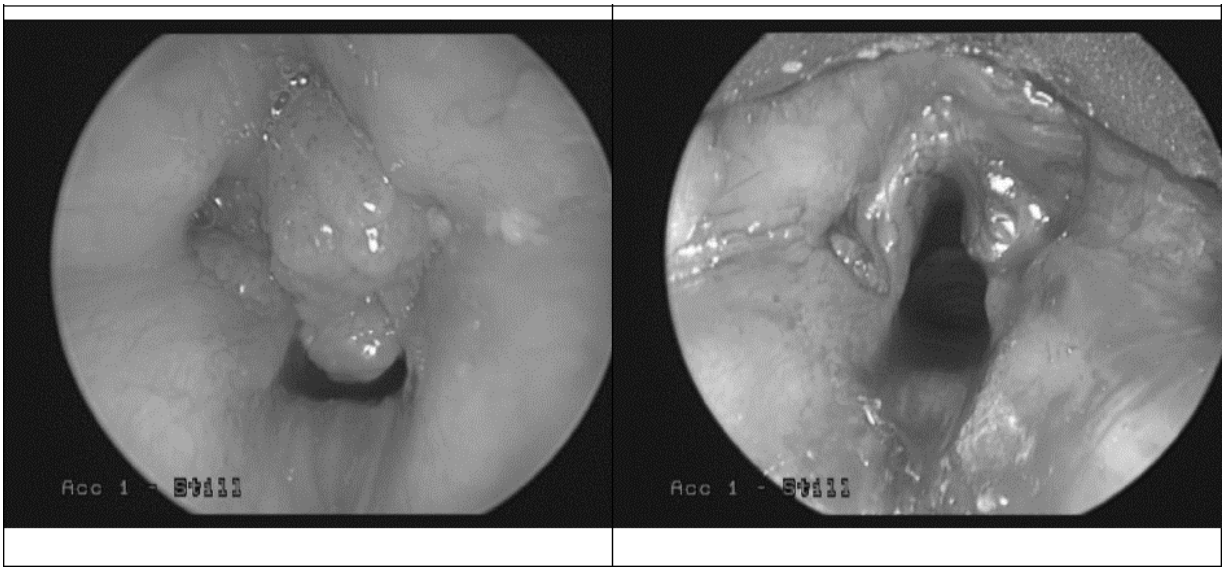


This figure shows polyps in a bronchus, some of which are partially occluding the bronchus. Photograph courtesy of Mathew Sitton, MD

Management: The only effective treatment for airway granulomas is removal of the lesions under direct vision, i.e., via direct bronchoscopy and laryngoscopy. Subglottic narrowing is managed by laser resection of the excess tissue or radial incisions into the larynx using a laser. Occasionally, the stenosis recurs and is treated with mitomycin-C, an anti-cancer drug, which is applied directly to the incised laryngeal surfaces to prevent re-adhesion of the freshly separated mucosal tissue.

Laryngeal Papillomas: Laryngeal papillomas are usually referred to as recurrent respiratory papillomas. They are small, assorted cystic-appearing masses that are found in the airways of children following transmission of human papilloma virus types 6 and 11 from the mother during passage through the birth canal at birth **Figure 17-8**. This occurs in approximately 4.5 per 100,000 births. There do not have to be active lesions in the birth canal at the time of delivery for the infectious agent to be transmitted to the child. Latent maternal infections with human papilloma virus can also cause airway papillomas in children.

Figure 17-8 Left: Laryngeal Papillomas at Glottic Opening. Figure 17-8 Right: Glottic Opening Post Laser Resection of Papillomas.



Left: papillomas are obstructing much of the glottis. Right: papillomas have been resected with a laser. The airway is much more open. Note the edema and mucosal bleeding following polyp removal. Photographs courtesy of John Jones, MD

Presentation and Pathophysiology: These airway masses are initially detected when a child develops respiratory distress and some element of airway occlusion. The amount of respiratory distress depends on how severely the airway is obstructed. Other symptoms include a weak cry, noisy breathing (stridor), chronic cough, and difficulty swallowing (dysphagia). Another indication of the severity of the airway obstruction is a voice that is barely audible. When this occurs, additional, signs of airway obstruction, such as suprasternal and intercostal retractions, may also be observed on physical examination.

Management: There is no permanent cure for polyposis. Some lesions spontaneously resolve, but some do not and are responsible for the patient's death, especially if the child's compliance with doctor visits was poor and he/she did not benefit from frequent airway examinations and/or resection of papillomas. Without regular follow-up and care, the papillomas usually grow and extend from the larynx or trachea into the tracheobronchial tree, at which point surgical management may be impossible. The anti-viral agent, interferon has been used for management of pappilomas but does not prevent recurrence of airway lesions. Surgical management involves resection of the airway lesions with carbon dioxide laser or coblation (cold cutting and coagulation) via direct laryngoscopy and bronchoscopy under general anesthesia.

Anesthetic Management: Following inhalation induction of anesthesia and application of routine monitors (electrocardiogram, blood pressure, pulse oximeter and temperature) intravenous access is secured. With the patient spontaneously breathing anesthetic and oxygen through a

facemask, the table is turned 90 degrees to facilitate direct laryngoscopy or suspension laryngoscopy by the surgeon. The anesthesia circuit is attached to the ventilating port of the bronchoscope to deliver anesthetic gas and oxygen during the procedure. Since the patient's mouth and the anesthesia system are open, it is usually necessary to give intermittent bolus of either ketamine or propofol to maintain the required deep plane of anesthesia. Alternatively, a continuous infusion of propofol 100-200mcg/kg/min can be used to maintain anesthesia. Intravenous lidocaine 2mg/kg will reduce airway reactivity or the surgeons can spray the airway with aerosolized lidocaine under direct vision, both of which decrease airway reactivity. Coblation or laser ablation of laryngeal papillomas can be performed with general anesthesia, spontaneous ventilation, and no tracheal tube. However, if frequent oxygen desaturation occurs, it may be necessary to intermittently insert a tracheal tube and ventilate the lungs with oxygen between laser treatments. At all times during this surgery, anesthesia providers and surgical team members must ensure that the inspired oxygen concentration is below 30% to prevent an airway fire (**See Chapter 8**). If higher oxygen concentrations are required, a special laser tracheal tube or a regular tracheal tube with aluminum foil wrapped around the exposed portion of the tube i.e., the part above the vocal cords. This decreases (but does not eliminate) the possibility of an airway fire. The dangers associated with using foil include the possibility of a piece of loose foil getting into the airway or an inadvertently exposed portion of the endotracheal tube catching fire during laser treatment.

Management of an airway fire includes immediate removal of the flaming object (tube, sponge), turning off the oxygen, and flooding of the airway with saline. The airway must be examined afterward with bronchoscopy to determine the extent of injury and the presence of airway debris that can be removed.

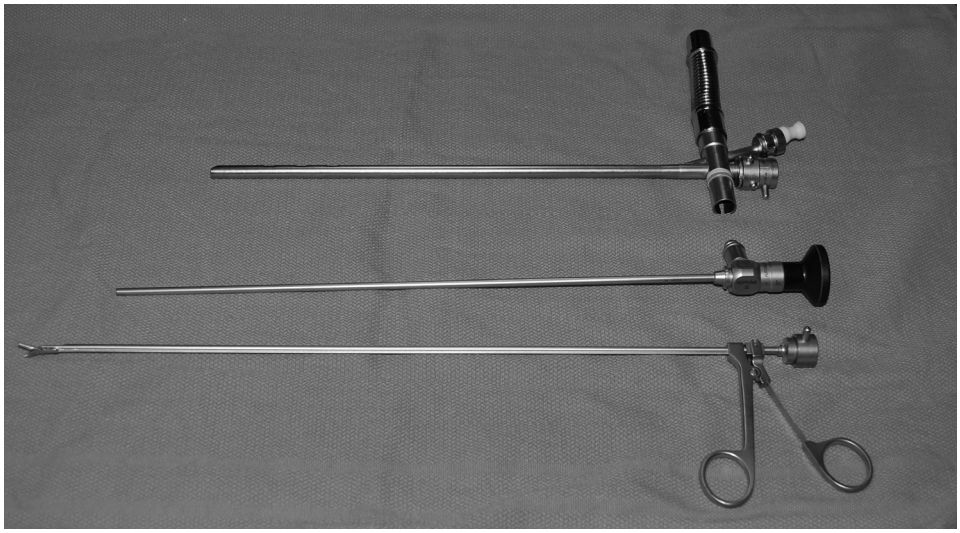
Foreign body aspiration is relatively common in the 1-5 year old children because they place objects in their mouths, including peanuts, seeds and toys that can be aspirated. Aspiration of small batteries is particularly dangerous because they contain corrosive matter that can severely damage the airway. Surgical removal of suspected batteries should be treated as an emergency.

Presentation: It is uncommon for someone to actually witness foreign body aspiration in children. Signs that this has happened include: sudden onset of drooling (caused by inability to swallow), wheezing, or persistent cough. In some instances, foreign body aspiration presents as pneumonia or reactive airway disease that is refractory to medical treatment.

Diagnosis: A chest X-ray may show that the lung on the affected side is collapsed. If the foreign body is lucent, it will be seen on X-ray. If it is not (plastic toys, glass) only the effects of airway blockage will be seen. Auscultation of the lung reveals decreased breath sounds on the affected side. In clinically stable patients, flexible diagnostic bronchoscopy is often performed to search for aspirated foreign bodies. However this requires two separate anesthetics because foreign bodies cannot be removed during flexible bronchoscopy.

Surgical management: Removal of the foreign body is indicated shortly after the diagnosis is made. Removal of objects from the airway is usually achieved during rigid bronchoscopy. The object is grasped with an airway forceps (**Figure 17-9**).

Figure 17-9: Ventilating Bronchoscope, Optical Telescope, and Foreign Body Grasper.



This picture shows a ventilating bronchoscope with an optical telescope (middle) and grasping forceps for removal of tissue or foreign objects.

Anesthetic management: Having an IV before surgery allows the anesthetist to administer anticholinergic agents to dry up watery secretions and improve visibility throughout the procedure. If no IV is available, once anesthesia has been induced and intravenous access is obtained, anticholinergic drugs can be given. Following induction of anesthesia and placement of routine monitors, the head of the bed is turned 90 degrees away from the anesthetist to allow the surgeon to have access to the patient's head.

Surgeons require an unobstructed view of the larynx for direct laryngoscopy and bronchoscopy. A rigid bronchoscope is inserted into the larynx and advanced into the trachea after the vocal cords are sprayed with lidocaine 3-5mg/kg or with nebulized lidocaine. These local anesthetics reduce vocal cord irritation during the procedure. Ventilation during the procedure can be provided either with a jet ventilator (if available) or bag connected to the side port of a rigid bronchoscope.

Jet ventilation administers high concentrations of oxygen at 50psi (pounds per square inch) down the larynx through a 14-16-gauge cannula. The high-pressure oxygen delivered down the narrow tube creates a negative pressure at the end of the cannula, which causes room air to be drawn (entrained) into the lungs. This system requires an unobstructed open glottis to allow the entrained air to be expelled during exhalation. Therefore, patients with complete airway obstruction are not good candidates for jet ventilation, as inability to exhale leads to barotrauma and pneumothorax. Other disadvantages of jet ventilation include inability to measure end-tidal

carbon dioxide and peak airway pressures. Gastric distension may also occur. Rigid bronchoscopy requires that the mouth remain open during the procedure to allow gases to escape. Therefore intravenous agents, boluses or infusions of propofol or ketamine, are useful for supplementing inhalation anesthetics. The side port of a rigid ventilating bronchoscope can be attached to the anesthetic circuit for delivery of inhaled anesthetic during bronchoscopy. Spontaneous ventilation and deep anesthesia are maintained while the surgeon is identifying the foreign body. A small grasping or biopsy forceps is inserted through the scope to retrieve the foreign body. In small children, the optical telescope used to visualize the airway may completely obstruct ventilation through the ventilating scope. If this occurs, the telescope must be removed intermittently and the proximal end of the ventilating scope occluded with a finger or thumb to allow the anesthetist to manually ventilate the patient's lungs. **Figure 17-9** shows the ventilating bronchoscope, optical style and grasping forceps. A small amount of non-depolarizing muscle relaxant or a bolus of propofol may be required just before the foreign object is retrieved to ensure that the patient does not move at this critical time. If the foreign body gets dislodged in the trachea as it is being retrieved, it is often best to push it back into the bronchi from which it was originally retrieved to prevent complete airway obstruction and inability to ventilate the lungs due to tracheal obstruction. Organic materials, such as peanuts, are particularly difficult to retrieve as they can fragment into many small pieces while being retrieved and require multiple attempts to retrieve and remove all of the pieces. In general, intravenous steroids, such as dexamethasone 0.5-1.0mg/kg, should be administered at the beginning of surgery to decrease mucosal swelling. Racemic epinephrine may also be required after surgery, depending on the degree of swelling and the presence of stridor.

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Chapter 18
PEDIATRIC AMBULATORY ANESTHESIA

George Politis, MD

Introduction & History

Many of the first anesthetics done in the mid 19th century were for dental extractions,[1] almost always on an outpatient basis, as were many of the earliest anesthetics for surgical procedures. The first known outpatient surgical clinic was established by James Nicoll in 1899 at the Glasgow Hospital for Sick Children, where approximately 1000 outpatient surgeries were performed each year, half of which were conducted on children under three years of age.[1] In the United States of America (USA), Ralph Waters established an ambulatory surgical clinic called the Down-town Anesthesia Clinic in 1919. This clinic improved access for patients and surgeons and provided economic benefits. Improved surgical techniques, anesthesia equipment, and anesthetic medications spurred the relatively small scale outpatient surgical clinics that began to appear after World War I to grow into organized large scale outpatient surgery facilities as early as 1959. The University of California, Los Angeles established an outpatient surgical clinic in 1962 and this clinic is considered to be the forerunner of modern ambulatory surgical centers (ASC).[1] John Ford and Wallace Reed founded the first successful freestanding ASC in Phoenix, Arizona in 1969.

Ambulatory anesthesia is practiced today in numerous settings, including hospital based and freestanding ASCs, surgical and dental offices, and hospital based out-of-OR locations. This chapter will primarily discuss ambulatory anesthesia within a hospital and in freestanding ASCs. Approximately 53 million ambulatory procedures were performed in the USA in 2006, with 57.3% of those taking place in hospitals, 42.7% in freestanding ASCs. The percentage in freestanding ASCs is growing rapidly.[1] Estimates of the percentage of procedures performed on an ambulatory basis in the USA exceeds 80%.[2] This explosion of ambulatory surgery has been driven by economic factors, and by surgeon and patient preference for the ambulatory model. Those preferences occur in part because ASCs allow surgeons to work more efficiently, and ASCs reduce the time patients spend away from home undergoing surgery, and they lower exposure to hospital-acquired infections. The increase in ambulatory surgery has been facilitated by development of short acting anesthetics and by advances in antiemetic and analgesic agents, including multimodal analgesics and especially the development and use of regional anesthesia. Multimodal analgesia uses different classes of analgesics with different sites of action to provide

pain relief with fewer analgesic-related side effects.

This chapter takes a close look at which pediatric patients and which procedures are appropriate for outpatient surgery. It also looks at preoperative considerations for pediatric ambulatory surgery patients, including patient screening, fasting guidelines, and whether or not to cancel an elective ambulatory surgical case when a child has an upper respiratory tract infection. Specific intraoperative and postoperative issues important for providing quality care and for providing efficient ASC patient throughput are discussed. Finally, this chapter will hopefully help existing ambulatory surgery programs to improve, and guide the development of an ambulatory surgery program for those that do not already employ this extremely useful surgical model

Procedure Selection Criteria

Whether a procedure is suitable for an ASC depends on several factors (**Table 18-**I). The procedure must be doable in the small operating rooms often found in ASCs. There should be no need for invasive monitoring (e.g., intra-arterial or central venous lines), little or no chance that a blood transfusion would be needed, and minimal chance of intraoperative or postoperative complications that could convert a stable patient into one who requires a higher level of care. The pain caused by ambulatory procedures must be easily and reliably managed with simple oral analgesics, with regional anesthesia, or with a combination of both. Post-discharge nursing care must be simple. In general, intrathoracic, intracranial, and major abdominal surgery is not appropriate for ambulatory surgery.

Table 18-1

Standard Criteria for Surgical Procedures to Qualify as Ambulatory
Can safely be performed within spatial constraints of the particular facility
No need for invasive monitoring
Little or no chance that a blood transfusion will be required
Minimal chance of surgical or anesthetic complications
Post-operative pain manageable by simple oral regimens or regional anesthesia
Post discharge nursing must be simple

An ambulatory surgical procedure should have a very low likelihood of requiring either escalation of needed care or overnight admission to a hospital. The level of acceptable risk depends in part on how difficult it will be to transfer the patient to a center where definitive care can be provided, and on the ASC's availability to provide laboratory, radiology, and respiratory therapy when needed. Duration of the procedure is generally not a limiting factor; though there may be local governmental rules about this.[3] Procedures commonly performed at the University of Virginia's Outpatient Surgery Center are listed in **Table 18-2.**

Table 18-2

Pediatric Procedures Commonly Performed in the ASC Setting	
Diagnostic Procedures	• Upper and lower endoscopy • Flexible bronchoscopy • Auditory evoked response
Urology	• Hernioraphy/Hydrocelectomy • Hypospadius repair • Orchiopexy • Cystoscopy for stent change or deflux injection • Circumcision • Cordaee repair • Meatotomy
Otorhinolaryngology	• Examination under anesthesia • Myringotomy tubes • Adenoidectomy • Adentonsillectomy • Direct laryngoscopy/bronchoscopy ± laser ablation, typically for papillomatosis
Dental	• Extractions and restorations
Plastic Surgery	• Excision of superficial lesions • Placement of tissue expanders for congenital nevus • Otoplasty

Table 18-2 (continued)

Orthopedic Surgery	• Syndactyly Release/Polydactyly removal • Hardware removal • Anterior cruciate ligament repair • Minor tendon releases • Arthroscopy • Cast changes (T&A) is commonly done, but many anesthetists believe patients undergoing T&A should stay in the hospital overnight if the child is less than three years old, has a syndrome or neuromuscular disease, or has severe obstructive sleep apnea
General Surgery	• Hernioraphy/Hydrocelectomy • Umbilical/Periumbilical hernia repair • Excision of superficial lesions/masses • Abscess incision and drainage • Removal of hardware after pectus repair
Ophthalmology	• Examination under anesthesia • Strabismus repair • Cataract removal, placement of intraocular lens • Lacrimal duct probing ± stenting • Glaucoma procedures: goniotomy, trabeculotomy • Excision of cysts

Often the combination of patient, procedure, and factors related to the facility are taken into account when deciding whether ambulatory surgery is appropriate for a given patient. For example, tonsillectomy and adenoidectomy (T&A) is performed on an ambulatory basis for most patients, but overnight stay is typical for children less than 3 years old, those with syndromes or neuromuscular pathology, and those with severe obstructive sleep apnea syndrome (OSAS). Those subgroups are prone to respiratory complications after T&A.[4-6]

Patient Selection Criteria

Suitability of patients for ambulatory surgery is just as important as the suitability of the procedure for the ASC. Both patient age and the presence of comorbid conditions are important. In general, children having ambulatory surgery should be healthy, but those with *stable* chronic diseases can also be good candidates for ambulatory surgery. Patient age, numerous common chronic pediatric conditions, and the patient's susceptibility to malignant hyperthermia are discussed below with respect to their effect on the appropriateness for ambulatory surgery for a given child.

Patient Age

Patient age is a limiting factor for ambulatory surgery, due to the risk of post-anesthetic apnea, which occurs most commonly in premature infants, defined as those born before 37 weeks gestation.[7] It is not advisable to perform ambulatory surgery in this population of patients before they reach 50-60 weeks postconceptional age (PCA). Those choosing a cutoff age of 50 weeks postconceptional age may justify their choice based on data by Coté that showed non-anemic, former premature infants were at very low risk of post anesthetic apnea if they were over 50 weeks PCA and did not develop apnea in the Post Anesthetic Care Unit (PACU).[7] My practice is to have the patient remain in the PACU for at least two hours after surgery if they were born before 37 weeks gestation and they are between 50-60 weeks PCA. If no apnea or oxygen desaturation develops during that time, they can be discharged from the PACU. Many institutions also have an age cutoff for ambulatory surgery for infants born after 37 weeks gestational age. We require that these infants are at least 44 weeks PCA and are at least four weeks old. Some states in the USA have established age cutoffs for doing surgery on infants in free standing ambulatory surgery centers.[3]

Obstructive Sleep Apnea Syndrome

Obstructive Sleep Apnea Syndrome (OSAS) is a breathing disorder characterized by frequent, repeated, partial or complete obstruction of breathing during sleep. OSAS is associated with hypercarbia and often with hypoxemia. In the worst cases, it is associated with pulmonary hypertension, right ventricular dysfunction, cor pulmonale, hepatic congestion, and peripheral edema. Patients with pulmonary hypertension should not undergo surgery in an ambulatory setting. The greatest concern regarding allowing OSAS patients to go home after surgery is the fact that both general anesthesia and opioids worsen OSAS. The Society for Ambulatory Anesthesia (SAMBA) published guidelines for adult patients in 2012 stating that ambulatory surgery is safe for adults with OSAS if they use previously prescribed continuous positive airway pressure (CPAP) postoperatively, have optimized comorbidities, have their postoperative pain managed predominately without opioids, and are otherwise good candidates for ambulatory surgery.[8] Guidelines for ambulatory surgery for children with OSAS do not exist, but elements of

the adult guidelines are easily applied to pediatric patients. For example, although children rarely present for surgery while using CPAP, those who do (often older children) should be required to use their CPAP after surgery.

Hypertrophied tonsils and adenoids are a major cause of OSAS or a milder form of sleep disordered breathing in children. The primary treatment for children with OSAS is T&A, and therefore a large percentage of the cases of OSAS encountered by pediatric anesthetists are children undergoing T&A. The majority of T&A procedures, with the exceptions noted above, are done on an ambulatory basis. Removal of hypertrophied tonsils and adenoids typically leads to significant improvement in the severity of the patient's apnea and her/his quality of life, but this cannot be expected to happen immediately after surgery. Postoperative T&A patients may continue to have some airway obstruction for weeks after their surgery. Furthermore, they may continue to have OSAS, especially if their preoperative OSAS was severe and if they are obese.[9,10] Therefore, morbidly obese children, and those with severe OSAS, should have their T&A performed in hospital, not on an ambulatory basis.

Pediatric patients with OSAS may come for ambulatory surgeries other than a T&A. OSAS is present in 1-3% of pediatric patients, so anesthetists should be on the lookout. The highest prevalence of OSAS occurs between 3-6 years of age, corresponding with the peak ages for hyperplasia of lymphoid tissue. The phenotype for OSAS differs in children and adults. Children with OSAS are frequently thin or even undernourished, unlike their obese adult counterparts. The global epidemic of childhood obesity[11] may be contributing to the increasing incidence of an adult type OSAS in pediatric patients.[12] If a child is obese, the anesthetist should be suspicious that he/she may have OSAS. Other physical features of a child that should raise suspicion for OSA include micrognathia, retrognathia, midfacial hypoplasia, and large tonsils. Pediatric medical conditions listed in **Table 18-3** should also prompt suspicion for OSAS. If during preoperative screening it is determined that the patient snores when asleep, he/she may have OSAS. The parents of snoring children should be asked the frequency of snoring and whether the child has night sweating, mouth breathing, and frank obstructive apnea. Affirmative answers to these questions improve the predictive value for OSAS.[13] Children with a high likelihood of having OSAS and who require general anesthesia for surgeries other than T&A, or for imaging studies, should be observed overnight unless preoperative polysomnography shows no OSAS.

Table 18-3: Syndromes and Other Diagnoses Associated with Pediatric OSAS

Syndromes and Sequences	Other Diagnoses
Beckwith Wiedemann syndrome	Achondroplasia
Craniofacial syndromes:	Arnold-Chiari malformation
Apert syndrome	Carney complex
Crouzon syndrome	Cerebral palsy
Pfeiffer syndrome	Choanal stenosis
Down's syndrome	Cleft palate following repair
Goldenhar syndrome	Craniometaphyseal dysplasia
Hallermann-Streiff syndrome	Cystic Hygroma
Klippel-Feil sequence	Hypothyroidism
Marfan syndrome	Myelomeningocele
Mucolipidosis (Sialidosis)	Obesity
Mucopolysaccharidoses:	Osteopetrosis
Hunter syndrome	Papillomatosis
Hurler syndrome	Pharyngeal flap surgery
Morquio syndrome	Sickle cell disease
Scheie syndrome	
Pierre Robin sequence	
Prader Willi syndrome	
Rubenstein-Taybi syndrome	
Schwartz-Jampel syndrome	
Treacher-Collins syndrome	

Syndromes and sequences associated with OSAS taken from Baum and O'Flaherty.[14] Other diagnoses associated with OSAS collected from multiple sources.[14-16]

Congenital Heart Disease, Acquired Pediatric Cardiac Disease, Cardiac Dysrhythmias, and Cardiovascular Implantable Electronic Devices

Children with complex congenital heart disease (CHD) are not candidates for ambulatory surgery. Those with more simple CHD, such as atrial septal or ventricular septal defects, may be reasonable candidates if their congenital heart defects have either closed spontaneously, been surgically repaired, or are hemodynamically insignificant. Generally speaking, children who have had CHD, cardiomyopathy, or myocarditis, are not candidates for ambulatory surgery unless they

no longer require pediatric cardiology follow-up care, except for patients with hemodynamically insignificant CHD.

Children with complex dysrhythmias, such as recurrent supraventricular tachycardia, who have not undergone successful ablation of the abnormal focus causing their tachycardia, are poor candidates for an ASC. Children with Long QT Syndrome should not have surgery in an ASC due to the possibility of anesthesia or surgery causing dangerous ventricular dysrhythmias.

Patients who have an implanted electronic device (CIED), such as a pacemaker (PM) or implanted cardioverter defibrillator (ICD), present a dilemma for anesthetists in ASCs. The Heart Rhythm Society (HRS) and the American Society of Anesthesiologists (ASA) produced a joint expert consensus statement in 2011 on perioperative management of patients with CIEDs.[17] Manipulation of PM or ICD function is generally not performed for patients undergoing surgery in ASCs. The HRS/ASA statement made it easier to preform ambulatory surgery in patients with CIEDs by relaxing requirements for reprogramming PMs and deactivating ICDs. Neither the HRS/ASA statement nor any other consensus statement address CIEDs in pediatric patients. Pediatric patients with CIEDs present unique problems, such as frequent lead malfunction[18] and closer proximity of electrocautery tip to the CIED by virtue of the fact that the patient is smaller. The HRS/ASA recommendation allows *monopolar electrocautery* use for procedures below the umbilicus, even when CIEDs have not been reprogrammed or deactivated. However, even surgery below the umbilicus can bring the cautery unit or its electrical exit path dangerously close to a small child's CIED. In general, children with CIEDs, other than teenagers who are otherwise healthy, are not good candidates for surgery in an ASC. The care of older children with CIEDs requiring surgery at an ASC should follow HRS/ASA guidelines for adults, including preoperative device interrogation and communication between the CIED and perioperative teams.

Diabetes Mellitus

Type 1, insulin-dependent, diabetes mellitus (T1DM), is more common than type 2, noninsulin dependent, diabetes (T2DM) in children; though prevalence of T2DM is on the rise due to the worldwide epidemic of pediatric obesity.[11] Preoperative fasting and stress make perioperative glycemic control challenging, especially if the child has T1DM. Patients with either form of DM can, however, be managed as ambulatory patients. However, the more difficult nature of perioperative glucose management dictates that special precautions are taken, especially in patients with T1DM (outlined in **Table 18-4**). Perioperative management of DM should be coordinated with an endocrinologist if one is available. As noted in **Table 18-4**, patients with T1DM are not good candidates for ambulatory anesthesia if they are very young, or if their blood glucose concentrations are chronically poorly controlled. Hemoglobin A1C is a good indicator of long-term glycemic control. A reasonable target range for hemoglobin A1C for children undergoing surgery in an ASC is 6-8.5% for 5-13 year olds and 6-8% for older children.[19]

Additionally, the child's glucose concentration should be well controlled when he/she presents to the ASC, and he/she should be able to remain in close proximity to a facility that can manage ketoacidosis if it occurs on the night of surgery. Children with DM should have their surgery early in the day (preferably first case) to minimize fasting time and to allow plenty of time for postoperative observation.

Table 18-4

Recommendations for Children with Type 1 DM Undergoing Ambulatory Surgery
Age should be older than 5 years and have minimal other health issues
Surgical procedure should be minor
Baseline glycemic control should be good (Hb A1C < 8-8.5% depending on age)
Glucose control should be adequate on the day of surgery
Fasting period should minimized (place as first case in AM when possible)
Maintain close perioperative glucose monitoring
Administer prophylaxis for postoperative nausea and vomiting
Extend PACU observation time (minimum of 2 hours)
Insure that family is capable of monitoring post-discharge glucose and will keep the child in close proximity to a facility that can manage diabetic ketoacidosis (if it occurs) during the 1st night after surgery

In 2011 the Society for Ambulatory Anesthesia published a consensus statement on perioperative blood glucose management for patients with DM who are undergoing ambulatory surgery.[20] This publication provides perioperative recommendations for administering insulin and non-insulin diabetic drugs. While this document does not specifically address pediatric diabetic management, this author believes that the recommendations can largely be applied to children.

Sickle Cell Disease and Thalassemia

Patients with all types of sickle cell disease (SCD), including those with HbSS, HbSC, and HbS beta thalassemia, have a high incidence of perioperative complications related to sickling of their red blood cells.[21-23] Life threatening complications, such as acute chest syndrome (ACS) and vaso-occlusive crisis (VOC), occur in 5% and 7% of SCD patients respectively, even when they undergo low-risk surgery.[23] The incidence of ACS is 4.2% with umbilical hernia repair and 2.3% with myringotomy tube placement. There is some disagreement about whether patients undergoing minor surgical procedures can have their surgery done in an ambulatory surgery unit. Firth suggests that select patients with SCD can safely undergo minor ambulatory surgery because the reported incidence of serious complications is low and because the need for preoperative transfusion in patients undergoing ambulatory surgery is unproven.[24] Others disagree and would

transfuse the patients preoperatively and keep them in hospital for overnight observation. Decisions regarding preoperative preparation and perioperative care of SCD patients should be made with a hematologist whenever possible. Criteria for excluding SCD patients from undergoing both large and small procedures in an ASC include patients who were hospitalized with VOC in the previous year and patients with pulmonary disease. Both of these conditions increase the risk for perioperative vaso-occlusive complications.[23] Airway procedures, including T&A, should require overnight hospital admission because they are not low risk in patients with SCD.[25] If patients with SCD are released from hospital on the day of surgery, they should remain for several days near a facility that can manage ACS because the average time for patients to present with ACS is three days after surgery.[26] ACS patients that are older than five years of age typically present with cough, fever, and with chest pain.[27] While patients with SCD require many precautions, those who have only sickle *cell trait* can undergo ambulatory surgery without taking any special precautions.

Thalassemias occurs in many variants and is the most common single gene disorder worldwide.[14] Thalassemia minor, the heterozygous form of beta thalassemia, causes mild anemia without any other specific changes that this author believes affect eligibility for ambulatory surgery. Thalassemia major (Cooley anemia) is the homozygous form of beta thalassemia. It manifests as severe anemia and extramedullary hematopoiesis in facial bones; the latter may make tracheal intubation difficult. Patients with thalassemia major may have chronic iron overload from extramedullary destruction of red blood cells and chronic blood transfusion. The iron overload predisposes the patient to cardiac failure, cardiac arrhythmias, and disturbances in myocardial electrical conduction.[14] Patients with thalassemia major should be evaluated for ambulatory surgery on an individual basis, including careful preoperative evaluation, laboratory work and possible assessment by a cardiologist. In general, patients with thalassemia major are poor candidates for ambulatory surgery at an ASC, but can undergo surgery in a hospital based ambulatory surgery setting. They may be appropriately discharged home if their perioperative course in a hospital based ambulatory center was uneventful. Alpha thalassemia with two alpha chain gene deletions causes minor anemia and no special considerations for ambulatory surgery. Alpha thalassemia with three gene deletions (Hemoglobin H Disease) has implications for ambulatory surgery similar to those for Cooley Anemia. Deletion of all four alpha hemoglobin genes is incompatible with life.

Reactive Airway Disease: Asthma and Cystic Fibrosis

In the U.S. asthma is present in 20-25% of patients presenting for surgery[3] and is one of the most common pediatric comorbid conditions. In Africa the incidence is 8-20%. Whether patients with asthma can be cared for in an outpatient setting depends on the severity of their asthma and on how well their asthma is controlled. It also depends on the nature of the outpatient surgical facility. Patients who have required frequent or recent hospital admissions for their asthma (especially during the previous three months) and those who required admission to an ICU

because of their asthma are not good candidates for surgery in an ASC[3] but could be discharged home after an uneventful perioperative ambulatory surgery in a hospital setting. Well-controlled, mild, or moderate asthma usually does not prevent patients from having ambulatory surgery.

Cystic fibrosis (CF) manifests itself with a range of pulmonary and gastrointestinal diseases and with variable degrees of airway reactivity. Patients with CF may be appropriate candidates for minor ambulatory surgical procedures in a freestanding ASC, but they have a relatively high risk for being admitted to hospital after more complicated and prolonged procedures (e.g., extensive endoscopic sinus surgery).[28] Careful consideration should be given to the extent of the patient's reactive airway disease. Patients with more severe airway reactivity and those with exacerbations of their pulmonary disease probably should not undergo surgery in a freestanding ASC.

Malignant Hyperthermia Susceptibility

Patients with a family history of malignant hyperthermia (MH) or children who are thought to have had MH in the past, may have ambulatory surgery at a free standing ASC if the anesthetist can provide a trigger free anesthetic and respond appropriately to a MH event. Agents that trigger MH include: Halothane, Enflurane, Isoflurane, Desflurane, Methoxyflurane, Cyclopropane, Sevoflurane, Ether, and Succinylcholine. The Malignant Hyperthermia Association of the United States (MHAUS) (http://www.mhaus.org) is an excellent source of information about MH and its treatment. Their website and their telephone "hotline" can be consulted whenever there is concern for MH. MHAUS also has a very useful smartphone application for management of MH that is downloadable from the *iTunes app store* for a small fee.

Most patients who develop MH are phenotypically normal and have no history of susceptibility to MH. Freestanding ASCs that administer agents that can trigger MH (whether MH susceptible or not) must have the ability to respond appropriately to MH if it occurs. The ability to appropriately respond to MH includes having all necessary medications and equipment immediately available, maintaining knowledge and skills regarding how to manage a MH crisis, and having a plan in place for transfer of patients with MH to a receiving hospital. Dantrolene is the specific treatment for MH, and must be available in a quantity needed to treat MH in an adult. Low resource nations may choose to share a Dantrolene supply by storing it in a centrally located hospital in order to lower cost and manage difficulties locating Dantrolene when outside of large cities. However, it is worth noting that MHAUS recommends that 36 vials be available within 10 minutes of the decision to treat MH. Yearly practice drills on the diagnosis and treatment of MH are a good way to maintain knowledge and skills that allow the operating room team to respond quickly and appropriately to MH. Each ASC should partner with a particular receiving hospital that will accept patients having a MH crisis. The Ambulatory Surgery Foundation and MHAUS recently developed a guide for transfer of MH patients from an ASC to a receiving hospital. These guidelines are meant to aid ASCs that want to create their own specific plan.[29]

Administering a trigger free anesthetic requires the anesthetist to have familiarity with procedures for eliminating volatile anesthetic from the specific anesthetic machine that will be used. Anesthesia machines differ in the procedure and the duration required to purge the very small amounts of volatile anesthetics known to trigger MH. Some anesthesia machines require two or more hours to adequately purge volatile anesthetics if charcoal filters are not available.[30] Anesthetic machine manufacturers typically publish guidelines that describe the best way to prepare their machine for a trigger free anesthetic. Patients susceptible to MH should have their surgery early in the day to allow for observation of the patient for two hours after surgery, as recommended by MHAUS.

Neurologic and Neuromuscular Disease: Myopathy, Cerebral Palsy and Seizure Disorder

Children with known muscle dystrophies and myopathies should have their surgical care in a hospital for the following reasons. Patients with Duchenne muscular dystrophy and those with many other myopathies have weak respiratory muscles and impaired cough, which makes them particularly susceptible to the negative respiratory effects caused by general anesthesia.[31] This leads to problems with postoperative gas exchange. Numerous forms of myopathies are also associated with cardiomyopathy and disturbances in cardiac electrical conduction. Mitochondrial myopathies are associated with a wide variety of conditions, including increased sensitivity to anesthetics, compromised renal or hepatic function, lactic academia, and glucose instability.[32] Some patients with mitochondrial myopathy do not tolerate normal preoperative fasting protocols because even small amounts of dehydration and reduced intravascular volume significantly affect their myocardial function.

Children with early myopathic symptoms may receive diagnostic muscle biopsy in an ASC. These children should be carefully screened for serious respiratory and cardiac symptoms. Particular attention must be paid to respiratory function and the other medical issues noted above. Volatile anesthetics are often used for anesthetic induction and maintenance of anesthesia for these patients; however, use of total intravenous anesthesia (TIVA) may offer advantages. By administering TIVA, volatile anesthetics are avoided, and the chance of needing succinylcholine to treat laryngospasm on induction is smaller. This may decrease the likelihood of a hyperkalemic crisis if the patient has an undiagnosed muscle dystrophy, and of MH if the patient has a myopathy that predisposes them to MH, such as Central Core Disease or Multi-Minicore Disease.[33]

Children with mild cerebral palsy (CP) are reasonable candidates for ambulatory surgery, including surgery at a freestanding ASC, so long as other comorbid conditions do not cause excessive risk. As noted in **Table 18-3**, children with CP are predisposed to obstructive sleep apnea syndrome. If airway surgery is planned (including T&A), even patients with mild CP should remain in hospital

overnight. Children with moderate to severe CP typically have weak respiratory muscles, scoliosis and restrictive lung disease, seizure disorders, and other comorbid conditions. Their surgical care should take place in a hospital, and they should remain in hospital overnight following general anesthesia.

Seizure disorders are common, but children who have seizures are eligible for ambulatory surgery in freestanding centers if their seizures are well controlled. Efforts should be made to continue their anticonvulsant medications throughout the perioperative period, though this can be a challenge in children who refuse to take medication without food. Intravenous formulations of anticonvulsants, if available, should be substituted for oral formulations when the anticonvulsant drug cannot be given orally.

Difficult Tracheal Intubation

Children who have a history of a difficult intubation or an airway examination that indicates a high likelihood of a difficult intubation are usually managed in a hospital. The determination of where to manage these children must be made on an individual basis, taking into consideration each ASC's availability of airway equipment, airway expertise, and capability to manage a situation where one cannot intubate and cannot ventilate (**See Chapter 4**). The latter depends on ability to create a surgical airway, and manage the aftermath and transfer of a patient when complications develop. An additional consideration is the extra time involved in securing a difficult airway, which impacts the efficiency of the ASC. There are no guidelines that indicate what airway equipment should be available in an ASC. ASCs usually do not have as full an assortment of airway equipment as a hospital operating room, but should keep on hand equipment for management of unexpected difficult tracheal intubations. Examples of emergency airway equipment that can be affordable options for resource poor nations include gum elastic bougies, non-disposable lighted stylets, and disposable videolarygoscopes..

Preoperative Management

Ambulatory surgery centers have gained popularity, in part, because they provide efficient service for patients and surgeons. Low cancellation rates on the day of surgery and rapid patient throughput are cornerstones of ASC efficiency. This section discusses preoperative patient evaluation and preparation because these help avoid delays and cancellations of cases on the day of surgery. Upper respiratory tract infection (URI) is one of the leading causes of cancellation of ambulatory pediatric cases on the day of surgery. This section therefore includes a discussion of the common preoperative dilemmas of whether to cancel elective surgery when a child has a URI.

Preoperative Evaluation and Preparation

Pre-anesthesia evaluation of ambulatory surgery patients may take place in a pre-anesthesia evaluation clinic (PAEC), by phone, or may first occur on the day of surgery. Most children coming for ambulatory surgery are healthy and can therefore be evaluated with a telephone call or by a visit on the day of surgery. Patients with comorbid conditions may need to be evaluated in a PAEC that is staffed by individuals who are familiar with anesthetic concerns. That visit can help determine whether the patient is appropriate for the planned ambulatory surgery. ASCs can benefit from creation of predetermined criteria that specify for surgeons which comorbid conditions require a visit to the PAEC and which conditions completely exclude them from having ambulatory surgery. A patient should visit the PAEC whenever there is any doubt regarding whether they are appropriate candidates for the planned ambulatory surgery. No matter where the pre-anesthesia evaluation takes place, it is useful to have the family complete an intake questionnaire that includes key elements of the patient history. Components of the ambulatory pre-anesthetic history and physical examination are the same as for inpatients.

Preoperative anesthesia evaluation prior to the day of surgery can avoid day of surgery surprises, save time on the day of surgery, and help patients to prepare psychologically for their surgery. Rules for fasting, discussed in detail below, are conveyed to patients and families. Necessary discussions with the patient and family can take place in advance, therefore saving time on the day of surgery. And, in-person visits offer an opportunity to psychologically prepare the child and family for the surgery. Psychological preparation was reported to be as effective as oral midazolam for providing a calm induction of anesthesia, and had the additional benefits of reducing emergence delirium, PACU analgesic requirements, and time spent in the PACU.[34] Two of the most important elements in that preparation were teaching parents ways to distract the child and encouraging them to practice with the child at home with a disposable anesthetic mask.[34]

Preoperative laboratory testing, if indicated, can be obtained at a PAEC or at the preoperative visit with the surgeon. Routine preoperative laboratory testing is not needed for healthy children undergoing standard ambulatory surgical procedures, but may be indicated according to a patient's comorbid conditions. For example, geographic areas with prevalent SCD should routinely screen for HbS in patients who have not previously undergone that screening. Criteria for preoperative laboratory testing are best developed by each center, based on local resources and on endemic diseases in each ASC's particular geographic region. Each ASC should develop a policy on pregnancy testing for menstruating females who will undergo general anesthesia or sedation. Opinions expressed by an ASA Joint Task Force on Pregnancy Testing and in the ASA Practice Advisory for Pre-anesthesia Evaluation suggest that every menstruating patient and her family should be offered a pregnancy test, but should not forced to have one.[35] Nonetheless, ASCs in some countries may decide not to offer a pregnancy test to every menstruating patient and her

family, and the decision to do so should depend on local prevalence of teenage pregnancy, medical legal considerations, and cultural mores. Unconsented pregnancy testing is *not ethical* when dealing with competent patients. Pregnancy testing may be done on the day of surgery with an inexpensive urine dipstick, and should be done as close as possible to the day of surgery.

All families should receive a preoperative phone call 1-2 days prior to the ambulatory surgery, even if their child has already undergone evaluation in the PAEC or by phone. The phone call 1-2 days before surgery can serve as a final check that the patient does not have any comorbid condition that would exclude them from ambulatory surgery. The phone call also serves as an important reminder of the surgical appointment, provides details regarding the facility location, gives a contact phone number in case the family needs to get in touch with the facility, and provides the following crucial information. Families should be informed of the need for a legal guardian to accompany the patient if signatures for consent for surgery and anesthesia will be required. They should be told to arrive 1 - 2 hours prior to the actual scheduled surgery time, which insures adequate time for patient preparation and allows an earlier start to surgery if the schedule permits. Instructions on whether or not to take medications should be given. Rules for preoperative fasting must be clearly conveyed to the patient and family during the preoperative telephone call, as NPO violations are a leading cause of case cancelation on the day of surgery. Our institution endorses the ASA's Practice Guidelines for Preoperative Fasting rules (**Table 18-5**).[36] It may be appropriate to add 1.5 hours to those fasting times (i.e., 3.5 hours for clear liquids) to allow flexibility if a patient's surgery time can be moved forward in the schedule. Clear liquids consist of water, electrolyte solutions, and fruit juices without pulp. They also include clear tea, black coffee, and carbonated beverages. Confusion and errors may occur if patients are given too many options for clear liquids. The preoperative phone call is also an opportunity to convey that patients should wait 36 hours after a general anesthetic before doing potentially dangerous activities that require coordination and/or judgment such as driving a vehicle or riding a bicycle.

Table 18-5: Minimum Fasting Periods for Healthy Patients of All Ages Undergoing Elective Surgery (adapted from ASA Guidelines[36])

Type of feeding	Minimum Fasting Time
Clear Liquid	2
Breast Milk	4
Infant Formula	6
Non-Human Milk	6
Light Meal*	6

**A light meal is considered to be a small amount of toast, plain rice, fruit, together with clear liquids, and without fried or other types of fatty food.*

The Child with a Upper Respiratory Tract Infection

Every day, pediatric anesthetists encounter children who present for surgery with respiratory infections. The dilemma of whether to cancel these infected patients is particularly common in ASCs, due to the sheer volume of patients they serve. Lower respiratory tract infections pose clear-cut contraindications for elective surgery. Wheezing or rales heard on auscultation of the chest imply bronchiolitis or pneumonia and should prompt cancellation of the surgery and referral of the patient to a pediatrician or family practitioner. Additionally, a child who is febrile or one who has a febrile illness and arrives for elective surgery on antipyretics, is best served by rescheduling surgery. A possible exception to cancellation of surgery for a child who is febrile would be the child who requires drainage of an infected area, such as chronic purulent otitis media or sinusitis. However, the anesthetist must be confident regarding the source of the child's fever; proceeding with surgery in a child who has fever that is part of an undiagnosed serious illness, may result in serious complications.

Pediatric viral upper respiratory tract infections (URI) are a leading cause for cancellation of cases on the day of surgery. Children under six years of age have 6-8 viral URIs every year; the number is higher for those <2 year of age and for those who spend time in day care with many other children.[37] Prior to the 1980s, anesthetists were taught to cancel elective surgery for children with URIs, mainly due to concern about possible intraoperative or postoperative respiratory complications.[38] Children who have URIs at the time of their anesthesia, and those who have had a URI up to 2 weeks before, are know to have increased risk for developing respiratory complications.[39] Some data imply that this increased risk persists for up to four weeks after a URI.[40] The risk of intraoperative bronchospasm nearly doubles and that of laryngospasm doubles or triples when a child with an active URI undergoes anesthesia and surgery.[39,41,42] However, Schreiner estimated that surgery would have to be cancelled in 130 children with URIs to prevent one laryngospasm, and that 8,000 cases would need to be cancelled in these same patients to prevent one unexpected overnight hospitalization for laryngospasm.[41] Pediatric anesthetists now recognize the additional risk of anesthetizing a child with a URI, but are far from cancelling all elective surgery for every child with a URI. Many factors go into determining when to cancel a child with a URI, including the anesthetist's comfort in managing complications, and these factors are discussed in detail below.

Viral URIs cause differing severities of disease, and occur in patients who are receiving numerous different types of surgery and who have many different comorbid conditions. Therefore, there are many possible combinations of URI severity, surgery type, and comorbid condition, and often no clear-cut answer for whether to proceed with surgery or reschedule it. Tait and his colleagues hoped to clarify the situation by identifying independent risk factors for adverse respiratory events in children with active URIs (**See Table 18-6**).[40]

Table18- 6

Independent Risk Factors for Adverse Respiratory Events in Children with Active Upper Respiratory Tract Infections
Copious secretions (e.g. productive cough or copious nasal secretions)
Need for a tracheal tube in a child under 5 years of age
History of prematurity (born at <37 weeks gestational age)
Nasal congestion
Parental smoking
History of reactive airway disease
Airway surgery

Once preoperative screening identifies a patient with an URI, anesthetists can use Tait's risk factors listed in **Table 18-6** to help identify the overall risk of proceeding. Tait's risk factors are helpful, but it is important to keep in mind that they mainly represent the least serious of respiratory events, such as coughing, breath holding, and oxygen desaturation to <90%. Those events occurred much more frequently than laryngospasm requiring succinylcholine (2.2%) and bronchospasm (5.7%). Comorbid conditions other than those on Tait's list (especially those related to the pulmonary system), whether the ambulatory surgery is urgent, and how difficult it will be to find a time when the child will be free of a URI for at least two weeks before surgery must all be considered. Patient safety should be paramount, but often factors unrelated to the patient come into play. These include how far the family traveled for the surgery, whether the patient's surgery has been cancelled before, and whether family members have taken time off from work to take the child to the hospital or ASC, The decision to proceed or postpone surgery must also take into consideration the anesthetist's comfort level and ability to manage predictable complications such as laryngospasm and bronchospasm. Generally, parents and surgeons can help with the decision to proceed or not to proceed after a brief discussion of potential complications and increased risk.

While the vast majority of children with URI's coming for ambulatory surgery can be safely anesthetized, it is difficult to predict which ones will have serious complications. If one proceeds with anesthesia and surgery in a patient with a URI, then it is important to avoid drugs that increase the risk of bronchospasm or laryngospasm, (e.g., thiopental, desflurane). A LMA or mask airway should be used rather than a tracheal tube, whenever appropriate, to avoid the increased risk of bronchospasm associated with tracheal intubation of patients with a URI.[43]

Anesthetic Management

Management of an anesthetic for ambulatory surgery is not much different from that for inpatient surgery. Details regarding anesthetic management can be found in appropriate locations throughout this textbook, including Chapter 7. Plans for treating pre-anesthetic anxiolysis should

be the same for patients coming for ambulatory surgery as they would be for all other children coming for surgery, and should include consideration for giving pre-anesthetic sedative medications (midazolam, clonidine, ketamine), the use of distraction techniques, and having a parent present during induction of anesthesia. If the parents will be present during the induction of anesthesia, they should be warned that the child may struggle, and is expected to demonstrate many features that differ from falling asleep at home, including involuntary movements, roaming or twitching eye movements, noisy breathing, abnormal breathing patterns, and body stiffening or sudden relaxation.

The ambulatory anesthetist must pay meticulous attention to prophylaxis for both postoperative nausea and vomiting (PONV) and emergence delirium (ED), and to provision of effective intra and postoperative pain management. Those elements are key for efficient throughput and quality care in the ambulatory surgery environment. Prophylaxis for PONV and ED are discussed individually in this section, while perioperative pain management is discussed subsequently in its own section and in **Chapter 20**.

Post Operative Nausea and Vomiting Prophylaxis

Postoperative nausea and vomiting (PONV) is ranked by adult patients as one of the most discomforting postoperative symptoms. PONV is also a leading cause of unanticipated hospital admissions for both children and adults who undergo ambulatory surgery.[44,45] Having PONV often increases the amount of time required for patients to recover from anesthesia, requires greater PACU nursing support, and causes patient dissatisfaction.[44] Data for children are usually limited to postoperative vomiting (POV) because young children are frequently unable to describe and report nausea.

Fortunately, prophylaxis for pediatric POV is highly effective. Single administration of either a 5-hydroxytryptamine-receptor antagonist or dexamethasone reduces relative risk (RR) of POV by 50-60%.[46] Droperidol alone decreases the relative risk of PONV by 40%,[46] but many anesthetists are reluctant to use it because of associated ventricular dysrhythmias. However, droperidol has rarely, if ever, caused this problem when administered in a dose of 10-15mcg/kg that is usually recommended for POV prophylaxis. The reductions in relative risk mentioned above compare favorably to reductions of just 25-26% in PONV relative risk seen in adults who receive single administration of either ondansetron, dexamethasone, or droperidol.[47] Children that receive prophylaxis with both dexamethasone and a 5-hydroxytryptamine receptor antagonist have an 80% reduction in the risk of POV,[46 making] it unnecessary to prophylax with additional drugs.

The incidence of POV in children is twice that of adults,[44] but not all pediatric patients require prophylaxis. A specific plan for POV prophylaxis should be created for each child based on his/her specific risk. Many pediatric risk factors are the same as for adults, including the use of nitrous oxide, the use of perioperative narcotics, a history of POV, the use of volatile anesthetics instead

of propofol, and the duration of surgery.[44,48] Strabismus repair is know to increase risk for POV.[48] Other specific pediatric procedures, including T&A, middle ear surgery, orchidopexy, hernia repair, and penile surgeries are believed to increase risk,[44] although evidence is lacking. Children less than two years of age were once believed to seldom develop POV, but that is not the case.[49] The risk of POV increases up to puberty and then decreases. Females become more likely than males to have POV only after puberty.[44] A simple risk assessment scale for pediatric POV has been developed by Eberhart and colleagues, and incorporates four risk factors that they found to correlate strongly with developing POV (**Table 18-7).** When their patients had 0, 1, 2, 3, and 4 of those risk factors, the incidence of POV was 9%, 10%, 30%, 55%, and 70%.

Table18-7

Pediatric Postoperative Vomiting (POV) Risk Factors
Eberhart's POV Risk Factors:[48] • **Strabismus surgery** • **Age of ≥3 years** • **Duration of surgery greater than 30 minutes** • **History of POV in patient, parent or sibling**
Risk Factors not included in Eberhart's list:[44] • **Nitrous oxide** • **Volatile anesthetic use versus propofol** • **Perioperative narcotics** • **Specific surgeries other than strabismus repair: T&A, orchidopexy, hernia repair, penile surgery, and middle ear surgery**

Eberhart's scale provides an easy way to approximate POV risk, and therefore facilitates decision making for POV prophylaxis. Patients with zero or one risk factor have a baseline risk of approximately 10%, so that single drug prophylaxis produces minimal benefit by decreasing risk to near 5%; the number needed to treat (NNT) to avoid a single case of POV would be near 20. Patients with two risk factors have a baseline risk of approximately 30% that decreases to near 15% after single drug prophylaxis (NNT ≈ 6), and marginally further (to near 6%) by adding a second drug. Children with three or four of Eberhart's risk factors may benefit from giving more than one drug for prophylaxis. The first drug will decrease the incidence of POV by 27-35% (NNT of 3 - 4) and the second will decrease it by an additional 16-21% (NNT 5 - 6). As noted above, risk factors other than those found by Eberhart might also require consideration.

When using a single drug for prophylaxis, anesthetists should consider that dexamethasone 0.05 - 0.15mg/kg (maximum of 4 mg for the purpose of POV prophylaxis) prevents POV as well as 5-hydroxytryptamine receptor antagonists and that dexamethasone is not an effective rescue

medication. Thus, dexamethasone is a logical first choice for POV prophylaxis. Ondansetron 0.05 - 0.1mg/kg (maximum of 4 mg) can be added if needed, or saved for rescue. Droperidol 10 - 15mcg/kg may be used to prevent or treat POV, but the anesthetist must take into account the need for post-administration cardiac monitoring.

Adequate hydration has been shown to reduce POV/PONV for both pediatric and adult patients.[44,50] Children 1-12 years of age that underwent strabismus repair and received no PONV prophylaxis, had a PONV incidence of 22% when they received 30ml/kg of lactated Ringers solution versus 54% after 10ml/kg.[50] The more generous hydration (30ml/kg) also reduced postoperative fever and thirst,[50] and may allow more time before patients who develop protracted POV become dehydrated.

Emergence Delirium Prophylaxis

Emergence delirium (ED), also referred to as emergence agitation, frequently occurs in children emerging from volatile anesthetics. Non-purposeful thrashing movements, uncontrollable crying, failure to make eye contact, and non-responsiveness are features that characterize ED.[51] Children with ED do not recognize familiar objects or persons. ED puts patients at risk of injuring themselves, dislodging their intravenous lines, damaging their surgical site, and may lead to a need for increased nursing support and additional recovery time. Parents who witness ED in their child may incorrectly conclude that the anesthetic was conducted improperly, may experience excessive stress, and may worry about permanent sequelae.[52]

Risk factors for ED are not as clearly delineated as those for PONV. Younger age, most notably the preschool age group, appears to have increase risk of ED,[53,54] although the potential for self injury, or injury to staff, is greatest when ED occurs in older, stronger children. Preoperative anxiety has been shown in two studies to elevate risk of ED,[55,56] but a large observational study found no relationship with the quality of separation or induction.[54] Otolaryngology procedures carry a higher risk of ED than other procedures, with a RR of 1.7.[54] Children that receive intravenous anesthesia with propofol have a very low incidence of ED.[57]

Prophylaxis for ED is highly effective, and pediatric anesthetists working in ambulatory settings should consider prophylaxis for all children who have received volatile anesthesia as their main anesthetic. Inadequately controlled pain contributes to agitated emergence from anesthesia.[58] Prevention of ED should involve strategies for insuring proper analgesia, including regional blocks, ketorolac, acetaminophen, and narcotics. Narcotics appear to have a prophylactic effect apart from their analgesic effect, as demonstrated by their ability to decrease the incidence of ED in cases where no surgery was performed; Cravero reported that the incidence of ED was decreased from 56%-to-12% by administering 1µcg/kg of fentanyl 10 minutes before emergence from sevoflurane.[59] Alpha-2 adrenergic agonists are highly effective prophylactic agents; dexmedetomidine 1µcg/kg was shown to decrease the incidence of ED from 33%-to-0% after

sevoflurane anesthesia for MRI scans, with dexmedetomidine given an average of 45 minutes prior to discontinuation of anesthesia.[60] Doses of dexmedetomidine as low as 0.15µcg/kg have also been shown to provide effective prophylaxis of ED, though not as effective as higher doses, and may be given closer to the time of emergence with less concern for delayed awakening. Clonidine 4µcg/kg given orally resulted in only 25% agitation in patients emerging from sevoflurane, compared with 60% in those that received oral midazolam 0.5mg/kg, both premedicants were given 30 minutes prior to induction.[61] Intravenous clonidine 2µcg/kg given just prior to the start of surgery reduced baseline risk of emergence agitation from 33%-to-14%, and severe emergence agitation from 10%-to-3%.[62] Propofol is also a useful prophylactic agent for ED; propofol decreased severe ED from 27%-to-5% when given intravenously in a dose of 1mg/kg at the time of discontinuation of sevoflurane.[63] Ketamine 0.25mg/kg given intravenously 10 minutes before the end of surgery decreased ED from 34%-to-17%,[64] and intravenous nalbuphine 0.1mg/kg has been shown to be a more effective prophylactic agent than the above dose of ketamine when given at the end of the surgical procedure.[65] A summary of useful prophylactic agents is given in **Table 18-8**. Agents used to treat ED include fentanyl, propofol, dexmedetomidine, and midazolam.

Table 18-8: Medications Commonly Used for Prophylaxis of Emergence Delirium in Pediatric Ambulatory Anesthesia

Medication	Route	Dosage	Timing
Fentanyl	IV	1µcg/kg	10 min PTE
Dexmedetomidine	IV	1µcg/kg	45-60 min PTE
		0.5µcg/kg	30 min PTE
Ketamine	IV	0.25mg/kg	10 min PTE
Clonidine	PO	4µcg/kg	Oral premed*
	IV	2µcg/kg	Post induction
Propofol	IV	1mg/kg	End of procedure
Nalbuphine	IV	0.1mg/kg	End of procedure

PTE = Prior to emergence
**Given 30 minutes prior to induction of anesthesia*

Perioperative Analgesia

Effective perioperative pain management is especially important in pediatric ambulatory anesthesia. Parent and patient satisfaction, and ability to efficiently move patients through the recovery process depend on successful perioperative analgesia. Inadequate postoperative pain control was the leading cause of unplanned hospital admission in a retrospective cohort of over ten thousand pediatric ambulatory surgeries.[45] Postoperative pain may play a role in problematic postoperative behavioral changes observed in nearly half of all children receiving surgery.[66]

Despite these serious consequences, parents often undertreat their child's pain after ambulatory surgery.[67,68]

Multimodal analgesia consists of administering two or more analgesic drugs acting by different mechanisms,[69] and is widely promoted for ambulatory anesthesia.[70] Multimodal strategies include combinations of opioids, non-steroidal anti-inflammatory drugs (NSAIDs), acetaminophen, local anesthetics (including regional anesthesia), ketamine, dexamethasone, alpha-2 adrenergic agonists, and gabapentinoids. Opioid analgesics remain an important component of perioperative regimens, but cause nausea, vomiting, pruritus, urinary retention, and impaired bowel function in some patients. Use of non-opioid analgesics, especially in combination, may allow opioids to be reserved for rescue analgesia. The ASA Task Force on Acute Pain Management recently updated their Practice Guidelines for Acute Pain Management in the Perioperative Setting, noting that consultants and ASA members "strongly agreed" that acetaminophen should be considered for multimodal strategies unless contraindicated. Additionally, they "agreed" that nonselective NSAIDs, COX-2 selective NSAIDs (COXIBs), and calcium channel alpha-2-delta antagonists (pregabalin and gabapentin) should also be considered unless contraindicated.[69] Those guidelines, which appear more geared for adults and for inpatients, "strongly recommend" around-the-clock (ATC) dosing of acetaminophen and NSAIDS/COX-2 inhibitors. Others note a lack of evidence for ATC dosing after ambulatory surgery. That decision should take into consideration surgery type, patient factors, risk factors of the particular drug, as well as difficulties administering nighttime doses, problems swallowing after T&A, and patient refusal of medication.[68] Analgesic medications and doses commonly used in pediatric ambulatory anesthesia are listed in **Table 18-9**.

Table 18-9: Analgesic Medications and Doses Commonly Used in Pediatric Ambulatory Anesthesia

Medication	Class	Route	Single Dosage [Maximum]
Fentanyl	Opioid	IV	0.5-1.0µcg/kg*
Morphine	Opioid	IV	0.025-0.05mg/kg*
Hydromorphone	Opioid	IV	3-8µcg/kg*
Tramadol[§]	Atypical Opioid	PO PR IV	1-2mg/kg [100mg] 1-2mg/kg [100mg] 1-2mg/kg [100mg]
Oxycodone	Opioid	PO	0.05-0.1mg/kg [10mg]
Codeine	Opioid	PO	0.5mg/kg [30mg]
Hydrocodone	Opioid	PO	0.1-0.15mg/kg [10mg]
Acetaminophen[§]	Para-aminophenol derivative	PO PR IV	10-20mg/kg [1 gm] 30-45mg/kg [1.3gm] 7.5-15mg/kg** [1gm]
Ketorolac	NS NSAID	IV/IM	0.5-0.75mg/kg [30gm]
Metamizole	NS NSAID	IV/IM PO	25-40mg/kg*** [1gm] 15/mg/kg [1gm]
Ibuprofen	NS NSAID	PO	5-10mg/kg [600mg]
Celecoxib[§]	COXIB	PO	250mg/meter^2 [400 mg]
Paracoxib	COXIB	IV	1mg/kg **** [40 mg]

IV = Intravenous; IM = Intramuscular; PO = Per Os; PR = Per Rectum; PRN = As needed; NSAID = Non selective non-steroidal anti-inflammatory drug; COXIB = Selective COX-2 inhibitor

** Titrate to effect and dose according to lean body mass to avoid overdosing*

*** Acetaminophen IV is licensed in the UK at a dose of 7.5 mg/kg (maximum of 30/mg/kg/day) for term neonates and infants up to one year of age, and at a dose of 15 mg/kg (maximum of 60mg/kg/day) for children over one year.*[71]

**** Only a single IV/IM bolus at this dose; subsequent IV/IM doses of 15 mg/kg Q8 hours. Administer by slow IV infusion over 15 minutes or more to avoid hypotension. IV administration not recommended for children <1 year old due to potential for hypotension.*

***** Recommended for children >2 years of age and over 10kg.*[72]

[§]Medications that may be given preoperatively in oral formulation as part of a multimodal strategy

Acetaminophen is typically included in pediatric perioperative multimodal strategies, and is frequently used at home postoperatively either ATC or as needed. It may be given orally, per rectum, or intravenously. Peak plasma levels are achieved 30 - 60 minutes after oral administration.[73] Acetaminophen 40mg/kg per rectum (PR) produces therapeutic plasma levels at 30 - 45 minutes and peak plasma levels at 2 - 3 hours after administration.[74,75] Analgesic effect depends on establishing cerebrospinal fluid levels, which occurs approximately one hour after plasma levels are established.[73] Rectal acetaminophen has a relative bioavailability of 80% compared to oral formulations, has erratic absorption and does not produce consistent therapeutic blood levels of the drug.[73,75] Nonetheless, rectal acetaminophen is widely available, cost effective, and has been demonstrated to work. Acetaminophen 40mg/kg PR administered shortly after anesthetic induction produced opioid sparing effects in the recovery room and during the first 24 hours after surgery following a variety of pediatric day-case surgeries, and also led to a substantial reduction in POV.[76] Subsequent doses of rectal acetaminophen must be decreased to 10-20 mg/kg every 6-8 hours after giving the initial large bolus noted above, and one must adhere to recommended daily maximum doses to avoid toxicity. Daily maximum doses for orally and rectally administered acetaminophen depend on age. The British National Formulary recommends a daily maximum dose for oral or rectal acetaminophen of 60mg/kg for term infants <3 months old. They recommend that infants >3 months of age receive a daily maximum of 90mg/kg for the first 48 hours and 60mg/kg thereafter. The US Food and Drug Administration (US FDA) recommends only 60mg/kg/day for children 1-2 years of age, and a daily maximum of 75mg/kg thereafter. Intravenous acetaminophen produces a rapid therapeutic level.[71] It is licensed in the UK at a dose of 15 mg/kg (every 6 hours, with a maximum of 60mg/kg/day) for children over one year old and at a dose of 7.5 mg/kg (every six hours, with a maximum of 30/mg/kg/day) for term neonates and infants up to one year of age. Pharmacokinetic studies suggest that infants 1 - 12 months of age may receive the same dosing as older children, and it is no surprise that a large percentage of UK pediatric anesthetists practice accordingly.[71] The US FDA has not approved IV acetaminophen for children under two years old. An epidemiologic association has been found between acetaminophen use and asthma prevalence and severity in children and adults. Some advocate avoidance of acetaminophen for children with asthma,[77] but no data exists regarding perioperative use and associated bronchospasm.

NSAIDs are amongst the most commonly used analgesic agents in ambulatory anesthesia and may be effective as a single agent when pain is mild or moderate. NSAIDs typically have anti-inflammatory properties that may also reduce edema. Ketorolac is a nonselective NSAID available in IV formulation, commonly administered in a dose of 0.5mg/kg IV or intramuscularly (IM). It has been advocated for numerous pediatric ambulatory surgeries, including as an equianalgesic intraoperative substitute for morphine in pediatric strabismus surgery.[78] Some studies demonstrate a reduction in opioid side effects when Ketorolac or other NSAIDS are used.[66,78] Ketorolac should not be given to patients having T&A because there is an association between its

perioperative use and post-T&A bleeding requiring surgical intervention (OR = 3.82, 95% confidence interval = 1.03-14.1).[79] Metamizole (also known as dipyrone) is a nonselective NSAID available in PO and IV/IM formulations. It is effective as both an analgesic and antipyretic but not as an anti-inflammatory agent.

Metamizole exerts its analgesic effect by central mechanisms and weak cyclooxygenase (COX) inhibition, and is well tolerated by the gastric mucosa.[80] It does diminish platelet aggregation, compared to a selective COX-2 inhibitor.[81] Metamizole is a popular pediatric postoperative analgesic worldwide, including in India, Indonesia, Thailand, and in many Latin American and European countries. Numerous countries (including the USA) removed it from the market due to an association with the potentially fatal complication of agranulocytosis, although the estimated risk is very low. Adult studies of single dose metamizole for postoperative pain show that 500mg PO is as effective as ibuprofen 400mg, and 2.5gm IV is as effective as Tramadol 100mg IV.[82] There is limited evidence for its use in pediatrics or in ambulatory surgery. Metamizole may cause hypotension when administered by rapid IV infusion, especially in infants. IV administration is best reserved for children >1 year of age. This drug should be slowly infused over at least 20 minutes. Recommended doses are listed in **Table 18-9**.

COXIBs provide similar analgesia to non-selective NSAIDS, with fewer concerns for inhibition of platelet function.[66,68] Celecoxib is a COX-2 inhibitor available for oral administration. It has been an effective component of multimodal pain regimens for adult ambulatory surgery,[83] but similar studies for children have not been performed. A pharmacokinetic study found that pediatric dosing of celecoxib 250mg/meter^2 of body surface area produced serum drug levels comparable to the accepted adult dosing of 400mg, and children metabolized the drug at twice the rate of adults.[84] No commercially available pediatric oral formulation of a COXIB exists. Parecoxib, an injectable precursor of its active metabolite valdecoxib, is the sole IV COXIB formulation. It appears to be available almost exclusively in the UK. Pharmacokinetic data for children 2 - 12 years old indicate that a parecoxib dose of 1mg/kg IV produces valdecoxib levels similar to adults given the accepted adult dose of 40mg/kg. Analgesic efficacy of parecoxib in children is unknown, and pharmacokinetic data for children <2 years old do not exist.[72]

Opioids are commonly used in pediatric ambulatory surgery, both intra- and postoperatively. They are indicated for severe pain, or for moderate pain when NSAIDs are contraindicated. Intraoperative opioids should be titrated to effect, using particularly small doses when dealing with patients who have, or may have OSAS. Commonly used intravenous opioids include fentanyl, hydromorphone, and morphine. Postoperative orally administered opioids commonly used include oxycodone, hydrocodone, and codeine, given alone or in formulations that combine acetaminophen. These medications are best given to children on an as needed basis (no sooner than every 6 hours) rather than ATC. Doses for both intravenous and orally administered opioids are given in **Table 18-9**. Dosing of opioids should be based on lean body weight rather than actual

weight to avoid overdosing obese children. Codeine is a pro-drug and depends on its metabolism to morphine by the liver. Ultra-fast metabolizers of codeine may develop dangerously higher morphine levels, and codeine is therefore a poor choice for postoperative opioid analgesia. Deaths have occurred postoperatively in children with OSA who received codeine after T&A, causing the US FDA to issue a recent black box warning and contraindication statement on use of codeine for postoperative pain management in children following tonsillectomy and/or adenoidectomy. Patients who receive intraoperative acetaminophen and who are prescribed postoperative combination opioid/acetaminophen drugs may benefit from a dose of oxycodone without acetaminophen prior to PACU discharge. Otherwise, they will not begin oral opioids until sufficient time (six hours) has elapsed from the time they received intraoperative acetaminophen. This may leave them with untreated pain. Opioid side effects include respiratory depression, PONV, pruritus, urinary retention, ileus, and constipation.

Tramadol is a centrally acting atypical opioid analgesic that can be administered by oral, rectal and intravenous routes. It produces analgesia as a result of weak μ-opioid agonist activity and weak inhibition of reuptake of serotonin and norepinephrine.[85] Tramadol is effective in children after adenoidectomy and after T&A,[86,87] and has negligible effect on respiration.[85] A cohort of children aged 1-8 years old undergoing T&A for OSAS had equivalent analgesia during the first 6 hours after surgery when given either 2mg/kg of IV tramadol or 0.1mg/kg of IV morphine. Those in the tramadol group had fewer decreases in oxygen saturation to <94% during the first 3 hours after surgery.[86] Overall, tramadol appears to have less opioid side effects than typical opioids, but is nonetheless associated with nausea, vomiting, pruritus and rash.[85] Tramadol is well absorbed when administered orally or rectally,[88,89] and the recommended dose for those routes is the same 1-2mg/kg used intravenously (**Table 18-9**). It is worth noting that ondansetron and other 5-hydroxytryptamine receptor antagonists may diminish the analgesic effectiveness of tramadol.[90] Also, caution is recommended when using tramadol in patients who are taking either serotonin reuptake inhibitors or monoamine oxidase inhibitors, because the combinations may lead to serotonin syndrome or seizures.

Regional anesthesia is particularly useful in ambulatory pediatric surgery. Placement of a block prior to the start of surgery permits pain control without opioids, allows the use of lower concentrations of volatile anesthesia during the surgery, and leads to rapid emergence from anesthesia with little or no pain. Techniques for regional anesthesia are discussed elsewhere in this textbook and are not elaborated on here (**See Chapter 21**). The most widely used pediatric regional technique is the caudal block. It provides excellent analgesia for lower abdominal and lower extremity surgery when using a volume of 1ml/kg, and for perineal surgery when using 0.5 - 0.75ml/kg of bupivacaine (Marcaine). Caudal block can be performed using 0.25% bupivacaine, though 0.125% has been shown to be just as effective for postoperative pain control after inguinal hernia repair and is associated with little or no muscle weakness.[91] The latter concentration results in earlier ambulation. Alternatively, 0.2% ropivacaine can be used, which also results in early ambulation.[92] A

test dose of 0.1ml/kg of local anesthetic containing epinephrine 5µcg/ml is 98% sensitive for detecting intravascular injection and should always be given prior to administration of the remaining injectate.[93] Other regional techniques commonly employed for pediatric ambulatory surgery include blockade of the dorsal nerve of the penis, and ileoinguinal/ileohypogastric block. The latter is best performed with ultrasound visualization of local anesthetic spread in the transversus abdominis plane when possible (**See Chapter 21**).[94] Brachial plexus blockade may be helpful in upper extremity or shoulder surgery when severe pain is anticipated. Femoral and sciatic blockade are commonly used instead of caudal blockade in lower extremity procedures for older patients (generally over seven years of age) or in situations when block duration of 12 - 24 hours is desired over the 4-6 hours typically provided by a caudal block. Large studies in the USA and Europe report that peripheral nerve blockade is safe; the Pediatric Regional Anesthesia Network reported 2,782 extremity blocks without any sequelae lasting longer than three months.[95] Using continuous catheters and elastomeric infusion devices can extend the duration of a peripheral nerve block. Some centers use continuous catheters for pediatric ambulatory patients. Continuous catheters are associated with more adverse events than single injections, particularly catheter related complications such as dislodgement, kinking and malfunction, as well as infection.[95] Continuous catheters are best placed in cooperative patients, who have parents who will be able to follow instructions and return easily to the facility should problems occur.

Discharge

Discharge criteria for ambulatory pediatric surgery patients include stable hemodynamic and respiratory statuses, well-controlled pain, adequately controlled nausea, and cognition at or near baseline. (**See Chapter 22**) Patients should be allowed to drink clear fluids in the recovery room if they wish, but should not be required to drink prior to discharge from the PACU.[96] Postoperative drinking should occur only when the child is ready to do so. Children also should not be required to urinate before discharge.

In general, patients should be discharged once criteria are met, rather than requiring a specific minimum PACU time. Some procedures and conditions do require more specific observation times. Most centers require a minimum of 2 - 4 hours after T&A, due to the possibility of early postoperative hemorrhage. The majority of that time can be spent in a step-down area without monitoring and with a reduced nurse-to-patient ratio. Patients who have received blood pressure support in PACU, naloxone to reverse narcotic effect, racemic epinephrine for post-intubation croup, or treatment for bronchospasm should remain in the PACU for at least one hour after the last treatment was given. Such patients should be discharged *after* the anesthetist has evaluated them. Patients should stay a minimum of a half hour after other intravenous medications. Guardians of pediatric patients should be given verbal and written instructions for postoperative care and contact information in case a complication develops. Guardians should be physically able to assist and mentally able to make decisions for the child's welfare.

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Chapter 19‡
ANESTHESIA FOR BURNS AND TRAUMA

Dean B. Andropoulos, MD, MHCM

Introduction

Burns and trauma remain a leading cause of hospitalization and need for anesthesia and critical care in the pediatric population, especially in resource poor countries. This chapter discusses anesthesia care of burned children by reviewing the epidemiology, pathophysiology, acute resuscitation and management, and anesthetic care of both initial burn procedures and longer-term reconstructive surgery. Next, anesthesia care for children who have suffered other types of trauma will be reviewed and etiology, acute assessment and management during trauma resuscitation, and trauma anesthesia management in the operating room will be discussed.

Burns

Burns are a significant cause of death and disability in children. In 2007, almost 500 children in the U.S. under the age of 14 died from accidental fires or burns, making burns the second highest cause of death for children 5-9 years of age, and placing burns in the top six causes of death for all other age groups. Approximately 15,000 children are hospitalized per year for burn injuries in the U.S., and 1,100 die of this injury. **Table 19-1** shows the top three causes of burn injuries for five age groups.

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‡ This chapter is modified from: Guffey PJ, Andropoulos DB. Anesthesia for Burns and Trauma. In: Gregory GA, Andropoulos DB. Gregory's Pediatric Anesthesia, 5th edition. Wiley-Blackwell, Oxford, United Kingdom; 2012: 896-918.

Table 19-1: United States National Burn Repository 2010: Burn Etiology

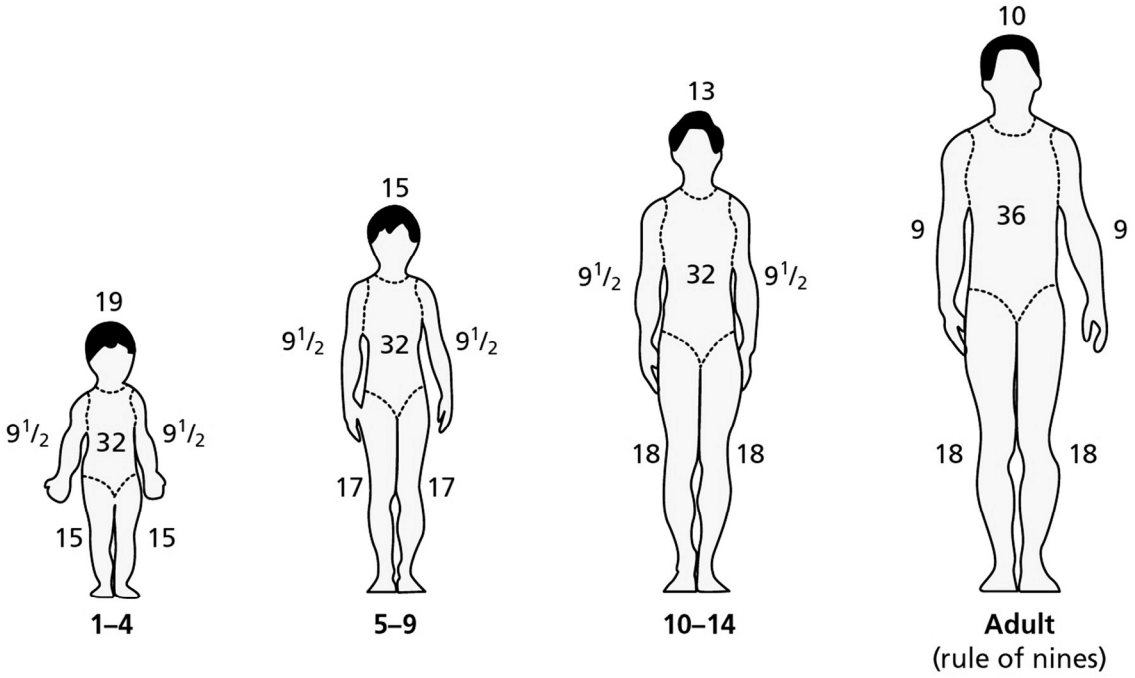
Age group	Etiology – top 3	Cases
Birth – 0.9 years	Scald	2422
	Contact with a hot object	1012
	Fire/flame	244
1–1.9 years	Scald	4636
	Contact with a hot object	2253
	Fire/Flame	444
2–4.9 years	Scald	4401
	Fire/flame	1462
	Contact with a hot object	1330
5–15.9 years	Fire/flame	5763
	Scald	3813
	Contact with a hot object	1036
16–19.9 years	Fire/flame	3348
	Scald	1469
	Other, non-burn	668

Source: National Burn Repository – 2010 Annual Report Version 6. American Burn Association. http://www.ameriburn.org/2010NBRAnnualReport.pdf28*; accessed February 28, 2011.*

Most pediatric burns are from a heat source, with scalds from hot water being most common until the age of five, and then direct contact with fire or flame becomes the leading cause until adulthood. Approximately 30% of burned children suffer inhalation injury with flames, superheated air, toxic gas, or smoke. Though children are less likely than adults to suffer from inhalational injury, children with inhalation burns have a significantly higher degree of morbidity and mortality, i.e., 3-10% without inhalational injury and 20-30% with it. Electricity, chemicals, and radiation may also cause burns. High voltage electrical burns are a serious problem, as outward appearance does not fully demonstrate the extent of damage to tissues and organs.

Burns are classified both by the percent of total body surface area (BSA) affected and by the depth of the injury. An estimate of the BSA involved is most often made from charts, which adjust surface area proportions for age (**Figure 19-1**).

Figure 19-1: Burn Area Chart With Body Surface Area (BSA) According to Age.



Numbers beneath the figures are age in years; other numbers are percent BSA in each region. Reproduced with permission from: Carvahal HF. Burn Injuries. (1992) In, Behrman RE, Kliegman RM, Nelson WE, Vaughn VC (eds.) Nelson Textbook of Pediatrics, 14th Ed. Philadelphia, WB Saunders Co.;1992:235.

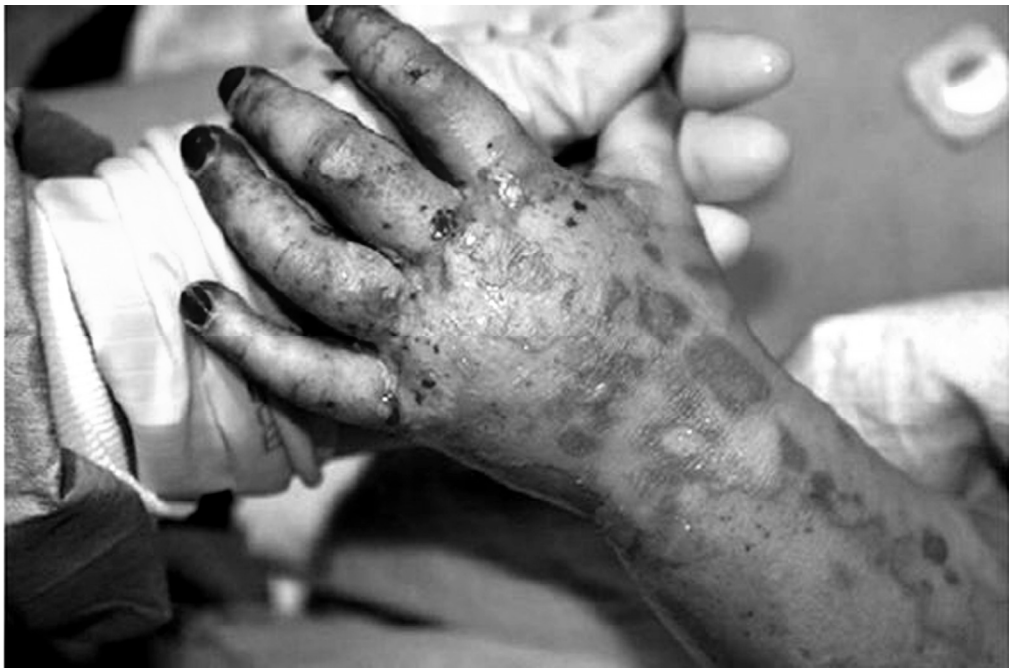
The head is relatively larger and the arms and legs relatively smaller in children than in teenagers and adults, causing the smaller surface area. Burn depth is classified as shallow (first degree—epidermis only); partial thickness – (second degree superficial—epidermis and shallow dermis); second degree deep—epidermis and deep dermis; or full thickness (third degree—epidermis and full-thickness dermis); or fourth degree—which includes the above plus fascia, muscle, and bone (**Table 19-2**).

Table 19-2: Depth of Burn Injury

Classification	Skin and tissue layers involved
First degree (superficial)	Epidermis
Second degree (partial thickness)	
Superficial second degree	Epidermis and superficial dermis
Deep second degree	Epidermis and deep dermis
Third degree (full thickness)	Epidermis and full-thickness dermis
Fourth degree (full thickness)	Fascia, muscle, and bone

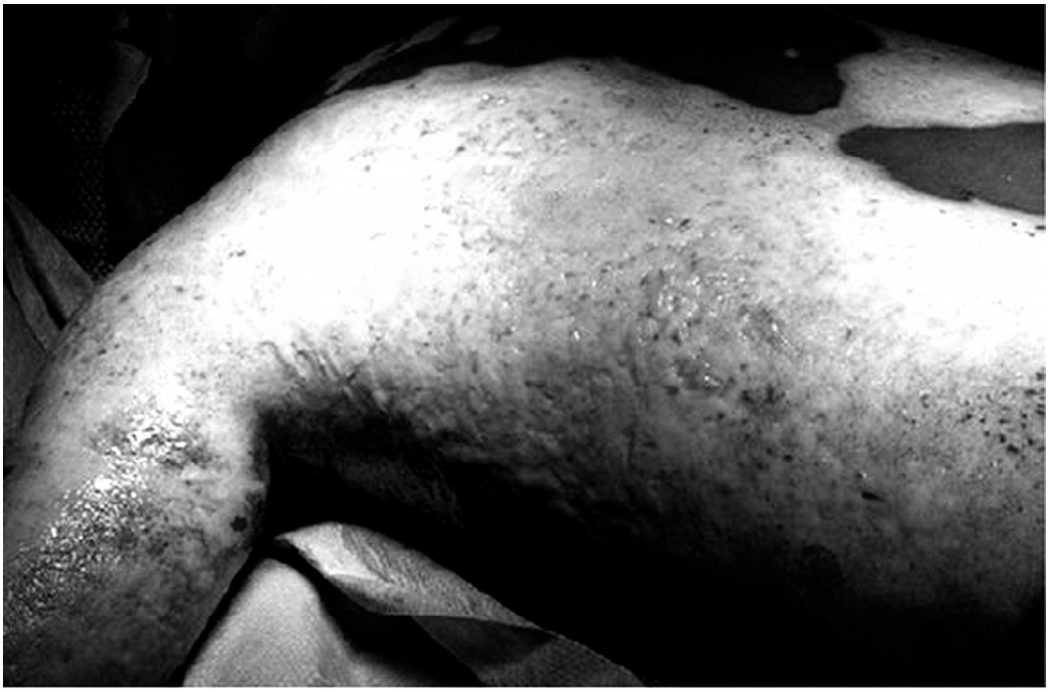
Shallow first-degree burns involve only the outer epidermal layer of the skin. There is not much tissue damage and the protective barrier functions of the skin remains intact, but these burns are often very painful due to innervation of the skin. Partial thickness (second degree) burns involve the entire epidermis of the skin and some parts of the dermis (inner layer of skin). There is blistering, weeping of tissue fluids, and severe pain. A shallow second-degree burn involves only the upper part of the dermis. Burns of this type heal rapidly with minimal scarring; the dermis is regrown with skin cells from hair follicles, sweat glands, and other areas in the deep dermis. A deep second-degree burn extends further into the dermis. With this type of burn, few living epidermal cells remain. Re-growth of the skin is very slow, and scarring occurs if the wound is not grafted. The local response to burn injury involves not only direct coagulation of burned tissue but also reactions of small blood vessels in the surrounding dermis, with poor blood flow and clotting. Fluid losses and the metabolic effects of deep dermal burns are similar to those of full thickness burns. Third degree burns involve destruction of the entire epidermis and dermis, leaving no skin cells to regrow the burned area. The burned skin is dry, leathery, and of firm consistency with black charring or pearly white color. The tissue has no blood supply, and a zone of ischemia exists between the dead tissue above and deeper living tissue below. Preventing the change of living tissue to scar tissue is a major goal during the resuscitation period. A fourth degree burn involves all of the skin structures plus the fascia, muscle, or bone underneath (**See Figures 19-2 and 19-3**).

Figure 19-2: Deep Second-Degree Burn in a Toddler



This is a major burn because it involves the hand. Reproduced with permission from: Fabia R, Groner JI. Advances in the care of children with burns. *Adv Pediatr 2009;56:219-48.*

Figure 19-3: Third-Degree Scald Burn of the Back and Arm of a Toddler.



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Chapter 19: ANESTHESIA FOR BURNS AND TRAUMA

Major burn injuries require special care in dedicated burn centers that have expertise in caring for pediatric patients, if such facilities are available. If these centers do not exist, following the principles outlined in this chapter should improve survival and outcome of patients who have major burns. Major burns are defined as third degree burns of greater than 10% total body surface area (TBSA) or a 20-25% second degree burn (15-20% in infants); burns involving the face, hands, feet, or perineum; inhalational burn injuries; chemical or electrical burns; burns associated with trauma; burns around the whole body or arm or leg, especially of torso; and burns in children with other medical problems. The initial classification of burn injury, both in TBSA and depth, often changes in the first several days following initial surgical excision of burned tissue; so it is best to assume the burn is larger than it really is during initial treatment when deciding whether to send the patient to a burn center or not.

Morbidity and mortality from burn injury increase with increasing size and depth of the burn and with decreasing age of the child. However, survival rates have improved in recent years with better care of burned children. Better understanding of resuscitation, support during the hypermetabolic phase, wound coverage, infection control, and treatment of inhalational injury are responsible for these improvements. Thirteen-to-eighteen year olds have the best survival rates. Recent reports from the year 2000 and later, indicate that survival of 80% TBSA burns in young children is now >60% in developed countries. Survival of children who have 99% TBSA burns who are over 14 years of age now exceeds 50% in major burn centers in these same countries.

Pathophysiology of Burn Injury

Major burn injury interferes with the protective functions of the skin. When skin blood vessels fail to work well, body heat is lost from the injured skin. Breakage of small and medium size blood vessels allows massive amounts of fluid and electrolytes to leak from the wound. Major burn injury initiates a large inflammatory response that results in both local and systemic inflammation. The stress hormone system is activated, and release of catecholamines and adrenocortical steroids occurs. This results in a hypermetabolic (great increase in calorie needs) state, which usually occurs 3-to-5 days after injury and normally lasts until the wounds are covered and healing. This hypermetabolic state slowly improves over weeks to months. All organs are affected by the inflammatory response and hypermetabolic state. So, anesthetists caring for these patients must carefully evaluate them for these changes and be prepared to treat any organ-specific problems.

During the acute phase (which last from a few hours to several days), burn patients may develop shock due to loss of fluid from both the burn injury site and from the capillary leak occurring elsewhere in the body caused by the inflammatory response. In addition there may be dehydration, low cardiac function, and increased resistance to blood flow associated with the

increased catecholamine release. Responses to stress hormones are low in this phase. After the acute phase, a hypermetabolic, stress-hormone driven response replaces the low heart function. Heart rate and cardiac output increase, and hypertension is often present.

The lungs may be affected directly by the burn itself and/or by inflammation. Inhalation of smoke, flames, toxic gases, or carbon monoxide can cause both heat damage and chemical irritation, which may cause swelling of the airways, pulmonary edema, and plugging of small airways. Laryngospasm, bronchospasm, infection in the airways and pneumonia, mismatching of ventilation-perfusion, and decreased respiratory compliance may develop. A condition similar to adult respiratory distress syndrome (ARDS), with pulmonary edema and hypoxemia may happen. Carbon monoxide (CO) inhalation is more common when fires occur in closed spaces like houses. Inhalation of CO is dangerous because it binds to hemoglobin 200 times more tightly than oxygen. CO levels that exceed 10-20% significantly reduce oxygen delivery to the tissues.

Endocrine and metabolic changes may occur with major burn injury and last for 9-12 months after the injury. Resting energy needs are increased, temperature is elevated (38-38.5°C is often the new “normal” temperature), burning of fat, wasting of muscle, and breakdown of liver carbohydrates occur. The degree of elevated energy requirement increases the larger the burn injury. Thyroid and parathyroid hormone levels decrease; vitamin D levels may be low.

Kidney function may worsen because of burn shock and not giving enough fluids, resulting in decreased urine production. Patients with electrical burn injuries, or those with crush injuries, may have significant muscle damage and myoglobin (muscle protein)-induced renal injury or failure.

Both reduced blood flow and inflammation affect the stomach and intestines early in major burns. Low oxygen to the intestines may cause bacteria and endotoxins to be released into the blood, causing sepsis, low intestinal function, and stomach ulcers. Poor liver function may occur due to the low liver blood flow and inflammation. Liver injury is diagnosed by elevated liver enzyme and bilirubin concentrations and by low liver protein production, e.g., elevated prothrombin time. Early feeding with adequate calories is required to meet the needs of the elevated metabolic state and for the rapid growth of young children. Failure to provide these calories makes wound healing more difficult.

The blood forming system is affected during the acute phase of a major burn. Dehydration-induced high hemoglobin concentrations are often the first problem. Following the initial treatment phase, patients are often anemic due to long-term illness, blood loss from skin debridements, and hemolysis of heat-damaged red blood cells (RBC). The platelet count is often low during the first week after a burn injury. After that it returns to normal or is higher than normal; in the late phase of the burn injury, a low platelet count may be an early indicator of infection. Prothrombin (PT) and partial thromboplastin (PTT) times may be high at first because of

liver and blood clotting problems, but they return to normal during the later weeks. Fibrin split products are initially high, and fibrinogen is low for the first several days after a burn, but both return to normal after the initial period. Blood often clots rapidly during the later phases of burn injury. The white blood cell count (WBC) of burn patients is often higher than normal, although applying wound dressings that contain silver sulfadiazine may cause the WBC to be low.

The brain of burned patients can be harmed in several ways, including low blood oxygen from lung injury or CO poisoning. Low oxygen can cause severe brain injury if it results in increased intracranial pressure, brain swelling, coma, and seizures (**See Chapter 10**). Burn encephalopathy (coma), which occurs in 14% of burn patients, includes bad dreams, delirium, personality change, seizures, and coma. High blood pressure (hypertension) can be severe enough to affect the brain and cause seizures in about 7% of those pediatric burn patients who have high blood pressure. Electrical burns can directly injure the spinal cord or brain, depending on the entry and exit sites of the current.

Loss of skin, infected burn wounds, tracheal or tracheostomy tubes, and increased gut permeability to bacteria are sources of infection for burned patients. In addition, indwelling foreign devices (central and bladder catheters, IVs) are also sites for infective organisms to enter the body. Burn injury lowers the patient's immune function, which increases her/his chance of infection. The temperatures and WBC counts of burned patients are high for a long time. Consequently, they often are not good indicators of infection in these patients. Feeding problems and decreasing platelet count frequently indicate the start of an infection. Culturing wounds, blood, and the respiratory and urinary tracts, and comparing the culture results to earlier cultures is one way to detect early infection. **Table 19-3** shows the changes in organ systems with a major burn injury.

Table 19-3: Organ System Changes with Major Burn Injury

Organ system	Early (acute) manifestations first 3–5 days after injury	Late (hypermetabolic) Until major wounds covered, may persist 9–12 months
Cardiovascular	Decreased CO: myocardial dysfunction Hypovolemia Increased SVR	Increased CO Tachycardia Systemic hypertension
Pulmonary	Airway obstruction and edema Carbon monoxide poisoning Laryngospasm and bronchospasm Tracheitis and pneumonia Adult respiratory distress syndrome	Tracheal stenosis Chest wall restriction Infection: pneumonia and tracheitis
Endocrine and metabolic	Increased catecholamines	Increased metabolic rate Increased core temperature (38–38.5°C) Increased protein catabolism Increased lipolysis Increased glucolysis Insulin resistance Decreased thyroid function Decreased parathyroid function
Renal	Decreased GFR Myoglobinuria	Increased GFR Tubular dysfunction from sepsis or nephrotoxins
Gastrointestinal/hepatic	Ischemia from decreased perfusion Stress ulcers, mucosal injury Ileus Endotoxemia Increased liver function tests, decreased prothrombin time	Stress ulcers Ileus
Hematological	Hemoconcentration Hemolysis Thrombocytopenia Elevated PT, PTT, fibrin split products Decreased fibrinogen	Anemia Elevated WBC Hypercoagulable state: elevated factor V,VIII, platelet count, fibrinogen
Central nervous system	Coma Seizures Cerebral edema Increased intracranial pressure	Hallucination Delirium Personality change Seizures Coma
Infection/immunity	Endotoxemia	Chronic immune dysfunction Burn wound infection Respiratory infection Bloodstream infection Urinary tract infection Endotoxemia Antibiotic-associated enterocolitis Infection with antibiotic-resistant organisms

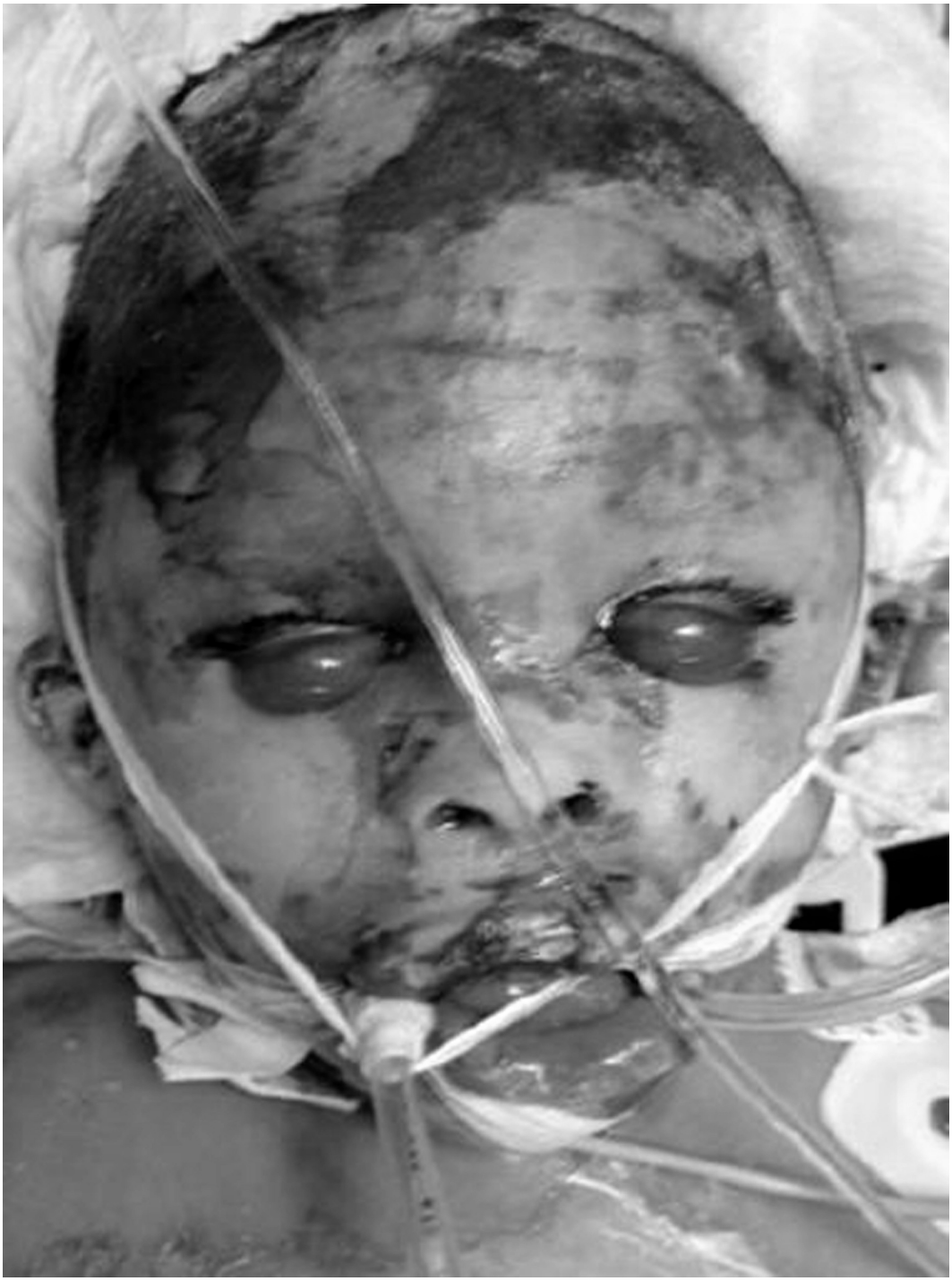
CO, cardiac output; GFR, glomerular filtration rate; PT, prothrombin time; PTT, partial thromboplastin time; SVR, systemic vascular resistance; WBC, white blood cell count.

Initial Evaluation and Resuscitation of the Burn Injured Child

Airway and Lung Management

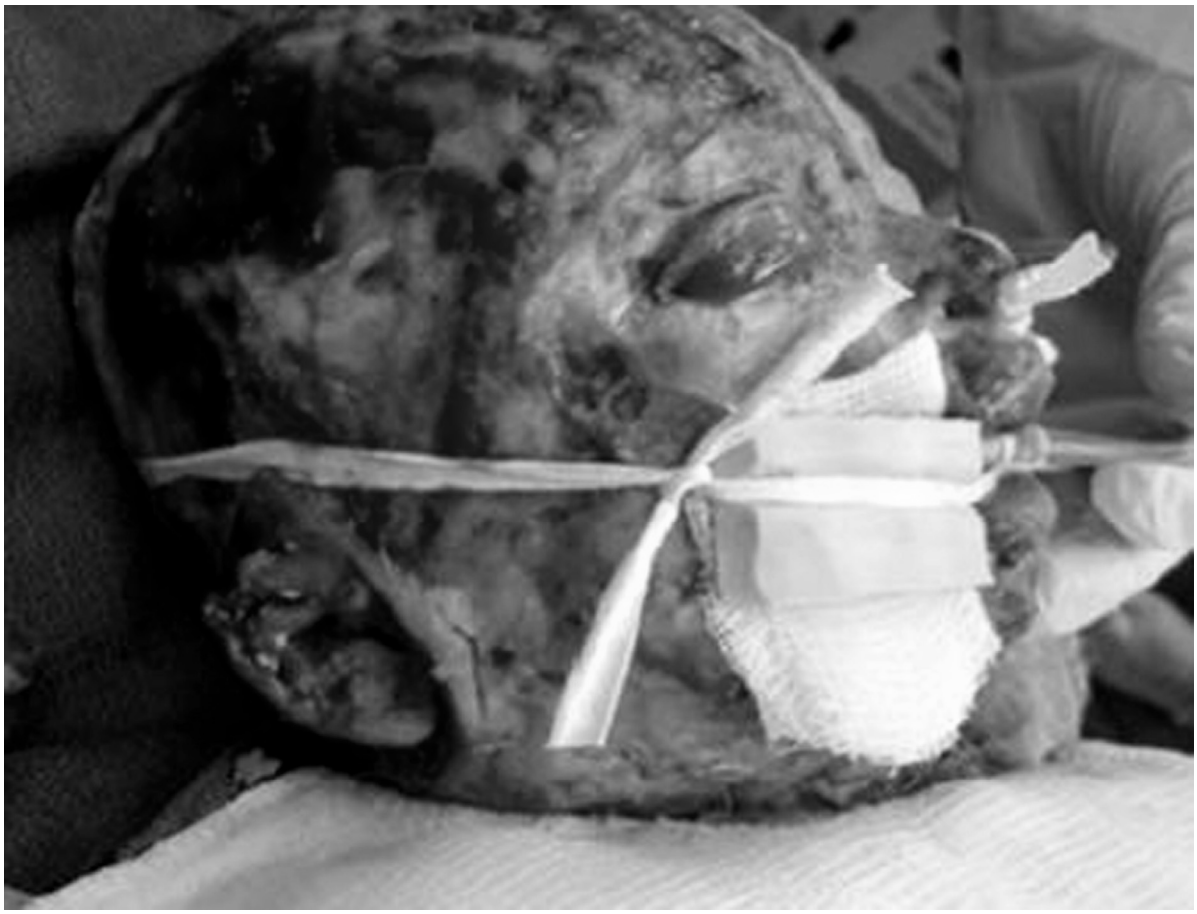
Focus on the airway, breathing, and circulation is the first responsibility of those resuscitating burn patients (**See Chapter 5**). Both respiratory and cardiac arrest requires oxygenation, ventilation of the lungs, and tracheal intubation. A history of a major burn, smoke inhalation, a burn on the face, stridor, soot in the nose or pharynx, respiratory distress (wheezing, dyspnea, cough, tachypnea, rales, rhonchi), a SpO_2 <95% (especially while breathing oxygen) means that there is a high chance the lungs are injured. Strong consideration should be given to early tracheal intubation, even if oxygen and ventilation seem good at first, because swelling of the pharynx, tongue, epiglottis, larynx, and trachea often make intubation much more difficult or impossible later. Oral intubation with a cuffed tracheal tube (TT) is the first choice because it permits ventilation with higher airway pressures and small gas leak if needed to treat severe lung injury. Use of a cuffed trachea; tube does not appear to cause more subglottic injury than an uncuffed TT in critically ill children (**Figure 19-4**). Burned children are considered to have a “full stomach” and can also be in shock. During the initial one-to-two days after a burn injury, succinylcholine may be used safely. After that it should not be used for fear of causing a cardiac arrest. Other neuromuscular blocking agents can also be used, but higher doses of drug may be required to produce paralysis. Ketamine, etomidate, fentanyl, and midazolam are usually excellent choices for tracheal intubation. In practice, normal tracheal intubation is not difficult in the very early burn phase because there is no significant airway swelling. Hours or days later, when significant swelling and airway blockage have occurred, special intubation techniques, including video laryngoscopy, laryngeal mask airways, fiberoptic bronchoscopy, cricothyrotomy, and the ability to place a surgical airway (tracheostomy), will be required. If these airway adjuncts are not available, early tracheal intubation is even more important. Special measures for securing the TT, such as wiring it to the upper teeth or jaw, or securing it with an umbilical tapes and tying the tape behind the head, may be required. Some patients with severe airway or facial burns require tracheostomy.

Figure 19-4A: Early Massive Facial Burn Injury.



Despite the significant facial swelling, this patient's trachea was intubated without problems. The tracheal tube is secured with an umbilical tape tied behind the head. An orogastric tube is in place to decompress the stomach. Reproduced with permission from reference 8: Fidkowski CW, Fuzaylov G, Sheridan RL, Coté CJ. Inhalation burn injury in children. *Paediatr Anaesth 2009;19:147-54.*

Figure 19-4B. Same Patient as in Figure 19-4A After Initial Debridement.



The extent of the facial burn is evident. The TT is tied in place with an umbilical tape. This type of burn is very painful, and the pain must be treated appropriately.

Worry about airway burn or smoke inhalation should lead to rapid examination and tracheal intubation. CO poisoning is a potentially treatable problem that is found by arterial blood co-oximetry or by some new pulse oximeters. One hundred percent oxygen should be administered immediately to patients suspected of having CO poisoning, as this reduces CO's half-life in blood. Patients with CO levels in excess of 20% have problems, such as fainting, nausea, and headache. Their true SaO_2 is >80%. Children with CO levels of 20-40% are confused, disoriented and/or lethargic. Those with CO levels above 40% will have coma, seizures, cardiopulmonary arrest, and death if the CO level is not rapidly reduced. When the FiO_2 is 0.21, the half-life of CO exceeds 4 hours; when the FiO_2 is 1.0, the half-life is 40-80 minutes. Bedside fiberoptic or rigid bronchoscopy may provide further evidence of airway injury. Bronchoscopy allows washing out and suctioning of the airway to clear them of soot, debris, or sloughed mucosa. Lung treatment, consisting of suctioning, saline lavage, and inhaled bronchodilators should be done to limit lung injury.

Fluid Resuscitation

When physicians determined that large amounts of normal saline are required for resuscitation, there were major improvements in the outcomes of burn patients. Following a major burn, there is a rapid decrease in blood volume, increased leakiness of the capillaries, and fluid accumulation in all tissues. This reduces intravascular volume. Loss of plasma volume is greatest during the first 4-to-6 hours post injury; the amount of loss significantly decreases by 18-to-24 hours post injury if the tissues have good blood flow. Protein loss is greatest during the first eight hours post burn. Smaller amounts of protein loss continue until the wounds are completely covered. Adequate initial volume resuscitation is critical for these patients to live and to prevent conversion of living deep thermal burns to dead tissue burn scars.

The goal of fluid administration is to maintain good blood flow to protect the burned tissue. Returning blood volume to normal should be done without overloading the circulation and compromising the heart. This is best done over a 24-hour period. The Parkland Formula is the most widely used formula to calculate fluid requirements for the first 24 hours after burn injury:

Total IV fluid for the first 24 hours = 4 ml x TBSA x weight (kg)

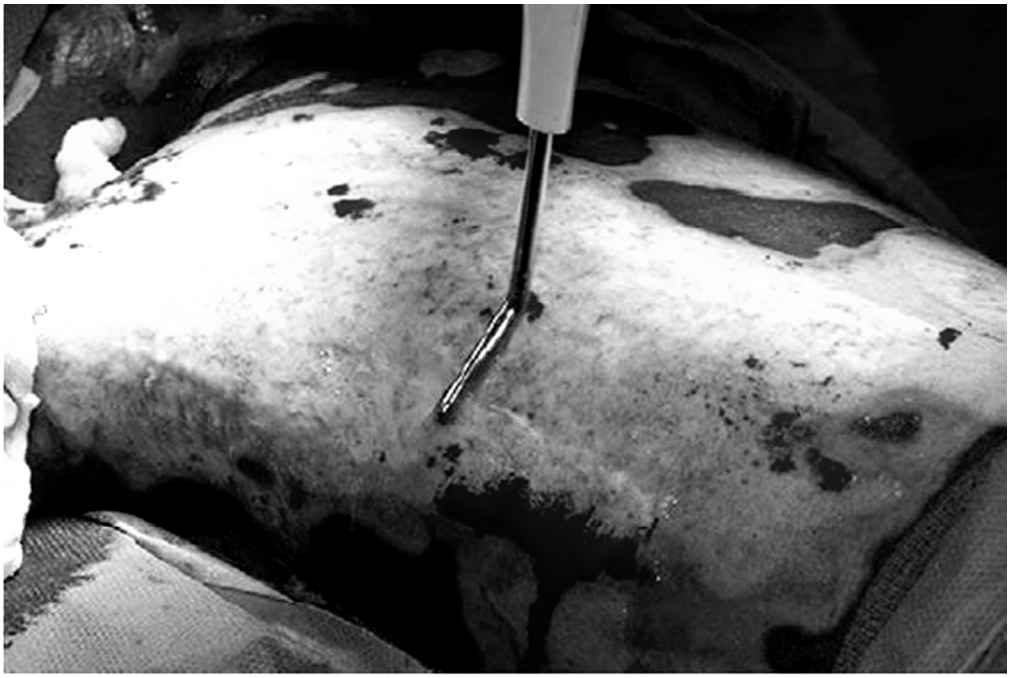
So, for a 10 kg child with a 50% TBSA burn, this would be 2000ml in the first 24 hours for burn fluid resuscitation. Lactated Ringer's solution is also used. Half of the total volume of fluid is given in the first eight hours; the other half is given in the remaining 16 hours. For patients weighing less than 20kg, additional IV fluids are required at maintenance rates. (Use the 4 + 2 + 1 rule for maintenance fluids: 4ml/kg/kg/hr for the first 10kg; plus 2ml/kg/hr for the second 10kg, plus 1ml/kg/hr for every kilogram above 20kg; a 10kg child needs an additional 40 ml/hr. If 5% albumin is available, it is very useful to use it to keep up with the protein losses in the burned tissue.

Each patient is different, and so is their response to the burn and the amount of fluid to maintain good blood flow to the tissues. Measuring heart rate and arterial blood pressure frequently, examining the arms and legs for pulses and circulation, feeling for the liver edge position as an index of central venous pressure, and measuring urine output, should be done but often (**See Chapters 1 and 2**). Sometimes these measures don't work well as monitors in the early phase of burn injury. Arterial blood gases and serum lactate concentration should be measured often when possible. An increasing negative base deficit or high serum lactate concentration is suggestive of low blood volume and tissue perfusion.

Early Burn Wound Excision and Grafting

Early excision and grafting of burn wounds improves survival of children and shortens their length of hospital stay. Grafting should be done as soon as the patient's condition is stable. For the first weeks after a serious burn, it is hard to tell whether her/his condition is stable enough for surgery. Fluid shifts and airway and lung injury must be thought about when deciding if their condition is stable. Burn wounds should be excised and covered to reduce the risk of scarring or infection if it is thought that the burns will not heal within 14 days. Two types of excisions are done, tangential (shallow) and fascial (deep). With shallow excision, the burn scar is cut in thin slices until bleeding tissue is reached so a graft can be placed. With shallow excision there is a lot of blood loss, which limits its use to 15 percent total BSA or less in very young children. Excision to the muscle fascia (deep) is faster and is usually associated with much less blood loss; but because fat and lymphatics are removed, the long term healing of the area is not as good. So, deep excision is only used for patients with burn wound sepsis and to limit blood loss (when the area to be excised is very large or the patient's condition is unstable). For large burns, total fascial (deep) excision can be done early, or tangential (shallow) excisions can be done every 3-to-7 days. The wound is closed with skin grafts with the patient's own skin if this can be done. **Figures 19-5 and 19-6** show these points.

Figure 19-5. Debridement of the Burn Patient Shown Above in Figure 19-3.



This is a tangential (shallow) excision; note the bleeding present where living, unburned tissue was reached. Reproduced with permission from: Fabia R, Groner JI. Advances in the care of children with burns. *Adv Pediatr 2009;56:219-48.*

Figure 19-6. The Same Burn After Coverage With Split-Thickness Skin Grafts.

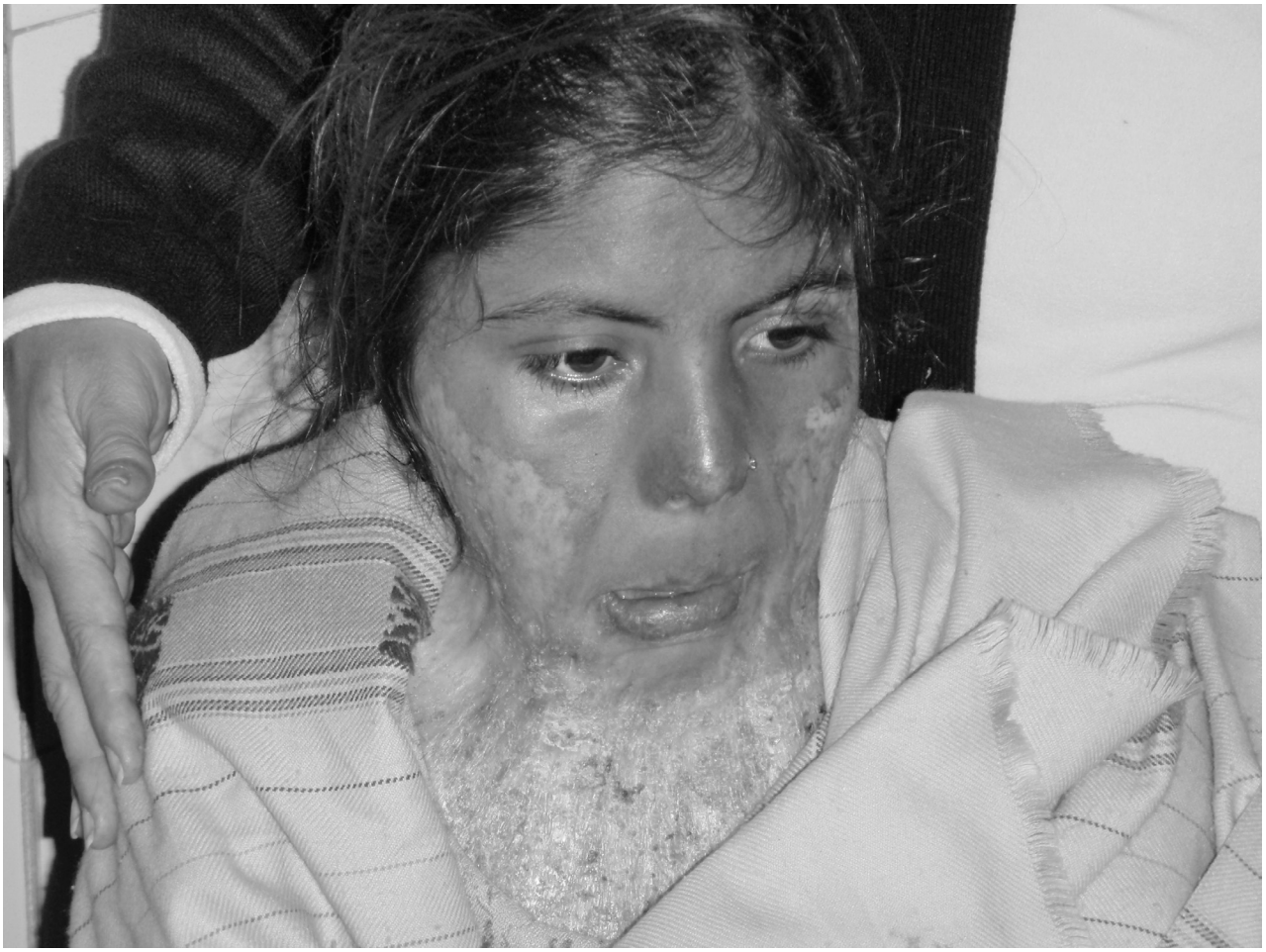
Burn Wound Dressing Changes

Dressing of burns and changing the dressings to look for evidence of viability of tissues or infection or to perform further debridement is a daily process in many burned patients. Many different dressing materials are available to aid healing and to prevent infection; the exact type used is different in every hospital. The regular dressing changes and the pain and distress this causes children is difficult for both patient, their parents, and anesthetists.

Long Term Burn Wound Reconstruction

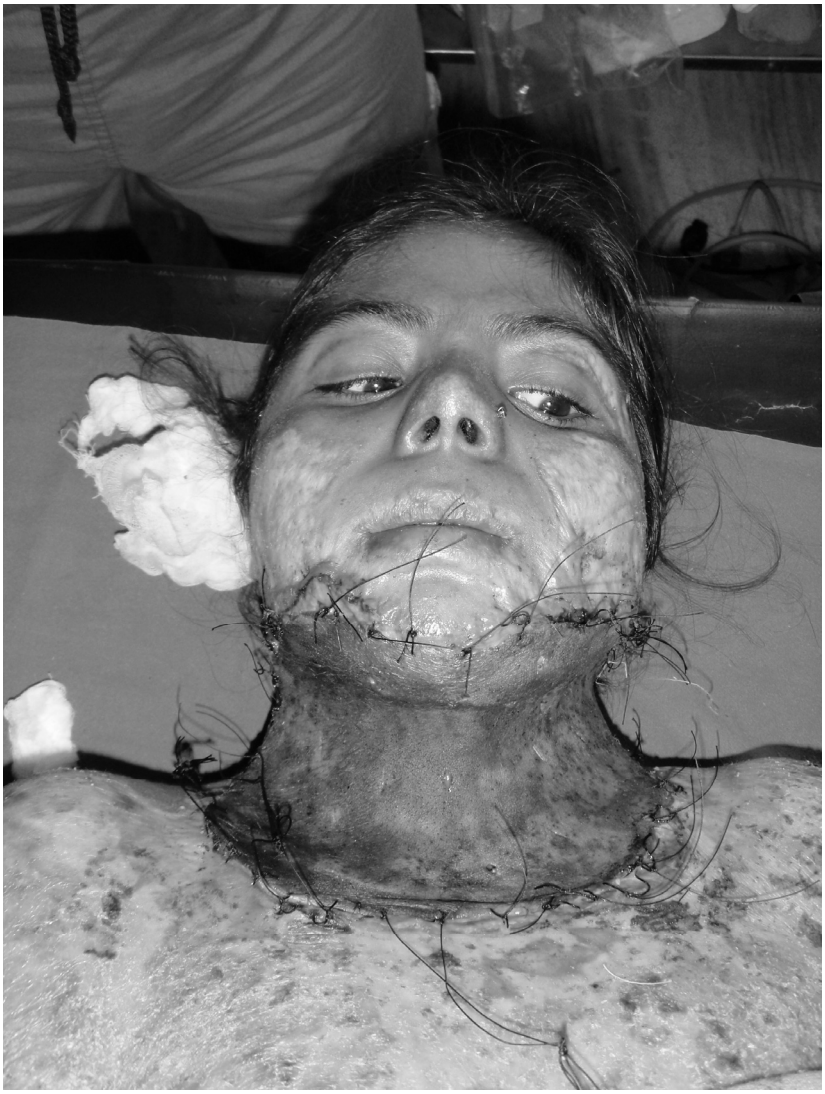
Patients with severe burns anywhere or burns in critical areas, such as the face, neck, hands, feet, or perineum, will return many times for reconstructive surgery to improve the function or appearance of the burned area, once the original burn wound has been treated and healed. These patients often require dozens of anesthetics in a relatively short period of time. Careful evaluation and anesthetic planning and discussion with the surgeon, particularly when the airway is involved, is important for these patients. Patients with burn scars that have shrunk, around the neck, chest, and face, may be very difficult to intubate and a careful examination and plan is very important with these patients (**Figure 19-7 A & B**).

Figure 19-7A: Burn of the Neck and Chest With Scaring of the Chin to the Chest.



This 16-year old female was burned by hot water she pulled off of a stove when she was five years old. She was not able to extend her head; tracheal intubation was impossible. Anesthesia was induced and maintained with ketamine and small doses of fentanyl. Oxygen was delivered during the initial phase of surgery through a nasal cannula connected to an anesthesia machine. Once the surgeons had released the scar, it was possible to intubate the trachea. Ketamine maintains ventilation and a patient airway. She was given 500mg of atropine to prevent ketamine-induced airway secretions.

Figure 19-7B: Neck and Head of Patient in 19-7A Following Removal of the Burn Scare and Skin Grafting



This is the same patient as in 19-7A following excision of the burn scar and grafting. Her head was now in a normal position. Following healing of the skin graft, she could extend her head normally. Blood transfusion was not necessary.

Preoperative Evaluation of Burn Patients

Good preoperative evaluation of burn patients is important when preparing for their anesthetic care. Is a severely burned patient having a first time surgery, or is he/she having long-term burn reconstruction and has had dozens of anesthetics? For the acutely burned patient, what is the airway examination? Will tracheal intubation be difficult? If so, special airway equipment and surgical backup must be immediately available (**See Chapter 6**). What is the condition of the lungs, heart, and circulation? Is the chest X-ray normal or abnormal? What are the mechanical ventilator settings, and blood gases and pH, if available? What IVs are present; do you need to

place additional IVs? What laboratory studies have been done—hemoglobin, white blood cell count, platelet count, electrolytes, kidney and liver function, or arterial blood gases? A good physical examination must be done to understand how much swelling is there, how much skin there is without burns that can be used to place monitors and IVs and obtain skin grafts. What medications is the patient taking, including antibiotics and pain medicines? What medical problems does the patient have, and what surgeries and anesthetics has he/she had? Are there drug allergies? It is important to talk to the burn surgeon about the goals of the surgery, how much blood loss to expect, from where the skin grafts will be taken, and what position the patient will be placed in for surgery. What is the patient's nutrition status? Does he/she take food by mouth, oral gastric tube, or gastrostomy? Talk to the parent and patient (when appropriate) about past anesthetic problems, and tell them all the anesthetic procedures, what the plans are for treating postoperative pain, and answer their questions. Many of these patients have had many previous surgeries, so it is important to listen to the child and parent and to understand if they want premedication and what kind of induction of anesthesia they want.

Intraoperative Management of the Burn Patient

Worried or fearful patients should be given premedication before transfer to the OR. Intravenous or oral agents, such as midazolam 0.5-1mg/kg PO (maximum dose 15mg) or 0.1mg/kg IV or ketamine 0.5-1mg/kg IV (not to exceed 2mg) work well. Having the parents present for induction of anesthesia (if possible) can really be helpful. Strict infection control technique must be maintained during all care.

Burned patients may have few unburned areas of skin for placement of ECG leads, pulse oximeters, and blood pressure cuffs. The anesthetist may have to search to find places to position monitors. Before placing any monitor or catheter in a non-burned areas, it is important to ask the surgeon if that is a site he/she plans to use for skin grafting. For major burn excision and grafting procedures, or for very sick patients, arterial and central venous monitoring are often used when possible because they allow beat-to-beat monitoring of blood pressure, blood sampling, measurement of central venous pressure, and good vascular access for giving large amounts of fluids and blood or drugs to keep blood pressure normal. The femoral (groin) area is often unburned and available for placement of central venous or arterial lines; but these areas are often the only areas available for harvesting skin for grafting. Urine output should be measured during major procedures because it is a way to monitor hydration status. Less than 1ml/kg/hr of urine output suggests volume depletion. Heat loss often occurs when there are large exposed burns and when large amounts of IV fluids are given. Consequently, the patient

S body temperature must be measured throughout surgery and controlled as much as possible with blankets and surgical drapes. The patient's temperature may increase during surgery if the surgeon excises infected tissue. Infection-induced hyperthermia must be differentiated from malignant hyperthermia (MH) (**See Chapter 23**).

The hardest things for the anesthetist during major excision and grafting procedures are keeping the patient's temperature normal, and keeping up with the blood loss. Talk to the surgeon often about these things. Uncovering the living tissue after excision of burned tissue rapidly lowers the patient's temperature if this is not prevented. Burn patients have a higher than normal core temperature (38-39°C) and are most comfortable in room temperatures of 30-31°C. The operating room should be as warm as surgeons and other personnel can stand, and should not have a draft or wind coming through it (e.g., an air conditioner). Heat lamps can be placed above the patient, when available, to maintain her/his body temperature, but care must be taken to assure that these lamps do not cause further burns. A warming blanket, forced air warmer, or a plastic or cloth cover, should be placed around operated sites. Arms and legs can be wrapped with cotton wadding or plastic wrap to reduce heat loss. All fluids that come into contact with the patient, including IV and irrigation fluids and blood products, should be warmed when possible. The inspired gases should be warmed and humidified when possible, or at the very least a condenser humidifier should be used. Surgery time should be made as short as possible by a well-coordinated team effort. If possible, one surgeon can excise burn scar while another harvests grafts. This allows wounds to be covered as quickly as possible and limits most procedures to about 1.5 hours of operating time.

Blood loss is rapid and massive for all but the smallest excisions. It is usually said that 3.5-5% of the blood volume is lost for every 1% of the body surface excised. Excising the scalp is a particularly bloody procedure. For small children, blood replacement with warmed, fresh whole blood should be started as soon as blood loss begins. Unfortunately, whole blood is increasingly difficult to obtain because of blood bank regulations. Because blood loss is difficult to estimate, arterial and central venous pressure should be measured when possible to help assess intravascular volume for major burn surgery. Several measures can be taken to reduce bleeding, including applying dilute epinephrine (1:10,000) soaked sponges to the wound. Tourniquets can be used in some circumstances. The volume of tissue excised should not exceed 10-15% of total BSA at one operation.

The problems of massive transfusion must be sought, especially acute citrate poisoning and hyperkalemia (high blood potassium). Burned patients often have low serum calcium concentrations and low blood pressure during rapid infusion of citrate-containing blood products because the excess citrate in the transfused blood reduces the concentration of ionized calcium necessary for myocardial and vascular muscle contraction. Giving calcium chloride 5-10mg/kg through a central venous catheter, or calcium gluconate, 30mg/kg through a peripheral IV will

return the blood pressure toward normal, but this increase may not last long. Hyperkalemia can occur with rapid transfusion of multiple units of packed red blood cells (RBCs), especially in small children. If possible, blood potassium concentrations should be measured during rapid infusion of blood, especially if the blood is more than two weeks old. Treatment of hyperkalemia with dextrose, calcium, and insulin, and with sodium bicarbonate and hyperventilation, plus stopping the surgery, may be necessary in extreme cases. Massive transfusion affects coagulation tests, especially the platelet count, which it reduces. Clotting factors, in the form of fresh frozen plasma, platelets, or cryoprecipitate, may also be needed. All blood products should be warmed with a properly designed fluid warmer when possible; a way to very quickly infuse blood products should also be available. Because bleeding and hypothermia go together, it is best to shorten the surgery if the anesthetist cannot keep up with massive bleeding.

Because of the hypermetabolic state and increased, there is a need for increased ventilation in the burned patient to remove CO_2. The patient's trachea is usually intubated and ventilation is controlled for all but very short and limited procedures. If the patient has a lung injury, even more ventilation may be required with higher inspiratory flows, peak pressures, tidal volumes, and positive end-expiratory pressure

Maintenance fluid requirements are higher than normal in burned patients and these increased volumes plus additional fluid for losses in the operating room should be given.

Pharmacokinetics of Drugs in Burned Patients

Major burn injuries alter the pharmacokinetics of many drugs, i.e., the way drugs are changed and removed in the body. In the early phase of a burn, cardiac output and tissue blood flow are lower than normal, which slows the metabolism and removal of drugs. Later, during the hypermetabolic phase, the opposite is true—drugs are metabolized and removed more quickly. The anesthetist must always look carefully at each patient's response to drugs, since it can change quickly.

Muscle relaxants are the drugs most commonly changed in burned patients. Burned patients need higher dose of muscle relaxants such as vecuronium, rocuronium, pancuronium, and atracurium. ***DO NOT USE SUCCINYLCHOLINE AFTER THE FIRST 24 HOURS FOLLOWING THE BURN, SINCE DOING SO CAN CAUSE HIGH BLOOD POTASSIUM LEVELS, CARDIAC ARREST, AND DEATH.*** Succinylcholine's ability to cause hyperkalemia can last as long as two years—so succinylcholine should not be used for at least this long. The need to use higher doses of the other muscle relaxants happens about one week after the burn and the larger the burn, the higher the doses of drug needed. If the burn exceeds 20% of the BSA, the dose muscle relaxant required is often 2-to-5-times larger than normal.

Ketamine and inhaled anesthetic agents are often used in burned patients, and repeated use of these agents appears to be safe. Total IV anesthesia with propofol and short acting narcotics is also useful. The dose of thiopental must be increased to 7-to-8mg/kg in burned children for at least one year after the initial injury.

Narcotics are the most commonly used pain medications, but the doses required are often higher than in non-burned patients. Many of these children have received long-term infusions or doses of narcotics, benzodiazepines (valium, lorazepam, or midazolam), or ketamine prior to surgery and require higher doses of these drugs. Dexmedetomidine, if available, works well for burned patients, but may cause transient reductions in cortisol.

Pain in the Burn Patient

Pain associated with burns is often severe and is worse with larger and deeper burns. The pain lasts at least until the burns are well healed. Burn pain is either background (constant) pain, or procedural, (dressing changes and wound debridement) pain. Both are commonly present. Treatment of background pain with acetaminophen and with oral or IV morphine administered every four hours by the clock can work well (**See Chapter 20**). Tolerance to morphine and other narcotics and the need for higher doses of these drugs often occurs.

Wound debridement and frequent dressing changes are needed for proper wound healing. The procedural pain associated with these procedures is often very, very severe. Ketamine, narcotics, and benzodiazepines are commonly used for sedation and pain relief for these procedures. Light to moderate sedation, deep sedation, and general anesthesia may be required. Ketamine is used because it gives excellent pain relief with little effect on breathing when given IV or orally. Repeated doses of a narcotic with long action, like morphine, provides good pain relief for dressing changes but may cause a lot of sleepiness when the pain from the dressing change is gone and the patient has returned to the ward. A combination of ketamine and a narcotic gives very good pain relief for the surgery, while lowering the narcotic dose needed and its side effects. Fentanyl also works well for burn patients but has to be injected repeatedly. Oral or IV midazolam or lorazepam are important adjuncts for allaying anxiety and producing at least partial amnesia for these events.

Outcome of Major Burn Injury in Children

Over the past several decades more pediatric patients with burns have lived because of better early care, intensive care, and better burn wound treatment. However, younger age, larger TBSA burned, and lung injury all worsen survival of pediatric patients. Lung damage is responsible for the death of about 25% of patients. Shock during the early hours after the burn is also a

significant cause of death. Later pneumonia and burn wound infection contribute to the death rate; low oxygen delivery to the brain is responsible for some deaths.

The long-term quality of life of young adults who had burns exceeding 30% of their body is often poor. Although most of them do pretty well with their physical strength, moving around, and eating, working, going to the bathroom, and sleeping, more than half have emotional problems, including depression and anxiousness. They think their quality of life is worse than normal, especially with friendships and marriage. The effects on the body and mind of a patient with a major burn are very important to think about when treating these patients.

Trauma

Trauma is a major reason for hospitalization in pediatric patients and the leading cause of death in children over the age of one year. The leading cause of injury-induced death in patients under one year of age is accidental suffocation due to choking or strangulation; motor vehicle accidents are the leading cause of death for children 1-19 years of age. In the 1-11 year age group, drowning is the second leading cause of death; in the 12-19 year olds homicide is the second leading cause of death, with the majority of deaths involving firearms. The statistics for nonfatal injuries parallel these causes. This section reviews anesthesia care for major trauma in children.

Organization of Trauma Services

A team of practitioners with interest and training in care of trauma patients is very important. A surgeon usually leads this team, but other types of physicians, like emergency medicine trained pediatricians also lead some teams. Anesthetists, nurses, radiology technicians, and intensivists are also important members of the team. Other very important requirements, if available, include neurosurgeons, radiologists, and a functioning blood bank. The exact role of the anesthetist is different in each hospital, but usually he/she will examine and manage the airway and help with the resuscitation of patients with severe trauma. Anesthetists also, provide sedation, analgesia, and anesthesia for diagnostic and therapeutic procedures during trauma evaluation and initial therapy. An anesthesia trauma box that contains the necessary drugs and equipment for airway management, including laryngoscopes and blades, oral airways, and a wide range of tracheal tubes and emergency airway management devices, such as laryngeal mask airways (LMAs) and equipment to perform an emergency cricothyrotomy should be readily available and restocked immediately after each use.

Initial Trauma Evaluation

Evaluation of major trauma begins with obtaining a history from the emergency first responders that includes how the patient was injured (motor vehicle accident, fall, drowning), time from

injury to treatment, extent of injury, vital signs, and what treatment(s) has been given, including vascular access, IV fluids administered, bag and mask ventilation or insertion of a laryngeal or pharyngeal airway or a tracheal tube. Neurological status is assessed and communicated to the team. It is important for one person, usually the trauma surgeon, to lead the evaluation and resuscitation. Initial evaluation begins with the primary survey of airway, breathing, and circulatory status. Cardiopulmonary resuscitation (CPR) is started or continued in the worst cases, realizing that the cause of the arrest may be massive hemorrhage from trauma (**See Chapter 6**). The patient's color, respiratory effort and distress, oxygen saturation, pulses, and arterial blood pressure are quickly evaluated, as is the number and type of IVs and whether there is ongoing hemorrhage. The neurologic system is quickly evaluated using the Glasgow Coma Scale that has been modified for Pediatrics (**Table 19-4**).

Table 19-4: Glasgow Coma Scale Modified for Pediatric Patients

	Response	Score
Eye opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Verbal	Coos, babbles	5
	Irritable	4
	Cries to pain	3
	Moans to pain	2
	None	1
Motor	Normal spontaneous movements	6
	Withdraws to touch	5
	Withdraws to pain	4
	Abnormal flexion (decorticate)	3
	Abnormal extension (decerebrate)	2
	Flaccid	1

The scale is made up of three tests: eye, verbal, and motor responses. The score for each is summed and a total score obtained. The lowest possible total GCS is 3 and is attained with deep coma or death; the highest score is 15 (a fully awake child).

After the primary survey and initiation of treatment, the secondary survey is done, which includes a head to toe examination that comprises the face, head, and neck, focusing on cervical spine injury. Careful removal of a cervical collar is necessary for complete central nervous system (CNS) assessment, as is removal of clothing for examination of chest, abdomen, back and spine, extremities, perineum, and rectum. Initial laboratory examination, including complete blood count, arterial or venous blood gases and pH (if indicated and available), serum electrolytes, blood urea nitrogen, creatinine, glucose concentrations, and blood for type and crossmatch are obtained. Additional vascular access is attained, the bladder is catheterized, and radiographs (including chest, abdomen, and the extremities) are ordered if indicated. Bedside ultrasound (see below) is a valuable diagnostic tool for initial trauma evaluation when available. CT scanning is ordered as indicated and available. However, the anesthetist must accompany the patient to the CT scanner because the patient's condition may worsen in the scanner and require resuscitation.

Airway Management in the Trauma Patient

The anesthetist is often the one called upon to evaluate and manage the airway and overall patient status to determine if the trauma patient needs emergent tracheal intubation. Indications for tracheal intubation include significant respiratory distress from airway or thoracic trauma, including rib fractures, pneumothorax, and flail chest from multiple broken ribs. Retractions, grunting respirations, hypoxemia, and unequal breath sounds all indicate the need for emergent tracheal intubation. In addition, tracheal intubation and mechanical ventilation may be required to provide adequate gas exchange and oxygen delivery to the brain of patients with CNS trauma or an altered mental status [Glasgow Coma Score (GCS) ≤8], which may indicate significant brain injury. The laryngeal reflexes of these patients are often absent and they cannot protect their airway from aspiration of gastric contents. CT scanning and transport to and from the scanner are thought to be safer when the airway is controlled. Low blood pressure and tachycardia from traumatic hemorrhage is often an indication for controlled ventilation. For lesser levels of respiratory distress and lesser levels of depressed consciousness or low blood pressure, high flow 100% oxygen by facemask with monitoring the oxygen saturation (SaO_2) by pulse oximeter and respiratory status may be enough. Because modern CT scanning is so rapid, additional sedation that might mandate tracheal intubation may not be required.

The condition of patients requiring tracheal intubation ranges from full cardiac or respiratory arrest, where no additional sedation or muscle relaxation is needed, to combative patients with normal cardiac function who require large doses of induction agents, analgesics, and muscle relaxants. In the absence of suspected CNS trauma, the cardiovascular status and possible intravascular volume deficit are of most important concerns. Patients who have hypovolemia are usually given a bolus of IV fluid, such as lactated Ringer's solution or normal saline 10-20ml/kg rapidly while the anesthetist is preparing for tracheal intubation. The airway is evaluated for

potential difficult facemask ventilation or difficult tracheal intubation. If difficulty is anticipated, backup methods, such as a video laryngoscope (when available) and surgical airway (cricothyrotomy or tracheostomy) must be immediately available. Preoxygenation and assisted facemask ventilation, if needed, is provided. Large bore suction should be readily available because many trauma patients have full stomachs. Intravenous induction drugs that keep heart function, blood pressure, and intravascular volume normal are preferable. Trauma patients with a lot of blood loss often tolerate etomidate, 0.2-0.4mg/kg or ketamine 1-2mg/kg IV better than thiopental or propofol because the latter two drugs can cause venodilation and hypotension. Muscle relaxation is frequently needed, and emergency tracheal intubation is an indication for using succinylcholine. If the anesthetist is sure tracheal intubation will not be a problem, other muscle relaxants that have few cardiovascular effects can be given. Rocuronium 1.2-2mg/kg, or vecuronium, 0.2-0.4mg/kg are often used. It should be assumed that the patient has a full stomach, and gentle cricoid pressure should be applied. If a cervical collar is present, it should be temporarily loosened or removed for airway management, but care must be taken not to move the head and neck during tracheal intubation. An assistant provides in-line stabilization (**not** traction, or pulling up on the head and neck) of the cervical spine. Because it may take 1-2 minutes for muscle relaxation to occur following administration of muscle relaxants, gentle, shallow mask ventilation with oxygen should be started rather than allow the patient to have a prolonged period of apnea before tracheal intubation. After gentle laryngoscopy and tracheal intubation, careful positive pressure ventilation starts. The heart rate and blood pressure are carefully monitored because positive pressure ventilation may reduce venous return to the heart and cause hypotension in patients with low blood volumes. Use of a styletted, cuffed tracheal tubes is the best choice because it makes ventilation easier and prevents gas leaks around the tube. Exhaled CO_2 detection should be used when available to be sure the tracheal tube is in the trachea. If no CO_2 is detected, a quick laryngoscopy will determine if the tracheal tube is in the esophagus or the trachea. If the tube is in the trachea, it is possible that cardiac output and pulmonary blood flow are so severely compromised that little CO_2 is being presented to the lungs (**See Chapter 2**). Additional narcotic and/or sedative drugs may be needed.

Tracheal intubation of patients with suspected or actual CNS trauma deserves special attention. Ketamine should not be used in this situation because it increases cerebral blood flow and intracranial pressure (ICP). If CNS trauma is the only problem and the arterial blood pressure and blood volume are normal, drugs such as thiopental 3-6mg/kg, propofol 1-2mg/kg, or etomidate 0.1-0.3mg/kg are more appropriate because they maintain normal or decrease ICP, cerebral blood flow, and brain oxygen needs. Adequate depth of anesthesia helps prevent unwanted increases in ICP. It is also important to prevent coughing by providing adequate muscle relaxation before attempting laryngoscopy. Pretreatment with IV lidocaine 1-2mg/kg can help prevent coughing and also stop increases in ICP.

Airway Trauma

Blunt or penetrating trauma to the face and neck should increase concern about injury to the pharynx, larynx, trachea, and bronchi. Possible complications of these injuries include swelling of the pharynx, glottis, and vocal cords. Damage to the larynx, including arytenoid cartilage dislocation, cricothyroid disruption, and vocal cord paralysis also occur. Although laryngeal fractures are less common in children than soft tissue injuries, they do occur and may involve any part of the laryngeal skeleton. The degree of injury can range from simple, nondisplaced fractures to comminuted fractures with loss of cartilage. Laryngotracheal separation is a particularly serious injury that is more common in younger children. Trauma of the face or neck with respiratory distress or subcutaneous emphysema suggests a hole in the digestive system, like the esophagus or pharynx. If such a patient needs tracheal intubation, be very careful before inducing anesthesia and giving muscle relaxation to avoid creating a problem where the anesthetist cannot ventilate the lungs and cannot intubate the trachea. Large bore suction must be available for clearing blood from the airway. Videolaryngoscopy (when available) and surgical backup for cricothyrotomy or emergency tracheostomy should be immediately available. If the patient does not require an emergency airway, CT evaluation or fiberoptic rigid laryngoscopy and bronchoscopy by an otolaryngological or trauma surgeon is indicated. Further surgical management, including repair of the injury via thoracotomy, is provided as indicated. It is important for the anesthetist to keep examining the patient for further airway problems.

Vascular Access

Adequate large bore peripheral IV access with at least two catheters should be provided as quickly as possible in patients with major trauma. Any peripheral vein can be used. However, if possible, access should be obtained in arms or legs that are without trauma. Place IVs in the arms in major abdominal trauma, or in the legs in major thoracic, upper extremity, neck or facial trauma. In the absence of accessible peripheral veins, insertion of an intra-osseous needle into the flat surface of the upper tibia will allow almost any fluid, blood product, or drug to be given (**See Chapter 2**).

Central Nervous System Trauma

Brain trauma is the leading cause of death among injured children and is responsible for 80% of all trauma deaths. Falls account approximately 40% of pediatric brain injuries, and motor vehicle accidents (MVAs) account for 11%. Walking or playing on the street is responsible for 17% and falls from bicycles account for 10% of childhood injuries. Infants and toddlers more often have falls from either a low height or from windows of one or more stories. School-age children are more likely to have sports related and MVA injuries. All ages can be victims of child abuse, but this

is seen more often in infants and young children. The teenage population is increasingly affected by penetrating brain trauma from gunshots.

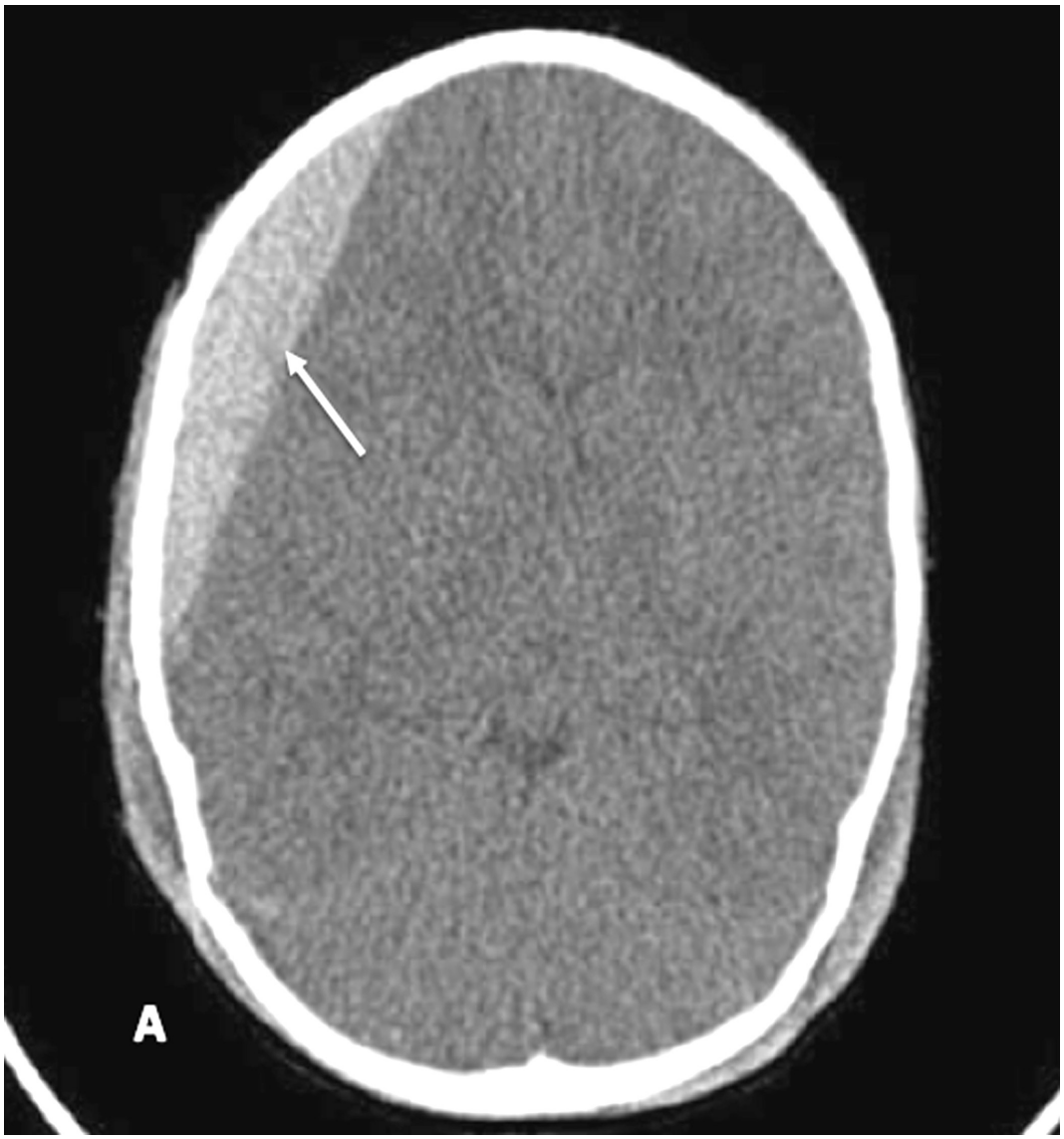
Head injuries in children range from minor scalp trauma and simple skull fractures to cerebral contusion with cerebral edema. They also include acute subdural, epidural, or intracerebral hemorrhage and penetrating brain trauma. The basic principles of examination include inspection of the pupils and determination of the GCS. Signs of serious brain injury and increased intracranial pressure include full fontanels and split cranial sutures (when present), depressed level of consciousness, irritability, or "sun-setting sign" of the eyes (pupils always looking down). Irritability and seizures may also be seen. In older children, headache, neck pain, sensitivity to bright light, vomiting, decreased levels of consciousness and seizures are also observed. Signs of impending brainstem herniation include Cushing's triad (hypertension, bradycardia, irregular respirations), and unequal pupils. Rapid examination, airway management, immediate CT scanning, and going to the operating room are most important when treating a significant head injury, especially when there is bleeding in and around the brain.

Maintenance of cerebral blood flow and oxygen delivery to the brain is the major goal of treating head trauma. Doing so lessens the death of brain cells. Cerebral perfusion pressure (CPP, which is mean arterial pressure minus intracranial pressure) must be kept at normal levels. CPP should be at least 40mmHg in the youngest children and 65mmHg in teenagers. However, high blood pressure is better than low blood pressure, so right after the injury it is probably better to maintain a mean arterial pressure of 60mmHg in young children and 85mmHg in teenagers to compensate for the fact that ICP is seldom measured. Fluid boluses or drugs to increase blood pressure may be needed. In the past, anesthetists and trauma physicians hyperventilated these patients to reduce cerebral blood flow, intracranial volume, and intracranial pressure. But this is now known to cause low blood flow and oxygen delivery to parts of the brain. The goal is to have good cerebral oxygenation and sufficient ventilation to maintain a $PaCO_2$ of 35-40mmHg. Hyperventilation should only be used if brainstem herniation is about to happen (unequal or unreactive pupils, decorticate or decerebrate posturing). The patient then needs a craniotomy to evacuate the hematoma. Once the hematoma is removed, ventilation should be adjusted to maintain a $PaCO_2$ of 35-40mmHg. The anesthetist's other goals include providing pain relief, sedation, and muscle relaxation to prevent further increases in ICP. Often narcotics (such as fentanyl), benzodiazepines (such as midazolam, and muscle relaxants are used in trauma resuscitation and transport to CT scan and the operating room.

These agents are also used during the initial craniotomy, but Isoflurane is frequently used because it maintains good cerebral blood flow and oxygen balance. Other goals of treatment of acute head trauma include using isotonic IV fluids like Ringer's Lactate or normal saline. It is important, however, to not give excessive amounts of fluid because this will worsen brain swelling. Keeping blood glucose concentrations and body temperature near normal (avoid hyperthermia) are also

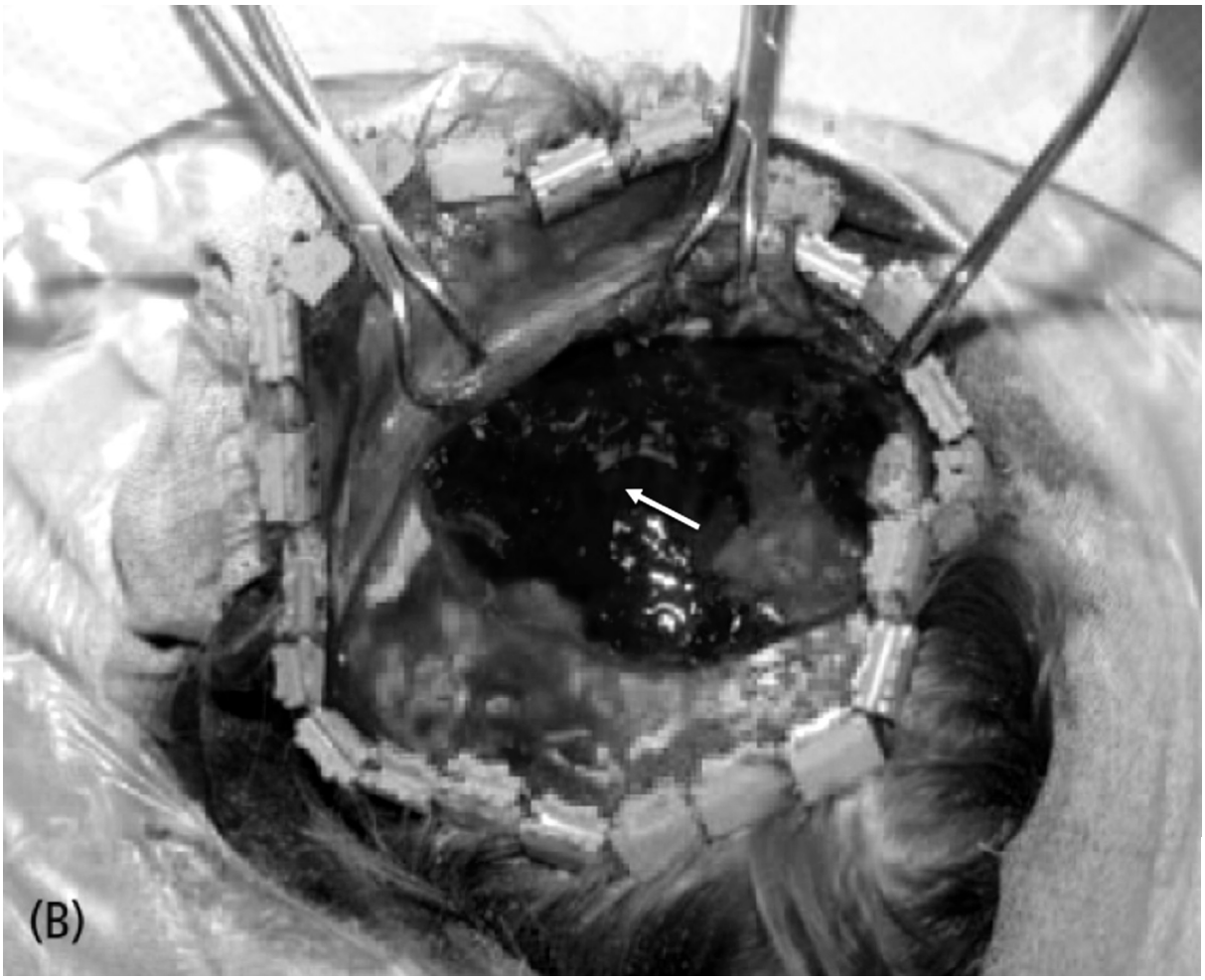
very important. If the patient has marked brain swelling, or there is worry about herniation, drugs, such as mannitol 0.5-1g/kg, or hypertonic (3%) saline, can be given to reduce the intracranial volume and pressure. Surgery includes craniotomy to drain epidural, subdural, or intracerebral hematomas, placement of ICP monitors or ventricular drains, or in severe cases, removing a skull bone flap to provide the brain with room to swell (**Figure 19-7A and 19-7B**).

Figure 19-7A: Epidural Hematoma in a Three-Year-Old



This is the CT scan of a three-year-old who fell from a second story window. A: The CT scan shows an, epidural hematoma (white arrow). There is brain swelling (cerebral ventricles are obliterated). Also, the lateral ventricles, and the difference between gray and white brain matter cannot be seen well.

Figure 19-7B. Acute Epidural Hematoma in 19-7A at Surgery



Epidural hematoma in a 3-year-old after falling from a second-story window. This is the same patient in 19-A after crainiotomy, and opening the dura. The white arrow shows the hemotoma.

Less than 10% of all spinal cord injuries occur in children, and these injuries, when they occur, differ from those of adults. Sixty-to-eighty percent of the injuries occur in the cervical spine. Spinal cord injury occurs without visible change on x-ray or CT scan in 30%-to-40% of pediatric patients who have spinal cord injuries. Cervical spine injures are rare in patients under three years of age and occur in less than 1% of patients with blunt trauma.

Patients under two years of age can have spinal cord injuries without bony injury being seen on plain x-rays. Radiologic studies obtained include plain radiographs in the trauma resuscitation room, CT scanning (if available) for more accurate diagnosis of bony and soft tissue injuries, and MRI scanning (if available) later on to help determine longer-term outcomes. Although cervical spine injury is unusual in young pediatric patients, it must always be ruled out when there is evidence of head injury. Patients with neck and head injuries should have a cervical collar in place

when they arrive at the hospital. As noted above, anesthetists must be very careful with airway management of patients with known or suspected cervical spine injury. If the cervical collar is removed for a short time to allow access to the airway, flexion or extension of the spine must be avoided. The head and neck are held in position without pulling up on the head and neck because this could worsen any spinal cord injury. Following successful tracheal intubation, the cervical collar is immediately replaced and x-rays can be obtained. In the case of quadriplegia, or paraplegia from high thoracic spinal cord injury, loss of sympathetic nerve tone in the arterial system may lead to spinal shock, which requires treatment with fluid boluses and vasopressors. High dose methylprednisolone (30mg/kg plus 5.4mg/kg/hr for 23 hours) is used in many trauma centers when there is significant spinal cord injury, because this thought to lessen the severity of the injury.

Chest and Heart Trauma

About 85% of major pediatric trauma is blunt trauma. Rib fractures and lung contusion occur in around 50% of patients with chest trauma. Twenty percent of patients have a pneumothorax (air in the pleura) and 10% have a hemothorax (blood in the pleura). Diagnosis of these injuries is usually made by either plain x-ray and by CT scanning; treatment is by placement of a chest tube. Thoracotomy is rarely indicated in the acute trauma setting. A tension pneumothorax that is accompanied by severe respiratory distress, low blood pressure, and greatly decreased or absent breath sounds on the side containing air in the pleura is treated acutely by inserting a needle through the second anterior intercostal space to remove some of the air. This is followed by insertion of a chest tube. Heart and great vessel trauma to the aorta, pulmonary artery, or vena cava is relatively rare and occurs in less than 5% of children with chest trauma. However, the problems caused by these injuries are severe and should not be forgotten in patients with major blunt trauma or penetrating chest trauma. Pediatric patients with heart trauma (contusion, coronary artery injury, or aortic dissection) also have injury to other organs almost 90% of the time. Premature ventricular contractions, ECG changes (such as ST segment elevation), and low arterial blood pressure and shock (due to poor heart function) may be seen. Plain x-rays and standard CT scans may not make the diagnosis of cardiac trauma. A diagnosis of cardiac tamponade (blood, fluid or air around the heart under pressure) or myocardial dysfunction can be made quickly at the bedside by echocardiography when available. Trauma-induced cardiac tamponade is rare in children, but when it is present the patient has low blood pressure and pulsus paradoxus (a wide swing in blood pressure with breathing in and out). These patients require emergent pericardiocentesis (drainage of fluid around the heart via a needle) followed by surgical drainage and repair of the cardiac injury. Chest CT scanning with contrast material is necessary to diagnose aortic injuries, such as aortic dissection. Patients with cardiac trauma who need emergency surgery on other organs need to be very carefully monitored for further problems with myocardial function.

Abdominal Trauma

Abdominal injuries occur in 10-15% of major pediatric trauma cases. Blunt trauma accounts for 85% of abdominal injuries and MVAs account for half of the blunt trauma. Injury to the spleen accounts for about 45% of intra-abdominal injuries in children. Other injuries include liver contusion or laceration, stomach or intestinal perforation, kidney, ureteral, or bladder trauma, and bleeding behind the peritoneum (in the retroperitoneal space) of the abdomen. Abdominal pain over the whole abdomen is common, making it difficult to determine the exact point of the trauma. Plain x-rays of the abdomen are seldom of help in these patients. Bedside portable ultrasound examination using the FAST protocol (***F***ocused ***A***bdominal ***S***onography for ***T***rauma) allows rapid scanning of all four quarters of the abdomen looking for free fluid, which, if found, is evidence of major trauma. Rupture of the spleen or a liver laceration is the usual cause. Even with ultrasound, it is hard to decide if the child needs surgery. CT scanning of the abdomen, if available, with IV contrast material is the best way to diagnose abdominal trauma and must be done as quickly as possible if there is concern for the presence of significant injury. Oral contrast material is often not used in the acute situation because it takes too long for the dye to traverse the entire gastrointestinal tract.

Most children do not need surgery for abdominal trauma, including most injuries to the spleen and liver. Ninety-to-ninety-five percent of patients are successfully treated without surgery, and it is rare to require surgery or blood transfusion for isolated Grade I or II injuries to the liver or spleen (bleeding under the covering [capsule] of the liver or spleen that involves ≤50% of surface area or a tear in the capsule of ≤3cm that does not involve a major blood vessel). If available, laparoscopy is often used instead of a full laparotomy to evaluate the amount of intra-abdominal trauma in some patients. It may be necessary to do an open laparotomy to control hemorrhage if there is rupture of a major blood vessel, perforation of the intestine, or other intra-abdominal trauma that cannot be treated without surgery. If surgery is needed, the anesthetist must prepare for major blood loss. Surgeons try to save organ function as much as possible and rarely do a splenectomy, liver resection, and nephrectomy.

Arm and Leg Trauma

Trauma to the arms or legs can involve both bones and soft tissue is often present in pediatric patients with multiple areas of trauma. However, arm and leg trauma is of less concern during the initial trauma examination and during treatment of life threatening problems. But, arm and leg trauma should not be forgotten. X-rays of injured arms or legs are done as soon as possible after initial treatment and stabilization of the patient's condition. Compartment syndrome from crush injuries is a major problem that may require the surgeon to make incisions in the fascia of the arm or leg to reduce the pressure caused by the swelling; these incisions help save as much tissue as possible. Release of muscle protein (myoglobin) from crush injuries may cause kidney failure. In

larger patients, the possibility of a fat or air embolus to should be considered. A large amount of blood loss from femoral and pelvic fractures is often hidden in the tissues, and the anesthetist must think about this when caring for patients with these injuries, especially if the blood pressure is low.

The Patient with Multiple Organ Trauma

Major or multiple trauma involves the entire body. When this occurs, the bodies stress response maintains blood flow to the most important organs, such as the heart and brain. The stress response may last for several days. A longer acting inflammatory response then occurs and produces many problems throughout the body. These include edema (swelling) throughout the body, including pulmonary difficulties, abnormal blood clotting, and poor cardiac function. When caring for trauma patients, the anesthetist must carefully examine the patient both during and after the initial resuscitation and treatment to assure that all problems are treated in a timely manner. Initial treatment of the patient with multiple trauma includes control of the airway and normalizing blood volume and hemoglobin to assure adequate tissue blood and oxygen delivery. Initial treatment must also include surgery to treat life threatening bleeding anywhere or hemorrhage or swelling of the brain. Afterwards, patients can usually be stabilized in an intensive care unit where more tests can be done if needed, and/or they can be returned to surgery for more treatment.

Management of the Trauma Patient During Surgery

Most pediatric patients who suffer major trauma do not require immediate anesthesia and surgery. Anesthetists commonly help manage the airway during initial treatment and during transport to a scanner for a 5-10 minute CT scan of the brain and abdomen. Then the patient is often taken to the ICU, where further monitoring and testing are done and further care is planned. The most common reason for immediate operative treatment is evacuation of an epidural or subdural hematoma from the brain. The next most common reason is for major abdominal trauma. In children, doing a laparotomy and craniotomy are rare, and so is penetrating trauma to the chest (gun shot wounds or stab wounds). On the other hand, children often require surgery at a later time for treatment of fractures, soft tissue injury, or for more surgery following initial trauma resuscitation.

For major trauma requiring surgery, it is very important to get the patient to the operating room and start the surgery as soon as possible, usually within 30-60 minutes from the time it is decided that he/she requires surgery. The anesthetist needs many people to help with the initial care. Ask the surgeons, emergency room or ICU personnel to help in the operating room (OR) if additional anesthetists are not available to help. The OR must be completely setup at all times with appropriate airway equipment, IV catheters, fluids, and warming devices for a variety of patients

from infants to adult sized teenagers. Pressure fluid infusers that deliver 500 ml per minute should be available. Catheters and transducers to measure arterial and central venous pressures should also be available. A nearby blood bank that has people to quickly provide large amounts of PRBCs, fresh frozen plasma, platelets, and cryoprecipitate is very important. If there is no time to crossmatch blood, type specific uncrossmatched blood (blood of the same blood type as the patient, for example A^+) is preferable and can be given within 10-15 minutes in most centers. In an emergency situation when blood products are required in less time and even type specific blood testing cannot be done, O negative uncrossmatched blood is used. Massive transfusion in a pediatric patient means transfusion of more than one blood volume over a 24-hour period (75ml/kg), but a better definition in the acute trauma setting is transfusion of 50% of the estimated blood volume in three hours. If massive transfusion with O negative blood is necessary, the anesthetist should continue giving O negative blood, even after the patient's blood type is known, because there is always a risk that the anti-A and anti-B antibodies found in some O negative blood will cause hemolytic anemia (**See Chapter 4**). Massive transfusion will also affect the blood clotting proteins, and the platelet count, so transfusion of PRBC alone can cause worse bleeding. One-way to prevent this is to use a 1:1:1 ratio of PRBCs, fresh frozen plasma, and platelets after identifying massive hemorrhage.

The anesthetist's most important job during acute trauma surgery is to make sure that blood flow and oxygen delivery to the tissues is as good as possible. The brain and heart are most important organs of concern, and the anesthetist attempts to assure their blood flow and oxygen delivery by maintaining an appropriate blood volume, hemoglobin concentrations, arterial blood pressure, cardiac output, oxygenation, and ventilation. An arterial catheter is often helpful in this setting, but one should not spend a lot of time inserting one of these catheters if doing so delays truly emergent surgery, such as removal of blood in or near the brain. Obtaining adequate large bore peripheral IV access is very important. If an arm or leg is accessible after draping for surgery, the anesthetist may be able to insert an arterial line during surgery; but this should not take the anesthetist's attention away from the more important job of monitoring and treating hemorrhage and low blood pressure. A central venous line can be placed when there is time to do so, often after the initial surgery. When possible, frequent blood gases, coagulation studies, and hemoglobin, electrolytes, ionized calcium, and lactate concentrations are very valuable in helping the anesthetist decide what to do next.

A frequent question asked is what anesthetic should be used and in what doses to provide anesthesia for very sick trauma patients with low blood pressures? An unconscious patient with head trauma will not wake up during the surgery; so resuscitation to a normal estimated blood volume should be the main priority. In general, narcotics, such as fentanyl, are good choices for trauma patients. It may be necessary to give 25-100mcg/kg of fentanyl for patients with major trauma. If there is brain injury, ketamine should *not* be used. However, ketamine is quite useful

for providing analgesia and amnesia in patients who have multiple trauma that does not involve the brain. Small doses of benzodiazepines, such as midazolam, often prevent the patient from being awake during surgery. Nitrous oxide (N_2O) should not be used in trauma patients because it may enlarge any air pockets they have in their chest, abdomen, or around their brain. Isoflurane, sevoflurane, and desflurane may be used in small doses, while paying careful attention to and treating low blood pressure. Halothane is not the best choice because it lowers blood pressure more than the other anesthetic gases. Scopolamine can be used in patients with severe trauma to make sure the patient does not wake up. Muscle relaxation during trauma surgery is always required, and drugs such as rocuronium, vecuronium, or cisatracurium are better because they do not alter heart rate or blood pressure very much.

Uncovering of the body, giving large amounts of cool IV fluids, transporting the patient to scanners, and performing a scan in cool environments often make trauma patients cold. To prevent this, the anesthetist must measure temperature, warm the OR and IV fluids, use forced air warming (when available), humidify and warm the inspired gases (if possible), and use radiant warming devices when needed to maintain a normal body temperature. Problems with blood clotting, poor tissue blood flow, and slow awakening from anesthesia are some of the difficulties occurring in cold patients. Every effort should be made to maintain the patient's core temperature above 35°C. Elevated temperatures (hyperthermia) are also dangerous because they increases the need for oxygen in vital organs, mostly in the brain, where it may reduce oxygen levels and increase the size of any brain injury. **Table 19-5** reviews the most important jobs of anesthetists caring for pediatric patients with major trauma.

Table 19-5: Most Important Jobs for the Anesthetist in Care of Patients With Major Trauma

Airway

- Rapid assessment
- Intubate trachea for GCS ≤8 or respiratory compromise, or need for significant sedation for diagnostic studies
- Rapid or modified rapid sequence with cricoid pressure
- Use small cuffed endotracheal tube
- Remove cervical collar and use in-line stabilization for suspected cervical spine injury
- Use CO_2 detection and auscultation to confirm placement
- Initiate positive pressure ventilation cautiously to minimize compromising venous return
- For trauma: have suction, surgical airway back-up; evaluation with CT or bronchoscopy

Central nervous system

- Manage airway pre-emptively to ensure adequate oxygenation and ventilation
- Hyperventilate only for impending herniation
- Maintain CPP (MAP – ICP) of 40–65 using volume infusion and pressors if necessary

Spinal cord injury

- In-line stabilization (not traction) for airway manipulation
- Anticipate and treat spinal shock: IV volume, pressors
- High-dose methylprednisolone per institutional protocol

Thoracic trauma

- Emergency airway intervention for significant distress
- Tube or needle thoracostomy for tension pneumothorax
- Tube thoracostomy for hemothorax
- Cardiac tamponade: pulsus paradoxus, pericardial effusion by echo – drain emergently
- Cardiac contusion or coronary injury: PVCs, ST elevation, myocardial dysfunction
- Aortic dissection: rapid CT scan with contrast and transport to OR

Abdominal trauma

- Evaluation by CT, non-operative management is the norm
- Prepare for massive transfusion if operative treatment necessary

Extremity trauma

- Compartment syndrome, myoglobinuria and renal failure in crush injury

Multiple trauma

- Neuroendocrine response
- Systemic inflammatory response syndrome:
- Capillary leak
- ARDS
- Multiorgan dysfunction
- Myocardial dysfunction
- Disseminated intravascular coagulation

ARDS, acute respiratory distress syndrome; CPP, cerebral perfusion pressure; CT, computed tomography; GCS, Glasgow Coma SCale; ICP, intracranial pressure; MAP, mean arterial pressure; OR, operating room; PVC, premature ventricular contractions.

Outcomes of Trauma Care in Children

As written earlier in the chapter, pediatric trauma is the leading cause of death in 1-19 year old patients despite major efforts over the past several decades to reduce the incidence of trauma. The best way to improve outcomes of pediatric patients with major trauma is to use the team approach, where surgeons, anesthetists, other physicians, nurses, radiology technicians, blood bank, and many others work together to determine the best ways in their own hospital to treat these patients.

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Chapter 20
PAIN MANAGEMENT

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INTRODUCTION

The International Association for the Study of Pain describes pain as, "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage"[1] Pain is sometimes beneficial (*e.g.,* withdrawing a hand from hot stove), at other times it is detrimental (*e.g.,* injury following burns, trauma, surgery) and can delay recovery from surgery, wound healing, and discharge from hospital. Pain can be acute (surgery, trauma, burn injury) or chronic (spine abnormalities, tumors, chronic infections). Anesthetists and surgeons are primarily concerned with the acute pain caused by surgery and trauma. Anesthetists are often asked to evaluate patients who have chronic pain and suggest possible therapy or provide treatment (including medical management as well as nerve blocks). It must be remembered that *just because a patient cannot communicate her/his pain (infants, patients with cognitive disabilities) does not mean he/she is not having pain!* Pain is subjective. What is not painful for one child may be very painful for another, based on previous experience with painful procedures (multiple IV insertions, surgeries), culture, and developmental stage. While consideration should be given to developing an acute and chronic pain clinics with people who are committed to reducing/eliminating pain, further discussion of doing so is beyond the scope of this chapter. For further information, several references have been included on how to establish these services.[2,3] This chapter discusses development of pain pathways and the response of infants and children to pain. It also discusses detection, evaluation, and treatment of acute pain. The use of regional anesthesia for treatment of postoperative or chronic pain is discussed in **Chapter 21**.

As the American Academy of Pediatrics indicated in the year 2000, pain has immediate and long-term consequences in infants and must be prevented or, if it cannot be prevented, it must be treated effectively.[4] Pain should be assessed before and after treatment to assure that the treatment provided (drug, dose, route) is effective. As far as possible, we have followed the World Health Organization (WHO) guidelines for treatment of pain. While the WHO's guidelines are primarily for chronic pain (usually from cancer), this framework is useful for treatment of acute postoperative pain. whqlibdoc.who.int/.../9789241548120 **Guid**... There are many drugs available to treat postoperative pain, many of them with similar effects. Consequently, it is best to limit the number of drugs used by any clinic or hospital (or country) to a few drugs and be sure surgeons, anesthetists, and nurses are familiar with the drugs, their effects, the potential complications of using these drugs, and the treatment of any potential complications.

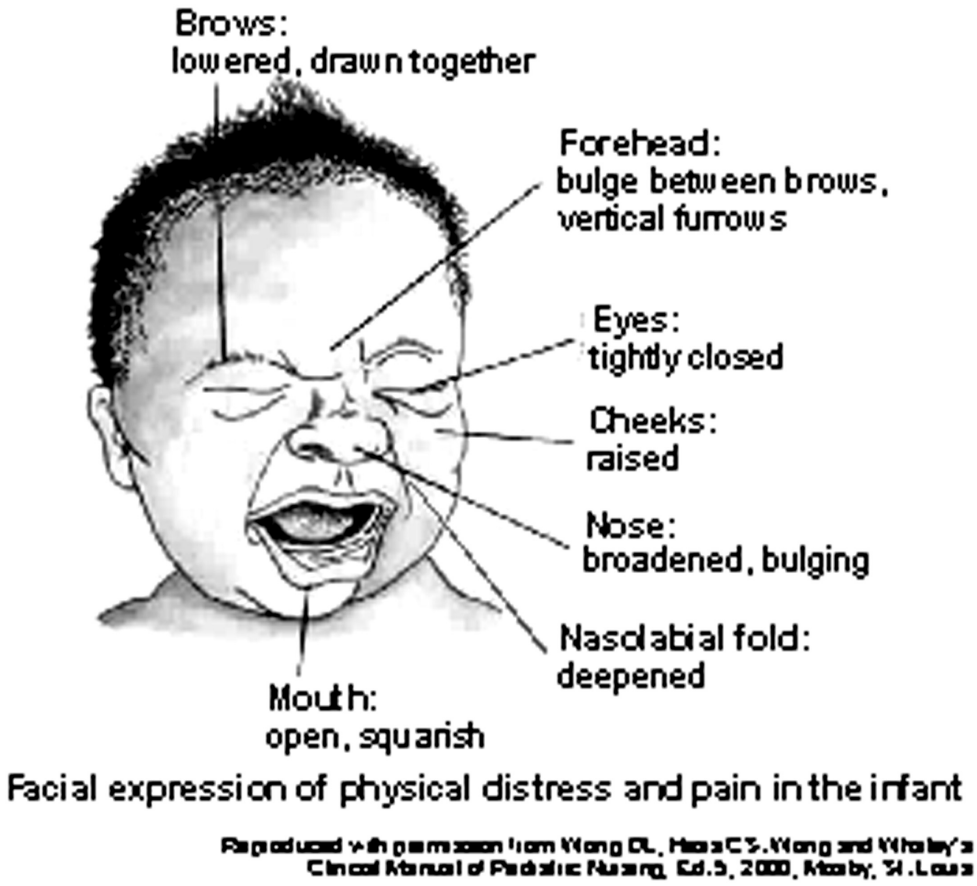
Pain Pathways

Sensory receptors appear within the first few week's of gestation, and integrated nociceptive cells (neurons that responds to potential tissue damage and send signals to the spinal cord and brain to perceive pain) are present by 24-28 weeks gestation and maybe earlier. There are cortical responses to noxious stimuli by at least 25 weeks gestation.[5] Premature infants of 24 weeks gestation clearly respond to painful stimuli (heel stick) in the author's experience. In fact, neonates born at 24-25 weeks gestation often exhibit exaggerated responses to stimuli (painful and otherwise), in part because brain-derived inhibitory pathways develop later.[6] Consequently, the young infant's response to pain is greater (more active) than in later life.

Ill babies requiring neonatal intensive care often undergo approximately 15 painful procedures every day, and these procedures have long-lasting effects. The repeated painful stimuli result in hyperalgesia (excessive sensitivity to pain) and increased responsiveness to pain and to touch (bathing, changing clothes and dippers). The hypothalamic-pituitary axis produces β endorphins in response to stress by 18 weeks gestation. Cortisol and noradrenaline are produced by 20 weeks gestation. The stress response has detrimental effects, but it is also necessary for maintenance of arterial blood pressure, cardiac output, and tissue oxygen delivery.

Most neonates respond to pain with increased heart rate, but very ill babies may develop bradycardia because illness decreases baroreceptors function. Their autonomic nervous system responds to pain with peripheral vasoconstriction (pale skin), duskiness, and cyanosis. In response to pain, their movements are disorganized; including splayed (spread out) fingers, extended arms and legs, frantic behavior, and jumpy movements. They frown, bring their eyebrows together, narrow or close their eyes, avoid eye contact, and sigh. Good evidence of pain in neonates is seeing their eyebrows go down and together, their nasal root (top of their nose where it meets the frontal bone) widen and bulge, their eyes close tightly, and their mouths become angular and squared (**Table 20-1**). In some cases they do just the opposite (they become completely limp).

Figure 20-1: Facial Response to Pain in Infants



See text for description. From Wong's Clinical Manual of Pediatric Nursing. With Permission

Bartocci et al.[7] reported that heal punctures significantly increased cerebral blood flow and oxygen delivery to the side of the brain being stimulated, while blood flow and oxygen to the other side decreased or did not change. The increase in flow was greater in males than in females, suggesting to some people that female babies felt pain less well than male babies. While this is *not* true, many nurses and doctors caring for neonates have administered less pain medication to female than to male patients for unknown reasons. Peterson et al.[8] found a correlation between the smaller brain volumes of ex-premature infants and their cognitive and behavioral outcomes. These changes were most prominent in somatosensory cortex and in the thalamic and hypothalamic regions of the brain. There is however, variation in the anesthetic requirement with age **Table 20-1**.

Table 20-1: Halothane requirement versus Age

Age	MAC
≤33wks	0.55 ± 0.05
Term – 1mo	0.87 ± 0.03
1mo – 6mo	1.20 ± 0.06

wks = weeks; mo = months; MAC = minimum anesthetic concentration (alveolar concentration of halothane at which 50% of patients move and do no move with a skin incision.

Repeated neonatal pain increases the response to future injury. Circumcision without anesthesia significantly increases responses to vaccinations months or years later.[9] Adequate anesthesia for circumcision ameliorates this response. Despite this knowledge, many infants still undergo circumcision without anesthesia. Children born with difficult births (forceps delivery, meconium) have increased pain responses as adults. Having a baby suck on a nipple containing sucrose reduces the response to heal stick but fails to prevent hyperalgesia later. There is still an increased stress response (cortisol release) to needle sticks at six months of age. Animals exposed to pain early in life had higher pain thresholds, impaired spatial learning, and exaggerated startle responses.[10] Acute pain causes the release of catecholamines, corticosteroids, glucagon. It also causes breakdown of fat and carbohydrates (stress response);[11] and stimulation of heart rate, arterial blood pressure, and respiratory rate, although many babies respond to pain by reducing respiratory rate and arterial blood pressure. Some babies appear to be "catatonic". In fact, some ex-premature infants have reduced pain later in life. Hypersensitivity may be the reason premature infants required higher levels of analgesia and anesthetics for sedation than term neonates. Increased sensitivity plus lack of inhibition may cause *windup* [i.e., an increase in pain strength following repeated painful stimuli (e.g., heel stick)]. Windup is thought to be the result of repeated stimulation of C fibers in the spinal cord posterior horn.[12] Premature infants who have repeated painful procedures may have inhibited brain growth and development.

Classification of Pain

Pain is classified in many ways. In this chapter it is primarily defined pathologically, i.e., as nociceptive and neuropathic (**Table 20-2**). *Nociceptive pain* occurs when nociceptors (pain receptors that respond to noxious stimuli) are stimulated by tissue injury. These stimuli include temperature (heat, cold), vibration, stretch, and chemical compounds released by hypoxia, acidosis, and inflammation. The pain is said to be either somatic or visceral, depending on where the pain arises. *Somatic pain* is usually the result of activation of skin, mucosa, bones, joints, muscle, and connective tissue. *Visceral pain* arises from organs within the chest and abdomen.

Table 20-2: Differentiating Features of Nocioceptive and Neuropathic Pain[13]

Types of Pain	Origin of Stimulus	Localization	Character	Referral and radiation of pain/sensory dysfunction	Examples
Nociceptive pain Superficial somatic pain	Arises from nociceptors in skin, mucosa of mouth, nose, urethra, anus, etc. Nociceptive stimulus is evident	Well localized	Usually sharp and may have a burning or pricking quality	None	• abscesses • postsurgical pain from a surgical incision • superficial trauma • superficial
Nociceptive pain Deep somatic pain	Arises from nociceptors in bone, joint, muscle and connective tissue. Nociceptive stimulus is evident	Usually well Localized with tenderness to palpation	Usually dull or aching or throbbing in quality.	In some instances, pain is referred to the overlying skin. No associated sensory dysfunction.	• bone pain due to metastasis • fractures • muscle cramps • sickle cell vasoocclusive episodes
Nociceptive pain Visceral pain	Arises from nociceptors in internal organs such as the liver, pancreas, pleura and peritoneum.	Poorly localized, diffused. Palpation over the site may elicit an accompanying somatic pain.	Usually vague, dull, aching, cramping or tightness, deep pressure, spasms, or squeezing or colicky in nature. Nausea, diaphoresis and emesis are frequently present.	In some instances, pain referred to skin supplied by same sensory roots that supply the diseased organ. There may be radiation of the visceral pain, but it will not be in a direct nerve distribution. No associated sensory dysfunction.	• pain from acid indigestion or constipation • pain due to stretching from liver metastasis, pleura stretching due to pleuritis, as in pneumonia or tuberculosis

Table 20-2 (continued)

Neuropathic pain	Is generated at various sites, and is not always stimulus dependent.	Poorly localized, diffuse pain in an area of sensory dysfunction in the area of anatomical distribution of nerve supply.	Difficult to describe and different words may be used in different populations: • burning, pricking or needle like pain; • sharp or shooting. The pain may be persisting or recurrent.	Neuropathic pain is perceived within the innervation territory of the damaged nerve. There may be abnormal radiation. The pain is associated with sensory dysfunction (dysesthesia, hypoesthesia, hyperesthesia, allodynia).	• central neuropathic pain due to spinal cord injury from trauma or tumour • painful peripheral neuropathies, due to HIV/AIDS, cancer or anticancer treatment pain (e.g. chemotherapy with vincristine) • phantom limb pain

From Ref 13. WHO. With permission.

Neuropathic pain, on the other hand, arises from nerve cell injury in the peripheral or central nervous system. It can be the result of trauma, tumors, infections, and inflammation. Fortunately, the common causes of neuropathic pain in adults (diabetic neuropathies, post-herpetic neuralgia, and trigeminal neuralgia) are uncommon in children. Table **20-3** lists the common features of neuropathic pain.

Table 20-3: Common Sensory Features of Neuropathic Pain13

Alodynia	Pain due to a stimulus that normally does not provoke pain (touch, clothing)
Hyperalgesia	Increased pain response to a normal painful stimulus (tactile or thermal (both are rare). Hyperalgesia to cold is more common than to heat
Hypoalgesia	Diminished pain response to a normally painful stimulus (tactile or thermal, both are frequent).
Paresthesia	Abnormal sensation to a stimulus that is normally not unpleasant, such as tingling, pricking or numbness. It may be spontaneous or evoked.
Dysesthesia	Unpleasant sensation. It may be spontaneous or evoked.
Hyperesthesia	Increased sensitivity to stimulation (tactile or thermal, both are rare).
Hypoesthesia	Decreased sensitivity to stimulation (tactile or thermal, both are frequent).

*From REF **13**. With Permission: WHO*

Mixed pain occurs when both neuropathic and somatic pain are present at the same time (e.g., burns).

Pain can also be defined as acute or chronic. *Acute pain* is of sudden onset, severe in intensity, and lasts less than 30 days (most surgical pain). *Chronic pain*, on the other hand, is either continuous or recurrent and last more than 30 days. Chronic pain wears on the patient and affects her/his activities, including school, playing, and family interactions. It also interferes with a child's ability to work and help support her/his family. Children with chronic pain are often depressed, are chronically fatigued, irritable, occasionally seek drugs (pain medications), and can exhibit problems with relationships.

Despite appropriate pain treatment, patients sometimes have intermittent increases in pain so called *breakthrough pain (*a temporary increase in pain beyond that which is present at baseline). When this occurs, it is usually necessary to increase the dose of her/his usual pain medications. It is important to have in place plans for giving additional doses of medication for management of breakthrough pain so the patient is not left with pain until he/she can contact someone. The patient should be told what to do (how much to increase her/his pain medications over and above the usual doses or whether he/she should take another pain medication in addition to her/his usual drug). Breakthrough pain is often treated by administering additional pain medication (often 10% of the daily dose). It is important to differentiate break through pain from *end of therapy* pain, which occurs at the time the effects of the patient's usual drug dose is

wearing off and he/she would normally take her/his usual dose of medication for pain. There is no need to increase the dose of drug in this situation. When patients who have pain must do something that will increase their pain (move, walk, climb stairs, physical therapy) it is important to give an additional dose of pain medication before this activity takes place.

Evaluation of Pain in Children

Evaluation of pain is often more difficult in children, especially young children, because they are unable to communicate the location, type (sharp, dull,) or whether anything makes the pain worse or better. As with all pain in young children, the parents can be the best source of information. Parents frequently can best interpret their child's pain when their child cannot do so.

Evaluation of pain in children begins with a comprehensive history (from the child if possible and from the parents/caretaker) and a complete physical examination. The history should include defining the word(s) used by the patient and family for pain. These words should be used when discussing pain with the child or family. How does the child respond to pain? Is it by withdrawing, crying, moving frantically, or in other ways? What makes the pain worse (movement, touching, clothing, hot or cold food or drink)? What makes it better? Does heat, cold, not moving, eating specific foods (milk) make it better? Has the family given the child medication for pain? If so, what medications were given? What doses were given? Sometimes parents tend to give more than appropriate amounts of medication to treat their child's pain. If so signs of drug toxicity should be sought. What was the response to the drugs given for pain? Did they improve/eliminate the patient's pain.

There are many methods of evaluating how much pain a patient is having, although these methods may not work well in children if they cannot communicate their symptoms effectively or at all. **Table 20-4** provides information regarding how infants and children express pain.

Table 20-4: Expression of Pain by Infants and Children

Age	Response to Pain
Infant	Facial expressions (See above), rigid body, arching of back, arms and legs extended, legs drawn up, crying/screaming, irritable, difficulty sleeping
Toddler (1-3yrs of age)	Loud mad cry, angry, withdrawn, guards painful area, difficulty sleeping
Preschool (3-6yrs)	Verbalizes pain, thrashes about in response to pain, pushes people away, clings to family and others, sleeplessness
School age	Verbalizes pain, nightmares, stalling behavior (e.g., "wait a minute"), rigid muscles, gritted teeth, closed eyes, frown, sleepless
Adolescent	Localize and verbalize pain, may deny pain around peers, sleeplessness, muscle tension, body control

Modified from ***Mazur A, Winnicki IR, Szxzepanski T. Pain Management in Children. Ann Agric Environ Med 2013;1:28-34****.*

Pain Assessment

There are multiple ways to assess pain in infants and children. **Table 20-5** is a very useful table from the WHO that lists appropriate questions to ask.[13]

Table 20-5: Questions to be Asked of Children and Parent About Pain[13]

Questions to be Asked by Health Care Providers
What words does the child and family use for pain?
What verbal or behavioral cues does the child use to express pain?
What do the parents and/or caregivers do when the child has pain?
What works best in relieving pain?
Where is the pain and what are the characteristics (sites, severity, character of pain as described by the child/parent, e.g., sharp, burning, aching, stabbing, shooting, throbbing?
How did the present pain start (was it sudden/gradual)?
How long has the pain been present (duration since onset)?
Where is the pain (single/multiple sites)?
Is the pain disturbing the child's sleep/emotional state?
Is the pain restricting the child's ability to perform physical activities (sit, stand, walk, run)?
Is the pain restricting the child's ability/willingness to interact with others, and ability to play?

***From WHO** with permission*

A child's ability to express pain is dependent on several things, including age, cognitive development, and cultural beliefs. Patients who are taught to be stoic may have significant pain and not express or be reluctant to express it because they were taught not to and that doing so is viewed as a sign of weakness. Most 2-4 year old children can verbally describe pain. By five years, they can describe pain and its strength (severity). By six years, they can differentiate levels of pain. By 7-10 years they can explain why something hurts.[14]

Clinically observing the patient in her/his response to pain is important. As stated in reference 13, the main behavioral responses to pain are:

- Facial expressions
- Body movement and body posture
- Inability to be consoled
- Crying
- Groaning, moaning

Their response to pain includes:

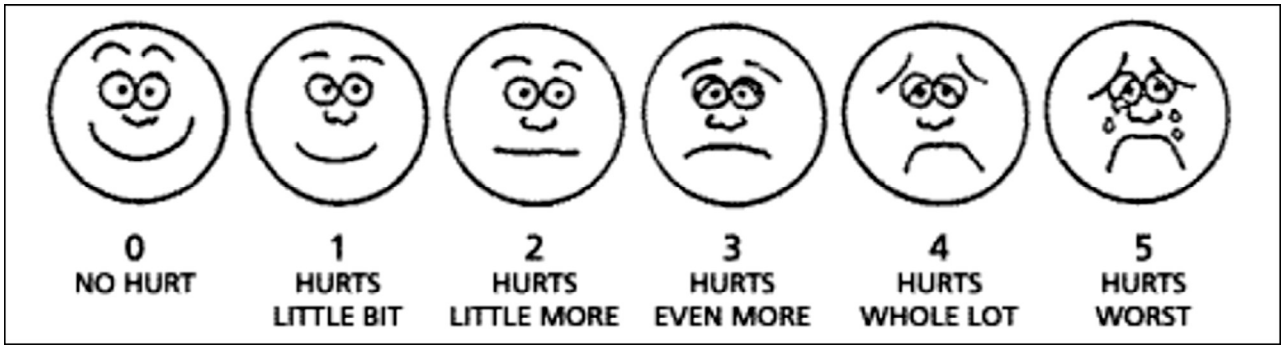
- Abnormal posturing
- Fear of being moved
- Lack of facial expression
- Lack of interest in surroundings
- Quietness
- Increased irritability
- Sleep disruption
- Anger/fear
- Change in appetite
- Poor school performance

However, as the American Society of Pediatrics stated in the year 2,000, patients who do not show signs of pain may still have significant pain.[4] The presence or absence of pain must be determined individually in every patient. It is also important to determine the patient's nutritional state. Children with poor nutrition may not respond to pain because they are under stimulated and/or developmentally delayed.

Tools for Measuring Pain

Methods (tools) have been developed to measure how much pain a patient is having. It is important to choose and use tools that are appropriate and easy of use in the society for which they are being used. They must also be appropriate for the child's age. One or two pain tools should be chosen and used so that nurses, parents, anesthetists, and surgeons become very familiar with their use. Two of these are the Wong's Pain Scale (**Figure 20-2**) and the Visual Analog Scale (**Figure 20-3**).

Figure 20-2: Wong-Baker Pain Scale for Infants and Young Children

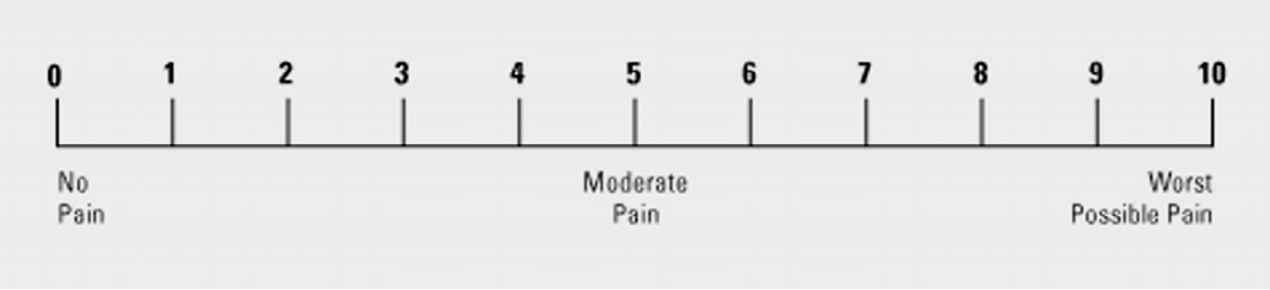


The faces are shown to the child and he/she is asked to show which face most shows how they feel. ***Wong's Essentials of Pediatric Nursing. With permission***

The Wong-Baker Scale is widely used, has been translated into many languages, and is easy to use. It was designed for children three years of age and older. The scale is used by having someone point to each face and describing to the child the pain intensity shown on each face. Then the child is asked to point to the face that best describes her/his own pain. The first face is described as no pain; each subsequent face is described as hurts a little more than the previous face until number five is reached. The fifth face is described as "hurts as much as you can imagine".

The *Visual Analog Scale* is another scale that is widely used for patients who are 7-8 years of age and older, including adolescents and adults. The scale, which consists of a straight line that is divided into 10 equal parts (**Figure 20-3**), has been translated into at least 12 languages. The results correlate significantly with the patient's and caregiver's rating of the child's pain. It is easy to use and is reproducible.

Figure 20-3: Visual Analog Scale



From: www.cancer.gov. The Visual Analog Scale is used by telling the child that zero (0) represents no pain. Five (5) is moderate pain, i.e., there is some pain but he/she can live with it. Ten is the worst pain possible. The child is then asked to point to or say what number is consistent with her/his pain. The scoring should be done before treatment and 30 minutes after treatment of the pain to be sure he/she is receiving adequate pain relief. If not, other treatment (medications) will be required.

Using these scoring systems allows for easy, repeated determinations of the child's pain and whether the treatment provided is effective or not. These are not linier scales, so four does not mean the pain is twice as severe as a score of two or that two is half of four. It does mean that the pain at four is greater than at two. The scores may be affected by outside factors (malnutrition, degree of illness, etc.). Doing the measurements at the same time each day and recording the scores, pain location, and what makes it better or worse makes it easier to determine the pain pattern and whether the treatment is effective.

Treatment of Pain

Regional anesthesia effectively treats and prevents pain. This is discussed in Chapter 21 and will not be discussed further here.

The basic principles for treatment of postoperative pain include, non-pharmacologic (distraction, placing the patient in the most comfortable position, swaddling of infants, preventing patient movement) and pharmacologic methods.

It is often best to use analgesic drugs that can be given by more than one route (oral, intravenous, subcutaneous, rectal, nasal). Intravenous or intramuscular (IM) administration of medication is the most common methods of treating postoperative pain on day one after surgery; oral drugs are commonly used thereafter. However, IM injections are painful, especially in the small muscle of infants. Clinicians should choose the most effective and least painful routes for administration of analgesics. Much higher doses of drugs are required (e.g., acetaminophen 40mg/kg as initial dose) are required to achieve adequate serum levels with rectal administration because the bioavailability of the drugs is poor by this route and because the drugs are differentially absorbed. Rectal administration of pain drugs provides only sporadic pain relief. Intramuscular and subcutaneous injections are usually avoided because they cause added pain and can sometimes cause painful sterile abscesses. Drugs given by the nasal route often cause burning and pain due to their pH (midazolam, ketamine, fentanyl). However, intranasal fentanyl may be useful for the rapid treatment of uncontrolled pain. Oral administration of analgesics is the most acceptable rout for medicating children. If an IV is already in place, many drugs can be given intravenously. IV administration of drugs assures that the entire drug dose gets into the blood stream. Drugs given orally, on the other hand, are absorbed to varying degrees. Once absorbed, as much as 50% of the drug absorbed from the stomach can be metabolized in the liver before it gets to the central circulation (midazolam). Thus, the effective dose is only one-half of that given. This is why larger doses of drug are required to get the desired effect.

WHO recommends treatment of pediatric pain by:[13]

- Using the two step method
- Dosing the drug at regular intervals, not just when the patient is having pain
- Using an appropriate and effective route of administration
- Adjusting treatment to the individual patient

The *two-step* approach to the treatment of pain was originally devised by the WHO for the treatment of chronic pain (e.g., cancer pain) but has been widely and effectively adapted for use with acute postoperative pain. This will be followed in the remainder of this chapter because their document is widely available.[13] Pain is divided into mild (step 1) and moderate/severe (step 2) pain, and the therapy provided is based on the degree of pain.

Step one provides treatment for mild postoperative pain (**Table 20-6**). Medications used in this step include prostaglandin synthetase inhibitors (acetaminophen, acetylsalicylic acid) and non-steroidal anti-inflammatory agents (NSAID – e.g., ibuprofen). It is often useful to use one of these drugs, e.g., ibuprofen, as the primary treatment for pain and another one, e.g., acetaminophen,

for break through pain when it occurs. Effective postoperative pain relief requires that pain medications be given around the clock, i.e., on a schedule every four or more hours apart, depending on the age of the child and the half-life of the drug. If we wait for the child to complain or demonstrate signs of pain, it is too late. With fixed interval dosing, pain scores are lower and the overall dose of narcotic (when used) is less. For children who can take medications orally, paracetamol (acetaminophen) 15mg/kg and ibuprofen 5-10mg/kg/dose every 6-8 hours are the drugs of choice. For younger children who are three months of age and older, a liquid form of these drugs is available that is easily swallowed. The maximum dose per day is 40mg/kg. Only paracetamol is available for patients less than three months of age. Like all drugs, paracetamol and ibuprofen have potential toxicity, which include gastrointestinal bleeding, and renal failure with ibuprofen and liver failure with paracetamol. Consequently, these drugs should be given cautiously to patients who have bleeding difficulties or renal and/or liver problems. These serious complications are infrequent when the drugs are given over the amount of time required for surgical and trauma pain, and if they are given in the appropriate doses, especially in children. The most common cause of liver failure from acetaminophen is an overdose of drug that occurs when a child ingests an overdose of the drug at home, not in hospital.

Table 20-6: WHO Recommended Initial Doses of Non-Opioid Analgesics[15]

Drug	Neonates 0-29 days	Infants 30d to 3mo	Infants 3mo to 12mo	Maximum daily dose
[a]**Paracetamol**	5-10mg/kg every 6-8hrs	10mg/kg every 4-6hrs	10-15mg/kg every 4-6hhrs	4 doses per day
Ibuprofen			5-10mg/kg every 6-8hrs	Child 40mg/kg/day

[a]*Over two years of age, a dose of 15mg/kg (maximum 40mg/kg) can be given every six hours.*

Step two provides treatment of patients with moderate or severe pain when paracetamol and/or ibupropfen do not completely relieve their pain. When this occurs, clinicians should consider administering narcotics to treat the patient's pain. Narcotic administration alone is more likely to cause unwanted complications (hypoventilation, apnea, itching, vomiting, nausea). To reduce the dose of narcotic used and still provide adequate pain relief, it is often useful to combine narcotics with paracetamol or ibuprofen. This reduces unwanted complications, but does not totally eliminate them. One regimen gives the dose of narcotic and then gives the dose of paracetamol or ibuprofen about two hours later. Remember not to exceed the maximum doses of either paracetamol or ibuprofen. **Table 20-7** gives the starting doses of two common narcotics. If these starting doses do not relieve the patient's pain, the dose of drug is increased in a stepwise manner until her/his pain is relieved. In the end, the appropriate dose of narcotic is the one that effectively relieves the patient's pain without causing unwanted side-effects. While the WHO

states that "there is no upper limit" on narcotic dose, it is important to be cautious and watch for opioid related side-effects such as respiratory depression and not to administer enough narcotic to cause severe adverse conditions. (**See below**). We agree that the appropriate dose is that which relieves the child's pain but still does not result in adverse side-effects. Giving enough narcotic to completely eliminate all pain may result in apnea and bradycardia. When administering narcotics, it is important to repeatedly evaluate patients for side effects. It is important to educate the parents about what side effect to look for and have them assist in the ongoing monitoring of the child, especially in countries that have few nurses and little technology. It is also important to determine if the patient has previously used pain-relieving medications, especially narcotics, because those who are narcotic naive may metabolize the drug more slowly and have a more profound response to the drug, including respiratory depression and apnea. It is often appropriate to intravenously administer half the usual dose of narcotic and determine its effect. Is the pain relieved? Is there evidence of hypoventilation? Fear that patients and hospital personnel will become addicted to narcotics is a common reason why narcotics are unavailable or avoided in some countries. Treating patients who have pain postoperatively with narcotics for several days does *not* lead to narcotic addiction, especially with morphine use.

Table 20-7: WHO Recommended Starting Doses of Narcotics for Infants.[15]

Medication	Route of Administration	Starting Dose
Morphine	IV, SQ	1-6mo: 25-50mcg/kg/dose every 4-6hrs
Fentanyl	IV	1-2mcg/kg/dose every 2-4hrs

IV = intravenous; SQ = subcutaneous; hrs. = hours; mcg = micrograms. Narcotics should be administered slowly over several minutes while observing the patient's respiratory rate and response to pain.

Two narcotics (fentanyl and morphine) are readily available in many countries for relief of postoperative pain (**Table 20-7**). Both effectively relieve severe pain, assuming the dose of drug given is adequate. *Fentanyl* is commonly used in Post Anesthesia Recovery Rooms (PACU) and the Intensive Care Unit (ICU) for acute treatment of pain, while *morphine* is used for longer-term treatment once the patient has left the PACU and ICU. *Methadone*, when available, may be a good choice of narcotic for longer-term use because its pain relieving effects last about 12 hours, and the same doses can be given orally and intravenously. Due to its longer half-life, methadone administration reduces the variation (peaks and valleys) in pain each day. Methadone also causes less respiratory depression than other potent narcotics.

Morphine is a very effective, inexpensive, widely available narcotic. However, in some countries its use is very tightly controlled. One of its main advantages is that it can be administered by multiple routes, including IV and by mouth (**Tables 20-8 and 20-9**). It can also be administered by continuous infusion. However, when given by continuous infusion, a properly calibrated infusion pump must be used. This reduces the chances that the patient will receive an inadvertent

overdose of narcotic. Consideration for ICU or monitored room administration is wise with trained nurses so resuscitation can begin immediately if the patient develops apnea. The response to morphine is also age dependent. Neonates and young infants have immature enzyme systems for metabolizing morphine. By six months of age these enzymes are usually present at adult levels, unless the child was born prematurely. Maturity of these enzyme systems affects how often narcotics can be given. In 1-10 year olds, morphine is given every four hours; in 1-3 month olds it is given every six hours; in newborns it is sometimes only required once or twice a day. If a child is malnourished or has liver or kidney dysfunction, drug clearance may be slower, and the time between narcotic doses may have to be increased.

Remember, it usually takes about five drug half-lives to reach a steady state. (A *half-life* is the amount of time needed for the concentration of a drug to decrease to half its initial value). For morphine, the half-life is about three hours. Thus, it takes about 15hrs for a steady state to be reached. Administering the drug every four hours is usually sufficient to attain adequate blood levels after the child is one year of age. Patients may require a higher or more frequent dosing of morphine the first day after surgery, due to pain intensity. Children who are having significant pain seldom are sedated or have respiratory depression. Only when their pain is well controlled (abolished) do these problems occur. Consequently, the goal of pain control may not be the complete abolition of pain (a pain score of zero). There may be increased safety with scores of 1 – 3. A realistic goal for optimal pain control is to give that amount of medication that optimizes analgesia and functional activity of the patient. Using adjuvants facilitates pain relief and reduces the amount of narcotic required. The latter reduces the incidence of narcotic side effects.

Fentanyl is commonly used for acute treatment of pain, but because of its short half-life, it must be given every 45-60 minutes or must be given by continuous infusion to provide adequate pain relief. Fentanyl is very helpful for treatment of pain in an ICU or the PACU, but is not a good choice of narcotic for long-term treatment of pain on the ward.

Pethidine (meperidine) is available in some countries but should be used with caution because it is metabolized to normeperidine, which can be neurotoxic and may cause seizures. If pethadine is the only narcotic available, then it should be used to treat moderate and severe pain. Otherwise there are better choices.

Oxycodone and hydromorphone are useful drugs for treatment of long-term pain but usually are not optimal for the first few days after surgery (unless the surgery is minor). Once the acute phase of surgical recovery has passed (3 -4 days), these drugs can be used effectively to treat pain, often when given in combination with NSAIDS. While oxycodone is the active agent in the drug that relieves pain and does not have to be metabolized to morphine, it has a metabolite (oxymorphone) that is active. It is excreted in part by the kidneys and can accumulate in blood and tissue in patients with renal failure.

All narcotics have side effects, including potentially causing excessive sedation without providing adequate pain control. Narcotics also cause nausea and severe itching. This problem of over sedation without adequate pain relief is often treated by reducing the dose of narcotic and by adding another drug (e.g., NSAIDS) to improve pain relief. Both nausea and itching associated with opioid administration can be treated by a reduction of dosage and sometimes by continuous infusion of low-dose naloxone. It is necessary to give naloxone by continuous infusion because the half-life of the naloxone is much shorter than the half-life of narcotics such as morphine. Also, with a continuous infusion, it is possible to titrate the dose of naloxone so that unwanted narcotic side effects are relieved and still not take away the patient's pain relief. The starting dose is usually 0.5mcg/kg/hr.; the dose is increased progressively to 2mcg/kg/hr. in 0.5mcg/kg/hr. steps if necessary. Naloxone is placed into a known volume of fluid (e.g., 2mg naloxone mixed in 250ml of NS) and run at the above infusion rates. If available, it is better to use an infusion pump to assure constant drug infusion. If the infusion rate is too high, the patient will have severe pain because naloxone will reverse the pain relieving effects of the narcotic. If morphine is being infused continuously, naloxone can be added to the morphine infusion, and the two drugs can be infused jointly. However, this requires determining the effective dose of naloxone and adding enough naloxone to the morphine solution to relieve the itching and nausea without removing pain relief. When the two drugs are mixed together, it is difficult to change the concentration of either drug without making a new mixture, which may be wasteful. Diphenhydramine (Benadryl) is not a good choice for the treatment of itching, since only 10% of itching from morphine and 0% from Fentanyl is due to histamine release.

Treatment of nausea is a complicated because nausea can be caused by five different receptors. Finding the right drug (other than naloxone) to treat the specific cause in a give patient is often by trial and error, i.e., multiple drugs must be tried until one is found that treats the problem. Nausea can sometimes be treated with Ondansetron (4-8mg)) or with metaclopromide (0.1-0.2mg/kg/dose every 6-8hrs). Children over 14yrs of age can receive 10mg total every 6-8hrs. Some patients respond to low-dose benzodiazepines, but these drugs also cause sedation. If sedation is not particularly desired, benzodiazepines may be a poor choice for the treatment of nausea.

Table 20-8: WHO Recommended Starting Narcotic Doses for 1 month to 1-Year Old Children[15]

Medication	Route of Administration	Starting Dose
Morphine	Oral (immediate release)	80-200mcg/kg every 4hrs
	IV or SQ	1-6months - 100mcg/kg/every 6hrs 6-12months – 100mcg/kg/every 4hrs (maximum dose 2.5mg/dose
	IV infusion	1-6 months – initial dose 50mcg/kg then 10-30mcg/kg/hr. 6-12 months – initial IV dose 100—200mcg/kg then 10-30mcg/kg/hr.
	IV injection and SQ	25-50mcg every 6hrs
	IV infusion	1-6mo initial dose 50mcg/kg then 10-30mcg/kg/hr. 6-12mo initial dose 100-200mcg/kg then 20-30mcg/kg/hr.
Fentanyl	IV Injection IV infusion	1-2mcg/kg every 2-4hrs Initial IV dose 1-2mcg/kg, then 0.5-1mgc/kg/hr.
Oxycodone	Oral	50-125mcg/kg every 4hrs

IV = intravenous; SQ = subcutaneous; mcg = micrograms; kg = kilograms; hr. = hour; mo. = month

Table 20-9: WHO Recommended Starting Narcotic Doses for 1-12 Year Old Children[15]

Medication	Route of Administration	Starting Dose
Morphine	IV	0.1-0.15mg/kg every 4 hours
	Subcutaneous	0.1-0.2mg/kg every 4 hours – maximum dose 2.5mg
	Oral	0.2-0.4mg/kg every 4 hours
	Infusion	Initial dose 0.1mg/kg then 0.1-0.2mg/kg/min – maximum dose 2-2.5mg
Fentanyl	IV	1-2mcg/kg every 30-60 minutes
	IV infusion	Initial dose 1-2mcg/kg then 1mcg/kg/hr.
[a]Methadone	IV and subcutaneous	0.1mg/kg every 4 hours for first two doses then every 6-12 hours.
	Oral	0.1mg/kg every four hours for the first two doses and then every 6-12 hours
Hydromorp-hone	IV or SQ injection	0.15mg/kg every 3-6 hour
	Oral	30-80mcg/kg every 3 hours – maximum dose 2mg/dose
Oxycodone	Oral	0.125-0.200mg/kg every four hours – maximum dose 5mg/dose

[a] dose of methadone is the same for all routes of administration. These are starting doses. They may have to be increased as needed to treat the patient's pain. IV = intravenous; mcg = micrograms; SQ = subcutaneous

Codeine is not recommended for use with children because of their varied response to the drug. About 10-15 percent of people (children and adults) lack the liver enzymes required to metabolize codeine to morphine (which is the primary source of pain relief with this drug). Failure to metabolize codeine to morphine prevents the drug from providing pain relief beyond that of the drug with which it is mixed (paracetamol, ibuprofen). On the other hand, some people (including children) are *hyper metabolizers* of codeine and much more rapidly convert codeine to morphine resulting in rapidly elevated morphine blood levels. This has caused respiratory depression, apnea, and death in some children.[16,17,18] Furthermore, for the first five years of life, children have only about 25% of the amount of the enzyme (CYP2D6) they have later in life. Thus, most children convert much less codeine to morphine and get much less pain relief form the drug. This also means that *all* drugs (codeine, tramadol, dihydrocodeine) that require conversion of the drug to morphine or another active ingredient for pain relief will not be as effective in children who lack this enzyme for drug conversion. This is why the WHO suggests morphine as the drug of choice for pain relief. Morphine's effects are well known, does not have to be converted to another drug to relieve pain, and is inexpensive.

Tramadol is also used for treatment of acute pain. It has two actions: 1) it acts as a weak narcotic and 2) it inhibits reuptake of serotonin-norepinephrine. The half-life of tramadol is 6-7hrs; its active metabolites have half-lives of 10-11hrs. Like many other drugs, the kidneys excrete one of the active metabolites. Consequently, renal insufficiency or failure prolongs its effects. The usual dose of tramadol is 0.5 – 1mg/kg every 3-to-4 hours (maximum dose 50mg/dose). It is often combined with other drugs (paracetamol) to improve its pain relieving characteristics. The pain relieving effects of tramadol last about six hours. Tramadol is thought to be about as effective as morphine for the treatment of moderate pain. The drug is only available in oral and IV forms. The complications associated with tramadol are considered minor and include nausea, dizziness, dry mouth, abdominal pain, vomiting, constipation, and drowsiness. Respiratory depression and constipation occur less often than with other narcotics. Since its metabolism depends on the CYP2D6 enzyme and several others, it should be used with care in patients who lack this enzyme or who are thought to be hyper metabolizers. Patients who lack CYP2D6 will get less pain relief from the drug. Pain relief can be improved by increasing the dose of the drug by about 30%. Since the kidney is responsible for removing a significant amount of the drug from the body, renal insufficiency or failure may affect dosing.

All narcotics should be used with care, and attempts should be made to prevent complications when possible. Monitoring the patient for drug side effects is the only way to determine the presence of potential complications.

Tolerance to the effects of narcotic may occur, especially to fentanyl. The dose that is effective today may not be affective tomorrow or the day after. This must be taken into account, and the patient must be evaluated several times a day to ensure he/she is still getting pain relief from the dose of drug being given. If this is not the case, it is often necessary to increase in the dose of drug administered. If the narcotic is suddenly withdrawn (especially fentanyl), the patient may show signs of drug withdrawal, including irritability, anxiety, sleeplessness, increased muscle tone, nausea, vomiting, diarrhea, and loss of appetite. Children undergoing withdrawal can also have evidence of tachypnea, tachycardia, fever, sweating, and hypertension. Such patients are often inconsolable.

What should we do if the patient receives an overdose of narcotic? As in all resuscitations, Airway, Breathing, and Cardiac support are the first steps **See Chapter 5**). Oxygen administration and ventilation of the lungs with a bag-and-mask are immediately provided. If there is severe bradycardia, it may be necessary to administer atropine 30mcg/kg (maximum dose 1mg) or to perform closed chest cardiac massage. If respiratory depression or apnea occurs, naloxone 10-20mcg/kg is given and its effect evaluated. The dose is increased by 1mcg/kg every three minutes until spontaneous breathing is resumed. Breathing usually returns before the withdrawal syndrome or lack of pain relief occurs. Once spontaneous breathing has resumed, it is necessary to infuse low dose naloxone to sustain breathing. Remember, the half-life of naloxone is much

shorter than the half-life of most narcotics. Consequently, it may be necessary to infused naloxone for several hours.

Summary

Pain is common, especially following surgery. It is incumbent on everyone caring for children to assure that the child's pain is adequately treated and that he/she is comfortable. For mild pain acetaminophen and ibuprofen are the first line drugs. For moderate and severe pain, morphine and fentanyl are appropriate drugs, with or without acetaminophen and/or ibuprofen. When narcotics are given, the patient must be monitored for hypoventilation or apnea. If either occurs naloxone administration is small doses is appropriate, but it must be given by IV infusion or by the anesthetist sitting with the patient and administering intermittent doses that are sufficient to allow normal breathing without taking away the patient's pain relief. Positioning the patient, massage, and distraction may reduce the pain. In some countries acupuncture or acupressure may effectively relieve pain.

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Chapter 21
PEDIATRIC REGIONAL ANESTHESIA

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Introduction

The modern practice of pediatric regional anesthesia has become complex. Ultrasound guidance, electrical stimulation, specialized needles and catheters, designer local anesthetics and adjuncts, etc., are now the *de facto* standards of care in the developed world. However, regional anesthesia has been practiced for many decades by a dedicated group of practitioners long before any of these modern developments occurred. In fact, some of the earliest anesthetics were regional anesthetics. While many nerve blocks have become more reliable, and somewhat safer with the application of new technology, many basic blocks are still manageable by traditional techniques and equipment.

Regional anesthesia is the generic term used to describe peripheral nerve, plexus, and neuraxial blockade, all of which are used to provide anesthesia and analgesia to a specific *region* of the body. Some of these techniques have existed for well over a century. However, as general anesthesia improved over that same century, and complication rates related to general anesthesia plummeted, the use of regional anesthesia, at least in the form of plexus and peripheral nerve blocks, waned significantly. While epidural and spinal anesthesia continued to be practiced routinely, plexus and peripheral nerve blocks struggled under the weight of imperfect success and modest, but significant, complication rates. The development of electrical nerve stimulation for nerve localization incrementally improved the success rate of peripheral nerve blocks, but still did not guarantee success. More recently the availability of portable, easy to use, and somewhat affordable ultrasound machines has fueled a wave of interest in performing peripheral nerve blocks. Affordable being a relative term of course. Ultrasound guided nerve blocks have a near perfect success rate in the hands of a well-practiced anesthesiologists. Some complication rates, however, have not measurably decreased despite this technology. And while certain incidents, such as intravascular drug injection, have decreased in the ultrasound era, other problems such as transient or permanent nerve injury, while very rare, still persist. Neuraxial blockade also improved gradually throughout this period, mostly due to equipment improvements, but also with increased knowledge of local anesthetic, opiate, and adjuvant drug pharmacology. While all these developments have clearly brought regional anesthesia into the mainstream of anesthetic practice, there continue to be challenges, especially where resources are limited.

This brief history, ignores the simple fact that much of the developing world is without even the most basic of anesthesia supplies, let alone special needles, catheters, drugs and equipment to localize nerves. Some sites may have an abundance of needles and local anesthetics, while others may have only ketamine or halothane available to provide anesthesia. And while a discussion of every conceivable combination of resources is not possible in this short chapter, my goal is to describe ways in which regional anesthesia may contribute to patient care, even when resources are limited.

This chapter will address the basic indications, contraindications, benefits, and risks of regional anesthesia in infants, children and young adults. It is not meant to be a step-by-step technical manual for the performance of every potential block, but will point out some important procedural details that differentiate these techniques from adult practice. Finally, this discussion is tempered by the knowledge that many sites are severely limited by the scarcity of equipment and human resources.

PEDIATRIC versus ADULT REGIONAL ANESTHESIA

The mantra of the pediatric anesthesiologist has historically been that "pediatric patients are not simply small adults." This principle can and should be applied when considering regional anesthesia as well. There are many anatomic differences other than simply "smaller." These differences necessitate alterations of technique when performing blocks in infants and children. Additionally, physiology, pharmacology, and behavioral issues specific to children will dictate many nuances. Block choice, drug choice, dosing, awake-versus-asleep block placement, likelihood of success, are some examples of decisions that are influenced by pediatric specific practice.

Pediatric anesthesiologists generally are accustomed to placing neuraxial, plexus and peripheral nerve blocks in patients under general anesthesia, or at minimum, when they are heavily sedated. This is a contentious topic with a great deal of opinion but little definitive evidence to support any position. Guidelines from the American Society of Regional Anesthesia (ASRA), do recommend performing both peripheral and neuraxial blocks in adults while awake. Recommendations from ASRA for children, however, agree with most pediatric anesthesiologists that careful application of regional anesthesia in anesthetized patients is safe and effective.[4] Large surveys documenting complications from pediatric regional anesthesia support this view. The complication rate of blocks performed on anesthetized pediatric patients does not exceed the complication rate of blocks performed on adults who are awake. Performance of most blocks in children, while certainly *possible* without heavy sedation or general anesthesia, can be a trying experience for the patient, staff, and anesthesiologist. Since most regional blocks are placed with the goal of

[4] *With the possible exception of interscalene nerve blocks, which ASRA suggests should never be performed on anesthetized patients.*

optimizing postoperative analgesia, most will be performed after general anesthesia is induced.

In some cultures and in this author's experience, many minor procedures, including regional anesthetic blocks, *can* easily be done with gentle persuasion and perhaps a hovering parent or authority figure on children as young as five years of age. This is an example of how local norms, when combined with patient and parent temperament, can influence the conduct of the anesthetic. Parental presence is more than enough persuasion to keep a school age child still in some parts of the world, while in other cultures this approach would have little hope of success. Regardless, even when the regional technique and conditions are perfect, most children cannot tolerate the psychologic demands of undergoing surgery while awake, and the stillness requirement is beyond all but the most calm and mature children. As a result, general anesthesia or heavy sedation is still usually required, and placement of blocks on children who are awake is rarely truly indicated or necessary.

Placement of nerve blocks and neuraxial block under moderate or heavy sedation is an alternative to general anesthesia in select patients. However, sedation can often have the opposite of the intended effect and result in an uncooperative, partially sedated or agitated child. Sedation often tends to be a more dynamic and difficult task than general anesthesia, and is usually best done in circumstances where another trained practitioner can dedicate him/herself solely to that task while the anesthetist can dedicate his/her attention solely to performance of the block.

BENEFITS OF REGIONAL ANESTHESIA

As discussed above, most regional anesthetics in children are placed with the patient heavily sedated or under general anesthesia. So if the patient needs a general anesthetic anyway, why bother adding a regional anesthetic? There are several significant benefits to adding regional anesthesia to the anesthetic plan, many of which have been demonstrated in adult studies. The pediatric literature is less clear, but despite this, some overall conclusions can be made about using regional anesthesia in children.

1. Use of regional anesthesia decreases the neuroendocrine stress response to surgery. There is a dramatic sympathetic, hormonal, and immunologic response to the barrage of afferent inputs resulting from the surgical insult. While high doses of opiates and general anesthesia attenuate the effects of these insults, the only reliable way to halt these responses is via regional blockade. It is likely that many perioperative complications, namely cardiovascular, pulmonary, coagulation related, and gastrointestinal events, are linked in part to the stress response. There is evidence for decreased morbidity and mortality in adults when regional anesthesia is used, though the evidence is not completely conclusive at present. However, even modest improvements in morbidity and mortality are relevant outcomes. Ongoing research continues

into the complex biology of the surgical stress response and its implications in both adult and pediatric patients.

2. Addition of regional anesthesia to general anesthesia can reduce the amount of general anesthetic agents required to maintain unconsciousness and stillness during surgery, and can significantly reduce the opiate requirements during and after surgery. Smaller doses and fewer medications generally mean less cost and fewer side effects. Opiates in particular, have a wide variety of side effects, which can be minimized by using regional anesthesia for postoperative pain control.
3. It is clear that regional anesthesia improves acute postoperative pain management. Improved pain control is a worthy goal in and of itself on the basis of humanitarian empathy. However, there may be benefits to decreased pain, other than patient comfort. Patients with less pain immediately after surgery are less likely to develop chronic pain syndromes. Neonates who are repeatedly exposed to painful events show evidence of detectable changes in behavior later in life. Finally, functional recovery from surgery is also a benefit of good acute pain management. Despite the fact that most functional recovery data come from the adult literature and rarely focus solely on regional blocks, better pain management certainly improves patient satisfaction and likely promotes more rapid and complete functional recovery. Fewer physiologic and behavioral complications are all likely benefits.
4. Economic benefits associated with regional anesthesia include avoidance of postoperative admission for pain management and decreased utilization of post anesthesia care units. The economic advantage of ambulatory surgery sometimes hinges on management of pain and the complications of that management. Given the cost of staffing post anesthesia care units, reduction in this need, even if only a few minutes per case, can have a significant cumulative cost saving.
5. Regional anesthesia, when used in lieu of general anesthesia, is indicated in a limited number of small, high-risk infants in whom general anesthesia carries an inordinately high likelihood of complications. In these cases, avoidance of a high-risk general anesthetic is of benefit. These specific situations will be pointed out below.

It is often difficult to objectively establish the risk-benefit ratio of such interventions. While complication rates have been published and reveal very low overall risk, the benefit is more difficult to quantitate, as there are few high quality studies. Practitioners who routinely use regional anesthesia have no doubts as to its benefits, especially with regards to its superior pain control. However, for some, it may be difficult to justify even rare potential complications for pain control alone. Since the experience of pain has such a profound emotional component, it is often difficult to prejudge the importance of good pain control for a specific patient or family. Additionally, pain is much more of an emotional event in children, and as such may take on a level of importance that is beyond what we can objectively understand. The risk-benefit ratio for every patient should be determined individually, and with as much input from the patient (if old

enough) and/or family members as possible. Again, cultural norms play a significant role in this regard. People from traditionally "stoic" cultures may be less willing to accept complication risk for the benefit of short-term pain alleviation, while those from other cultures may have significantly different attitudes on this subject.

GENERAL RISK CONSIDERATIONS

Infectious Complications

All neuraxial and peripheral nerve blocks are invasive procedures that carry a small risk of infection. While infectious risk is quite low, infections, especially those occurring in the neuraxis, are potentially catastrophic. Meningitis and epidural abscess have both been reported after neuraxial anesthesia. Fortunately, basic sterile technique is usually all that is required to prevent most infections. Obviously, needles and catheters must be sterilized. Skin preparation with iodine, chlorhexidine, or alcohol-based solutions is adequate when used appropriately. Alcohol and chlorhexidine solutions must be allowed to dry in order to be effective anti bacterial agents, and there is a theoretic risk of neurotoxicity if they are transported by the needle and neural structures are exposed to these agents. Since some neuraxial infections can be caused by respiratory flora, it is appropriate for the anesthetist to wear a facemask when performing neuraxial blockade. When inserting neuraxial and peripheral indwelling catheters, the operator should maintain a sterile field and wear sterile gloves. Single shot blocks, however, can usually be performed safely using skin prep, followed by a "no touch" technique using a sterile needle.[5]

Bleeding Complications

In otherwise normal patients, the risk of bleeding is quite low. Epidural hematoma after neuraxial blockade has been reported in otherwise normal adults but is extraordinarily rare. There are no equivalent reports in pediatric patients. However, patients with bleeding disorders are at a significantly higher risk for epidural hematoma, and *should not receive any sort of neuraxial block*. This includes patients with hematologic or systemic disorders that lead to bleeding, or patients being treated with anticoagulant medications. Most case reports of epidural hematoma after neuraxial blockade are in adult patients being treated with low molecular weight heparin. Epidural hematoma must be treated aggressively and rapidly, usually with neurosurgical intervention to decompress the spinal cord, in order to avoid permanent neurologic deficit. There

[5] In the *"no touch" technique an area of skin that has been prepped with antiseptic remains untouched by any non-sterile items, including hands, gloves, or equipment for the entire performance of the procedure. Since infective agents do not "jump" from non-sterile areas to sterile areas without direct contact, this technique is considered acceptable to prevent inoculation of a puncture site.*

are few data in the pediatric population, but the safest approach for patients know to have bleeding disorders is to avoid spinal or epidural anesthesia altogether.

Plexus and peripheral blocks are a bit more of a gray area. The risk of bleeding from most peripheral blocks is relatively low, even in patients with inherent or acquired coagulation defects. A vessel puncture is usually well tolerated and easily dealt with. Some peripheral blocks may have a higher incidence of vessel injury and hematoma formation than others (e.g., axillary versus ilioinguinal block.) Additionally, peripheral blocks have a far lower potential for injury, even if a large hematoma is created during the block, as the hematoma does not typically occur in non-compliant spaces. The use of ultrasound (when available) is the best way to avoid puncturing vessels during plexus and peripheral nerve blocks. For those not using ultrasound, the most conservative approach would be to avoid peripheral blockade in patients with a known bleeding diathesis. The benefit of a peripheral block in a coagulopathic patient must be balanced against the risk of bleeding on a case per case basis.

Nerve Injury

Several very large studies in children have documented a very low rate of temporary nerve dysfunction or permanent neurologic deficits from any regional anesthetic. The permanent deficit rate from nerve injury in adults undergoing neuraxial anesthesia is estimated to be 0.04%, whereas in children, the rate is reported as essentially zero. Despite this overall safety record, there *are* several case reports (outside of the above mentioned large scale studies) of severe permanent neurologic deficits due to spinal cord injury occurring during placement of regional anesthetics in children (both epidural and interscalene blocks). After peripheral blockade in adults, the temporary nerve dysfunction rate is significantly higher (0.3 - 3%), with the vast majority of those dysfunctional symptoms disappearing within several weeks to months. A similar low rate of neurologic complications has been demonstrated for children undergoing peripheral nerve blocks, with few if any true permanent injuries.

Post block nerve dysfunction may be the result of local anesthetic toxicity rather than mechanical injury from needle trauma or pressure injury from injection. The most compelling evidence to support this is that the relative rate of nerve injury in the pre versus post ultrasound era appears to be similar. It is highly likely that before ultrasound was used routinely, passage of needles through nerve bundles was common, if not desired (i.e. paresthesia seeking techniques). Additionally, when using ultrasound and nerve stimulation together, we often see that contact of the nerve by the tip of the needle does not always result in a motor response or a paresthesia. Again, this implies that prior to using ultrasound, nerve-needle contact was likely common while seeking muscle twitches. Pressure injuries which are caused by direct injection of drugs into nerves has been speculated to occur; we would expect this type of event to be far less likely in the ultrasound era and there are studies which place significant doubt on this mechanism of damage.

Ischemia from epinephrine containing solutions has also been suspected as an additional possible mechanism of nerve injury, though the data are not conclusive. And finally, there are numerous case reports associating preservatives used in local anesthetic preparations (especially benzyl alcohol) as the causative agent in nerve injury after neuraxial blockade. Most of these observations indirectly implicate the local anesthetic itself as the problem. At present, however, there is not enough evidence to support one particular mechanism for nerve injury over another. Thus, it would seem prudent to avoid multiple needle passes, high-pressure injections of drugs, very large doses of local anesthetics, and medications containing preservatives.

LOCAL ANESTHETIC PHARMACOLOGY

Local anesthetics are most easily classified by their chemical structures, that is amides or esters. However, it is far more useful to classify these drugs by their clinical properties, the most relevant being onset time, duration of action, and potential for toxicity. A few of their clinical differences are of particular interest to us. Lidocaine and bupivacaine are the most commonly used local anesthetics and are readily available throughout the world. While there are locations where tetracaine, chloroprocaine, ropivacaine, levobupivacaine and mepivacaine may be available, this chapter will focus primarily on lidocaine and bupivacaine.

Table 21-1: Most Common Local Anesthetics Classified by Duration of Action

Short	Medium	Long
chloroprocaine procaine	prilocaine lidocaine mepivacaine	tetracaine bupivacaine ropivacaine levobupivacaine

All local anesthetics impart their biologic effects, both therapeutic and toxic, by binding to voltage gated Na^+ channels in nerve and other specialized tissue (e.g., conduction fibers in cardiac muscle). Local anesthetic systemic toxicity is usually the result of an inadvertent intravascular injection of drug, but occasionally can result from over dosage and uptake of drug. This is a feared complication of most types of regional anesthesia. The tighter range of doses and narrow therapeutic window for these drugs in neonates, infants and small children magnifies the problem. Additionally, limited clearance of local anesthetics by neonates and infants, as well as decreased serum protein binding, further narrows the therapeutic window of these drugs. Great care should always be taken to calculate the absolute maximum dose of the drug when administering local anesthetics.

Table 21-2: Maximal Allowable Doses for Common Local Anesthetics

Drug	Single Dose (mg/kg)	Continuous infusion rate in patients > 6 months old (mg/kg/hr.)	Continuous infusion rate in patients <6 months old (mg/kg/hr.)
bupivacaine	2.5-3	0.4-0.5	0.2-0.25
lidocaine	4-7	1.6	0.8
lidocaine w/epi	6-10	n/a	n/a

Epinephrine has little influence on plasma levels for bupivacaine, probably because bupivacaine has some inherent vasoconstrictor effect on its own. There is a notable idiosyncrasy about lidocaine in patients with right-to-left shunting congenital cardiac lesions. Since lidocaine is normally heavily sequestered in pulmonary tissue, plasma lidocaine levels are significantly higher in patients with right-to-left cardiac or pulmonary shunts. The wide range of maximal allowable doses reflect the fact that anesthetic uptake is significantly different with different blocks. Intrapleural and intercostal blocks, for example, are associated with the highest uptake of drug. Conversely there is little drug uptake following wound infiltration with local anesthetics or spinal blocks. Plexus blocks and epidural blocks are somewhere in between these extremes.

In neonates and infants, incomplete myelination of most nerves allows the use of lower doses of local anesthetic than those required for a similar block in an adult. After two years of age the dose of drug required (per kg) appears to approach that required for older children and adults. Neonatal animals also appear to be relatively resistant to the direct cardiac toxicity of these drugs. Whether this resistance to direct cardiotoxicity also exists in human infants is not known, so it is probably best to assume there is no difference in the response of adults and infants to the systemic effects of these drugs. The practitioner should always take appropriate precautions to prevent inadvertent intravascular injection of local anesthetic, and to avoid administering local anesthetics in excess of the recommended dose ranges.

While measuring drug levels is potentially useful, it is expensive, and seldom available. The diagnosis of local anesthetic toxicity is primarily clinical. All local anesthetics demonstrate the same basic sequence of toxicity when the plasma level rises to toxic levels, with the notable exception that bupivacaine's cardiac toxicity is particularly difficult to treat. The central nervous system manifestations of local anesthetic toxicity have a typical prodrome. These symptoms and signs, in order of lowest to highest plasma concentration of local anesthetic, are vertigo/dizziness,

anxiety, tinnitus, circumoral numbness, tremors, myoclonic movements, seizures, and coma. Cardiovascular toxicity usually occurs at approximately three times the plasma concentration required for seizure activity. At lower drug plasma concentrations, CNS excitement is associated with tachycardia, hypertension, and increased cardiac output. As plasma concentrations rise, true cardiovascular depression ensues with the occurrence of hypotension, decreased cardiac output, arrhythmias and finally cardiac arrest.

It is important to remember that this typical sequence of events is not absolute. It is possible, for instance, to see seizures or arrhythmias as the first sign of local anesthetic toxicity, especially when the plasma concentration rises rapidly. Treatment of local anesthetic toxicity has traditionally been supportive: manage the airway, control seizure activity with benzodiazepenes (e.g. diazepam, lorazepam, midazolam) or a hypnotic (e.g. barbiturate, propofol), and treat specific cardiovascular issues as they arise. More recently it has been shown that many manifestations of toxicity can be halted or reversed by the administration of a 20% intralipid emulsion intravenously (i.e., 1.5 ml/kg followed by an infusion of 0.25 ml/kg/min; rebolus 0.5 ml/kg/min of intralipid if necessary). In the developed world, the standard of care is to have intralipid immediately available in any setting where regional anesthesia is performed. Even though intralipid emulsion is inexpensive, its availability may be limited in some parts of the world. Propofol is not an acceptable alternative to intralipid (even though propofol preparations contain intralipid), since the volume of the preparation required to attain the appropriate intralipid dose will result in a gross overdose of propofol. When intralipid is available for the treatment of local anesthetic systemic toxicity, it is recommended that treatment be initiated at the first signs toxicity.

Unfortunately, the use of local anesthetics can occasionally cause toxicity, even when they are injected appropriately *(see nerve injury discussion above.)* Most of the reports of nerve dysfunction after plexus blockade have occurred in adults, and implicate bupivacaine most commonly. However, since bupivacaine is the most commonly used drug for plexus blockade, we would expect complications to be more commonly reported following its use. The true incidence of nerve dysfunction does not, however, appear to favor any particular local anesthetic. One exception, though, may be lidocaine and its use in spinal anesthesia. Lidocaine is no longer used for spinal anesthesia in most developed countries, due to a number of case reports of the drug causing transient neurologic symptoms after its use. No equivalent cases have been reported in children. However, minor transient neurologic sequelae are not likely to be discovered in infants and preverbal children, so the incidence of injury may be higher than reported in these age groups. Despite these concerns, it is important to recognize that very large surveys of regional anesthesia in pediatric patients reveal startlingly low rates of either temporary or permanent nerve injury.

Since intravascular drug injection can easily lead to systemic toxicity, it is always prudent to aspirate through needles and catheters prior to any injection of local anesthetic and then to slowly give incremental injections of the total dose. The first increment should serve as the "test" dose to detect signs of an intravascular drug injection. However, negative aspiration tests *never* guarantee that the needle or catheter is not intravascular. In children it is recommended that test doses contain epinephrine at 5 mcg/ml (1:200,000). Test doses with only local anesthetic are not interpretable in an anesthetized patient, or in preverbal/immature children who cannot give feedback on subtle neurologic prodromal symptoms (See above). Even frank seizure activity may not be detectable during general anesthesia. Heart rate and blood pressure responses to an epinephrine test dose can be quite variable in anesthetized pediatric patients, especially when a halogenated vapor anesthetic is the primary agent used to produce general anesthesia. Pretreatment with atropine may make heart rate and blood pressure responses more reliable. A more sensitive indicator, however, is an increase in the T wave amplitude on a continuous electrocardiogram (with a 25% increase in amplitude considered a positive test). If continuous ECG monitoring is not available, then an increase of 10 beats per minute in heart rate or a 15mmHg increase in systolic or mean blood pressure is also considered a positive test. Lack of capability for ECG monitoring is a relative contraindication to the use of doses of local anesthetics that approach toxic levels. That risk-benefit analysis should be made on a case-by-case basis.

NEURAXIAL TECHNIQUES

Spinal Anesthesia (Subarachnoid Block, SAB*)*

Spinal anesthesia is a reasonable means of providing anesthesia for surgery below the umbilicus when general anesthesia is undesirable (i.e. the very high risk premature or ex-premature infant). It is also useful for providing analgesia as high as the thoracic levels. Occasionally, mature pre-teenage and teenage children may undergo SAB as a primary anesthetic, but the incidence of post dural puncture headache is significant in this age group. While small diameter pencil tip needles (e.g. Sprotte, Whitacre) will lessen the incidence post Dural puncture headache in the older age group, these needles are much more expensive and less available than the traditional Quinke type needle. Otherwise equipment needs for SAB are minimal and as such, this technique is a useful way to provide surgical anesthesia and postoperative pain relief in resource-limited environments.

Use of SAB is often used in preterm and former preterm infants to decrease the real risk of apnea after general anesthesia. The risk of post anesthetic apnea is elevated until at least the post gestational age of 60 weeks, especially if the infant was born before 36 weeks of gestation, and/or has a hemoglobin concentration of less than 10g/dl. While using SAB, rather than general anesthesia for these patients, appears to decrease the rate of postoperative apnea, it does not *eliminate* that risk. Since patients who are less than 60 weeks post-gestational age should be

strictly monitored for apnea after a spinal anesthetic anyhow, resource utilization is not necessarily improved through the use of SAB. However, the benefits of SAB may extend beyond simple apnea reduction. Some infants with significant lung disease may benefit from avoidance of general anesthesia and endotracheal intubation. In this circumstance the use of regional anesthesia to avoid tracheal intubation often can significantly reduce the level of postoperative care required. While SAB provides very dense sensory and motor blockade, the duration of a spinal anesthetic with bupivacaine or tetracaine is significantly shorter in neonates and infants than in older children and adults (90-120 versus >120 min).

There are some procedural details that need to be considered when performing a SAB in infants. First, SAB (or any neuraxial block) rarely leads to hypotension in infants, and consequently, some anesthesiologists will perform the SAB first and then place an IV in an insensate lower limb. Second, the conus medullaris of the spinal cord in infants ends at L2-3 and not L1, as in older children and adults. It is prudent to use the lowest interspace possible when inserting a needle into the intrathecal space of infants to avoid damaging the spinal cord. Third, while positioning of the patient for a SAB is by preference of the anesthetist, there are significant advantages to performing the block in a seated upright position. The sitting position, with the head/neck slightly flexed, is somewhat easier for an assistant to maintain, especially in the case of a vigorously squirming infant. But it is important that the assistant and anesthetist be cognizant of the infant's ventilation and oxygen saturation throughout the procedure. In a neonate with lung disease, even slight airway obstruction due to neck flexion will cause rapid oxygen desaturation. Whenever possible, the patients oxygen saturation should be measured during performance of a SAB. The sitting position also adds a hydrostatic pressure gradient to the CSF at the lumbar level, facilitating CSF flow through the needle.

A small skin wheal with 1% carbonated lidocaine[6] injected through the smallest needle available (30 gauge if possible) and/or the use of a topical local anesthetic (such as EMLA cream)[7] is helpful, especially when a 22 gauge spinal needle must be used for the block. A short (30mm) x 22-to-25 gauge Quinke tip spinal needle is typically used when available. While longer "adult" needles can be used, they are very awkward to use and have a significant amount of dead space, which can affect detection of CSF flow and can allow part of an infant's anesthetic dose to be left in the needle and not be injected into the subarachnoid space. Smaller gauge spinal needles can be used, but CSF flow can be very sluggish in this age group and smaller needles exacerbate this

[6] *Adding sodium bicarbonate to neutralize the pH of lidocaine significantly decreases the initial sting from infiltration. Typically 1 mEq of NaHCO3 will neutralize the pH of 10cc of 1% lidocaine.*

[7] *EMLA cream is a eutectic mixture of lidocaine and prilocaine, which is effective at producing topical anesthesia through intact skin. In needs to be applied at least 45 minutes before anticipated need in order to be effective.*

problem. If it is felt the intrathecal space has been accessed with the needle but there is no CSF flow, *gentle* aspiration of the needle with a 1cc syringe is suggested. When injecting medication, it is important to be aware of the volume of the needle and either adjust the total dose volume in the syringe to compensate for drug remaining in the needle, or to flush the needle with 0.1-0.2cc of sterile preservative free saline after the local anesthetic is injected. After a SAB, the infant should be kept supine until the extent of cephalad spread of the anesthetic is stable. Placing the baby in a head down position, even transiently, can cause rapid cephalad spread of the hyperbaric local anesthetic solution and result in a very high or total spinal.

Table 21-3: Dosing Guidelines for Neonatal Spinal Anesthesia.

Hyperbaric Local Anesthetic	Dose Range	Duration
0.5% tetracaine	0.8-1.2 mg/kg	90-177 minutes
0.5% bupivacaine	0.8-1.2 mg/kg	90-177 minutes
2.5% lidocaine*	5 mg/kg	60-90 minutes

**There are numerous case reports of transient neurologic symptoms when using lidocaine for spinal anesthesia in adults. While no similar reports exist in infants or children, lidocaine should probably be avoided for SAB.*

The SAB technique in older children does not vary from that in an adult patient, with the exception that, depending on the age and temperament of the child, he/she may need significant sedation or general anesthesia prior to placing the block.

Spinal Analgesia

The use of intrathecal opiates has some utility for postoperative analgesia in ~~major~~ spinal, abdominal, thoracic and cardiac surgery. For example, addition of intrathecal opiates has been shown to be of significant benefit after major spinal surgery (e.g. scoliosis correction). These operations are associated with severe postoperative pain and a single intrathecal dose of morphine (10-25mcg/kg) placed by the surgeon under direct vision near the end of the procedure provides significant postoperative pain relief. *Preservative free* morphine is the most commonly used spinal opiate and doses of 10-30mcg/kg are typically used. Delayed respiratory depression can occur for as long as 24 hours post injection, especially when higher doses of narcotic are used. Respiratory depression is also more likely to occur when parenteral opiates are used in addition to the intrathecal morphine. When postoperative respiratory monitoring is not possible, the use of neuraxial opioids is contraindicated.

Epidural Anesthesia

While the caudal approach is primarily used for epidural analgesia in the pediatric population, there are several uses for caudal epidural anesthesia as the sole anesthetic in high-risk neonates and infants. Whenever one might consider a spinal anesthetic in an infant, a caudal epidural anesthetic is a good alternative, as it is often technically easier to perform and may have less potential for complications. It is reasonable to use a single shot caudal anesthetic for simple, short duration lower extremity or lower abdominal surgery (e.g., inguinal hernia repair, circumcision, rectal biopsy, achilles tenotomy, etc.).

Caudal injection is the simplest and most reliable way to access the epidural space in neonates, infants and children up to about 5-7 years of age. Up to this age, the sacral hiatus is relatively superficial and easy to identify with simple palpation at the apex of an inverted equilateral triangle drawn between the posterior superior iliac spines. Traditionally, the presence of a sacral dimple has been considered a relative contraindication to any caudal injections, due to their association with spinal dysraphism and other cord anomalies. More recent studies, however, have found this association to be quite weak.

Bupivacaine, 0.375%, is most commonly used drug for surgical anesthesia by the caudal route. By using the maximum permissible dose of 2.5-to-3mg/kg, it is possible to achieve approximately 90 minutes of surgical anesthesia up to the L1 level. Lidocaine or chloroprocaine are acceptable alternative drugs to use, but in all but the shortest of procedures (<45 minutes), use of these agents requires placement of a catheter in the sacral hiatus to permit repeated intermittent injection or continuous infusion of drug. Placement of a temporary epidural catheter is easily accomplished by inserting a small IV catheter (22 gauge) into the epidural space via the sacral hiatus, attaching a short extension tubing, and securing the catheter with adhesive dressing. This permits administration of intermittent boluses of local anesthetic during the surgical procedure. One useful dosing regimen, when using 3% chloroprocaine, is to administer 1 ml/kg of the drug initially, followed by 0.3 ml/kg boluses of the drug solution until the desired block level is achieved. Subsequently, approximately 1ml/kg/hour of drug is given, either by intermittent injection or continuous infusion. This dose scheme, while differing from previously described dosing limits for chloroprocaine, was shown in a small study to result in very low plasma levels of local anesthetic and no complications. Onset of caudal epidural anesthesia when bupivacaine is used may take 10-to-15 minutes to attain a dense enough block to perform surgery. The presence of flaccid lower extremities is usually a strong indication that the anesthetic will be adequate to perform the surgery.

There are numerous formulas for determining the drug volume required for a desired spread of a drug in the epidural space. The easiest and most consistent formula to use, in this author's opinion, is 0.05-ml/kg per spinal segment. This works well when discussing spread from the tip of

an epidural catheter. However, practically speaking, 1.2-ml/kg of drug administered by caudal injection should reach up to mid thoracic dermatomes. One ml of drug per kg of body weight is usually sufficient to provide a block up to the high lumbar/low thoracic levels. This applies for both anesthesia as well as analgesia.

Other approaches to epidural anesthesia, such as lumbar and thoracic, are rarely used for surgical anesthesia in children. They can, however, be used to provide epidural analgesia.

Epidural Analgesia

Single shot caudal blocks for postoperative analgesia are the most common regional anesthetics utilized in children. It is a relatively simple procedure, has few equipment requirements and has an extensive safety record. It is indicated for lower extremity, genital, perineal, anal, and lower abdominal procedures. While it can be used for procedures above the umbilicus, its duration of analgesia at those dermatomes is limited. Single shot caudal blockade with 0.125-0.25% bupivacaine can be expected to provide about 4-8 hours of analgesia. Urinary retention and lower extremity motor weakness are potential complications that are rarely issues in infants, but could present problems for an ambulatory toddler.

If analgesia is desired for longer than 4-8 hours, one option is to add preservative free morphine (30-100-mcg/kg) or the α-2 agonist clonidine (1-2-mcg/kg) to the local anesthetic; both of these adjuvants are given as a single dose and not repeated. The result is a synergistic improvement in pain control and a longer duration of pain relief. However, complications such as sedation, nausea, pruritus, urinary retention, and respiratory depression can occur and may limit the use of these adjuvants, especially if medications and staffing to treat those problems are not available. Additionally, adding any opiate to the local anesthetic requires that postoperative respiratory monitoring is available and used. When morphine is added to the local anesthetic, respiratory monitoring is required for up to 24 hours after the final dose of morphine is administered.

Placing an indwelling epidural catheter is perhaps the best option for prolonged neuraxial analgesia. Since infusion pumps (either electronic or elastomeric) are often not available in settings with limited resources, intermittent manually administered boluses of drug may be given through these catheters. It is very important to maintain good sterile technique when catheters are repeatedly accessed for bolus drug injections. Depending on the surgery type, the final position of the catheter tip, and the drugs chosen, dosing schedules could be as frequent as every 3-4 hours or as infrequent as every 12 - 24 hours. When repeated bolus dosing of local anesthetics is used, motor blockade is a more likely than when the drugs are continuously infused. The patient must be prevented from falling if he/she is ambulatory, as some degree of motor weakness is common with epidural administration of local anesthetic. While the use of epidural catheters is very effective for pain control, they have relatively high resource requirements, including the catheter and infusion equipment itself, the staffing needed for both

intermittent bolus administration of drugs and for respiratory monitoring when opiates are used. As a result epidural analgesia is less often used when resources are scarce.

However, if sufficient equipment and staffing are available, continuous epidural analgesia is a good option for postoperative pain relief. When this is the case, the following details should be considered. Caudal, lumbar, or thoracic catheters can be used, but caudal catheters are the most widely used in this author's experience. In fact, catheters can be successfully threaded blindly from the sacral hiatus to the thoracic dermatomes with modest success rates in children under 5 years of age. The epidural fat is very loosely packed in such patients, which permits catheters to be advanced from the sacral hiatus up to thoracic levels with little resistance. This is especially true in infants, and when a styletted catheter is used. Methods for determining the level of the resultant catheter tip include ECG monitoring (via the catheter), electrical stimulation, ultrasound, and epidurogram, all of which require significant additional equipment and resources. They do, however improve ones ability to place the tip of the catheter in the desired location for the block. Lumbar placement of catheters is also a reasonable choice in most infants and children, but placement of thoracic epidurals is best left to those with significant experience, as the potential for spinal cord injury can be much higher. If a loss of resistance technique is used for epidural access in a pediatric patient, the syringe should be filled with saline rather than with air, as inadvertent intravenous injection of significant amounts of air is possible. Such a venous air embolism can have catastrophic consequences in patients with right-to-left shunting lesions, and is significant even in otherwise normal infants. Placement of the epidural catheter tip near the dermatomal level of the expected surgical pain reduces the amount of local anesthetic required and/or allows one to successfully use lipophilic opiates, such as fentanyl or sufentanil, for pain relief. If the catheter tip cannot be placed at the correct dermatome, or many dermatomes must be covered, the only viable option is the use of more hydrophilic opiates (e.g., morphine, hydromorphone.) If it is available, the addition of clonidine to the anesthetic solution can be very useful for covering dermatomes distant from the catheter tip.

Drug choice for epidural analgesia depends highly on the location of the catheter tip. If the catheter is sited at or near the relevant dermatome, a dilute bupivacaine solution with fentanyl is an excellent combination of drugs for pain relief. If the catheter tip is far from the surgical dermatome (e.g., when using a lumbar catheter for thoracic surgery) there is little reason to use local anesthetic, as it is unlikely to reach the appropriate dermatome. In this circumstance, a hydrophilic opiate, such as morphine or hydromorphone, is a better choice of drug, as these opiates tend to spread significantly better than the lipophilic opiates fentanyl and sufentanil. Epidural hydromorphone and fentanyl appear to be associated with lower rates of nausea, urinary retention, and pruritus than morphine. Morphine, however, is universally available, inexpensive, and has a longer duration of action. As discussed before, all drugs used epidurally should be preservative free preparations.

Dosing of epidural drugs is complex and depends on a number of factors, including catheter location, intermittent versus continuous infusion of drugs, drug combination, number of dermatomes to cover, and of course the patient's age and size. Continuous infusion of drugs is usually the better approach, as it avoids large variations in the concentration of drug at the effect site and may result in fewer side effects. However, infusion pumps are quite expensive and are not universally available, so intermittent dosing of an epidural block may be the only choice. Notice the wide range of doses and the intervals between doses in the table below (**Table 4**). It is appropriate to adjust subsequent drug doses and intervals on the patient's response to the previous dose. Combining a local anesthetic and an opiate allows lower doses of either drug to be used than if they were used alone.

Table 21-4: Intermittent Epidural Dosing Guidelines

Drug	Dose	Interval
morphine	30-100 mcg/kg/dose	12-24 hours
hydromorphone	5-10 mcg/kg/dose	6-12 hours
bupivacaine 0.1-0.25%	0.2-0.5 ml/kg/dose	2-6 hours

Continuous epidural drug infusions can contain either single or multiple agents. It is typical to calculate the amount of local anesthetic to be infused and then to mix in an opiate to achieve the desired final dose of both agents (**Table 5**). For example, a 1 year old, 10kg child for whom we would like to infuse 0.4mg/kg/hr. of 0.1% bupivacaine would require an infusion of 4 ml/hour. By then adding fentanyl 2mcg/ml to the anesthetic solution, the child would also receive 0.8mcg/kg/hour of fentanyl.

Table 21-5: Continuous Epidural Dosing Guidelines

Drug	Concentration	Dose
morphine	20-50 mcg/ml	5-15 mcg/kg/hour
hydromorphone	3-20 mcg/ml	1-5 mcg/kg/hour
fentanyl	1-5 mcg/ml	0.5-2 mcg/kg/hour
bupivacaine	0.0625-0.2 %	0.1-0.5 mg/kg/hour*
lidocaine	0.5-1%	0.5-1.6 mg/kg/hour*

**Age dependent maximum doses should always be observed when calculating epidural dosing regimens.*

PLEXUS AND PERIPHERAL NERVE BLOCKS (PNB's)

As pointed out previously, most PNB's are usually placed for the management of postoperative pain. There may also be the circumstance where a PNB can be used as the primary anesthetic in a mature teen or pre-teenage patient. Postoperative pain is usually best managed by using a multimodal approach[8] with regional blockade at the cornerstone. Single shot drug injections are the most common form of PNB, but they only provide anesthesia/analgesia for a limited period of time. Single shot blocks are usually relatively simple to perform and require relatively little equipment. Unfortunately, most single injection PNB's last for 24 hours at best, and often for significantly less time. Anecdotally, this author has noted that even blocks performed with 2.5-3mg/kg doses of 0.5% bupivacaine usually last less than 12 hours when used for brachial plexus blocks in school age children or toddlers. Infants and adults experience somewhat longer duration of pain relief with most blocks. There is growing evidence that adding dexamethasone to local anesthetics for peripheral nerve blocks extends the duration of analgesia approximately 30%, although there are few data in pediatric patients, and safety data for adding this adjunct drug is still lacking. Epinephrine does increase the duration of blocks performed with lidocaine, but only modestly so when bupivicaine is used. There is no convincing evidence that any other adjunct drug consistently improves the quality or duration of PNB's.

Placing plexus catheters, if equipment and staffing are available for doing so, allows postoperative pain relief until the drug infusion is stopped and the catheter is removed, typically for up to five days. While the use of this technique extends the duration of blocks, it also adds significant complexity and cost to the procedure, and as such, is rarely used in environments with limited resources.

Dosing of PNB's is somewhat dependent on the location of the block and the desired effect. If the PNB is used solely for postoperative analgesia, lesser concentrations of local anesthetic can be used, usually in the 0.125%-to-0.25% range for bupivacaine. If, however, the goal is to minimize the surgical stress response or to guarantee muscle relaxation, a surgical anesthetic dose of 0.375-0.5% bupivacaine is required. It should be noted that the duration of drug action is directly correlated with the amount of drug injected, so in order to maximize the duration of analgesia from a single shot PNB, it is appropriate to administer the largest recommended safe dose.

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[8] *Multimodal analgesia refers to the use of several different drugs and techniques targeting different parts of the pain perception pathway. For example, by combining a regional anesthetic, a systemic opiate receptor agonist, a non-steroidal anti inflammatory (e.g. ibuprofen), and acetaminophen, a profoundly effective pain management strategy results. In combination each individual component can be minimized in order to lessen side effects.*

Plexus and Peripheral Nerve Localization

Nerve localization for PNB's relies on a thorough understanding of nervous system anatomy, motor and sensory innervation patterns, and surface landmarks. Injection of local anesthetic based solely on surface landmarks, however, has mediocre success rates at best, and is routinely found to be lacking in efficacy when compared to the use of ultrasound or electrical stimulation methods. A reproducible, reliable, safe, and inexpensive method for localizing the appropriate nerve and injecting medication as near to it as possible is the holy grail of peripheral and plexus regional anesthesia. The technique of seeking paresthesias in patients who are awake is not generally applicable to pediatric practice.

The present state of the art in nerve localization is to use real time ultrasound imaging to visualize the target nerve and the needle and/or catheter used to deliver the medication. Small stand-alone systems, however, can cost upwards of $70,000 USD. It is possible, however, to adapt a typical hospital owned ultrasound machine that is used for echocardiography or obstetrical imaging by acquiring a single high-resolution linear ultrasound probe. These linear array probes are usually 25-50 mm long, and operate at frequencies of 5-15 MHz. They cost a fraction of what a total system would cost. Even when the equipment is available, ultrasound guided regional anesthesia still requires a long and involved training period before any degree of proficiency with the technique can be attained. Medical missions often take portable ultrasound machines to do PNB's for postoperative pain. This is a great opportunity for local anesthetists to get exposure and training in the use of these techniques for PNB's. Ultrasound imaging is also an invaluable tool for teaching anatomy and other nuances of regional anesthesia to practitioners using alternative methods of nerve localization.

Since most practitioners trained in the last 20 years are familiar with motor *nerve stimulation* for localization of nerves, and the equipment needs are modest, this is probably the most viable option for doing blocks in resource-limited environments. The basic principle behind electrical nerve stimulation is that delivery of a small intermittent current through the block needle (i.e. the cathode) will usually depolarize the intended target nerve and elicit a motor response in that nerve's motor distribution. All other variables being equal, the amplitude of the motor response is inversely proportional to the distance of the needle tip from the nerve (i.e., the closer the needle tip is to the nerve, the greater the response). When a motor twitch is still present at a very low current, the needle tip should be near to the nerve, and injection of local anesthetic can proceed. Using surface mapping to locate nerves can also be helpful, and is particularly effective in pediatric patients due to the relatively small distance between nerves and the skin surface.[9] The use of surface mapping for this purpose requires a somewhat higher current setting on the

[9] *Surface mapping is a noninvasive nerve localization technique accomplished by application of electrical current on the skin overlying a nerve plexus or peripheral nerve.*

stimulator (1-5 mA). The cathode must be applied directly to moistened skin above the anticipated injection site. Again, muscle response (twitching) is most prominent when the cathode-to-nerve distance is smallest. Microprocessor controlled peripheral nerve stimulators are commercially available throughout the world and are far less costly than an ultrasound system. Finally, while any needle can be used for electrical stimulation of nerves, the best results occur when specifically designed block needles with blunt tips, insulated shafts, and a simple connector designed for use with the particular brand of nerve stimulator are used.

While many blocks rely on specific localization of the nerve with a nerve stimulator or ultrasound, field blocks or infiltration blocks using anatomic landmarks are also used. Anatomic landmarks and tactile feedback during needle advancement are used to get the needle tip close to the target nerves. Blocks that rely on sensing "fascial clicks"[10] with advancement of the needle are best achieved with a blunt needle (known as a B-bevel needle), as sharp needles tend to pass through tissue with little observable resistance. A possible secondary benefit of short bevel needles is that there is potentially less nerve damage if the needle makes direct contact with a nerve. This theoretic advantage has never been proven, however. Short bevel needles of many differing lengths and gauges are manufactured specifically for performing nerve blocks, but may or may not be available depending on resources. Needles designed specifically for use with a nerve stimulator are usually insulated along the shaft of the needle to concentrate the highest current density near the tip of the needle. A Tuohy needle can be used if a blunt needle is not available. Tuohy needles give very obvious sensory feedback to the operator as the needle passes through fascial planes, and these needles are particularly useful for deeper blocks as they tend to track better due to their rigidity. They are, however, usually much larger in diameter and may be inappropriate due to their size. Unfortunately, electrical stimulation and landmark localization blocks have higher failure rates than ultrasound-guided blocks, even in the most experienced of hands. Consequently, they should always be carried out with a good backup plan for providing anesthesia or analgesia should the block fail.

Step by step performance of most routine peripheral and plexus blocks is well documented elsewhere, so the following sections will simply address the indications, contraindications, and a few caveats specific to each block. An excellent free source of information is the New York School of Regional Anesthesia website (http://www.nysora.com).

Brachial Plexus/Upper Extremity Blocks

The brachial plexus is a complex neurologic structure originating at the C4 through T1 nerve roots

[10] *A fascial click is a sensation noted by the practitioner as the block needle is advanced through tissue. It is best described as an increase in resistance followed by a sudden "give" as the needle punctures a relatively dense fascial structure.*

and enervating the upper extremity, shoulder, and a portion of the neck and chest. Techniques for localizing the brachial plexus in pediatric patients include landmark, infiltration, nerve stimulation, and ultrasound. This discussion will assume the practitioner has a basic understanding of the sensory and motor innervation patterns of the brachial plexus. Proximal plexus blocks described in this section will require at least a nerve stimulator be used for plexus localization. Some more distal blocks can be accomplished with only a needle and local anesthetic. Ultrasound guidance, if available, will always yield the best overall success rates with the least risk.

The choice of block should be very specific to the sensory coverage needed for the surgery proposed. For surgery of the distal clavicle, shoulder and proximal humerus, the *interscalene* approach is most useful technique and is well-described using both nerve stimulation and ultrasound. Deltoid, or more distal muscle twitches, are acceptable endpoints when using a nerve stimulator. This approach frequently spares the lower roots of the brachial plexus (C8-T1) and as such, blockade in the distribution of the ulnar nerve will be unreliable. Nearly 100% of the time, the ipsilateral phrenic nerve will be blocked with the interscalene brachial plexus block. Additionally, the cervical sympathetic blockade that usually occurs produces an ipsilateral Horner's syndrome (ptosis, hyperemia of the conjunctiva, nasal congestion). Neither of these side effects is of much consequence except for in patients with significant pulmonary disease. In these patients, loss of unilateral diaphragm strength can cause acute respiratory failure, and consequently, interscalene block should be used very cautiously in patients with pulmonary issues. Since morbidly obese individuals frequently have reduced pulmonary reserve, caution is also advised when using this block in these patients. It should be noted that the American Society of Regional Anesthesia recommends against using interscalene brachial plexus blockade in anesthetized adults or pediatric patients, due to several case reports of spinal cord injury. These case reports occurred before routine use of ultrasound guidance for blocks and may not reflect the risk when using an ultrasound-based technique. Nevertheless, except for the occasional mature preteen or teenager in whom the block can be performed under mild or no sedation, this block should be used with great care, and perhaps avoided completely in anesthetized pediatric patients.

For surgery from the mid humerus to the hand, *supraclavicular* or *infraclavicular blocks* are both reasonable choices. Both approaches can be done using either nerve stimulation or ultrasound. When a stimulator is used for localization of the brachial plexus, success rates are greatest when twitches of the hand are achieved. The block is usually of lower quality when only more proximal twitches are used as an endpoint. A supraclavicular block occurs at the level of distal roots and divisions of the brachial plexus, while the infraclavicular block occurs at the level of the cords. Both levels are proximal to the take off of the musculocutaneous nerve and reliably block this nerve. When a nerve stimulator is used to localize the brachial plexus during supraclavicular block, the reported incidence of pneumothorax as high as 6%. Proper use of ultrasound almost

completely eliminates this risk. Additionally, while the incidence of phrenic nerve block is significantly reduced with a supraclavicular block compared to that with an interscalene block, it is not completely eliminated. Because of the significant risk of pneumothorax (with a nerve stimulator technique) and small but present risk of phrenic nerve block, the supraclavicular approach may not be the best choice of block for patients with limited pulmonary reserve. Additionally, if the resources to perform chest tube drainage for a pneumothorax are not available, alternative block methods should be seriously considered. Infraclavicular block also caries some risk of pneumothorax, albeit much less so than with a supraclavicular block, especially when the lateral (sub coracoid) approach is used. This is a relatively "deep" block and can be difficult to accomplish in larger patients (even with ultrasound). The success rate is good, however, when ultrasound is used, or if stimulation of the posterior cord of the brachial plexus is achieved (i.e. radial nerve mediated wrist extension) when using electrical stimulation.

*Axillary block*ade can be used for surgery that will be done distal to the elbow and is the one brachial plexus block technique that can be accomplished with nothing more than a needle and some local anesthetic. While a nerve stimulator or ultrasound technique can be used, the trans arterial approach (injecting an adequate volume of local anesthetic deep to and superficial to the axillary artery) is simple and relatively effective. Some practitioners simply use a "field block" technique by depositing drug below, above, and superficial to the axillary artery. Both of these techniques rely on the close proximity of the radial, median and ulnar nerves to the easily palpable artery. An additional injection of local anesthetic into the body of the coracobrachialis muscle can be done to anesthetize the musculocutaneous nerve, but the success rate with this block is inconsistent. Unfortunately, not blocking the musculocutaneous nerve during an axillary block is common and often negates the efficacy of this block. Since purposeful or inadvertent arterial puncture often occurs with axillary block, formation of an axillary hematoma is a common, but easily managed complication.

Simple field injections near terminal branches of the brachial plexus may sometimes be useful, especially to rescue an otherwise incomplete brachial plexus block. The median nerve can be blocked in the antecubital fossa, just medial to the brachial artery, or more distally between the flexor carpi radialis and the palmaris longus tendons. The ulnar nerve is best blocked by infiltrating local anesthetic under the flexor carpi ulnaris tendon and on the medial side of the ulnar artery. Performing a field block lateral to the radial artery near the anatomic snuffbox can sometimes be used to blindly block the radial nerve. Ultrasound guidance to block the median, ulnar and radial nerves in the forearm results in near 100% success rates, as these are very easy nerves to image. *Digital blocks* of the fingers are easy and safe with the caveat that epinephrine should never be added to the local anesthetic due to the risk of digital ischemia from this adjunct.

Lumbar and Sacral Plexus/Lower Extremity

Approaches to blockade of a lower extremity are essentially divided into two major groups*: lumbar plexus blocks* (T12-L4) and *sacral plexus blocks* (L4-S3). Lumbar plexus innervation of the lower extremity is comprised of the femoral nerve and its terminal saphenous branch, obturator nerve (anterior and posterior division), and the lateral femoral cutaneous nerve. These nerves are responsible for sensory innervation of the inguinal area, the anterior and lateral thigh, some portions of the medial thigh, a portion of the knee, and a strip of skin down the medial leg to approximately the medial malleolus. The sacral plexus innervation is comprised of the sciatic nerve and its terminal peroneal and tibial nerves, which supply the rest of the lower extremity. All these nerves can be blocked either individually or in groups.

A posterior lumbar plexus block ideally provides anesthesia and analgesia for hip surgery. The traditional posterior lumbar plexus block is a deep block in which local anesthetic is deposited within the body of the psoas muscle where the lumbar nerve roots travel. This is one of the few blocks where nerve stimulation is the preferred localization technique with quadriceps contraction being the desired endpoint prior to injection. This block is also the only reliable way, other than neuraxial techniques, to block all three major terminal branches of the lumbar plexus with one injection. Retroperitoneal hematoma, renal injuries, and injuries to the great vessels are high morbidity complications that make the risk-benefit ratio of this block somewhat questionable in this authors opinion. Consequently, experienced practitioners should only perform it. The lumbar plexus can also be approached anteriorly (e.g. 3-in-1 block, fascia iliaca block), but success with these anterior approaches is inconsistent. For the *3-in-1 block*[11], the needle is the same as if one were doing a *femoral nerve block*, but a larger volume of local anesthetic (typically 20-to-50ml in adults) is injected while pressure is applied to the tissues just distal to the needle. The *fascia iliaca block* is similar, but the needle is place more laterally, with the best success achieved with ultrasound guidance. For a landmark guided fascia iliaca block, a blunt needle is advanced while feeling for fascial “clicks”, since no specific nerve is targeted. The second click occurs when the needle pierces the fascia iliaca and a large volume of anesthetic is then injected. Theoretically, both these anterior approaches rely on local anesthetic tracking into the pelvis under the fascia iliaca, and into the psoas compartment, to block the lumbar plexus. Neither, however, reliably achieves full lumbar plexus blockade, and the obturator nerve commonly remains unanesthetized. As such, these anterior lumbar plexus blocks are not terribly reliable for surgical anesthesia, but can be useful as part of a multimodal analgesic plan in hip surgery.

For procedures on the mid/distal femur, thigh and knee, an isolated *femoral nerve block* can be done using electrical stimulation or ultrasound. Innervation of the knee is quite variable and

[11] *The 3-in-1 block was originally named as such because it is an attempt to anesthetize the femoral, obturator, and lateral femoral cutaneous nerves with one anterior injection.*

includes contributions from the sciatic, femoral, and obturator nerves. For analgesia purposes, femoral nerve block alone is quite effective for reducing both pain and opiate consumption. When using the nerve stimulator to perform this block, it is important to achieve contraction of the quadriceps muscle as an endpoint and not accept a sartorius muscle twitch. Seeing the patella move up and down is the most reliable way to identifying quadriceps contraction. Injecting drug when only sartorius muscle twitch is present usually results in an incomplete or failed femoral nerve block. The most significant side effect of any femoral nerve block (including those achieved with lumbar plexus blocks) is quadriceps weakness, which lasts for the duration of the block. Falls have been reported when a patient who feels very comfortable after surgery (no pain), tries to bear weight on the partially paralyzed limb. A more distal variant of the femoral nerve block is the *adductor canal block*. This block produces modest analgesia of the knee while minimizing quadriceps weakness. The use of a knee immobilizer, or enforced non-weight bearing, for the duration of these blocks is crucial to prevent injuries. This may limit the usefulness of this block, depending on the circumstances.

It should be noted that heavy sedation plus a femoral nerve block, and/or lateral femoral cutaneous nerve block is a reasonably good option as an anesthetic for quadriceps muscle biopsy in patients who are potentially susceptible to malignant hyperthermia. The susceptibility to malignant hyperthermia is perhaps one of the few indications for “awake” regional anesthesia during childhood and may be the most cost effective option when resources are scarce. Infusions of non-triggering general anesthetic agents (i.e. propofol) may be very costly or completely unavailable. And while a ketamine/narcotic/barbiturate-based general anesthetic is certainly possible and inexpensive, it is a less than elegant alternative.

Anesthesia and analgesia of the posterior thigh, lower leg or foot can be accomplished with *sciatic nerve blockade*. This block can be accomplished via the classic posterior gluteal and sub gluteal approaches, or it can be done more distally in the popliteal fossa, where the nerve is relatively more superficial and easier to localize. Either ultrasound or nerve stimulation is appropriate for localization of the nerve. While selective blockade of the peroneal or tibial nerve is possible, it is rarely necessary. When using a nerve stimulator to localize the sciatic nerve in the popliteal fossa, it is typical to see either a plantar flexion-inversion (of the foot) response (tibial component) or dorsiflexion-eversion response (peroneal component). If, for example, surgery on the lateral aspect of the foot is planned it can be beneficial to look specifically for a peroneal muscle twitch, as this will ensure appropriate sensory block and have a quicker onset of blockade in that sensory area. Additionally, since the location of the split of the sciatic nerve into its peroneal and tibial components varies in person to person, and can be as proximal as mid thigh, it is prudent to do popliteal sciatic blocks as high up as possible if one wishes to anesthetize the entire nerve. It needs to be pointed out that the terminal sensory branch of the femoral nerve, the saphenous nerve, does supply some innervation of the medial aspect of the lower leg, the skin over the medial ankle and a variable amount of the medial foot. When ankle or foot surgery is planned

within these areas, *saphenous nerve block* is also required for complete analgesia/anesthesia. Either a field block at the knee, near the saphenous vein, or a low dose femoral nerve or adductor canal block is typically done. The femoral nerve/adductor canal block is significantly more effective and reliable than the field block, but will often result in quadriceps weakness (as described above.) The sciatic nerve is large and consequently it is not uncommon for full onset of sciatic nerve blockade to take 30+ minutes when using long acting local anesthetic. Sciatic nerve blocks can last for upward of 36 hours. Again, partial lower extremity weakness or paralysis is likely and must be accounted for during postoperative care to prevent injury if patients try to walk.

For isolated foot surgery, the *ankle block* is simple and nearly risk free. Isolated nerves can be blocked individually to suit the particular need. The target nerves at the level of the ankle are the saphenous, sural, superficial peroneal, deep peroneal, and the tibial nerves. These blocks are usually performed by using specific landmarks for each nerve, or by performing field blocks, and require no equipment other than a needle and some local anesthetic.

Truncal Blocks

Thoracic paravertebral blocks rely on the existence of a paravertebral space bordered anteriorly by the parietal pleura, and posteriorly by the bony transverse processes of the spinal column. This block is useful for reducing/preventing post thoracotomy pain and for breast surgery in adults. This block is probably as effective as a thoracic epidural for pain management, with the added benefit of fewer side effects. “Walking” a needle off the transverse process of the appropriate vertebra and advancing it slightly while seeking a loss of resistance has traditionally been used to localize the paravertebral space. Since the risk of pneumothorax is significant when using this blind technique, the block should not be performed in environments incapable of managing a pneumothorax. Even with the use of ultrasound, which is the localization method of choice, there still is a modest risk of pneumothorax. If thoracotomy is planned, then the resources to manage this complication are available and this block is a reasonable choice. Additionally, the anesthetist can place catheters in the paravertebral space preoperatively, or the surgeon can place it under direct vision intraoperatively.

The *transversus abdominus plane block, or TAP block*, is an excellent block for abdominal surgery when neuraxial analgesia is otherwise contraindicated or impossible to perform. The terminal branches of the low thoracic (T8 - 12) and lumbar nerve roots consistently lie in the plane between the transverses abdominus muscle and the internal oblique muscle. Depositing local anesthetic in this plane reliably blocks abdominal wall sensation. The use of ultrasound for identification of this space is easy and reproducible, but performing the block using landmarks has met with variable success rates. The *rectus sheath block* is the most distal variant of the TAP block and covers only midline abdominal wall structures. As in the TAP block, ultrasound is the

localization method of choice for this block, though landmark approaches are also well described. A very specific version of the TAP block, *the ilioinguinal nerve block,* is often used for surgery in the inguinal area. The ilioinguinal and iliohypogastric nerves are the terminal branches of the T12 and L1 nerve roots, respectively, and supply the skin over the inguinal area. Blockade of these nerves is especially useful for inguinal hernia surgery, inguinal orchipexy, hydrocele surgery, etc. Since these nerves run in the plane between the internal oblique and transversus abdominus muscles, the method typically described for identifying this plane is feeling for fascial "clicks" as the needle is advanced through the fascial layers. The efficacy of the block is significantly better when ultrasound is used for localization of the nerves. If an ultrasound machine is not available, it is still reasonable to attempt this block, as the rare complications are injection of drug into the peritoneal space or bowel, and an occasional femoral nerve block, both of which are of little consequence. Alternatively, the surgeon can place this block under direct vision during the surgical procedure.

Penile nerve block is often used for circumcision or other penile surgery in male infants and children. The traditional block relies on insertion of a needle below Scarpa's fascia and injection of local anesthetic. Since there is midline septation of this space, it is important to do this block bilaterally. Alternatively, subcutaneous injection of local anesthetic around the base of the penis, otherwise known as a *ring block*, is simple, but not quite as effective as a penile nerve block. Hematomas are a common complication of these blocks. As in digital blocks, it is important to avoid epinephrine-containing solutions because epinephrine has been implicated in causing vascular compromise of the penis after penile nerve block.

Conclusions

Regional anesthesia is one the most satisfying and elegant approaches to anesthesia and analgesia in pediatric surgical patients and there are numerous benefits to its skillful application. The challenges in the pediatric patient, while significant, should not be a barrier to its use by anesthetists accustomed to caring for infants and children. And while many of the most modern approaches to regional anesthesia in the developed world revolve around advanced and expensive technology, it is important to recognize that a good portion of the field was developed before many of these technologies were available and, as such, can be used by any practitioner willing to learn and use them. Your patients will thank you!

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Chapter 22
THE POST ANESTHESIA CARE UNIT (PACU)

Susan Fossum RN, CPAN, Maria Pedersen RN, and George A Gregory, MD

Introduction

Within a year of anesthesia's discovery, it was recognized that patients recovering from anesthesia develop problems that require care in a specialized unit. However, it took many years for this concept to spread to most of the developing world. It only did so when Beecher and Todd documented fewer deaths in hospitals that had PACUs.[1] All hospitals in these countries now have a PACU staffed by nurses specially trained to care for patients recovering from anesthesia and surgery. This training is important because it is the PACU staff that provides the minute-to-minute postoperative care and must understand how patients recover from anesthesia. Even with this knowledge, in many parts of the world today, patients still recover from anesthesia in their bed on the ward, not in a post anesthesia care unit (PACU), with family providing the postoperative care. Many low resource countries have yet to establish a PACU in their hospitals, but should do so because more than half of perioperative cardiac arrests occur in the few hours after anesthesia and surgery end.[2] If this occurs on the ward, resuscitation will be less effective.

The goal of this chapter is to provide information on the PACU care of infants and children and to provide information for people who wish to develop a PACU.

Organization of the PACU

The purpose of a PACU is to provide a safe environment where potential or real complications of anesthesia and surgery can be detected immediately and treated. This requires a room dedicated to the early postoperative care of patients. This room should be located near the operating rooms. Most importantly, the room must be continuously staffed with enough well trained nurses who are expert in interpreting and responding to the events that occur during recovery from anesthesia. PACU nurses should be trained in airway management, basic life support, and training specific to caring for the special needs of postoperative patients emerging from anesthesia. Each PACU requires a readily available source of oxygen and a means of delivering that oxygen to multiple patients at the same time when necessary. A bag valve mask must be readily available for airway management, along with masks of different sizes. Drugs (including those needed for resuscitation from cardiac arrest) must also be readily available to everyone providing care in the

PACU. Nurses should be trained to start IVs because children sometimes dislodge their I.V.s. Maintenance of body temperature is also important, which means a means of warming and cooling a patient is required. Since vomiting is a common occurrence after anesthesia and surgery, a suction device and a supply of suction catheters are needed to remove vomited material from the mouth and upper airway. Electronic monitors are important as they help nurses detect and treat problems before they become serious.

PACUs require a nursing and medical director (usually an anesthetist) to assure efficient and effective functioning of the unit. The two directors are not only responsible for the day-to-day running of the unit; they are also responsible for the education of the staff. They also must interact with the hospital administration to obtain needed personnel, medications, and equipment.

Equipment

The most important monitor in the PACU is the nurse who looks at the patient and interprets what he/she sees. Other monitors provide information that aids nurses in assessing the patient's condition, but they certainly do not replace the nurse. Important considerations when purchasing any monitor includes whether the monitor can be repaired immediately if it breaks, who will repair it, and how long will it take to do the repairs? Can the repairs be done in the city/town/hospital where the monitor is used? Monitors should not be purchased unless the answers to these questions are clearly known. Otherwise, when the monitor breaks it will sit in a closet and never be used again. The following is a discussion of monitors commonly used during the PACU care of patients.

Pulse Oximeters

Trying to determine the level of oxygenation by looking at the patient's skin color is not very effective. Physicians and nurses are no better at this than untrained family members. The oxygen saturation (SpO_2) must be 80%-85% before everyone will agree that the patient is hypoxemic. If looking at skin color is the only method available for assessing oxygenation, the accuracy of the method can be improved by assessing the color of the mucus membranes of the lower eyelid, not the skin. The mucus membranes of the eyelids are less pigmented and have a larger number of capillaries, therefore, there are fewer effects on tissue color.

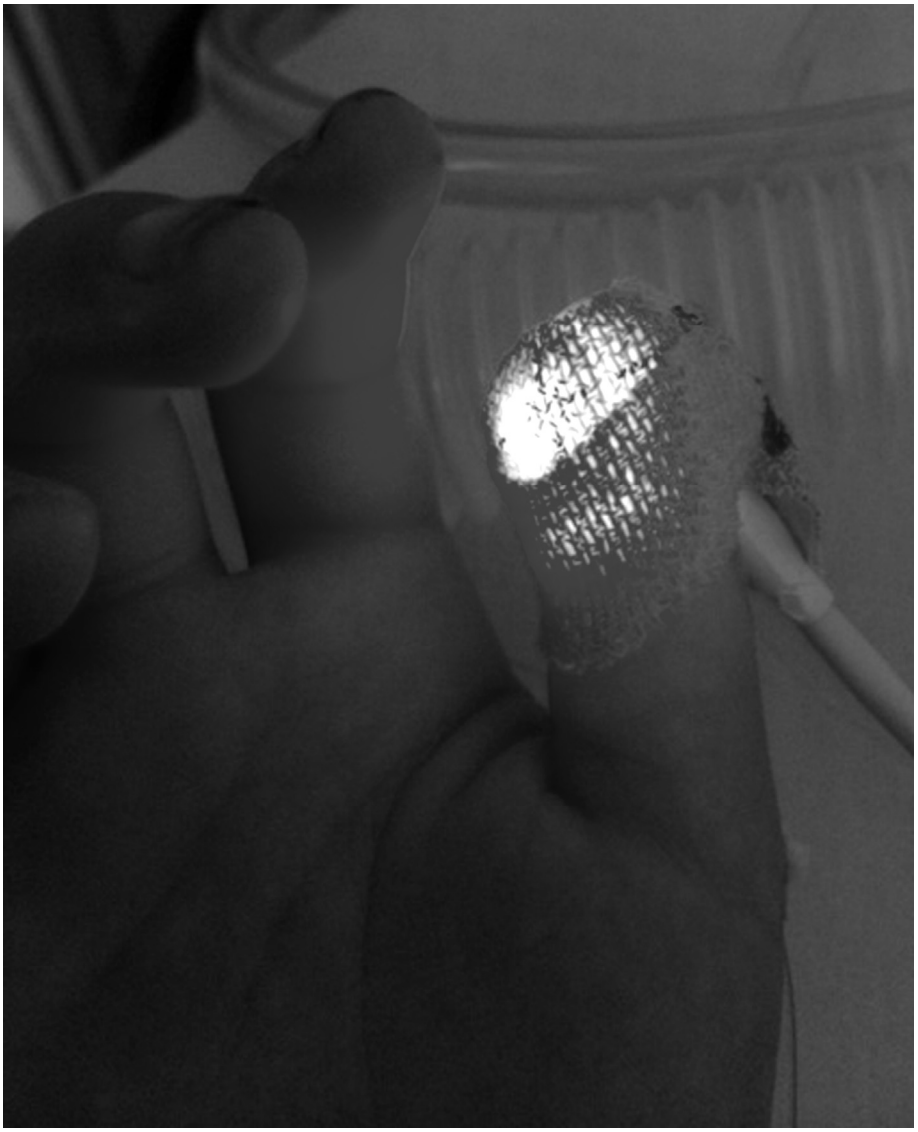
The presence of hypoxemia is best determined by measuring SpO_2 with a pulse oximeter. These are small, lightweight, sturdy, and noninvasive devices that provide immediate information about oxygenation. They allow nurses and others to quickly respond to decreased levels of oxygen, correct its cause, and prevent serious injury to the patient. For these reasons, they are widely used in operating rooms, PACUs, ICUs, and patient wards to assess oxygenation. They seldom break down but must have their batteries replaced occasionally. Most oximeters are inexpensive

and, depending on the manufacturer, work on both batteries and electricity from the wall. While SpO_2 monitors are not affected by skin color or jaundice, they are severely affected by low cardiac output and hypothermia. Both low output and hypothermia reduce blood flow under the probe, and this reduces accuracy of the SpO_2 measurement. To measure SpO_2, these monitors are usually placed on a finger, ear, or, in the case of infants, a hand or foot.

How Pulse Oximeters Work

Pulse oximeters consist of a computer processor, two diodes (LEDs) that emit light at different wavelengths, and a photodiode that collects light that passes through the finger, ear, hand or foot (**Figure 22-1**). One wavelength is 660nm (red) and the other is 940nm (ultra violate). Oxygenated (arterial) and unoxygenated (venous) blood absorbs light at different wavelengths. The photodiode opposite the LEDs measures the amount of light that passes through and is not absorbed by the tissues between the LEDs and the photodiode. This measurement fluctuates over time because the amount of arterial blood present between the two sensors increases slightly with each heartbeat. By subtracting the minimum amount of light transmitted from the maximum amount of light transmitted, the processor can correct for the effects of tissues other than blood vessels that are between the LEDs and photodiode. The ratio of red light to infrared light (which represents the ratio of oxygenated hemoglobin to deoxygenated hemoglobin) is calculated by the processor and converted to SpO_2.

Figure 22-1: Pulse Oximeter Probe on Hand of an Infant



The pulse oximeter probe is loosely wrapped around the child's hand without creating enough tension to obstruct blood flow to the hand. The oximeter cord is facing towards the arm. If tension is placed on the cord, the oximeter will tend to be pulled on to the hand. If the cord is facing the opposite direction (down), it will tend to pull the oximeter probe off of the hand.

A low measurement of SaO_2 may be seen on the monitor if the patient's SpO_2 is below 50%. Inaccuracies also occur with rapid movement of the extremity on which the monitor is placed because the oximeters cannot distinguish between pulsating arterial and venous blood and motion of the extremity. Motion artifact results in under estimation of SaO_2. Many manufacturers have added a program to the oximeter that compensates for motion artifact. Motion artifact results in false alarms, and after a while people may stop paying attention to them. This often results in real hypoxemic events being missed.

Pulse oximeters only measure hemoglobin oxygen saturation. They provide no measure of ventilation, work of breathing, or acidosis [carbon dioxide, pH, base deficit, or serum bicarbonate (HCO_3^-)] concentrations. SpO_2 only tells us the amount of oxygen in hemoglobin, which, however, is about 99% of all of the oxygen in the blood. During severe anemia, the amount of oxygen in blood (oxygen content) will be reduced, but the SpO_2 will be normal because the amount of hemoglobin the patient has is fully saturated with oxygen. See **Chapters 1 and 2** for further discussions of oxygen content and SaO_2s determinations.

Most normal children have a SpO_2 of 95%-to-100%. The SpO_2 of preterm infants is usually maintained between 90%-to-95% because high levels of oxygen in their blood can cause blindness (*retinopathy of prematurity*).

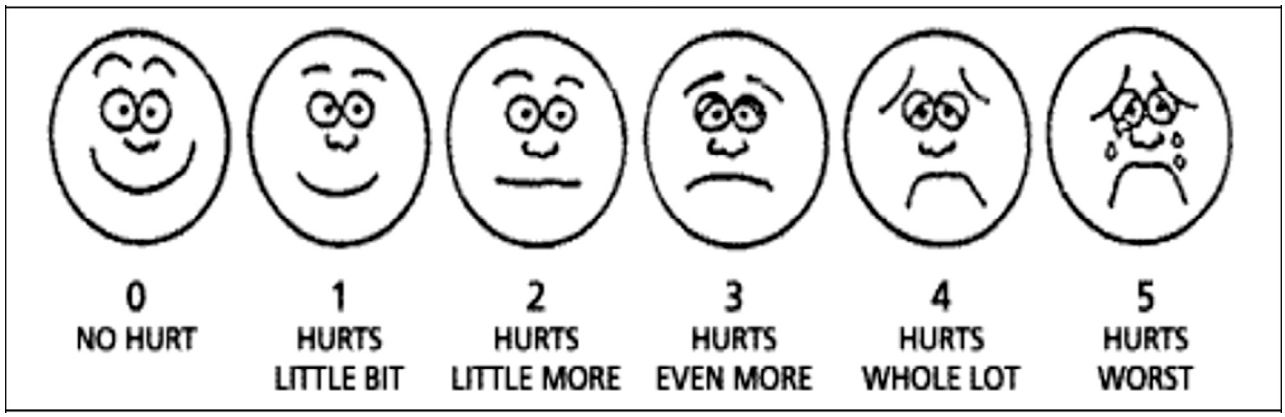
Vital Signs Monitoring

Measuring heart rate (HR), arterial blood pressure (BP), and evaluating capillary refill time, arterial pulses, and skin temperature are a vital part of PACU assessment and help to determine the appropriate care needed. Electronic vital sign monitors are commonly used because they provide continuous heart rate and electrocardiogram (ECG) data and intermittent arterial blood pressures. Many of these monitors provide continuous SpO_2 and end-tidal carbon dioxide monitoring also. These monitors are expensive and at times need repair.

If electronic monitors are not available, heart rate is measured by the time-honored method of counting the patient's pulse for 60 seconds. It is common for the HR of normal patients to vary with pain, movement, and fear, all of which can occur in the PACU and must be frequently evaluated and treated. If an electronic monitor is not available, blood pressure should be measured with a cuff and sphygmomanometer. Normal values for HR and BP are shown in **Appendix 1**. When the skin of a finger or toe is compressed, the skin blanches. When the pressure is released, it takes less than two seconds for the capillaries to refill with blood in adults and less than three seconds for this to happen in babies and infants. This is called capillary refill and is a valuable method of determining vascular stability of a patient. Normal patients of all ages have pulses in all of their extremities. Absent or markedly reduced pulses often indicate poor tissue perfusion, as does cool skin temperature (**See Chapter 1**). The heart tones should be easily heard and crisp. Distant or weak heart sounds, suggest reduced myocardial contractility. Lung assessment is done by listening to the breath sounds. Lung sounds are either normal or abnormal (wheezing, crackles and rhonchi).

Pain assessment is often regarded as the fifth vital sign. It is an objective measurement rather than subjective. It is important for PACU nurses to assess pain frequently using age appropriate scales. These pain scales may include: simple descriptive, the numeric scale, the faces scale, or the FLACC for infants. (**Figure 22-2)**

Figure 22-2: Wong Pain Scale for Infants and Young Children



The faces are shown to the child and he/she is asked to show which face is most shows how they feel. ***Wong's Essentials of Pediatric Nursing. With permission***

Other equipment:

Each PACU must have a way of quickly removing vomit, blood, and secretions from the mouth, pharynx, and nose. If a suction machine is not available, suctioning can be done with a bulb syringe, but this is not as effective as using a suction machine. Placing a child on her/his side during recovery from anesthesia reduces the likelihood of aspirating blood, secretions, or vomit. The signs of aspiration are sudden gagging, coughing, rapid breathing, and in many cases a sudden decrease of SpO_2. Suction catheters are available in sizes that correlate with the age/size of a patient and if possible, different sizes should be available for use.

The temperature of PACU patients should be measured frequently. Otherwise it is impossible to know if they are cold or febrile. The goal is to maintain body temperature within the normal range (36°C and 37.4°C), because cold children metabolize drugs and awaken from anesthesia more slowly and are more likely to be acidotic. Post anesthesia shivering is caused by exposure to a cold environment in the operating room and anesthesia drugs that may depress the CNS and cause peripheral vasodilation. Post anesthesia shivering can increase the patient's oxygen consumption 400 – 500%. It is important to continue providing oxygen to these patients until shivering stops and/or normothermia is achieved. Furthermore, cold children may be more likely to vomit and aspirate vomited material into their lungs due to delayed emptying of their stomachs. Febrile children, on the other hand, have increased consumption of oxygen that may lead to oxygen desaturation if the child's lung function is marginal. Febrile children are often unhappy and difficult to console, which may lead to excessive amounts of drugs being administered to calm the child. If a febrile child has a cardiac arrest, it is often more difficult to resuscitate her/him from the arrest, and if resuscitated he/she is more likely to have brain damage. It is important to remember that some cases of malignant hyperthermia (**See Chapter 23**) first occur in the PACU, not in the operating room. Unless the patient's temperature is measured, abnormally high

temperatures may be missed until it is too late, and the patient will either die or suffer severe brain injury.

Assessment on Admission to the PACU

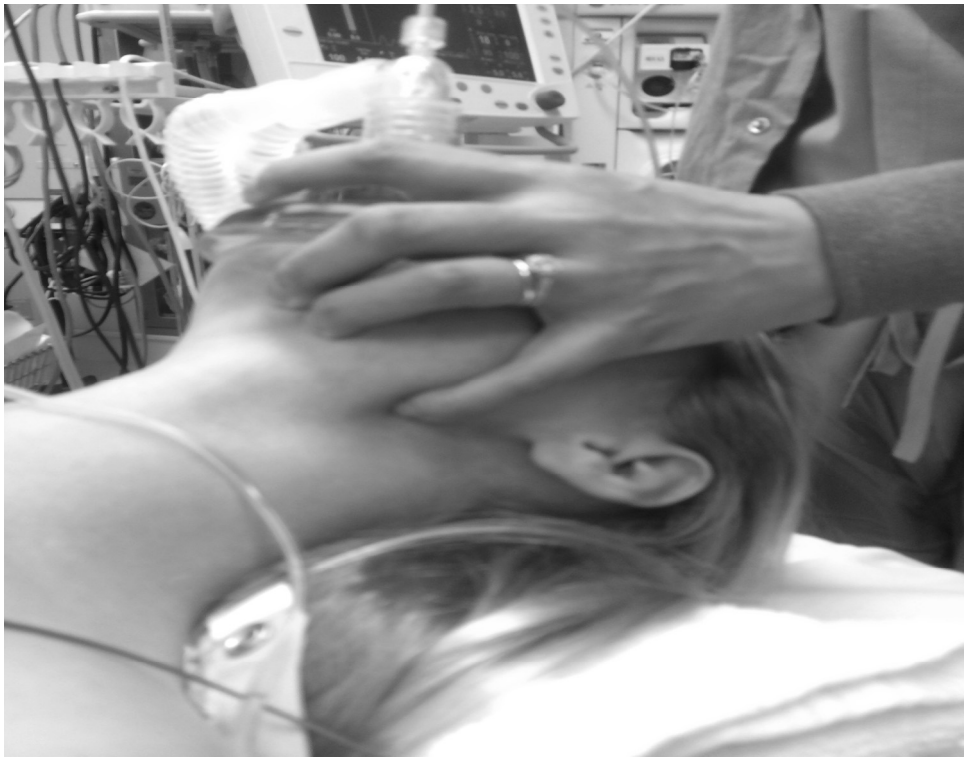
When a patient arrives from the operating room, the anesthetist should provide the nurse with a description of what occurred during surgery. This report should include: the surgery performed and any surgical complications that occurred, the form of anesthesia used (sedation, general, block, spinal, epidural) and any complications that occurred, the drugs administered (including anesthesia drugs, antibiotics, muscle relaxants, narcotics or other drugs for pain relief) and their route of administration. Many drugs given during anesthesia last into the postoperative period and can affect the patient's recovery from anesthesia. All surgeon-specific needs, including limitations or restrictions on patient positioning after surgery, dressings and their care (if any), the care of drains (if present), whether there is concern about circulation of blood to the surgical area (e.g., blood flow to a limb) should be reported to the patient's nurse. It should be clear if arm restraints are necessary or can be used if required. The anesthetist should inform the PACU nurse of the type and volume of fluid administered during surgery (glucose or no glucose, normal saline, Ringer's Lactate, or other balanced salt solution). The anesthetist should also report how much blood loss occurred, and if known, the patient's hemoglobin concentration and the urine output. Before the anesthetist leaves the PACU, the child's nurse should be completely satisfied that he/she understands what occurred in the operating room and that he/she has a way to immediately communicate with the anesthetist and get help when needed. The anesthetist is responsible for writing orders for the postoperative care of the patient in the PACU so there will be no confusion as to what the orders are. These orders should include the need for oxygen and for how long, the type of medication and drug dose for pain relief, the amount and type of intravenous fluids (ml/kg) per hour, and the time to re-administer antibiotics if necessary. Any other patient or surgery specific orders should be written at this time, including NPO status.

Initial Assessment

Initial evaluation of patients in the PACU starts when they arrive. Assessment includes an over all visual inspection of the patient including evaluation of the airway, breathing, cardiac function, surgical site, and skin color. This is done while monitors are being applied to the patient and oxygen is being administered. Oxygen can be provided using a simple facemask or by blowing oxygen by the patient's face ('blow by') if the mask is not tolerated. The concentration of oxygen delivered as well as the length of time needed should be patient specific. Signs of difficult breathing are evaluated. The ribs, sternum, or the supra or infra sternal notches should show no evidence of in drawing (retraction) during inspiration, nor should one hear breathing noise during inspiration or expiration. These are signs of airway obstruction and/or increased work of breathing and should lead to immediate correction of whatever is causing the obstruction. Upper

airway obstruction is the most common reason for hearing noise during inspiration, and is frequently due to the tongue falling back into the throat. This often occurs because anesthesia blocks or reduces activity of the muscles that hold the tongue away from the pharyngeal wall (genioglossus and hyoglossus). Tone in these muscles returns as the patient awakens from anesthesia. When this happens, the obstruction disappears and the patient can breathe without obstruction. Snoring is a common sign of obstruction. Treatment of this type of upper airway obstruction requires moving the tongue away from the pharyngeal wall. To do this, a rolled towel is placed under the patient's shoulders and the head is extended. If this fails to relieve the obstruction, the little fingers of ones hands are place in the angle of the jaw and the jaw is pulled forward (**Figure 22-3**). Since the tongue is attached to the posterior mandible, pulling the mandible forward pulls the patient's tongue out of the pharynx. Inserting an oral or nasal airway also helps. However, oral airways occasionally cause vomiting or injure the teeth. Airways inserted through the nose can cause bleeding if they tear infected, friable adenoids tissues of young children; nasal airways are frequently avoided for this reason in young children. Oxygen should be administered by facemask when attempting to relieve the airway obstruction and should be continued until the patient can maintain a normal airway without signs of obstruction or oxygen desaturation. Retractions of the chest wall and sternum will stop and airflow within the chest will be free when the obstruction has been relieved. A noise that occurs during expiration (wheezing) is usually caused by airway obstruction within the chest (e.g., asthma). This obstruction may need to be treated with drugs (Albuterol) given by a nebulizer. Stridor is a high-pitched sound caused by a narrowed or obstructed airway. This may occur in children with croup, laryngeal edema (from a tracheal tube), or with the presence of a foreign body. Stridor is treated by providing positive pressure ventilation by mask with 100% O_2. The O_2 flow rate should be 10-15 liters/ min. Treatment for a partial occlusion of the airway that is caused by edema may include the use of racemic epinephrine. *Any airway issue is a true emergency, and an anesthetist must be notified*

Figure 22-3: Jaw Thrust in a Child



The anesthetist is performing jaw thrust by placing one of her hands on each side of the child's head. She has placed the tips of her little fingers in the angles of the patient's jaw behind the mandible and is lifting the jaw forward (upward) while slightly extending the patient's head. The facemask is being held on the patients face with the thumb and index finger of the anesthetist's left hand. Oxygen is being delivered through the facemask. I necessary she can ventilate the patient's lungs.

Grunting during exhalation is evidence of reduced functional residual capacity (FRC), which is the volume of gas within the chest at the end of expiration. The FRC's function is to reduce the work required to inspire gas and to allow oxygen and carbon dioxide to be exchanged during both inspiration and expiration. When the FRC is low, the concentration of oxygen varies between inspiration and expiration. When the FRC is normal oxygenation remains relatively constant during both inspiration and expiration. The patient's respiratory rate should be determined and should be normal for the patient's age. (**See Appendix 1**) If it is not, the reason for this abnormality (pain, fever, acidosis, sepsis, hypoxemia, low FRC) should be sought and corrected when possible.

The pattern of respiration is important. Normally, the abdomen and chest move in the same direction during breathing, in with inspiration and out with expiration. During airway obstruction, the chest moves inward and the abdomen outward with inspiration. Patients who contract their chest and abdominal muscles during expiration usually have airway obstruction (asthma, tumor, mucus, blood). Movement of the head (head bobbing during breathing) and nasal flaring (widening of the nostrils during inspiration) also indicate airway obstruction during inspiration.

Upon arrival in the PACU, the patient's level of consciousness is evaluated. Several questions should be answered. Does the patient respond to voice or stimulation? Those who fail to respond are more likely to have airway obstruction and to vomit and aspirate than patients who are awake when they arrive. Awake patients, on the other hand, while less likely to have airway obstruction and aspiration, need to be watched closely to prevent self-harm. IV's, tubing and dressings need to be watched so the patient does not remove them. The goal is to have a calm, responsive patient, which requires her/him to be free or nearly free of pain. Children who have pain and are partially anesthetized often are "wild" and hard to control. One-to-one patient care may be needed until the child is calmer. It is important that gurneys have working brakes and side rails for patient safety. Treating a child's pain allows them to rest and recover more easily and slowly. How much pain relief is required depends on the amount of pain the child has and on the amount of analgesic given during surgery. Close observation is needed to evaluate if pain is being experienced. It is more effective to begin providing pain medication before pain is experienced. Once they have significant pain, much higher amounts of drug are required to control the pain. Many times having a parent or family member at the bedside during wake up and recovery allows familiar comfort measures (holding, familiar voice, rocking, etc.) to be used, which may decrease the need for pain medication.

Table 22-1 gives the doses of commonly available analgesic drugs. Fentanyl 1-2 micrograms/kg (mcg/kg) effectively relieves pain for 30-60 minutes and then requires additional doses of ⅓-½ of the original dose of drug. Morphine provides 1-2 hours of pain relief. It is best to titrate the dose of analgesic drugs to achieve the effect desired (pain relief without depression of respiration). Doing so usually avoids giving too much drug. Other drugs, such as acetaminophen 15 mg/kg I.V. or orally or 40 mg/kg by rectum (as a one-time dose), often provides good pain relief for hernia repair and similar surgeries. The combination of oral acetaminophen and codeine was commonly used in the past but is seldom used at present because 15%-to-20% of patients do not get pain relief beyond that due to the acetaminophen. Codeine works because the liver converts it to morphine. Some patients, especially those in Africa and Japan, can metabolize more codeine to morphine than others, and this has caused both respiratory and cardiac arrests.[3] Some of these children have died.

All drugs, including those used to relieve pain, cause unwanted side effects in some patients. Narcotics (fentanyl, morphine) cause nausea, vomiting, itching, and constipation in some patients. These drugs can also depress respiration that leads to hypoxemia. Furthermore, narcotics can reduce the tidal volumes and the respiratory rates of some patients, which may cause them to retain carbon dioxide and develop respiratory acidosis. If the respiratory depression (hypoventilation) is severe enough, apnea and cardiac arrest may occur. Naloxone (0.01 mg/kg for hypoventilation, 0.1mg/kg for respiratory and/or cardiac arrest due to narcotic overdose) - up to a total of 1 mg) is given and may be repeated every 2-3 minutes as needed to restore breathing in a patient who has significant hypoventilation and/or apnea from narcotics. It must be remembered

that the effect of naloxone only lasts 30-45 minutes. Therefore, those patients given naloxone must be observed closely for signs that naloxone's effects are wearing off and hypoventilation is recurring. Very large doses of acetaminophen injure the liver and may cause the patient to die. Intravenous ibuprofen relieves pain, but some surgeons do not want to use it because it has been said that it causes bleeding at surgical sites; however, this is unproven. Tramadol, another frequently available and effective analgesic, is also metabolized to morphine by the liver and has been reported to cause apnea in patients, especially those who have previous sleep apnea and when the drug is give intravenously. Respiratory depression is uncommon when the drug is given orally. The most common side effects of tramadol are nausea, vomiting, dizziness, dry mouth, and sedation. These side effects also occur less frequently when the drug is given orally. Allergic reactions have been reported but are rare. Cardiac and central nervous system effects are rare after giving normal doses of the drug.

Nerve blocks or spinal or epidural analgesia provide excellent postoperative pain relief (**See Chapters 20 and 21**). However, due to their effects, the stay in PACU may be prolonged. The anesthetist should evaluate the patient for stability of her/his condition before discharging the patient to the ward or the ICU.

Table 22-1. Drugs commonly Used in the PACU to Relieve the Pain of Patients

Drug	Dosing	Administration Route	Time Between Doses	Maximum Dose per 24 hrs.
Fentanyl	1-2 mcg/kg	IV or IM	45 min-1 hr.	25-30 mcg/kg
Morphine	0.05-0.1 mg/kg 0.3 mg/kg	IV or IM Orally	< 1mo 1 x/d 1-3mo 2-4 hrs. 1-10 y 4 hrs.	0.1 mg/kg 0.2 mg/kg 0.3 mg/kg
Meperidine	1 mg/kg	IV or IM	2-4 hrs.	10 mg/kg
Ketorolac	0.5mg/kg	IV	6-8 hrs.	2mg/kg
Acetaminophen	15 mg/kg 40 mg/kg	PO or IV Rectally 1st dose 40mg/kg 2nd dose 20 mg/kg	6 hrs. 6 hrs.	400-600 mg/kg
Ibuprofen	10-40 mg/kg	Orally	6-8 hrs.	40 mg/kg
Tramadol (Ultram)	1-2 mg/kg	Orally	4-6 hrs.	8 mg/kg/d

mcg = micrograms, mg/kg = milligrams per kilogram, IV = intravenous, IM = intra muscular, PO = by mouth, hr. = hours, min = minutes

Nausea and Vomiting

Nausea and vomiting are common complications after anesthesia and surgery and can be distressing to families and patients. Both nausea and vomiting occur commonly after narcotics or tonsillectomy. Emptying the stomach of blood and secretions and administering antiemetic drugs to patients before the end of surgery reduce the occurrence of nausea and vomiting but does not

eliminate them. Zofran (ondansetron) 0.45mg/kg – maximum dose 4 mg/dose - is an effective antiemetic. If needed, the dose of ondansetron 0.15mg/kg can be repeated every 4 hours. Reglan (Metacolpromide) 1-2mg/kg every 2-6 hours and Compazine (procloparazine) can also be used to treat nausea, but their side effects may limit their usefulness. Decadron (dexamethasone) 0.25-0.5mg/kg, (4-8mg total) when given during surgery, significantly reduces the incidence of postoperative nausea and vomiting and reduces airway swelling that may add to postoperative pain. There is little evidence that this one-time dose of Decadron increases postoperative bleeding or other complications. Specific surgeries (e.g., tonsillectomies, cleft palate repair) may bleed after surgery and cause blood to be swallowed, which may increase the incidence of postoperative nausea and vomiting. Patients who drink liquids before going home often vomit on the way home due to movement of the vehicle, especially if the drive home involves curvy roads. However, it is important to be sure the patient can drink without vomiting before a patient is discharged home from the PACU. It is best to start with water or juice and see how the patient does. If there is no nausea or vomiting, they can be discharged after warning the parents that the patient may vomit with excessive movement.

On Going Assessment

The patient's condition must be reevaluated throughout her/his PACU stay. This includes adequacy of the airway/respiration, blood pressure, heart rate, peripheral perfusion (capillary filling, skin temperature), and pulses. It is recommended that vital signs be recorded in the patient's chart on a nursing form every five minutes for 15 minutes, then every 15 minutes for one hour; every 30 minutes for two hours, then every hour or until the patient is discharged from the PACU. Patients who are unstable require more frequent vital signs/observation. Vital signs are recorded so that if problems occur, those caring for the patient can hopefully determine what caused the problem. The purpose of recording data in a patient chart is to evaluate the patient's condition over time and whether treatment is improving the patient's outcome.

Intravenous Fluid Management

Management of intravenous fluids during recovery from anesthesia is an important part of PACU care, since surgery often causes fluids to shift from one body compartment to another. This can cause the patient's intravascular volume to become inadequate. Thus it is important for nurses to obtain as much information as possible about the surgery performed, the amount of blood lost during surgery, and the amount and type of fluid given to replace losses. Part of the assessment should include examining the patient to determine if the intravascular volume is adequate or not. (**See Chapter 1**) The *fontanel* of babies younger than three months of age is usually open and is a useful guide to the evaluation of the patient's state of hydration. Skin over the fontanel should be even with the outer bone of the skull. If the skin is at the level of the inner bone of the skull, the patient is 5% dehydrated; if it is below the inner bone of the skull, hydration is reduced 10% or

more. The *mucus membranes* of the mouth should be wet when felt. If they are not, this is a sign on of dehydration. Babies who are less than three months of age do not make tears when they cry. Older patients do. Thus, when an older patient cries without making tears, this is a sign of dehydration. When the skin is picked up (*tented*) it should immediately return to its normal position if hydration is normal. If the hydration is not normal, the skin will remain tented for a few seconds and slowly return to its normal position. *Pulses* should be present and normal in all limbs. If the pulses are reduced or absent in the wrists and feet, the patient is about 5% dehydrated; if the popliteal or brachial pulses are absent or decreased, volume is reduced by 10%; if the femoral and axillary pulses are reduced or absent, the child's intravascular volume is reduced by 15% or more. The arterial blood pressure should be within normal limits for the patient's age. If it is more than 20% lower than normal for age, the child is probably hypotensive. Most children produce 1 ml/kg/hr. of urine or more. If their urine production is less than this, hypovolemia is often the cause. Finally, the examiner should place a warm hand on the child's chest and move her/his hand out along the shoulders and arms to the patient's fingers. The patient's skin should be warm all the way out to the fingertips. If the skin of the forearm suddenly becomes cold, the volume is reduced by 5%; if the skin of the upper arm gets cold as the hand moves over it, the patient's volume is depleted by 10%; if the entire arm is cold, the patient's volume is reduced by 15% or more. When all of these factors are taken together, it is easy to estimate the patient's intravascular volume. The information gained from this examination can be used to guide the amount of fluid needed to correct hypovolemia. Following fluid administration, the patient's volume status must be reevaluated to determine if sufficient fluid has been administered or if more is needed. Before discharge from the PACU, the patient's intravascular volume and perfusion should be normal.

Discharge From the PACU

Part of on going patient assessment is to determine when the patient is ready for discharge from the PACU. In general, patients are ready to leave when they are awake, responding to voice and commands, able to maintain a normal SpO_2 in room air, and have little pain. **Table 22-2** is the modified Aldrete Score, which is widely used to determine if patients can be discharged from the PACU to their hospital room or to home.

Postoperative instructions are given prior to discharge from the PACU to family members and/or nursing staff. These instructions should include information on diet, pain control and surgery specific instructions (dressings, when to shower/bathe, daily activities). Follow up appointments should be provided.

Table 22-2: The Modified Aldrete Score

Patient Variable	Scored values	Score
Activity	**Patient can move**:	
	All extremities	2
	Two extremities	1
	No extremities	0
Respiration	**Patient can:**	
	Breath deeply and cough	2
	Short of breath or decreased breathing	1
	Apneic or obstructed breathing	0
Circulation	**Arterial blood pressure:**	
	± 20% of pre-anesthesia BP	2
	± 20% to 49% of pre-anesthesia value	1
	± 50% of pre-anesthesia value	0
Level of Consciousness	**Patient is:**	
	Fully awake	2
	Responds to name	1
	Unresponsive	0
Oxygen Saturation (SpO2)	**SpO_2:**	
	>92% in room air	2
	>90% with added oxygen	1
	<90% with added oxygen	0

Each variable of the Aldrete Score is scored from 0-to-2. The numbers are added together to get a total score. If the total score is 9 or 10, the patient is ready for discharge from the PACU. If the score is less than 9, the patient should be observed until it is at least 9. ***With permission from: Aldrete JA. The post anaesthesia recovery score revisited [letter]. J Clin Anesth 1995;7:89– 91***

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Chapter 23
CRITICAL CARE MEDICINE IN THE DEVELOPING WORLD

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"An accident of latitude often determines whether a child lives or dies… this is not the nightly news, this is a crisis of our world and of our time – history will judge us and our success". Bono.

Economic Disparities in Critical Care Medicine

Around the world, over 10.1 million preventable deaths occur per year in children less than five years of age. Most of these are caused by infections and could be prevented with improved access to immunization and primary care. When a country's mortality rate for children who are under five years of age is low (fewer than 20 deaths per 1,000 live births), death from infections are lower and access to intensive care unit (ICU) care can lead to better survival from diseases like trauma, asthma and congenital heart disease. However, when the mortality rate of this same population is high, (greater than 30 per 1,000 live births) many deaths are caused by infections, i.e. pneumonia and diarrhea (**Table 23-1**) and ICU care is less certain to lead to better survival.

TABLE 23-1: Leading Causes of Death in Children younger than 5 years – 2011*

Cause	% total		
	Neonatal (0-27 days)	1-59 months	Total (0-4 years)
All causes	43	57	100
Pneumonia	5	13	17
Prematurity	15	2	17
Birth asphyxia	10	1	11
Diarrhea	1	9	9
Malaria	0	7	7
**** Some figures do not add up to 100% because of rounding.***			

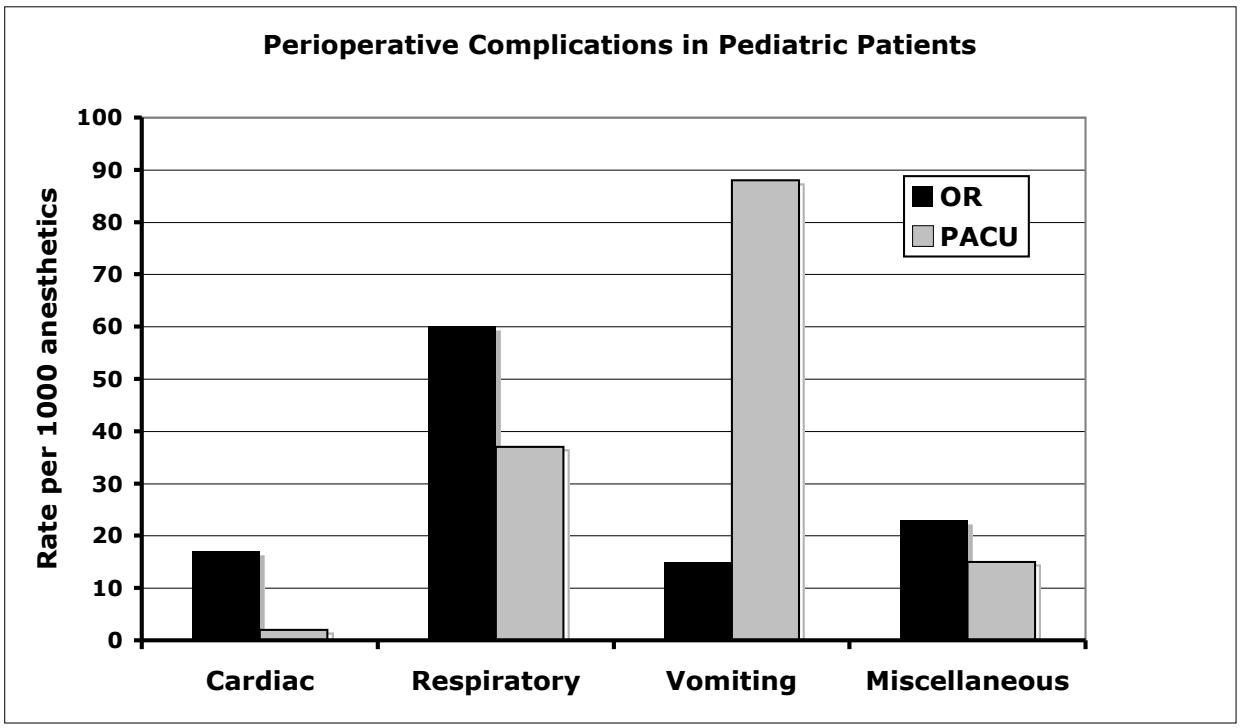
Adapted from Bryce J, et al. WHO estimates of the causes of death in children. Lancet 2005; 365: 1147–52.

Even though the money spent on ICU care could be spent on preventative treatments, is it fair that children needing ICU care not receive treatment given to children in wealthier countries? An important goal for every child in the world is to have available ICU care that can provide oxygen delivery, fluid resuscitation, tracheal intubation, mechanical ventilation, and support of the cardiovascular system.

Surgical (Perioperative) Problems – Most Common Needs:

Most perioperative problems are acute and can be corrected with time and good medical care. Since the first post-anesthetic care units (PACU) were opened in the 1940's, anesthetists have played an important part in stabilizing patients and improving the outcomes of surgery. The extension of PACU services after immediate post-anesthetic recovery was the start of critical care medicine. If needed, the same PACU environment may be used for anesthetic recovery and for ongoing ICU care. In many studies of post-anesthesia problems in adults, respiratory failure and hypoxia are common and serious problems, which can lead to cardiac arrest. The surgical or anesthetic-related death rate is as high as 1,063/100,000 in some countries. This is 2-to-3 times greater than that observed in wealthier countries. (1) The same post-surgery problems are likely to be even more common in pediatric surgery. Murat et al. reviewed intraoperative versus postoperative adverse events in a tertiary hospital of Africa and found respiratory problems to be more common in the operating room after the surgery, than in the recovery room (**Figure 23-1**). This may be because of the common need to "recover" pediatric patients in the OR rather than taking them to a PACU with limited resources.

FIGURE 23-1: **Common Perioperative Complications in Pediatric Patients**



Adapted from Murat et al (2)

Perioperative Respiratory Insufficiency or Failure

It is very important during recovery from anesthesia to recognize problems with breathing, cardiac function, and brain function, in order to decide which patients need ICU care. Since there are many physiologic changes in pediatric patients as they grow older, a table of normal heart rate, blood pressure, and respiratory rate is shown (**Table 23-2**).

Table 23-2: Important Physiologic Parameters for Respiration and Blood Pressure

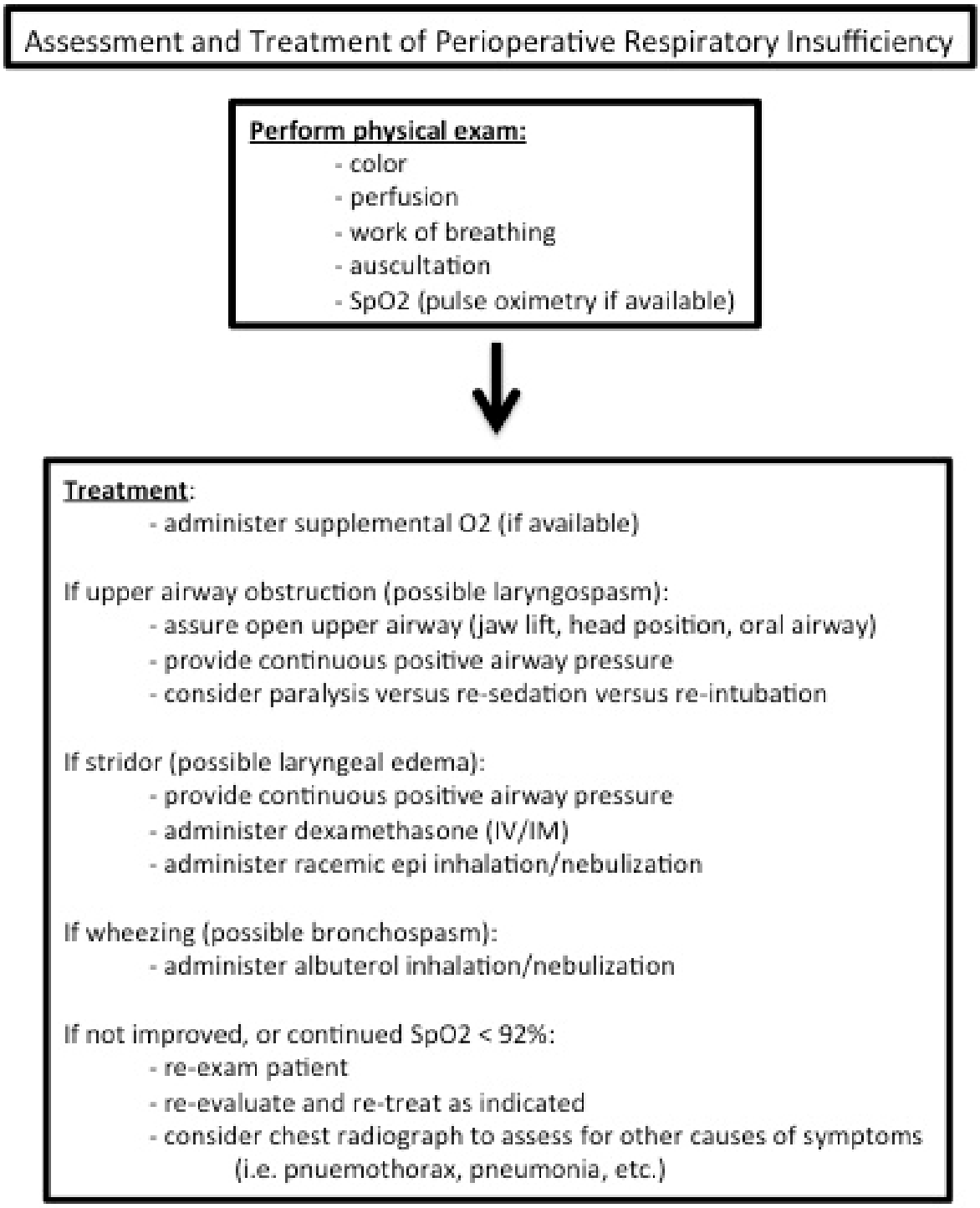
Heart Rate (rate/min)		
Age	**Awake Rate**	**Sleeping Rate**
Newborn to 3 months	85 to 205	80 to 160
3 months to 2 years	100 to 190	75 to 160
2 to 10 years	60 to 140	60 to 90
>10 years	60 to 100	50 to 90

Respiratory Rate (breaths/min)	
Age	**Rate**
Infant	30 to 60
Toddler	24 to 40
Preschooler	22 to 34
School-age child	18 to 30
Adolescent	12 to 16

Definition of Hypotension by Systolic Blood Pressure and Age	
Age	**Systolic Blood Pressure**
Term neonates (0 to 28 days)	<60 mm Hg
Infants (1 to 12 months)	<70 mm Hg
Children 1 to 10 years (5th BP percentile)	<70 mm Hg + (age in years x 2) mm Hg
Children >10 years	<90 mm Hg

Breathing problems are common in children, and we recommend a standard way to examine airway and breathing, and ways to treat the child with a breathing problem. We listed the order of problems, from the most common operating room issues to most common PACU issues. Frequent examination of the patient is required to decide the cause of hypoxemia and respiratory distress because the cause(s) is always changing; the anesthetist must check the patient again and again to make the right decisions (**Figure 23-2**).

FIGURE 23-2: Algorithm for Management of Respiratory Insufficiency



Laryngospasm, *which* is the involuntary contraction of the glottic muscles (vocal cords) that leads to tight closure of the glottis, occurs frequently during pediatric anesthesia and can result in blockage of the airway, admission to the hospital, need for a long time in PACU, and sometimes tracheal intubation and mechanical ventilation. (3) If laryngospasm is very severe, it can induce negative pressure pulmonary edema (rapid onset of rales, respiratory distress, and hypoxia). (4) Laryngospasm can happen any time during or after an anesthetic, but the risk of laryngospasm is highest immediately after tracheal extubation and may be increased by stimulation (e.g., airway suctioning) of the patient during emergence from anesthesia. Laryngospasm is more common in younger patients. It can occur in the PACU or ICU as well as in the OR, especially when the tracheal tube is removed "deep" and the patient emerges from anesthesia in the PACU. The first treatment of laryngospasm is continuous positive airway pressure (CPAP) via facemask with 100% oxygen. This often is all that is required to correct this problem. Some patients require deepening of the anesthetic (a bolus of propofol or other induction agent) in addition to CPAP. The action that almost always relieves laryngospasm is the administration of a small dose of succinylcholine IV 0.1mg/kg or IM 0.3mg/kg to relax the vocal cords. When the laryngospasm breaks, the anesthetist can support breathing with bag and mask ventilation, but sometimes the patient's trachea must be intubated.

Post-extubation stridor is a changing inspiratory upper airway obstruction that can occur in any age group, but happens more often in pediatric patients due to anatomic differences of the airway. Because the subglottic region (below the vocal cords) is the narrowest portion of the pediatric airway, an endotracheal tube that has been inserted easily through the true vocal cords may still cause pressure edema and/or necrosis of the subglottic mucosa. Also because the pediatric airway is smaller than that of the adult, when there is mucosal edema, even if mild, the airway obstruction can be severe (**See Chapter 6**). This is made worse by the soft cartilaginous portions of the pediatric airway that collapse easily. This is also made worse by negative intrathoracic pressure, which leads to respiratory distress and inspiratory stridor. In its severe form, the anesthetist can hear expiratory sounds as well; in patients with little to no ventilation (movement of almost no air), there is often no stridor. The first treatment for this condition is inhaled vasoconstrictor (e.g. nebulized racemic epinephrine) to decrease tissue edema (**See Chapter 4**). Giving racemic epinephrine (2.25%) 0.25mL in 3cc normal saline via high-flow nebulization and repeating up to three times with humidified oxygen in between treatments is the first treatment. Corticosteroids may also be administered for a longer-acting, anti-inflammatory effect. Those patients who receive multiple treatments with racemic epinephrine and/or corticosteroids are often admitted to the hospital for observation. Dexamethasone 0.5mg/kg/dose IV every six hours for four-to-six doses (24-36 hours) often works well. Treating patients with a history of croup or subglottic narrowing who are at high risk for stridor may help. Not every small child with mild stridor needs treatment. (5) Rarely,

patients may require re-intubation for significant obstruction. If re-intubation is needed, a smaller tracheal tube than the one placed for surgery should be used, and dexamethasone should be used for 24 hours prior to again attempting to remove the tracheal tube. If stridor happens again, evaluation by an ear, nose, and throat surgeon (if available) by bronchoscopy may be needed to examine the glottis and trachea for other abnormalities (such as arytenoid dislocation or other trauma, tracheitis, or airway granuloma).

Bronchospasm, (wheezing) or reversible bronchiolar smooth muscle constriction leading to air-trapping, respiratory distress, and whezing. These are the main signs of an asthma exacerbation. Bronchospasm is most commonly seen in known asthmatics, but it can also be seen in any patient because endotracheal intubation and extubation irritate the airway. The first treatment for wheezing is inhaled beta-agonist therapy (e.g. albuterol). However, anesthetic gases also work very well to relax airway smooth muscle and may be used (if blood pressure is not to low) to treat severe wheezing in both intubated and non-intubated patients. Other drugs, like anticholinergic therapy, such as inhaled ipratropium bromide, can be used in select patients (6), although these drugs may not work as well as the beta-agonists. If the wheezing is not too bad, inhaled bronchodilators may be sufficient. The patient with moderate to severe bronchospasm may require additional treatment; corticosteroids are next. Drugs like prednisone 2mg/kg orally once daily or methylprednisolone 0.5-to-1mg/kg/dose IV every 6 hours, maximum dose 80mg/day, work well. Other drugs for very severe wheezing include magnesium sulfate for its airway smooth muscle relaxation (usual dose 75mg/kg/dose IV, maximum dose 2g) (7) and subcutaneous epinephrine (10mcg/kg/dose subcutaneously, max dose 0.5mg). If all these treatments do not work, continuous infusions of adrenergic agents (e.g., terbutaline, epinephrine) are often used and work most of the time.

***Persistent oxygen requirement*:** Giving oxygen after emergence from anesthesia and tracheal extubation is the standard of care in most of the world. Many patients need oxygen throughout their PACU stay and sometimes also require oxygen on the ward. In parts of the world where oxygen cannot be given on the ward, patients with increased work of breathing and cyanosis after discontinuing oxygen should remain in the PACU or ICU until they no longer require oxygen. Oxygen can be given by nasal cannula, facemask or blow-by oxygen (8). The cause of oxygen desaturation or hypoxemia (SpO_2 < 92%) may include mild atelectasis with mismatched ventilation and perfusion, reduced functional reserve capacity (lung volumes), or limited inspiratory effort with mild hypoventilation due to pain (i.e. “splinting”) or left over anesthetic action. Some pediatric patients are receiving added oxygen preoperatively or have relative hypoxia at baseline (e.g. congenital heart disease patients with intra-cardiac shunts – **See Chapter 5**). However, more serious causes of hypoxemia must always be considered. These include significant hypoventilation (which may be masked by supplementary oxygen), pulmonary edema, pneumothorax, and significant ventilation/perfusion mismatch as in pulmonary embolism (see below). Persistent perioperative hypoxia has been associated with

postoperative confusion in adults following cardiac surgery (9). Thus, although not every patient will return to baseline oxygen requirement prior to PACU discharge, the need for added oxygen should be evaluated and provided in all patients requiring it. If available, a chest x-ray can diagnose lung collapse and pneumonia. If pneumonia is suspected, treatment for pneumonia (see section on Pneumonia) is needed.

***Postoperative central apnea*:** Young infants particularly premature infants, are at risk of postoperative central apnea. This occurs with both general anesthesia and regional anesthesia (10), although fewer patients have apnea after regional anesthetics. Risk factors for postoperative apnea in infants include anemia, history of apnea, younger gestational age, and younger post-conceptual age (gestational age plus ex-utero age). Cote et al. showed that the chances of postoperative apnea were five percent in non-anemic infants with a post-conceptual age of 48 weeks or less monitored in the PACU (11). Most pediatric anesthesiologists believe that after 50-to-60 weeks post-conceptual age, the chances of apnea are zero. We recommend that any infant with anemia or a history of apnea, any infant less than 56 weeks post-conception, and any infant with apnea demonstrated in the PACU should be admitted for postoperative monitoring with pulse oximetry for 24 hours. Preoperatively, plans should be made for the care of patients with these problems, and all young infants who have outpatient surgery require longer observation for apnea in the PACU before discharge.

Perioperative Hypovolemia

Before we discuss hypovolemic shock, we will discuss baseline daily fluid requirements and how to determine fluid deficits (**See Chapter 3**). Various methods have been used for determining the daily free water and electrolyte needs of children and infants. (12). **Table 23-3** reviews this information.

TABLE 23-3: Administration of Maintenance Fluids in Infants and Children

Body Weight (kg)	Fluid per day (ml/day)	Hourly Rate
<10 kg	**100-120 ml/kg**	**4 to 5 ml/kg/hour**
10-19 kg	**90-120 ml/kg**	**3 to 5 ml/kg/hour**
>20 kg	**50-90 ml/kg**	**2 to 3 ml/kg/hour**

TABLE 23-3 (Continued)

For Example:		
2 kg infant	**220 ml/day**	**9 ml/hour**
4 kg infant	**440 ml/day**	**18 ml/hour**
10 kg infant	**1100 ml/day**	**45 ml/hour**
14 kg child	**1400 ml/day**	**58 ml/hour**
20 kg child	**1800 ml/day**	**75 ml/hour**
24 kg child	**2000 ml/day**	**83 ml/hour**
26 kg child	**2100 ml/day**	**87 ml/hour**

Adapted from WHO Pocket Book for Hospital Care for Children. Guidelines for the management of common childhood illnesses, 2nd Edition, 2013; ISBN 978 92 4 154837 3.

Keep in mind that these methods of determining fluid deficits work well for healthy children with intact skin and mucosal surfaces, but may not work as well for the post-operative child with increased insensible fluid losses, ongoing surgical losses, or malnutrition. The intraoperative and perioperative course may have both evaporative losses through the surgical wound (which may be in excess of 8-to-10ml/kg/hour for large abdominal incisions) and ongoing blood loss, in a patient with underlying medical illness and increased fluid requirements. These problems can quickly lead to hypovolemic shock in the pediatric surgery patient. Multiple studies have determined that hypovolemic shock is a common perioperative cause of pediatric cardiac arrest. For instance, Desalu et al. reviewed postoperative cardiac arrest (POCA) in children at a single Nigerian center and they identified hypovolemia as its most common cause (13). The diagnosis is often hard becasue pediatric patients in shock will usually not be hypotensive (have low arterial blood pressure) until very late when circulatory collapse happens. Instead, they often have tachycardia and constriction (narrowing) of the small blood vessels to the skin of the arms and legs (which makes capillary refill time longer) to maintain normal arterial blood pressure for age. This means that a small number of pediatric patients will arrive in the PACU with unrecognized, but compensated, hypovolemic shock. If they then have more fluid loss from surgical bleeding, tachypnea and increased insensible fluid losses thorough the airway, and vomiting, they can develop severe shock and cardiac arrest. It is very important that postoperative team (whether PACU or ICU) examine all patients on admission to see if they have compensated or decompensated circulatory shock, and rapidly work to treat any cause of hypovolemia they find. This should include not only measurement of heart rate and arterial blood pressure and comparing them to normal values for the child's age, but also examination of capillary refill time, which should be less than two seconds in normal pediatric patients (three seconds in infants). The

type and amount of fluid resuscitation should take into account whether the patient is malnourished, because "over-resuscitation" (giving too much fluid) by modern shock management results in poor outcomes in malnourished children, probaboly because they have reduced concentrations of serum proteins. (14)

***Postoperative Surgical fluid losses*:** Depending on what surgery was done and how much bleeding there is, postoperative bleeding may be a reason for emergent return to the operating room (for example, a patient after cholecystectomy with 10ml/kg/hour of frank blood in a surgical drain), an indication for intensive monitoring (a multi-level spinal fusion patient who is expected to continue bleeding for up to 24 hours postoperatively and should be monitored in the ICU), or may be of little concern (the exploratory laparotomy patient with 0.5ml/kg/hour of serosanguinous drainage who has stable arterial blood pressure and heart rate). When in doubt, always speak to the surgeon about the amount of drainage. However, even if the patient does not need to return to the OR, excessive fluid or blood losses should be replaced with an appropriate fluid. Electrolyte composition of the lost body fluid may be measured to help guide therapy if necessary.

Patients with severe surgical bleeding can also have dilutional hypofibrinogenemia (low fibrinogen level), coagulopathy (especially Factors V and VIII), and thrombocytopenia (low platelet count) due to crystalloid and packed red blood cell replacement without giving plasma and platelets (**See Chapter 4)**. Patients who receive large volume transfusions (greater than one blood volume) may also be at risk for hypothermia, breakdown of their blood clots, platelet dysfunction, and electrolyte abnormalities. These patients should have laboratory tests for complete blood count, platelet number, and blood clotting times (prothrombin time-PT, partial thromboplastin time-PTT) followed by blood component replacement with platelets, fresh frozen plasma, or cryoprecipitate as needed to correct ongoing bleeding (**See Chapter 4**). (15)

Extracellular Fluid Sequestration means edema formation in the tissues with fluid that comes from the plasma. Also called "third space fluid," this volume of fluid is the cause of a lot of postoperative hypovolemia. It is very hard to measure the amount of third space fluid since it is not in direct connection with the circulatory system. Bowel edema and fluid leakage after major intra-abdominal surgery is the most common source of "third space fluid" loss, but pleural effusions, peripheral edema, or swelling at any other site of capillary damage may be the cause. All patients have some fluid leakage after major intra-abdominal surgery, but since the anesthetist cannot measure the amount of fluid lost, physical examination and measuring arterial blood pressure and heart rate again and again are very important when deciding how much fluid to give. Replacing these losses with a balanced crystalloid solution that contains electrolytes like sodium and potassium that are in the same concentrations as human blood, is important. Ringer's Lactate is often used because it has the same electrolyte concentrations as the leaking fluid and as blood plasma. However, it contains no proteins. Because of this (and for other reasons) even giving as much as 20ml/kg/hour of crystalloid often is not enough, and it can produce even more edema. (16).

Temperature Instability

Although the usual surgical teaching regarding the causes of postoperative fever (temperature > 38^{o}C) is that common problems that occur in the first 24-48 hours postoperatively (for example, surgical site infection, atelectasis/pneumonia, urinary tract infection, deep venous thrombosis are usually not the cause of fever in the PACU. If the patient was afebrile preoperatively, patients who have a fever on arrival to the PACU or during their PACU stay must be carefully evaluated to determine the cause of the fever. Most of the time, the cause of a new fever in infants and small children will be due to an OR that is too warm (i.e., lack of air conditioning in the OR). But, serious problems must always be thought about. Most important among these are malignant hyperthermia and sepsis.

Malignant hyperthermia (MH) is a rare but potentially fatal disease of skeletal muscle that causes a large increase in muscle metabolism after exposure to a drug that triggers MH. These drugs include succinylcholine and inhalational anesthetic agents, with halothane being the worst culprit for doing so. It is very important for the pediatric anesthetist to realize that the first signs of MH may not occur until the patient is in the PACU or even after discharge from the PACU. Patients have tachycardia, increased oxygen consumption, and increased carbon dioxide generation (as measured by end-tidal CO_2 monitoring), very stiff muscles and rigidity, and rapid development of high fever (often in excess of 42^{o}C). Common signs and symptoms, along with the management of MH, are outlined in **Table 23-4**. Progression to rhabdomyolysis (breakdown of muscle tissue) is common, and the disease carries an extremely high death rate if not recognized and treated quickly. Treatment requires critical care and has five parts, which are treated simultaneously: 1) Give dantrolene IV (2.5 mg/kg)[12], 2) hyperventilate with 100% oxygen, 3-4) give enough IV fluids and manage the electrolytes, and 5) alkalinize the blood with sodium bicarbonate IV (1-2mEq/kg – more may be needed).[13] The drug causing the MH—usually anesthetic gases—must be stopped immediately. The anesthetist can telephone the 24-hour Malignant Hyperthermia Hotline at (800) MH HYPER (644-9737) or 001-1-315-464-7079 (outside the U.S. and Canada) for help with MH crisis management. Alternatively, information is available on line at http://www.mhaus.org/healthcare-professionals/managing-a-crisis.

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[12] Dantrolene is expensive and seldom used. In a city with several hospitals, the hospitals can go together and buy an appropriate amount of dantrolene to treat a teenage child and store it in one central hospital. When needed it can be quickly sent by messenger to the hospital requiring the drug.

[13] 50meq of sodium bicarbonate produces 1,250cc of CO_2. Consequently, controlled ventilation must be assured before giving sodium bicarbonate to prevent causing worse respiratory acidosis.

TABLE 23-4: Clinical Symptoms and Management of Malignant Hyperthermia

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Clinical Signs of MH:

Increased temperature (temp >42^{o}C) tachycardiaI, arrhythmias, increase in end-tidal CO_2, desaturation, fasiculations, muscle rigidity

Laboratory Tests Abnormalities Associated with MH:

Increased serum potassium, metabolic acidosis (pH < 7.2), myoglobinuria

Treatment of MH:

1) Call for help, call the MH hotline

2) Hyperventilate and increase FiO_2 to 1.0

3) Administer dantrolene 2.5mg/kg IV [Repeat as frequently as needed until the patient responds with a decrease in $ETCO_2$, decreased muscle rigidity, and/or lowered heart rate. Large doses (>10mg/kg) may be required for patients with persistent contractures or rigidity]; then give 1mg/kg IV every 6 hours once the patient's condition is stable.

4) Stop surgery and anesthesia.

5) Cool the patient: iced intravenous fluids, lavage stomach, bladder, rectum with cold solutions. External cooling with ice packs to axilla, groin and head. Goal temp <38^{o}C.

6) If metabolic acidosis is present, administer Nabicarb 1-2mEq/kg IV (if available) and repeat as needed.

7) If the concentration of potassium is elevated, administer glucose, insulin and calcium.

8) Keep urine output at 2ml/kg/hour using IV hydration, mannitol and diuretics if necessary.

9) Avoid calcium channel blockers.

Bacteremia (sepsis) is seen much more often than MH (i.e., bacteria in the blood) and may be just as deadly in the pediatric patient, particularly when not clinically suspected. Very often bacteria are released into the bloodstream following certain surgical procedures, such as exploratory laparotomy (particularly if the surgical technique involves "running the bowel"), and this can cause sepsis and septic shock in some patients. The clinician must decide how much testing to do

for a simple fever. A study by de la Torre et al. of women undergoing exploratory laparotomy for gynecologic surgery found a fever greater than 38.0^{o}C in 29% of patients after surgery. But, real infection was seen much less often and was mostly seen in those with a history of cancer, intraoperative bowel resection, a higher fever, high white blood cell counts, and prolonged fever. Because serious infection does not occur very often, testing for fever should be done only on those patients who have other signs of infection, for other patients the testing is not worth the cost. (17). But during anesthesia or during immediate recovery from anesthesia, pediatric patients might not demonstrate the usual signs of septic shock, like fever, tachycardia, poor perfusion (as measured by capillary refill time), and hypotension, which is a late finding. So, if the anesthetist is worried about sepsis then broad-spectrum antibiotics should be given urgently. At the same time, blood, urine and sputum cultures should be sent to diagnose the cause of the sepsis.

Shivering often happens right after surgery (18). While it is usually not serious, it can be uncomfortable for the patient and concerning for the family. Lyons et al. (19) found that 14.4% of children had shivering postoperatively, and low postoperative temperature was the main cause. A larger study (20) of 1,507 children found a much lower number of shivering children (3.5%) and saw that an age of more than six years, a long surgery procedure, and use of an intravenous induction drug were often associated with shivering. In both studies, primary treatment was forced air warming of the child. The most important treatment of shivering is warming the child.

Delayed Emergence and Emergence Failure

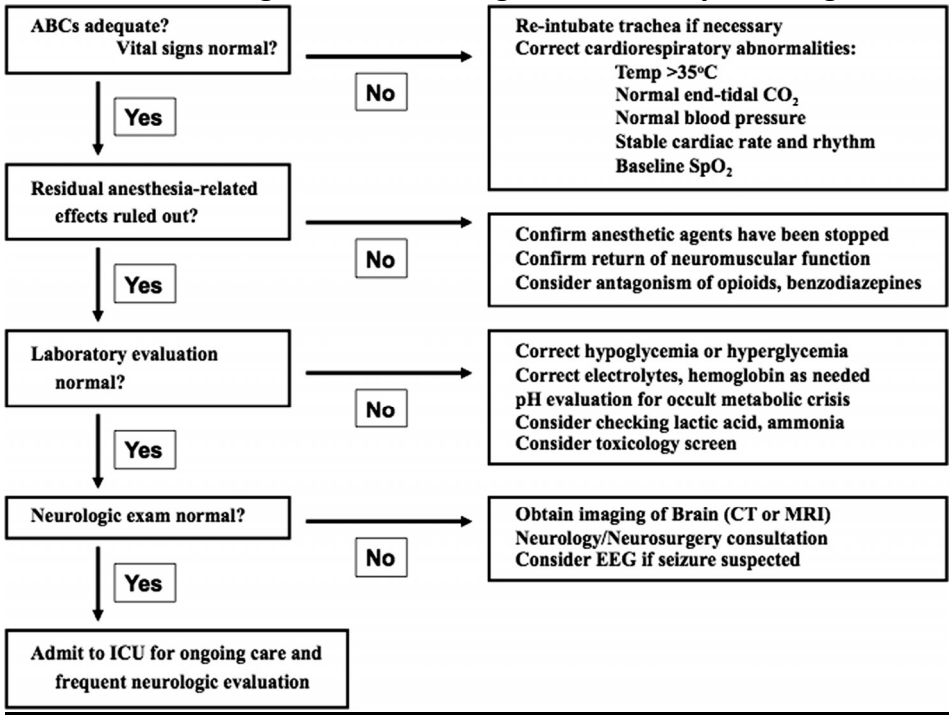
At the end of surgery, anesthetics are stopped and the patient emerges (wakes up) from anesthesia as described above. We have written about some of the frequent and serious complications of emergence that may be seen in the OR as well as the PACU. However, sometimes even with a well-conducted anesthetic, some patients might not wake up quickly. In this event, the anesthetist should run through a checklist to decide on the cause and treatment (**Figure 23-3**). The first steps are to be sure that oxygenation, ventilation, and circulation are all normal. Check the pupils next. If they are of equal size, small to medium size, and react quickly to light, the problem is not due to the result of a serious problem with the brain. Take the child's temperature again to be sure he/she does not have hypothermia or severe hyperthermia.

Next, the anesthetist should check to determine if too large a drug dose was given or a there is a leftover anesthetic effect. Look carefully at total doses of both intravenous drugs and inhaled anesthetic gases. Examination of pupils (see above) may help to decide if the patient received excessive opioid, e.g., morphine or fentanyl. If the pupils are very small ("pinpoint"), the dose may have been large. Reversal of muscle relaxation should be checked with a twitch monitor when possible (**See Chapter 2**).

The next thing to check is for glucose, electrolyte, and other metabolic problems. A blood sample should be sent to determine the glucose concentration, arterial blood gases and pH, and electrolyte measurements. Once in a while, unrecognized severe anemia may present as coma, and so the hemoglobin concentration should be determined as well. The anesthetist should be aware that rare genetic diseases might cause a metabolic crisis and changes in the patient's mental status. This might happen for the first time in the pediatric patient during a stressful event, such as an anesthetic and surgery.

If all of these tests fail to provide a cause for delayed awakening from anesthesia, the patient should have a more thorough neurologic assessment by a pediatric neurologist or neurosurgeon, if available, and radiographic imaging [i.e. computed tomographic (CT) scan or magnetic resonance imaging (MRI)] followed by ICU admission for ongoing evaluation and management.

FIGURE 23-3: Algorithm for Management of Delayed Emergence



Tracheal Intubation and Mechanical Ventilation

Children who present with respiratory distress, fail to improve with supportive measures, or whose condition deteriorates during care may require tracheal intubation and mechanical ventilation. Recognizing impending respiratory failure and triaging care to a facility with resources to provide this degree of respiratory support can be life saving. However, access to clinicians and working mechanical ventilators appropriate for infant and/or children can be challenging even in developed countries outside of major population centers. The decision to intubate the trachea of an infant or child should begin with a declaration that this is a life-threatening emergency, call for help to assist in assessment of the patient and gathering of resources, and once the decision is

made to place an artificial airway, the most experienced person should perform the orotracheal intubation or tracheostomy (i.e., an anesthetist or surgeon). Typically, endotracheal tubes should be available for insertion that are smaller and larger than the size required (**Table 23-5**).

TABLE 23-5: Tracheal Tube Size by Age

Age (years)	TT tube size (I.D. – mm)
Premature	2.5-3.0
Term Newborn	3.0
1	4.0
2	4.5
3-4	5.0
5	5.5
6	6.0
7-8	Cuffed 5.0
9	Cuffed 5.5
10	Cuffed 6.0

Abbreviations: TT = tracheal tube; I.D.- internal diameter of TT General rule for TT size selection in children older than 2 years: (16 + age in years)/4 General rule for depth of tube insertion (at the lip): ETT size x 3 in centimeters

The other equipment essential for safe and successful tracheal intubation include: laryngoscope, suction, oxygen, mask, ambu-bag and medications for safely sedating and immobilizing the patient. Once an tracheal tube is placed into the trachea, it should be secured with tape after verification of correct function and position by using the ambu-bag to provide positive pressure ventilation via the tracheal tube. Clinical signs of successful tracheal intubation include: improvement in color (i.e. blue-to-pink), observed symmetric rise and fall of the chest with bag ventilation, condensation in the tracheal tube, presence of equal air entry in both sides of the chest, with absent or reduced sounds and no distention over the abdomen. Chest radiography, if available, should be performed to confirm the position of the tracheal tube in the trachea. Ideally, exhaled end-tidal CO_2 monitoring will be available to help differentiate tracheal versus esophageal positioning, along with improved saturation by pulse oximetry (**See Chapter 2**).

Prior to and after tracheal intubation, positive pressure ventilation can be provided with a self-inflating (Ambu®-type) bag, Effective bag-mask ventilation can be clinically assessed by a rise of the chest with compression of the bag, and a fall of the chest with release of the bag. Auscultation should demonstrate adequate bilateral breath sounds as air enters and exits the chest. If available, a pulse oximeter can be used to evaluate improvement in oxygen saturation. With an artificial airway (either tracheal tube or tracheostomy) the patient will require continued medical care and observation by personnel familiar with common problems and complications from these procedures. Bag ventilation via the tracheal tube or tracheostomy can be sufficient to maintain and stabilize the child's condition.

If available, mechanical ventilation can be used to effectively ventilate and oxygenate patients through an artificial airway (i.e. an tracheal tube or tracheostomy). A complete discussion of

mechanical ventilation is beyond the scope of this chapter; however, worldwide this is one of the major aspects of critical care of infants and children. Modern anesthesia machines have all the capabilities of critical care ventilators. However, older anesthesia machines lack this ability. Like anesthesia equipment, only those experienced and familiar with safely applying this technology should use them. Again referral to a center with these resources may be warranted, or consultation with an anesthesiologist or surgeon may be helpful. There are two major types of gas delivery when mechanically ventilating a patient – either *volume controlled* or *pressure controlled.* With volume controlled ventilation a tidal volume is set (8-10cc/kg), and the peak inflating pressure varies. With *pressure controlled* ventilation, the peak inflating pressure is set and the delivered tidal volume varies from breath to breath and with changes in compliance of the lung. There are theoretic advantages and disadvantages to both modes of ventilation, suffice it to say – it must be possible to determine in which mode the ventilator is functioning to reliably make adjustments that will benefit the patient and reduce risks (pneumothorax). Alternatively, continuing to assist the patient's spontaneous ventilation or to provide ongoing bag ventilation can be equally effective and safe if questions about the function of an available mechanical ventilator can't be resolved. The basic range of ventilator settings and outputs relative to the dynamic changes in respiratory physiology of the pediatric patient are provided in the following **Table 23-6**.

TABLE 23-6: Initial Mechanical Ventilation Parameters

Parameter	Infant	Child	Adult
FiO_2	1.0*	1.0*	1.0*
Tidal Volume (ml/kg)	10	10	10
Peak Inflating Pressure (cm H_2O)	20	20	20
PEEP (cm H_2O)	3-5	2-4	0-3
Respiratory Rate (bpm)	30-40	20-30	12-20
Inspiratory time (seconds)	0.4-0.6	0.6-1.0	1.0-1.5
I:E ratio	1:1.5-1:2	1:2-1:2.5	1:2-1:3

Abbreviations: FiO2 = fraction of inspired oxygen; PEEP = positive end expiratory pressure; bpm = breaths per min; I:E = inspired to expiratory ratio

Common Medical Issues requiring ICU care

Pneumonia and dehydration are seen often and can be fatal in the developing world, but these diseases should be survivable when adequate ICU care is available. Rapid identification of pneumonia and transfer to an ICU capable of caring for high-risk pediatric patients is essential. The World Health Organization (WHO) guidelines for deciding who needs a higher level of hospital care for children lists the highest risk children (**Figure 23-4**) and helps care providers to identify patients who will need hospital care. The most common medical issues of neonatal asphyxia, pneumonia and hypovolemia from diarrhea will be discussed.

FIGURE 23-4: Triage of the High-Risk Child for Hospitalization and Care

Assessment	Findings	Primary Treatment	Secondary Treatment
Priority Signs for Prompt assessment	-Tiny infant (< 2months) -Temperature high -Trauma -Pallor (severe) -Poisoning -Pain (severe) -Respiratory distress -Restless and irritable -Referral (urgent) -malnutrition (severe) -Edema of both feet and face -Burns (major)	-Identifying these patients is the first priority for assessment and treatment -Call for help to assess and treat -Get surgical help for all trauma or suspected surgical problems	-Proceed to systems based assessment and treatment of: Airway Breathing Circulation Dehydration
Airway and Breathing	-Obstructed breathing OR -Central cyanosis OR -Severe Respiratory distress	Manage airway and assess for foreign body	- Manage airway -Give oxygen -Warm child

FIGURE 23-4 (Continued)

Circulation	Capillary refill > 3 seconds AND Weak and fast pulses	-Stop any bleeding -Give oxygen -Warm child -Assess nutritional status	If no malnutrition: -Insert peripheral IV and begin rapid fluid administration If severe malnutrition and lethargic: -Give IV glucose -Insert peripheral IV and give fluids If severe malnutrition and not lethargic: -Give glucose orally -Assess and treat without peripheral IV
Severe Dehydration	Diarrhea plus two of these symptoms: -Lethargy -Sunken eyes -Very slow skin pinch -Unable to drink or drinks poorly	-Warm child -Assess nutritional status	If no malnutrition: -Insert peripheral IV and begin rapid fluid administration If severe malnutrition and not lethargic: -Give glucose orally -Assess and treat without peripheral IV

Adapted from WHO guideline for triage of hospital care for children 2013.

Neonatal asphyxia is the failure to establish effective breathing at birth and development of tissue hypoxia. It accounts for an estimated 900 000 deaths each year and is one of the primary causes of early neonatal mortality. Neonatal hypoxia leads to multiorgan injury, most of which are reversible if treated early. However, central nervous system injury; with hypoxemic/ischemic injury, seizures, and intracranial bleeding is not reversible. Most of these severe neurologic

problems can have devastating consequences, such as cerebral palsy, mental retardation, and learning disabilities. While there are many causes, issues of labor and delivery care and good initial treatment of infants has been the focus for reducing the mortality rate in many countries. Diagnosis of neonatal asphyxia can be made for in-hospital births based on the following: profound acidosis (pH<7.0) on umbilical cord arterial blood sample; Apgar Score of <3 for more than five minutes; and neurologic problems in the immediate neonatal period like seizures, hypotonia (low muscle tone), coma, or hypoxic ischemic encephalopathy. Education to improve hand washing, umbilical cord care, and early breast-feeding may help reduce early infant death through improved post-partum infant care; however, birth asphyxia is impacted primarily by improvements in the quality of care during both the delivery and the resuscitation periods. Guidelines for neonatal resuscitation, such as those endorsed by WHO and the American Academy of Pediatrics, should be followed for resuscitating asphyxiated newborns (**See Chapter 5**). This includes early resuscitation of the low-Apgar infant with positive pressure, bag mask ventilation with room air to assist the initiation of breathing and establishment of the functional residual capacity (FRC). Expansion of the lungs has effectively reduced mortality rates, even in very sick infants. Tracheal intubation and mechanical ventilation are often needed, but if this can not be done quickly, air flow (5-to-6l/min) via a nasal cannula may help establish breathing and FRC. If oxygen is available, it can also be used to improve oxygenation. The goal is to keep oxygen saturation above 90% but not above 95%. Poor cardiac output is common with asphyxia, and can be caused by poor heart function or low blood volume. Careful IV fluid administration and inotropic support with a drug like dopamine or epinephrine may be needed. But this also might mean an infection is present and may potentially cause severe injury because the infant is very sick. In developed countries, cooling the neonate's body temperature to about 35.5°C is done to protect the brain and other organs. This has become common practice; this might not be possible in many countries because of the technology required.

Pneumonia is the leading cause of death in children worldwide. Approximately 151 million new episodes of community acquired pneumonia (CAP) occur every year, and an estimated two million children die of this disease every year - more than AIDS, malaria and tuberculosis combined! *Streptococcus pneumoniae* and *Haemophilus influenzae* type-b remain the two most common causes of CAP in developing countries. The most common bacterial causes of pneumonia by age are summarized in **TABLE 23-5**.

TABLE 23-5: Common Bacteria Causing Community Acquired Pneumonia in Developing Countries

Bacteria	Birth to 1mo	1-to-3 months	3-mo-to-5-years	5-to-18 years
Streptococcus pnuemoniae	+	+++	++++	+++
Haemophilus influenza	+	+	+	±
Streptococcus pyogenes	-	+	+	+
Staphylococcus Aureus	++	++	+	+
Streptococcus agalactiae	+++	+	-	-
Eschericha coli	++	+	-	-
Mycoplasma pnuemonia	-	+	++	++++
Chlamydiophyla pneumonia	-	++	-	-
Chlamydia trachomatis	+	++	-	-
Bordetella pertussis	±	++	+	+

++++, very common; +++, common; ++, relatively common; +, rare; ±, very rare, - absent

These organisms can reside in the child's oropharynx and be inhaled into the lungs to cause pneumonia; or pneumonia can be caused by direct spread via air-borne droplets. In addition, pneumonia may occur by spread of bacteria through the blood, especially during and shortly after birth. Children at highest risk for pneumonia are newborns with low birth weight (≤ 2,500g), malnourished children, non-exclusive breastfeeding (during the first four months of life), lack of immunization during the first year of life, environmental pollution, and overcrowding or overpopulation of an area.

Diagnosis of pneumonia is usually made by history and physical examination. If available, laboratory testing (including blood and sputum for gram stain, cultures, and radiographic testing) can be used, but these tests often have low yield. Most often, additional testing is indicated in patients with severe pneumonia or in those patients who fail initial antibiotic therapy. Pneumonia and severe pneumonia are usually encountered in the critical care settings. The proposed WHO criteria to identify patients with pneumonia are based on clinical symptoms (**Table 23-6**).

TABLE 23-6: WHO Classification of Severity of Pneumonia

Signs or symptoms	Classification	Treatment
Cough or difficulty breathing with: -Oxygen saturation <90% or central cyanosis -Severe respiratory distress (e.g. grunting, severe chest indrawing) -Signs of pneumonia with a general danger sign (inability to breastfeed or drink, lethargy, or unconsciousness)	Severe pneumonia	-Admit to hospital -Give oxygen if saturation <90% -Manage airway as needed. -Give antibiotics -Treat high fever if present
-Fast breathing: - >50 breaths/min in a child aged 2-11 months. - >40 breaths/min in a child age 1-5 years. -Chest indrawing	Pneumonia	-Home care -Give antibiotics -Advise on appropriate course and symptoms that warrant re-assessment -Re-evaluate in 3 days.
No signs of pneumonia or severe pneumonia but increased work of breathing.	No pneumonia – cough or cold	-Home care -No antibiotics but symptomatic remedies - Advise on appropriate course and symptoms that warrant re-assessment -Follow up in 5 days if not improving.

Adapted from WHO guideline for triage of hospital care for children, 2013.

Management of pneumonia is based on the severity of the pneumonia. For the most severe symptoms, early administration of antibiotics can be life saving. Given the probability of certain organisms, the following guidelines assist in type and duration of antibiotic therapy.

WHO Guidelines for Antibiotics Treatment for Pneumonia:

a) In settings with high HIV rates, children with pneumonia (chest indrawing) should be treated with oral amoxicillin at least 40mg/kg/dose twice daily for five days.

b) In settings with low rates of HIV/AIDS infected children, give oral amoxicillin at least 40mg/kg/dose twice daily for 3 days.

Antibiotic Treatment for Severe Pneumonia:

a) Children with severe pneumonia should be treated with parenteral (IV) ampicillin (or penicillin) and gentamicin as a first line treatment.
— Ampicillin: 50mg/kg every 6 hours for at least five days **OR** Benzyl penicillin: 50,000 units/kg IM/IV every six hours for at least five days
— Gentamicin: 7.5mg/kg IM/IV once a day for at least five days

b) Ceftriaxone 80mg/kg IM or IV once daily for 5 days should be used as a second line treatment in children with severe pneumonia when the first line treatment fails to adequately treat the pneumonia.

c) If child does not improve within 48 hours and staphylococcal pneumonia is suspected, switch to Cloxacillin 50mg/kg IM/IV every six hours and continue the Gentamicin.

Additional supportive therapy with supplemental oxygen therapy should be considered when available and the pneumonia symptoms are severe. Oxygen therapy can be life saving in patients with severe pneumonia. Children with hypoxemia should receive oxygen therapy, guided by pulse oximetry, if available. Oxygen can be provided from oxygen cylinders or oxygen concentrators (**See Chapter 7**). Supplying oxygen from cylinders is expensive and requires frequent filling and transportation of the cylinders. On the other hand, oxygen concentrators are about 25-50% more cost effective than oxygen from cylinders, with no need for frequent refilling. However, concentrators require electrical power to function (**See Chapter 7**).

Oxygen can be delivered via nasal prongs, nasal catheter or nasopharyngeal catheters (NP). For nasal prongs, the typical flow rate is 1–2 liters/min (0.5 liters/min in young infants) to deliver an inspired oxygen concentration of 30–35%. For nasal catheter delivery of oxygen, a size 6 or 8 French catheter is passed to the back of the nasal cavity and taped in place; a flow rate of 1-2 liters/min is used to achieve an inspired oxygen concentration of 35-50%. The appropriate depth of catheter insertion can be determined by inserting the catheter a distance equal to that from the side of the nostril to the inner margin of the eyebrow. When in place, the tip of the catheter should NOT be visible below the uvula (**Figure 5**). Neither nasal cannulas or nasal catheters

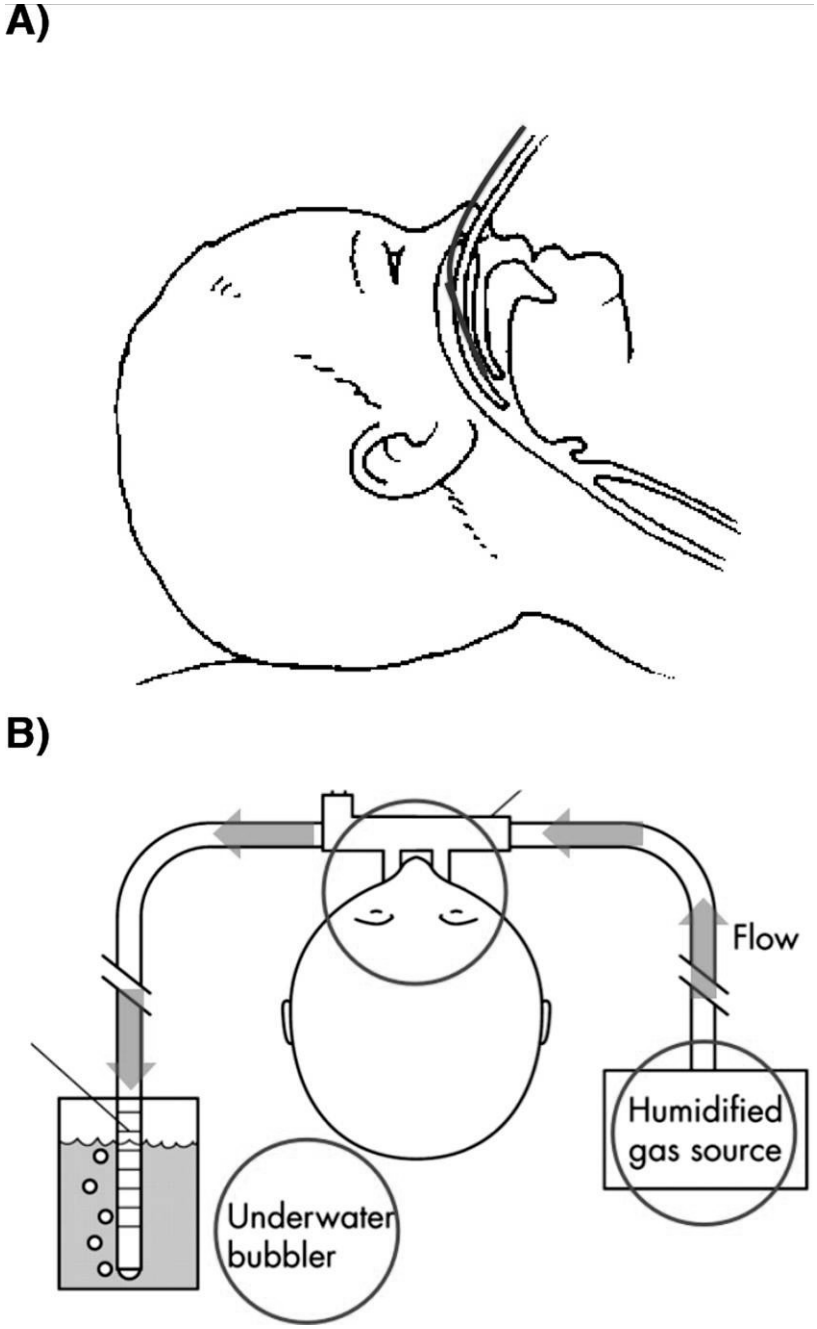
require humidification. Nasopharyngeal catheters are 8F catheters that are inserted 1cm less than the distance from the side of the nose to the front of the ear. These catheters supply higher concentrations of oxygen and greater positive end-expiratory pressure (PEEP) at a given flow rate than nasal prongs. Nasal catheters should be cleaned every 12 hours and must be carefully observered because they have higher complication rates than nasal prongs. Because of this, nasal prongs are the preferred method for administering oxygen, especially for patients less than five years of age. An illustration of bottle CPAP using nasal prongs and the nasopharyngeal method are provided in **Figure 23-5**. The following table lists the relative benefits and ease of placement **Table 23-7**).

TABLE 23-7: Comparison of Oxygen Delivery Methods

Method	Max inspired Oxygen	Humidification	Change in effective O_2 with mouth breathing	Obstruction of airway with mucous	Chance of device dislodgement	Risk of gastric distention
Nasal prongs	**30-35**	**Not required**	**+++**	**+**	**++**	**None**
Nasal catheter	**35-40**	**Not required**	**++**	**++**	**+**	**+**
Nasopharyngeal catheter	**45-60**	**Required**	**+**	**+++**	**++**	**+++**
Nasal bubble CPAP	**45-80**	**Required**	**++**	**+**	**++**	**+**

Key: + = least change/risk; +++ = most change/risk

FIGURE 23-5: Nasal and Nasopharyngeal Oxygen Delivery and Bubble CPAP Configurations



Schematic drawings of supplemental oxygen delivery. A) demonstrates nasal oxygen flow (red catheter) and nasopharyngeal oxygen delivery (white catheter). Note that the red catheter remains above the soft palate, while the white catheter resides just below and can be visualized in the retropharynx when looking into the mouth. From WHO Guideline for Hospital Care of Children, 2nd Edition, 2013. B) demonstrates nasal bubble continuous positive airway pressure (CPAP). This system allows for 5cmH_2O pressure to be created when continuous high gas flow is administered via a nasal cannula system. Kawaza K, Machen HE, Brown J, Mwanza Z, Iniguez S, et al. (2014) Efficacy of a Low-Cost Bubble CPAP System in Treatment of Respiratory Distress in a Neonatal Ward in Malawi. PLoS ONE 9(1): e86327. doi:10.1371/journal.pone.0086327

Nutrition and Fluid Support: Nutritional support in infants and children with pneumonia is very important because many patients with pneumonia are under nourished or malnourished. Only with adequate nutrition and caloric intake can tissue-breakdown states be helped and early support for recovery from the infection can begin. Full enteral feeds should be provided orally or via a nasogastric tube when possible. In patients with severe respiratory distress, feeding via a nasogastric tube plus intravenous dextrose containing fluids is important and should be administered when possible. Deciding how much fluid to give patients who have respiratory distress is a problem. Care should be taken to avoid fever and excessive fluid losses. Fluid administration should be carefully administered, and any evidence of increased work of breathing during fluid administration may mean increasing fluid in the lungs. Clinical examination looking at skin turgor (ability of the skin to snap back into normal shape when pinched and elevated), heart rate, arterial blood pressure, and urine output should be monitored to assess for hydration status and to avoid dehydration or fluid overload in patients who may have fluid retention as a result of increased antidiuretic hormone secretion caused by pneumonia or surgery.

Acute Gastroenteritis and Hypovolemic Shock

Acute diarrheal illnesses claim an estimated 1.4-to-2.5 million lives every year and are the second leading causes of death in children in many countries around the world. World wide there is a 1.5 billion diarrheal episodes every year, and children less than three years of age average three episodes per year. During the past three decades, the death rate from acute diarrhea has decreased due to widespread distribution and use of oral rehydration solutions (ORS), improved rates of breastfeeding, improved nutrition, better sanitation and hygiene, and increased rates of measles immunization.

Pathophysiology of diarrheal illness and the development of dehydration are the result of abnormalities in gastrointestinal tract fluid homeostasis and depends on the secretion and reabsorption of fluid and electrolytes by the bowel. In acute infectious diarrhea either secretion, or reabsorption, or both are abnormal, leading to movement of fluids from the intravascular space to the intestinal lumen. When there is increased secretion, the organism causing the diarrhea release poisons that cause the release of chloride ions, fluids, and diarrhea. When the impairment impacts resorption of fluids and electrolytes, direct damage to the villous brush border of the intestine causes malabsorption and osmotic diarrhea. Most of the time, reabsorption remains intact, allowing for efficient reabsorption or absorption of salt and water. This is how oral rehydration treatments work. ORS are effectively absorbed.

Diarrhea can be classified into “acute diarrhea”, which is three or more loose watery stools within 24 hours; “persistent diarrhea”, which consists of one or more daily episodes of diarrhea lasting more than 14 days; and finally, “dysentery”, which is bloody acute or persistent diarrhea with visible blood and mucus present in the stool.

All types of diarrhea can result in dehydration. Signs and symptoms of dehydration are decided by clinical examination (**Table 23-8**). History, physical examination, current and previous weight (if available) are very important in deciding how severe the dehydration is. Fever is common and can be caused by invasive bacteria. Bloody stools are usually caused by invasive and cytotoxin releasing bacteria (i.e. shigella, E-coli), while vomiting with watery stools are usually caused by virusses or bacteria producing toxins (i.e., *Staph aureus*).

TABLE 23-8: WHO Classification of the Severity of Diarrhea by Clinical Symptoms

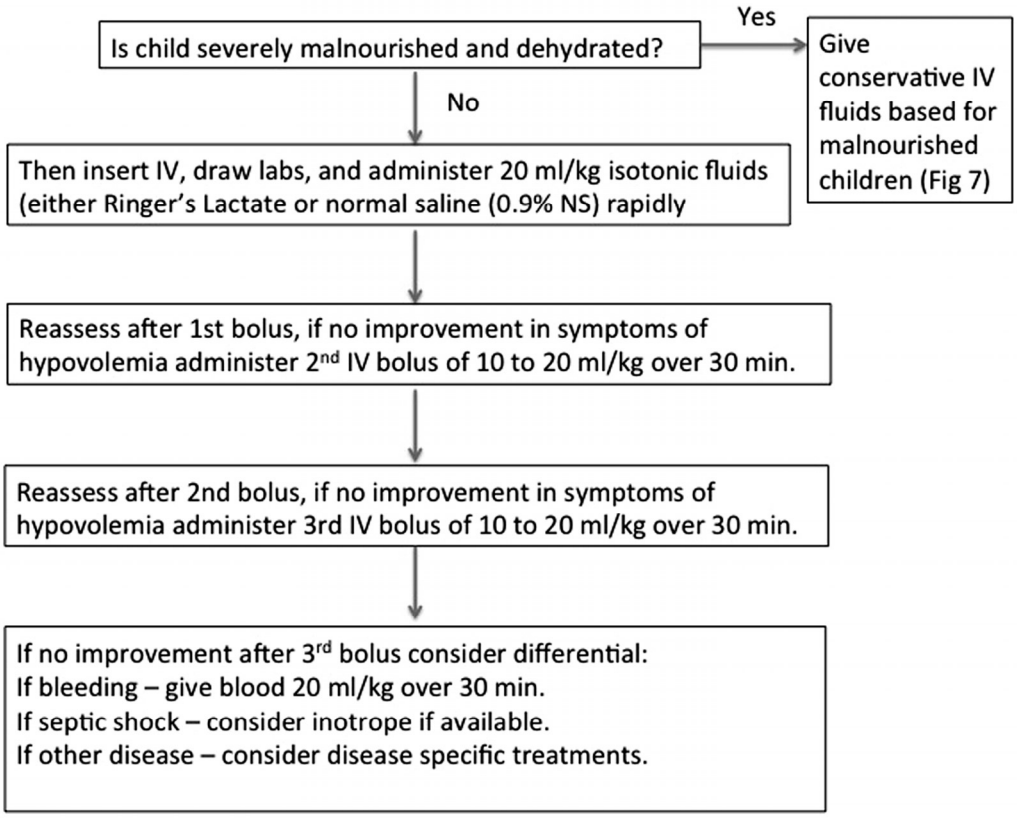
Classification	Signs or symptoms	Treatment
Severe dehydration	**Two or more of the following signs:** **-Lethargy or unconsciousness** **-Sunken eyes** **-Unable to drink or drinks poorly** **-Skin pinch goes back slowly (> 2 seconds)**	**Give fluids for severe dehydration**
Some dehydration	**Two or more of the following signs:** **-Restlessness and irritability** **-Sunken eyes** **-Drinks eagerly, thirsty** **-Skin pinch goes back slowly (< 2 seconds)**	**-Give oral fluids and foods for some dehydration with observation** **-After observed re-hydration, give plan for home therapy and symptoms**
No dehydration	**Not enough signs to classify as some or severe dehydration**	**-Encourage oral fluid and food intake at home.** **-Provide parent a list of symptoms requiring re-evaluation at hospital**

Acute gastroenteritis can cause hypovolemic shock, electrolyte abnormalities, acid-base abnormalities and secondary bacterial infections. The severity of dehydration symptoms depends on the rate and amount of fluid losses. Clinical signs of dehydration occur when about 30-40ml/kg of fluid is lost from the body (i.e., 3-4% dehydration). Diagnosis and treatment of dehydration is

based on frequent examination of the child (every 15–30min) until fluid replacement has produced a strong radial pulse. After that has occurred, reassess the child by checking skin turgor (by pinching and lifting the child's abdominal skin), level of consciousness, ability to drink, and return of urine output; do so at least every hour in order to make sure that hydration is improving. Sunken eyes recover more slowly than other signs of dehydration and do not help as much for determining the response to rehydration therapy.

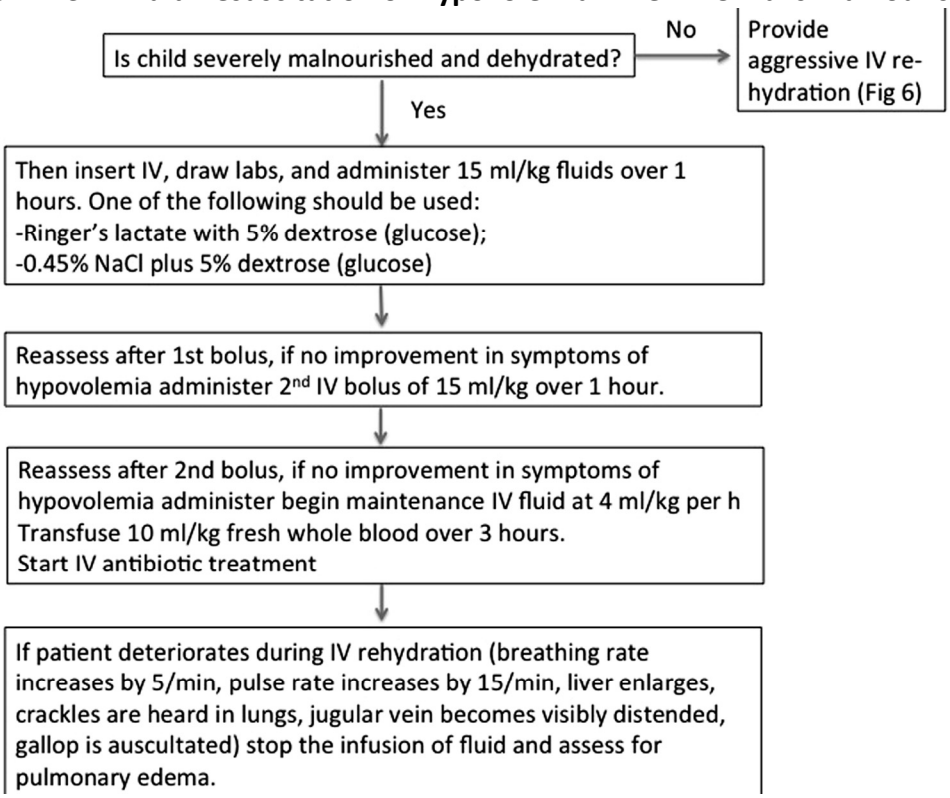
Before a large amount of IV fluid is given quickly, check the child's nutritional status. If the child appears well nourished, follow the resuscitation guidelines for severe dehydration (**Figure 23-6**). If the child is malnourished, follow a more slow and careful fluid resuscitation approach for severe dehydration for the malnourished (**Figure 23-7**).

FIGURE 23-6: Fluid Resuscitation of Hypovolemia in Children Without Malnutrition



In box two, "rapidly" means to give the fluid over 10-15 minutes.

FIGURE 23-7: Fluid Resuscitation of Hypovolemia When A Child is Malnourished



When the full amount of IV fluid has been given, again check the child's hydration status, in general by following the WHO guidelines 2013:

- If signs of severe dehydration are still present, repeat the IV fluid infusion outlined earlier. Persistent severe dehydration after IV rehydration is unusual; it usually occurs only in children who pass large watery stools frequently during the rehydration period.

- If the child is improving but still shows signs of some dehydration, discontinue IV treatment and give ORS solution for 4h. If the child is usually breastfed, encourage the mother to continue breastfeeding frequently.

- If there are no signs of dehydration, encourage the parent to provide the child frequent fluids and feeding, or if breastfeeding encourage more frequent breastfeeding. Observe the child for at least 6h before discharge, to confirm that the mother is able to maintain the child's hydration.

Very important for recovery, all children should start to receive some ORS solution (about 5ml/kg/hr.) by cup when they can drink without difficulty (usually within 3–4 hours for infants and 1–2 hours for older children). ORS provides additional base and potassium, which may not be adequately supplied by available IV fluids. When severe dehydration is corrected, prescribe zinc. Zinc is an important nutrient for a child's overall health and development but is lost in greater

quantities during diarrhea. Replacement increases the rate of a child's recovery, reduces the duration and severity of the diarrheal episode, and lowers the incidence of subsequent diarrheal illness in the next 2–3 months. WHO guidelines recommend the following guidelines:

≤ 6 months: half tablet (10mg) per day for 10–14 days

≥ 6 months: one tablet (20mg) per day for 10–14 days.

Oral rehydration therapy (ORT) can be used to treat mild to moderate dehydration. It can be given orally or via nasogastric tube in patients with persistent vomiting. There are different ORT solutions; the WHO solution contains sodium 90meq/L, potassium 20meq/L, base 30meq/L, carbohydrate 111mMol/l (2%). Deficits and ongoing losses should be replaced over 2-4 hours and maintenance continued afterwards. Again, breast-feeding is advocated as soon as tolerated.

Dehydration can cause poor heart function or shock. Hypovolemic shock usually exists with severe dehydration (**Table 23-9**). Initiation of rehydration therapy is often delayed is in some parts of the world because other causes of dehydration and shock are commonly. Current recommendations for resuscitation are: 1) Patients require intravenous or intraosseous vascular access, with 2) rapid administration of crystalloid solutions. Plasmalyte or Ringer's lactate solutions are preferred over 0.9% normal saline to avoid giving an excess chloride load that can worsen the patient's metabolic acidosis. 3) The shock state should be corrected over 10-15 minutes and rechecked frequently to determine the response to the fluid administration response. 4) Other treatments, like oxygen and inotropic support (dopamine or epinephrine), may be necessary. Monitoring heart rate, respiratory rate, arterial blood pressure, and urine output are necessary to guide therapy.

TABLE 23-9: Differential Diagnosis in a Child with Shock

Diagnosis or underlying cause	Supports diagnosis
Bleeding Shock	**-History of trauma** **-Bleeding site**
Dengue Shock Syndrome	**-Known dengue outbreak or season** **-History of high fever** **-Purpura**
Cardiac Shock	**-History of heart disease or murmur** **-Enlarged neck veins and liver** **-Crackles in lung fields**
Septic shock	**-History of febrile illness** **-Very ill appearance** **- Skin may be cold or warm but blood pressure low** **-Purpura may be present** **-History of meningococcal outbreak**
Shock associated with dehydration	**-History of profuse diarrhea** **-Known cholera outbreak**

Note: General symptoms of shock include lethargy, fast breathing, cool skin, prolonged cap refill, fast weak pulses and may have low blood pressure as a late sign.

After intravascular resuscitation is completed, fluids are continued to provide maintenance and replace ongoing fluid losses. Electrolyte problems are very common with severe, acute diarrhea. Serum electrolytes should be monitored if possible as long as diarrhea episodes and fluid replacement continue. If diarrhea continues, it may be difficult to determine if the child is urinating, and a urinary catheter or bag may be required.

Electrolyte abnormalities from diarrhea-induced dehydration are common. Sodium abnormalities are very common. Patients often develop hyponatremia (low sodium) or hypernatremia (high sodium). Hyponatremia is usually associated with intake of too much low sodium containing fluid, or with excessive sodium losses (as with cholera, E-coli and rotavirus infections). Correction of

hyponatremia should be done no faster than 0.5mEq/hour, with determination of sodium levels every four hour.

Hypernatremia occurs in about 10% of patients with severe dehydration, and is more common during the first year of life. It is usually related to excessive water loss, or excessive salt intake (usually wrong formula for oral rehydration solutions or formulas). Hypernatremia can cause severe brain injury and even death. After initial fluid resuscitation and correction of the hypovolemia, fluid deficits should be corrected over 48-72 hours, with monitoring of the serum sodium when possible. Correction of hypernatremia should not occur more rapidly than 0.5 mEq/hour with 0.45% NS intravenous solution or an oral rehydration formula.

Potassium abnormalities are common in severely dehydrated patients. Hypokalemia (low potassium) may not be present initially but is often present after initial resuscitation. If there is adequate urine output, potassium supplements can be added to the hydrating solution. With significant hypokalemia, patients may develop bradycardia and muscle weakness. Slow and careful extra enteral potassium should be started with frequent checking of serum potassium levels. Hyperkalemia can also occur with severe dehydration if there is severe metabolic acidosis and acute renal failure. In addition to fluid replacement and minimal urine output, consider insulin and glucose if potassium levels are more than 6.5mEq/l and evidence of electrocardiographic changes (tall, peaked T waves) are present. However, the T waves may be normal in young children who have hyper- or hypokalemia. Thus, the ECG cannot be relied upon as evidence of either condition. If the T waves are abnormal, that is helpful, but if they are normal, the patient may still have hyper- or hypokalemia.

The development of metabolic acidosis is very common with severe dehydration. Though this usually improves with adequate fluid resuscitation and appropriate enteral replacement, ongoing bicarbonate loses from the gastrointestinal tract create a non-anion gap acidosis. (The anion gap is calculated by subtracting the serum concentrations of chloride and bicarbonate (anions) from the concentrations of sodium and potassium (cations): = $([Na^+] + [K^+]) - ([Cl^-] + [HCO_3^-])$. If metabolic acidosis persists despite adequate hydration or a gap acidosis is present, other causes for the metabolic acidosis should be sought.

Critical Care and Medical Missions:

When travelling abroad on medical missions there are two types of humanitarian mission, those that allow the anesthetist to practice within the medical system of the host country and others where resources and multi-specialty teams travel into the country to provide a developed world standard of care. Often local resources are limited and the delivery of critical care has been adapted to provide for those individuals with the greatest chance for survival. Availability of oxygen, tracheal tubes, and mechanical ventilation (either invasive or non-invasive) can be life

saving. Unfortunately, health systems that allow for triage and transport of critically ill children and adults are limited in the majority of developing countries. When travelling to a developing country, anesthesiologist and critical care clinicians can work on developing systems that improve the triage and management of the sickest patients, even if the resources are not plentiful.

When an international medical mission is providing surgical care and anesthesia of the developed world standard, efforts should be made to provide the equipment and medicines that meet the same standard. While not a cost-saving or efficient approach, it allows for the visiting clinicians to practice in a manner more similar to their home medical practices. For instance, trips designed to perform cardiac surgery on the simplest lesions (i.e. VSD and ASD) still require that cardiopulmonary bypass, mechanical ventilation, inotropic support and invasive vascular access and monitoring equipment be available. Often visiting groups will ship all this equipment to the surgical site for the purposes of creating an "ICU" for the increased volume of surgery and complex perioperative care that the group will perform. While expensive, it provides for a standard and type of critical care that is essential to meeting "developed world" standards, otherwise the surgeries would not and should not be performed in this environment. Regardless, the right to provide a standard of care to all children remains a major driving principle behind the creation of triage and critical care services in developing countries (**Table 23-10**).

TABLE 23-10: What the Developed World Critical Care Community Can Contribute

1) Acknowledgment of global disparities in critical illness.
2) Contact and collaborate with colleagues in developing countries by sponsoring mutual knowledge transfer programs, including mutual travel exchanges of qualified intensivists and trainees.
3) Donate time, knowledge, and resources to organizations already doing work in the developing world.
4) Advocate for less expensive medications, including newer generation antibiotics, analgesia and sedation, as well as less expensive medications for prevention of critical illness, such as vaccination and anti-retroviral.
5) Mandate that our professional societies and funding agencies consider a global perspective in research and education support and create a section within medical professional society task forces on global disparity with the specific aim of reducing disparities through education, research, and mutual knowledge transfer.
6) Sponsor attendance of acute care professionals at an upcoming conference or your own intensive care unit.

Ethics – Local Allocation of Limited Resources:

One important aspect of providing intensive care and resources to a critically ill child is providing valuable time to see if the patient will recover. In developed countries, there is typically no limit

to access so there are no rationing of resources to acutely decompensated patients. However, when the chances for survival become questionable, or the potential for a recovery to the pre-morbid state is questionable, these consistently pose ethical issues to the treatment team and the patient's families. In developing countries the ethical discussion is often complicated by political and social decisions on the provision of health resources to children. This can result in potential conflicts amongst the health care providers (i.e., nurses and physicians) as well as amongst the family members of the critically ill child.

Conclusions

Though there has been a decline in perioperative mortality in many parts of the world, the overall all rate of perioperative and anesthetic-related mortality remains 2-to-3 times higher in some countries than in others. Further, the mortality related to common conditions, such as diarrhea and pneumonia, remains very high because of the lack of access to immunizations, antibiotics and sufficient medical care. This finding is partially related to limited access to adequate critical care resources to provide necessary respiratory and hemodynamic support. A global priority should be to build and provide the necessary resources to make available critical care support and improve the outcome of infants and children from surgically and medically treatable conditions.

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Appendix 1:
NORMAL HEART RATES, BLOOD PRESSURES, OXYGEN SATURATIONS, AND CENTRAL VENOUS PRESSURE FOR INFANTS AND CHILDREN

Age	Range of normal heart rates (beats per minute)	Range of normal systolic blood pressures, (mmHg)	Range of Normal Respiratory Rates, (Breaths per minute, spontaneous respiration)	Normal Pulse Oximeter Saturation, Room Air, (SpO_2%)	Range of Normal Central Venous Pressure (mm Hg, spontaneous ventilation)
Neonate (<30 days)	120–160	60–75	30-50	93-98	5-12
1–6 months	110–140	65–85	24-40	94-99	4-10
6–12 months	100–140	70–90	20-36	≥ 95	4-8
1–2 years	90–130	75–95	20-32	≥ 95	4-8
3–5 years	80–120	80–100	20-30	≥ 95	4-8
6–8 years	75–115	85–105	18-26	≥ 95	4-8
9–12 years	70–110	90–115	14-20	≥ 95	4-8
13–16 years	60–110	95–120	12-16	≥ 95	4-8
>16 years	60–100	100–125	10-16	≥ 95	4-8

Appendix 1:NORMAL HEART RATES, BLOOD PRESSURES, AND CENTRAL

Appendix 2:
PEDIATRIC ANESTHESIA DRUGS AND OTHER TREATMENTS IN THE PERIOPERATIVE PERIOD

Dean B. Andropoulos, M.D.

Drug doses and treatments are those commonly recommended; each patient's treatment must be individualized, drug doses double checked for accuracy, and drug concentrations and modes of administration used according to local guidelines. Information is current at the time of publication; however, the practitioner must always be aware of new recommendations, and is responsible for determining the best course of treatment. Consult the textbook, hospital formulary, or authoritative internet resources, for a complete listing of indications, contraindications, interval dosing schedules, and side effects of these drugs and treatments. All drugs are intravenous unless otherwise noted. Drugs denoted with an asterisk (*) are not U.S. FDA approved as of February 2014. Source for dosing information is the Texas Children's Hospital Drug Formulary, current as of February 2014, except where otherwise noted in the references.

Category	Drug/ Treatment	Bolus/ Loading Dose	Infusion/ Continuous Dose
Anesthetic , Sedative, and Analgesic Agents			
Opioids	Fentanyl	1-10 mcg/kg; 50-200 mcg/kg total dose	5-20 mcg/kg/hr
	Remifentanil	0.25-1 mcg/kg	0.05-2 mcg/kg/min
	Sufentanil	0.1-5 mcg/kg	0.1-3 mcg/kg/hr

Appendix 2: PEDIATRIC ANESTHESIA DRUGS AND OTHER TREATMENTS

	Morphine	0.03-0.2 mg/kg	0.01-0.05 mg/kg/hr
	Meperidine(shivering)	1-2 mg/kg	NA
	Methadone	0.1 mg/kg	NA
	Hydromorphone	0.02 mg/kg	0.006 mg/kg/hr
Benzodiazepines	Midazolam IV	0.03-0.1 mg/kg	0.05-0.1 mg/kg/hr
	Midazolam PO	0.5-1 mg/kg	NA
	Lorazepam	0.25-0.1 mg/kg	NA
	Diazepam IV	0.05-0.3 mg/kg max 10 mg	NA
	Diazepam PO	0.04-0.3 mg/kg max 10 mg	NA
Barbiturates	Thiopental	1-6 mg/kg	NA
	Pentobarbital	1-6 mg/kg	NA
	Methohexital	1-3 mg/kg	NA
Other Sedative/Analgesic Agents	Ketamine IV	1-2 mg/kg	NA
	Ketamine IM	5-10 mg/kg	NA
	Etomidate	0.1-0.3 mg/kg	NA
	Propofol	1-3 mg/kg	50-200 mcg/kg/min
	Scopolamine	10 mcg/kg	NA
	Dexmedetomidine	0.3-1 mcg/kg	0.3-0.7 mcg/kg/hr
Neuromuscular Blocking Drugs	Vecuronium	0.1-0.3 mg/kg	0.05-0.1

and Reversals			mg/kg/hr
	Rocuronium IV	0.6-1.2 mg/kg	NA
	Rocuronium IM	2 mg/kg	NA
	Atracurium	0.4-0.5 mg/kg	NA
	Cisatracurium	0.15-0.2 mg/kg	NA
	Pancuronium	0.1-0.2 mg/kg	NA
	Succinylcholine IV	1-2 mg/kg	NA
	Succinylcholine IM	4 mg/kg	NA
	Neostigmine	40-80 mcg/kg	NA
	Glycopyrrolate	8-16 mcg/kg	NA
	Sugammadex*[1]	2-4 mg/kg	NA
Vasoactive Drugs			
Inotropes/Vasoconstrictors	Epinephrine	0.5-10 mcg/kg	0.03-0.1 mcg/kg/min
	Atropine IV	10-20 mcg/kg	NA
	Atropine IM	20-40 mcg/kg	NA
	Phenylephrine	0.5-3 mcg/kg	0.05-0.5 mcg/kg/min
	Ephedrine	0.05-0.2 mg/kg	NA
	Calcium chloride	10 mg/kg	5-10 mg/kg/hr
	Calcium gluconate	30 mg/kg	NA
	Dopamine	NA	3-20 mcg/kg/min
	Dobutamine	NA	
			3-20 mcg/kg/min

	Milrinone	25-75 mcg/kg	0.25-0.75 mcg/kg/min
	Norepinephrine	NA	0.05-0.1 mcg/kg/min
	Vasopressin	NA	0.02-0.05 units/kg/hr
	Isoproterenol	NA	0.01-0.1 mcg/kg/min
	Levosimendan [2-4]	6-12 mcg/kg	0.1-0.2 mcg/kg/min
	Triiodothyronine (T3)[5]	NA	0.05 mcg/kg/hr
Vasodilators/Antihypertensives	Sodium nitroprusside	NA	0.3-5 mcg/kg/min
	Nitroglycerine	NA	0.3-5 mcg/kg/min
	Prostaglandin E_1	NA	0.0125-0.05 mcg/kg/min
	Prostacyclin (Flolan, epoprostenol, PGI_2)	NA	2-5 ng/kg/min
	Nesiritide	NA	0.01-0.03 mcg/kg/min
	Fenoldepam	NA	0.025-0.3 mcg/kg/min initially, titrate to max 1.6 mcg/kg/min
	Nicardipine	NA	0.1-0.3 mg/kg/hr; max

			15 mg/hr
	Hydralazine	0.1-0.2 mg/kg	NA
	Phentolamine	0.1-0.2 mg/kg on CPB	NA
	Phenoxybenzamine	0.25 -1 mg/kg on CPB	
	Labetalol	0.25-0.5 mg/kg	NA
	Enalaprilat	5-10 mcg/kg	NA
	Sildenafil[6,7]	0.35-0.44 mg/kg over 1-3h	0.067 mg/kg/hr
Antiarrhythmics/β-blockers	Lidocaine	1-2 mg/kg	20-50 mcg/kg/min
	Procainamide	10-15 mg/kg	20-80 mcg/kg/min
	Esmolol	250-500 mcg/kg	50-300 mcg/kg/min
	Propranolol	0.01-0.1 mg/kg	NA
	Amiodarone	5 mg/kg over 10-15”, may repeat x 2 to max 15 mg/kg	NA
	Verapamil	0.1-0.3 mg/kg	NA
	Adenosine	25-50 mcg/kg – double if ineffective	NA

Appendix 2: PEDIATRIC ANESTHESIA DRUGS AND OTHER TREATMENTS

	Magnesium sulfate	25-50 mg/kg over 30-60”	NA
	Digoxin	8-10 mcg/kg 1st loading dose	NA
Antibiotics	Cefazolin	25 mg/kg max 1 g	NA
	Ampicillin	50 mg/kg, max 1 g	NA
	Vancomycin	15 mg/kg max 1 g	NA
	Gentamycin	2.5 mg/kg max 120 mg	NA
	Nafcillin	50 mg/kg max 2 g	NA
	Clindamycin	10 mg/kg	NA
	Cefuroxime	25-30 mg/kg	NA
	Cefoxitin	30-40 mg/kg max 2 g	NA
	Pipericillin/Tazobactam	100 mg/kg piperacillin component, max 4 g	
Non-Steroidal Anti-Inflammatory Drugs/Non-Opioid Analgesics	Ketorolac	0.5 mg/kg max 30 mg	NA
	Acetaminophen PO	15 mg/kg max 1000 mg	NA
	Acetaminophen PR	20-30 mg/kg (one time)	NA
	Acetaminophen IV	12.5-15 mg/kg max 1000 mg	NA
	Ibuprofen IV	5-10 mg/kg; max	NA

		400 mg	
	Ibuprofen PO	5-10 mg/kg; max 400 mg	NA
Malignant Hyperthermia Treatment	Dantrolene	2.5 mg/kg; may repeat x 3 up to 10 mg/kg	NA
Local Anesthetics	Lidocaine	Max 5 mg/kg	NA
	Bupivacaine	Max 2.5 mg/kg	NA
	Levobupivacaine	Max 2.5 mg/kg	NA
	Ropivacaine	Max 2 mg/kg	NA
	Tetracaine (spinal—infants < 6 months)	0.4-0.8 mg/kg	NA
Local Anesthetic Toxicity	20% Intralipid[8]	1 ml/kg; repeat x 2 to max 3 ml/kg	0.25 ml/kg/min
Corticosteroids	Methylprednisolone	1-30 mg/kg depending on indication	5.4 mg/kg/hr x 23 hours for spinal cord injury
	Dexamethasone	0.25-1 mg/kg. max 20 mg	NA
	Hydrocortisone	1-2 mg/kg	NA
Anticoagulants	Heparin	CPB: 300-400 units/kg	NA
	Bivalrudin[9,10]	Interventional cardiac cath: 0.75 mg/kg CPB: 1 mg/kg; plus 1 mg/kg CPB prime	Cath: 1.75 mg/kg/hr CPB: 2.5 mg/kg/hr

Appendix 2: PEDIATRIC ANESTHESIA DRUGS AND OTHER TREATMENTS

	Argatroban	CPB:35-100 mcg/kg (ACT >400 sec)	2-10 mcg/kg/min
	Antithrombin III	50 units/kg; target levels 80-120% of normal	NA
Hemostasis Agents	ε-aminocaproic acid	75 mg/kg patient; 75 mg/kg CPB prime	75 mg/kg/hr
	Tranexamic acid	10-100mg/kg	1-10 mg/kg/hr
	Recombinant factor VIIa	30-90 mcg/kg; may repeat x 2	
Diuretics	Furosemide	0.5-1 mg/kg max 40 mg	0.1-0.4 mg/kg/hr
	Bumetanide	0.015-0.1 mg/kg max 2.5 mg	NA
	Mannitol	0.25-1 g/kg	NA
Perioperative Nausea & Vomiting/Gastrointestinal Prophylaxis	Ondansetron	0.1 mg/kg max 4 mg	NA
	Granisetron	10-20 mcg/kg	NA
	Metaclopromide	0.1-0.2 mg/kg max 10 mg	NA
	Promethazine (over age 2 years only)	0.25-0.5 mg/kg Max 25 mg	
	Sodium citrate PO	30 ml	NA
Antihistamines	Diphenhydramine	1-2 mg/kg max 50 mg	NA
	Ranitidine	1 mg/kg max 50	NA

		mg	
	Famotidine	0.5 mg/kg, max 40 mg	NA
Alkalinizing Agents	Sodium bicarbonate (dilute 1:1 sterile H_2O for neonates)	1-2 meq/kg	NA
	Tromethamine (THAM)0.3 M solution	3-6 ml/kg	NA
Inhaled Agents/Bronchodilators			**Inspired dose**
	Sevoflurane		1-8%
	Isoflurane		0.5-3%
	Desflurane		2-12%
	Nitrous Oxide (N_2O)		50-75%
	Nitric Oxide (NO)		5-20 ppm
	Levalbuterol	8-12 MDI puffs per ETT; 0.075-0.15 mg/kg nebulized in 3 ml normal saline	NA
	Racemic epinephrine	0.25-0.5 mL of 2.25% racemic epinephrine in 3 mL normal saline	NA
	Prostacyclin (Iloprost, PGI_2)[11]	2.5-5 mcg nebulized in 3 ml normal saline	NA
Electrolytes/Dextrose	25% Dextrose in Water (50% dextrose diluted 1:1)	0.25-0.5 ml/kg	NA

Appendix 2: PEDIATRIC ANESTHESIA DRUGS AND OTHER TREATMENTS

	Potassium chloride (KCl)	0.5-1 meq/kg	NA
	3% NaCl	3-5 ml/kg	NA
Insulin (regular)	Insulin (regular): Dose based on plasma glucose levels	0.02-0.1 units/kg	0.02-0.1 units/kg/hr
Sedation/Analgesia Reversal	Naloxone	1-10 mcg/kg	NA
	Flumazenil	1-5 mcg/kg; repeat as needed, max dose 1 mg	NA
Immunosuppressants (Transplant)	Basiliximab	<35 kg: 10 mg; >35 kg: 20 mg	NA
	Mycophenolate	15 mg/kg max 1.5 g	NA
Miscellaneous Drugs	Caffeine citrate	10-20 mg/kg	NA
Transfusions	Packed red blood cells	10-15 ml/kg	NA
	Whole blood	10-15 ml/kg	NA
	Platelets	1 unit/5 kg will increase plt count by 50,000; 1 pheresis unit=6 random donor units	NA
	Cryoprecipitate	1 unit per 5 kg; max 4 units	NA
	Fresh Frozen Plasma	10-20 ml/kg	NA
Intravascular Volume Expansion	5% albumin	10-20 ml/kg	NA
	25% albumin	2-4 ml/kg; 0.5-1 g/kg	NA

	6% hetastarch	10-20 ml/kg, max 15 ml/kg/24 hr	NA
Direct Current Cardioversion/Defibrillation	External synchronized cardioversion	0.5 J/kg; increase to max 1 J/kg; max 100 kg	NA
	External defibrillation	2-5 J/kg; increase if ineffective; max 200 J biphasic; 360 J monophasic	NA
	Internal defibrillation	5 J; increase to 10 if ineffective	NA
	Internal synchronized cardioversion	2 J; increase to 5 J if ineffective	NA

Abbreviations: mg, milligram; mcg, microgram; ng, nanogram; max, maximum; CPB, cardiopulmonary bypass; NA, not applicable; J, joules; meq, milliequivalents; IV, intravenous; IM, intramuscular; PO, oral.

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Appendix 3:
NORMAL LABORATORY VALUES

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Source: Texas Children's Hospital Clinical Laboratory, Houston, Texas

Updated 9/15/2010

Legends: **(A)** = Arterial **(M)** = Male

(B) = Blood **(P)** = Plasma

(C) = Capillary **(S)** = Serum

(CSF) = Cerebrospinal Fluid **(SWT**) = Sweat

(F) = Female **(U)** = Urine

(WB) = Whole Blood

For conversion of conventional (U.S.) units to SI units, see:

http://www.soc-bdr.org/rds/authors/unit_tables_conversions_and_genetic_dictionaries/e5196/index_en.html

All practitioners are urged to consult the normal laboratory values for their local laboratory, as these may differ from those listed below. They are also advised to continually check for updated normal ranges.

TEST	REFERENCE RANGE (United States Conventional Units)
Albumin (CSF)	10 – 30 mg/dL
Albumin (S,P)	
Age	**g/dL**
0 – 30 days	2.9 – 5.5
1 – 3 mos	2.8 – 5.0
4 – 11 mos	3.9 – 5.1
≥ 1 yr	3.7 – 5.5
Albumin (Random Urine)	< 37 mg/L or < 3.7 mg/dL
Albumin/Creatinine Ratio (Random Urine)	< 16 mg/g
Alanine Aminotransferase(S,P)	
Age	**M/F (U/L)**
0 – 11 mos	6 – 50
1 – 3 yrs	6 – 45
4 – 6 yrs	10 – 25
7 – 9 yrs	10 – 35
	Male (U/L) **Female (U/L)**
10 – 11 yrs	10 – 35 10 – 30
12 – 13 yrs	10 – 55 10 – 30
14 – 15 yrs	10 – 45 6 – 30
16 – 18 yrs	10 – 40 6 – 35
≥ 19 yrs	21 – 72 9 – 52
Alkaline Phosphatase (S,P)	
Age	**M/F (U/L)**
0 – 5 days	110 – 300
6 days – 11 mos	110 – 320
1 – 3 yrs	145 – 320
4 – 6 yrs	150 – 380
7 – 9 yrs	175 – 420
	Male (U/L) **Female (U/L)**
10 – 11 yrs	135 – 530 130 – 560
12 – 13 yrs	200 – 495 105 – 420
14 – 15 yrs	130 – 525 70 – 230
16 – 18 yrs	65 – 260 50 – 130
≥ 19 yrs	38 – 126 38 – 126
Ammonia (P)	
Age	**µmol/L**
0 – 7 days	54 – 94
8 – 30 days	47 – 80
1 – 12 mos	15 – 47
1 – 15 yrs	22 – 48
≥ 16 yrs	9 – 26
Amylase (S,P)	30 – 115 U/L
Antistreptolysin-O (ASO)	< 250 IU/ml
Antithrombin (ATIII)(P)	85 – 130 %
Normal Ranges for healthy full-term infants	
Day 1	63% (39-87%)
Day 5	67% (41-93%)
Day 30	78% (48-108%)
Day 90	97% (73-121%)
Day 180	104% (84-124%)

Asparate Aminotransferase (S,P)			
Age	**M/F (U/L)**		
0 – 5 days	35 – 140		
6 days – 3 yrs	20 – 60		
4 – 6 yrs	15 – 50		
7 – 9 yrs	15 – 40		
	Male (U/L)	**Female (U/L)**	
10 – 11 yrs	10 – 60	10 – 40	
12 – 15 yrs	15 – 40	10 – 30	
16 – 18 yrs	10 – 45	5 – 30	
≥ 19 yrs	17 – 59	14 – 36	
Bilirubin (S,P)	**Premature mg/dL**	**Full-Term mg/dL**	
Age	**Total**	**Total**	
Up to 23 hrs	1 – 8	2 – 6	
24 – 48 hrs	6 – 12	6 – 10	
3 – 5 days	10 – 14	4 – 8	
≥ 1 month	mg/dL		
Bc Conjugated	<0.35		
Bu Unconjugated	<1.0		
Total	0.2 – 1.0		
Blood Gases			
pH			
Capillary/Arterial			
Age			
Newborn	7.33 – 7.49		
1 day	7.25 – 7.43		
2 – 30 days	7.32 – 7.43		
1 mo	7.34 – 7.43		
2 mos – 1 yr	7.34 – 7.46		
≥ 2 yrs			
Male	7.35 – 7.45		
Female	7.36 – 7.44		
Venous			
All ages	7.32 – 7.42		
Blood Gases pCO_2			
Capillary/Arterial			
Age	**mmHg**		
0 – 1 mo	27 – 40		
2 mos – 1 yr	26 – 41		
≥ 2 yrs			
Male	36 – 46		
Female	33 – 43		
Venous			
All ages	40 – 50		
Blood Gases pO_2	**mmHg**	**mmHg**	**mmHg**
Age	**Capillary**	**Arterial**	**Venous**
0 – 1 yr	**60 – 70**	**65 – 76**	**25 - 40**
≥ 2 yrs	80 – 90	88 – 105	40 - 47
Oxygen Saturation	85 – 100%		
Ca++ (Ionized) (WB)			
Age	mmol/L		
0 – 30 days	0.90 – 1.45		
1 – 5 mos	0.95 – 1.50		
≥ 6 mos	1.10 – 1.30		

Calcium (S - Total)		
Age	**mg/dL**	
0 – 11 mo	8.0 – 10.7	
1 – 3 yrs	8.7 – 9.8	
4 – 11 yrs	8.8 – 10.1	
12 – 13 yrs	8.8 – 10.6	
14 – 15 yrs	9.2 – 10.7	
≥ 16 yrs	8.9 – 10.7	
Chloride (S,P)	95 – 105 mmol/L	
Chloride (CSF)	122 – 132 mmol/L	
Chloride (U) — Random	No Reference Range	
CO_2 Content (S,P)		
Age	**mmol/L**	
0 – 15 yrs	20 – 28	
≥ 16 yrs	25 – 35	
Cortisol (S)		
Age	**µg/dL**	
1 – 7 days	2 – 11	
1 – 12 months	2.8 – 23	
1 – 16 yrs (8 AM)	3 – 21	
≥ 16 yrs(8 AM)	8 – 19	
(4 PM)	4 – 11	
Creatine Kinase (CPK)(S,P)		
Age	**M/F (U/L)**	
0 – 3 YRS	60 – 305	
4 – 6 yrs	75 – 230	
7 – 9 yrs	60 – 365	
	Male (U/L)	**Female (U/L)**
10 – 11 yrs	55 – 215	80 – 230
12 – 13 yrs	60 – 330	50 – 295
14 – 15 yrs	60 – 335	50 – 240
16 – 18 yrs	55 – 370	45 – 230
≥ 19 yrs	55 – 170	30 – 135
Creatine Kinase MB band(S,P)	Normal: <5 ng/mL Borderline: 5-10 ng/mL Abnormal: >10 ng/mL	
Creatinine Clearance (U)		
Age	**mL/min**	
0 – 30 days	25 – 55	
1 – 5 mos	50 – 90	
6 – 11 mos	75 – 125	
≥ 1 yr	90 – 150	
D-Dimer (P)		
Adult	≤ 0.40 µg/mL FEU	
Neonatal Reference Range from Cord Blood	≤3.40 µg/mL FEU	
Fibrinogen (P)		
Adult	220 – 440 mg/dL	
Neonatal Reference Range from Cord Blood	135 – 283 mg/dL	
Fibrin Split Product (FSP)(P)	1:2 Dilution = Negative (<5 ug/mL)	
Gamma-Glutamyl Transferase (GGT)(S,P)		
Age	**M/F (U/L)**	
0 – 5 days	34 – 263	
6 days – 2 mos	10 – 160	
3 – 11 mos	11 – 82	
1 – 3 yrs	10 – 19	
4 – 6 yrs	10 – 22	

7 – 9 yrs	13 – 25
	Male (U/L) **Female (U/L)**
10 – 11 yrs	17 – 30 17 – 28
12 – 13 yrs	17 – 44 14 – 25
14 – 15 yrs	12 – 33 14 – 26
16 – 18 yrs	11 – 34 11 – 28
≥ 19 yrs	10 – 78 10 – 78
Glucose (S,P)(WB): Glucose conversion factor for mg/dL to mmol/L: divide mg/dL value by 18	
Age	**mg/dL**
1 – 12 hrs	40 – 65
12 – 23 hrs	40 – 80
1 day	50 – 58
2 days	58 – 60
≥ 3 days	70 – 110
Glucose (CSF)	50 – 70% of serum glucose
Glucose (U) — Timed	<500 mg/24 hrs
Glucose (U) — Random	<30 mg/dL
Glycosylated Hemoglobin (WB)	Non-diabetics 4.0 – 6.8% Diabetics 6.0 – 22.0%
Hematocrit (B)	
Age	**%**
0 – 30 days	44 – 70
1 mo	32 – 42
2 – 6 mos	29 – 41
7 mos – 2 yrs	33 – 39
3 – 6 yrs	34 – 40
7 – 12 yrs	35 – 45
13 – 18 yrs/Female	36 – 45
13 – 18 yrs/Male	37 – 49
≥ 19 yrs/Female	36 – 46
≥ 19 yrs/Male	41 – 53
Hemoglobin (B)	
Age	**g/dL**
0 – 30 days	15.0 – 22.0
1 mo	10.5 – 14.0
2 – 6 mos	9.5 – 13.5
7 mos – 2 yrs	10.5 – 14.0
3 – 6 yrs	11.5 – 14.5
7 – 12 yrs	11.5 – 15.5
13 – 18 yrs/Female	12.0 – 16.0
13 – 18 yrs/Male	13.0 – 16.0
≥ 19 yrs/Female	12.0 – 16.0
≥ 19 yrs/Male	13.5 – 17.5
International Normalized Ratio (INR) (P)	
Adult	0.8 – 1.2
Neonatal Reference Range from Cord Blood	1.0 – 1.4
Lactate (P,WB,CSF)	Mmol/L
Plasma (venous)	0.2 – 2.0
Plasma (arterial)	0.3– 0.8
CSF	0.6 – 2.2
Whole Blood	0.2 – 1.7
Lactate Dehydrogenase (LDH) (S,P)	
Age	**M/F (U/L)**
0 – 5 days	934 – 2150
6 days – 3 yrs	500 – 920

4 – 6 yrs	470 – 900	
7 – 9 yrs	420 – 750	
	Male (U/L)	**Female (U/L)**
10 – 11 yrs	432 – 700	380 – 770
12 – 13 yrs	470 – 750	380 – 640
14 – 15 yrs	360 – 730	390 – 580
16 – 18 yrs	340 – 670	340 – 670
≥ 19 yrs	313 – 618	313 – 618
LDH (CSF)		
Age		
0 – 30 days	2.3 – 8.4 U/L	
≥ 1 mo	Approximately 10% of serum value	
Lipase (S,P)		
Age	**U/L**	
0 – 9 yrs	25 – 120	
10 – 13 yrs	15 – 110	
14 – 18 yrs	25 – 110	
≥ 19 yrs	23 – 300	
Magnesium (S,P)		
Age	**mg/dL**	
0 – 6 days	1.2 – 2.6	
7 – 30 days	1.6 – 2.4	
1 mo – 1 yr	1.6 – 2.6	
2 – 5 yrs	1.5 – 2.4	
6 – 9 yrs	1.6 – 2.3	
10 – 13 yrs	1.6 – 2.2	
≥ 14 yrs	1.5 – 2.3	
Methemoglobin (WB)	<2% of tHb	
Osmolality (S,P)	275 – 295 mOsm/kg H_2O	
Osmolality (U)	300 – 1000 mOsm/kg H_2O	
Phosphorus, Inorganic (S,P)		
Age	**mg/dL**	
Premature	5.6 – 8.0	
Term	5.0 – 7.8	
0 – 3 mos	4.8 – 8.1	
4 – 11 mos	3.8 – 6.7	
1 – 4 yrs	3.5 – 6.8	
5 – 7 yrs	3.1 – 6.3	
8 – 11 yrs	3.0 – 6.0	
12 – 16 yrs	2.5 – 5.0	
≥ 17 yrs	2.3 – 4.8	
Plasma Hemoglobin (P)	≤4 mg/dL	
Platelet Count (B)	150,000 – 450,000 µL	
Potassium (S,P)		
Age	**mmol/L**	
0 – 30 days	4.5 – 7.0 (venous or arterial) 4.5 – 7.5 (heel stick)	
1 – 2 mos	4.0 – 6.2	
3 – 11 mos	3.7 – 5.6	
≥ 1 yr	3.5 – 5.5	
Potassium (WB)		
Age	**mmol/L**	
Premature	4.5 – 7.0	
0 - 11 mos	5.0 – 5.7	
≥ 1 yr	3.5 – 5.5	
Protein, Total (S,P)		

Age	**g/dL**
0 – 30 days	4.4 – 7.6
1 – 3 mos	4.2 – 7.4
4 – 11 mos	5.6 – 7.2
≥ 1 yr	6.0 – 8.0
Protein (CSF)	
Age	**mg/dL**
Premature	40 – 300
0 – 30 days	<100
≥ 1 mo	15 – 45
Prothrombin Time (PT)(P)	
Adult	12.2 – 15.5 seconds
Neonatal Reference Range from Cord Blood	12.9 – 16.9 seconds
Partial Thromboplastin Time (PTT) (P)	
Adult	26.5 – 35.5 seconds
Neonatal Reference Range from Cord Blood	28.7 – 53.7 seconds
Red Blood Cell Count (RBC) (B)	
Age	**$x10^6$/µL**
0 – 30 days	4.1 – 6.7
1 mo	3.0 – 5.4
2 – 6 mos	2.7 – 4.5
7 mos – 2 yrs	3.7 – 5.3
3 – 6 yrs	3.9 – 5.3
7 – 12 yrs	4.0 – 5.2
13 – 18 yrs /Female	4.1 – 5.1
13 – 18 yrs /Male	4.5 – 5.3
≥ 19 yrs/Female	4.2 – 5.4
≥ 19 yrs/Male	4.7 – 6.0
Reticulocyte Count % (B)	
Age	**%**
0 – 2 days	3.0 – 7.0
3 – 4 days	1.0 – 3.0
> 4 days	0.5 – 1.5
Reticulocyte Count Absolute (B)	
Age	**$x10^6$/µL**
0 – 2 days	0.140 – 0.220
3 – 4 days	0.040 – 0.110
> 4 days	0.020 – 0.080
Sedimentation Rate (B)	0 – 20 mm/hr
Sodium (S,P) (WB)	
Age	**mmol/L**
Premature	132 – 140
0 – 11 mos	133 – 142
≥ 1 yr	136 – 145
Sodium (U) — Random	No Normals
Thromboelastogram (TEG)with Kaolin	
Reaction Time	4.4 – 11.0 minutes
Angle	49.1 – 74.7 degrees
Max Amplitude	53.6 – 70.3 mm
Fibrinolysis	0.0 – 7.5%
Clot Strength	5.2 – 11.3
Thrombin Time	15.0 – 19.0 seconds
Triglycerides (S,P)	20 – 150 mg/dL
Tropronin I (S,P)	<0.15 ng/mL
Urea Nitrogen (S,P)	
Age	**mg/dL**
0 – 1 yr	8 – 28

2 – 15 yrs	5 – 25
≥ 16 yrs	5 – 20
Uric Acid (S,P)	2.0 – 6.2 mg/dL
Urinalysis (U)	
Specific Gravity	1.001 – 1.035
PH	4 – 9
Protein	Neg
Glucose	Neg
Ketone	Neg
Bilirubin	Neg
Urobilinogen	< 2.0
WBC	0 – 4 / HPF
RBC	0 – 4 / HPF
EPI (Epithelial cells)	0 – 4 / LPF

White Blood Cell Count (WBC) (B)

Age	**x10^3/µL**
0 -- 30 days	9.1 – 34.0
1 month	5.0 – 19.5
2 – 11 mos	6.0 – 17.5
1 – 6 yrs	5.0 – 14.5
7 – 12 yrs	5.0 – 14.5
13 – 18 yrs	4.5 – 13.5
≥ 19 yrs	4.5 – 11.0

Age	Seg%	Band%		Lymphs%		Monos%	
		EOS%		BASO%		ANC	
0 – 30 days	32-67	0-8	25-37	0-9	0-2	0-1	6.0-23.5
1 month	20-46	0-4.5	28-84	0-7	0-3	0-1	1.0-9.0
2 – 11 mos	20-48	0-3.8	34-88	0-5	0-3	0-1	1.0-8.5
1 – 6 yrs	37-71	0-1.0	17-67	0-5	0-3	0-1	1.5-8.0
7 – 12 yrs	33-76	0-1.0	15-61	0-5	0-3	0-1	1.5-8.0
13 – 18 yrs	33-76	0.1.0	15-55	0-4	0-3	0-1	1.8-8.0
≥ 19 yrs	33-76	0-0.7	14-54	0-4	0-3	0-1	1.8-7.7

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