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 5th 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 guidewires; 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 steriley 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

<table>
<thead>
<tr>
<th>Patient Weight</th>
<th>IJ/Subclavian Vein</th>
<th>Femoral Vein</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 10 kg</td>
<td>4 Fr, 2 lumen, 8 cm</td>
<td>4 Fr, 2 lumen, 12 cm</td>
</tr>
<tr>
<td>10-30 kg</td>
<td>4 Fr, 2 lumen, 12 cm</td>
<td>4 Fr, 2 lumen, 12-15 cm</td>
</tr>
<tr>
<td>30-50 kg</td>
<td>5 Fr, 2 lumen, 12-15 cm</td>
<td>5 Fr, 2 lumen, 15 cm</td>
</tr>
<tr>
<td>50-70 kg</td>
<td>7 Fr, 2 lumen, 15 cm</td>
<td>7 Fr, 2 lumen, 20 cm</td>
</tr>
<tr>
<td>&gt;70 kg</td>
<td>8 Fr, 2 lumen, 16 cm</td>
<td>8 Fr, 2 lumen 20 cm</td>
</tr>
</tbody>
</table>

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

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

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 proceed 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 pneumothoraces 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 steriley 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.
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 8th-to-10th thoracic vertebra. Problems with UACs include ischemia of the intestines and occlusion of a renal artery with subsequent hypertension.
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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

<table>
<thead>
<tr>
<th>Weight</th>
<th>Radial/DP/PT/ulnar arteries</th>
<th>Brachial Artery</th>
<th>Femoral Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2kg</td>
<td>24 g</td>
<td>Not recommended</td>
<td>Not recommended</td>
</tr>
<tr>
<td>2-5 kg</td>
<td>22 g</td>
<td>24 g</td>
<td>22-24 g</td>
</tr>
<tr>
<td>5-30 kg</td>
<td>22 g</td>
<td>22 g</td>
<td>22 g</td>
</tr>
<tr>
<td>&gt;30 kg</td>
<td>20 g</td>
<td>22 g</td>
<td>20 g</td>
</tr>
</tbody>
</table>

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.
Chapter 2: VASCULAR ACCESS AND MONITORING

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 occur? Is there stridor that is audible with the ear or stethoscope?
Large and small precordial stethoscopes are displayed. These can either 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 SaO2 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 PaCO2. 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 (SpO₂) 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 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

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

Pulse Oximetry

Pulse oximetry uses the light absorption characteristics of oxygenated and deoxygenated hemoglobin to estimate the oxygen saturation (SpO₂) 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 SpO₂, 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)
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 SpO₂s below 90%. At SaO₂s below 70%, oximeter accuracy is greatly affected.

Cyanotic congenital heart disease (CHD) is a common problem encountered by pediatric anesthetists. Several studies have compared SpO₂ to co-oximeter determined arterial oxyhemoglobin saturations in these patients. Although some newer devices are more accurate at lower SpO₂ ranges, in general, the lower the oxygen saturation the less accurate the SpO₂. 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 SpO₂ is below 70-80%. When the SpO₂ is rapidly decreasing, it lags behind true SaO₂, because signal averaging occurs over 15 seconds. This means that the patient’s SaO₂ is often lower than 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 SpO2. The strongest predictor of inaccurate SpO2 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 SpO2. 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 hemoglobin, hyperbilirubinemia has little effect on pulse oximeter accuracy. Fetal hemoglobin also has little effect on SaO2. Carbon monoxide (CO) falsely elevates SpO2 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%), SpO2 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 SpO2 of patients without CHD or significant lung disease exceeds 94%. However, it must be understood that a SpO2 of 95-96% is associated with an arterial PaO2 (oxygen tension in the blood) of 60-80mmHg when breathing room air (FiO2 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 SpO₂ is 99%-100%, the PaO₂ can range from 80 – to - >600mmHg. In premature infants, the SpO₂ 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 PaO₂s than children six months of age or older. For example, at a SpO₂ of 95%, neonates have PaO₂s of about 50mmHg, whereas older children have PaO₂ of about 70mmHg.

**Capnography**

Monitoring end-tidal carbon dioxide (CO₂) 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 CO₂ in the exhaled gases. For pediatric patients, these devices work best when the exhaled CO₂ is measured as close to the tracheal tube (ETT) as possible. It is also important to minimize the space between the tracheal tube and CO₂ 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 CO₂ sampling more accurate: pediatric circle system; small infant condenser-humidifier proximal to sampling line; minimum dead-space (0.5 ml) connector. The CO₂ 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 CO₂ waveform is often present with esophageal intubation or from CO₂ introduced into the stomach during mask ventilation. However, in both instances the concentration of CO₂ is low, and no CO₂ will be detected within 5-6 breaths. CO₂ 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 CO₂ detected during cardiac arrest or very low cardiac output is extremely low or absent because there is insufficient pulmonary blood flow to deliver CO₂ to the lungs. Low end-tidal CO₂s 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.
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$_2$. 

FiCO$_2$  | etCO$_2$  
---|---
1 | 35 mmHg 
0 | 10 mmHg 
16 | 41 mmHg 
0 | 30 mmHg
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 ETCO2 is essential when a LMA is used. Finally, a divided CO2 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 CO2 values may occur during equipment malfunctions or failure. A partially disconnected CO2 sampling line, or cracked connector can entrain room air and artificially lower end-tidal CO2 values. An occluded CO2 sampling line (moisture or secretions) will detect little or no end-tidal CO2. Automatic machine calibration at inopportune times, e.g., immediately after tracheal intubation, prevents CO2 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 ETCO2 waveforms, go to http://www.capnography.com.

**Anesthetic Agent, Ventilation, and FiO2 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 CO2 and FiO2. Monitoring respiratory gases, including the anesthetic agent, CO2, and FiO2, 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 CO2 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, positive 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 15-20cmH2O and positive end-expiratory pressures (PEEP) of <10cmH2O 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, PaCO₂, and PaO₂. 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). PaCO₂ measures the partial pressure of carbon dioxide in arterial blood (plasma) and is primarily a measure of respiratory function. The PaCO₂ is normally 35-45mmHg. PaO₂, 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 PaO₂. Normal PaO₂ values while breathing room air vary with age. During the first hours and days of life, premature and full term neonates have normal PaO₂s 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 PaO₂ 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, PaCO₂ is converted to hydrogen ion (H+), which decreases pH. If the only cause of a low pH is CO₂, 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 PaCO₂. During anesthesia and spontaneous ventilation, volatile anesthetics or opioids often depress respiration and elevate PaCO₂. 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 PaCO₂ 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 +2 mmol/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 -2 mmol/L.

Respiratory alkalosis is defined as a PaCO₂ < 35 mmHg 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 PaCO₂ is < 25 mmHg, there is significant risk of cerebral ischemia, especially when the patient is also hypotensive.

Metabolic alkalosis occurs with a pH > 7.45 and normal PaCO₂. 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 PaCO₂ > 45 mmHg 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 PaCO₂ < 35 mmHg and base deficit in excess of -2 mmol/l. This usually occurs as compensation for metabolic acidosis. Normal values for pH, PaCO₂, and PaO₂, 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

<table>
<thead>
<tr>
<th>Condition</th>
<th>pH</th>
<th>PaCO₂ mmHg</th>
<th>PaO₂ mmHg</th>
<th>Base deficit/excess mmol/L</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>7.35-7.45</td>
<td>35-45</td>
<td>50-80 (neonate); 90-110 (older infant/child)</td>
<td>-2 to +2</td>
<td>Healthy infant under anesthesia</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>&lt;7.35</td>
<td>&gt;45</td>
<td>Variable</td>
<td>-2 to +2</td>
<td>Asthma, surgical retraction, inadequate ventilator rate/tidal volume</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>&lt;7.35</td>
<td>35-45</td>
<td>Variable</td>
<td>&gt;-2</td>
<td>Anemia, hypovolemia, low cardiac output</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>&gt;7.45</td>
<td>&lt;35</td>
<td>Variable</td>
<td>-2 to +2</td>
<td>Excessive tidal volume/ventilator rate under anesthesia</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>&gt;7.45</td>
<td>35-45</td>
<td>Variable</td>
<td>&gt; +2</td>
<td>Long term diuretic use; pyloric stenosis</td>
</tr>
<tr>
<td>Mixed respiratory/metabolic acidosis</td>
<td>&lt;7.30</td>
<td>&gt;45</td>
<td>Variable</td>
<td>&gt;-2</td>
<td>Inadequate tidal volume/ventilator rate with large blood loss</td>
</tr>
<tr>
<td>Mixed respiratory/metabolic alkalosis</td>
<td>&gt;7.50</td>
<td>&lt;35</td>
<td>Variable</td>
<td>&gt; +2</td>
<td>Excessive ventilator rate/volume with long term furosemide</td>
</tr>
</tbody>
</table>

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 CO₂ and add oxygen, the normal PvCO₂ is 45-55mmHg, and the PvO₂ 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 SaO₂.

**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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**Bibliography**


Chapter 2: VASCULAR ACCESS AND MONITORING