

CHAPTER 4

CARDIOVASCULAR PHYSIOLOGY AND SUPPORT

Mark W. Newton

John R. Gosche

Laura Boomer

Introduction

Failure of the circulatory system leads to organ dysfunction and ultimately to death. A basic understanding of the physiologic principles of cardiovascular control is essential for early recognition and appropriate treatment of cardiovascular dysfunction.

Cardiac Structure and Function

The force required to pump blood throughout the circulatory system is generated by the heart. The arrangement of the four chambers of the human heart results in two parallel pumping mechanisms (an atrium plus a ventricle, each supplying a separate circulation) that are arranged in series. Due to this series arrangement, failure of one side of the heart usually ultimately results in dysfunction of the other. The force required to pump blood is provided by the contraction of the cardiac muscle, and the valves between the cardiac chambers and at the outflow of the ventricles assure that blood flows in the proper direction. Thus, failure of any of the cardiac valves due to either acquired or congenital defects can severely impair cardiac function.

Cardiac output is the quantity of blood pumped by the heart per unit of time. Cardiac output varies with body size and is proportional to body surface area. Thus cardiac output is frequently normalised to body surface area, which is referred to as the cardiac index. The normal cardiac index per square meter of body surface area for the adult is approximately 3.0 l/min. Normal cardiac index in the newborn infant is approximately 2.5 l/min. This value rapidly increases during early childhood to about 4 l/min by 10 years of age.

Cardiac output is the product of heart rate (contractions per minute) and average stroke volume (ml per contraction) over a time period. Stroke volume, in turn, is affected by changes in preload, afterload, and contractility. During periods of inadequate cardiac output, alterations in all of these variables should be sought and addressed to optimise cardiac function.

Preload is the amount of blood in the ventricle at the end of diastole and reflects the venous return to the heart. Under normal circumstances, the heart pumps whatever amount of blood enters the right atrium without a backup of blood in the atria. This physiologic ability to increase cardiac output is referred to as the Frank-Starling relationship and reflects improved interdigitation of actin and myosin filaments, resulting in optimal force generation during contraction. This ability to increase contractile force even occurs in the weakened heart. Thus, increasing blood volume by giving a fluid bolus or transfusion may improve cardiac output and perfusion even in patients with known cardiac dysfunction.

Of course, there are physiologic limits beyond which increasing end diastolic volume results in excessive stretch of the myocardial fibres and decreases contractile force. This circumstance is seldom observed in patients with normal cardiac function, but may develop in patients with cardiac failure due to ischaemia, valvular disease, myocarditis, or congenital cardiac anomalies. In the absence of valvular disease and

pericardial disease, end diastolic right ventricular filling pressure in the right ventricle is equivalent to diastolic atrial pressure and is reflected by central venous pressure. In practice, unfortunately, direct measurements of venous pressure may not always be available. Then indirect indicators such as jugular venous distention and changes in blood pressure with changes in patient position (i.e., orthostatic hypotension) should be looked for, as they may reflect increased or decreased central venous pressures affecting preload.

Afterload is the pressure against which the ventricles must contract to eject blood from the heart. Thus, the afterload on the ventricles is the pressure in the aorta for the left ventricle (or pulmonary main for the right ventricle) throughout systole. In the normal heart, changes in systolic pressure over the physiologic range do not significantly affect cardiac output. Only at extremes of pressure does afterload impair cardiac output in the normally functioning heart. However, congenital anomalies that result in obstruction of blood flow (e.g., coarctation of the aorta, pulmonic stenosis) may create excessive afterload on the heart and impair cardiac output, resulting in heart failure. Furthermore, in patients with poor cardiac function (e.g., myocarditis or valvular heart disease), the judicious use of vasodilators to decrease afterload may significantly increase cardiac output.

Contractility refers to the strength of cardiac muscle contraction and is measured as the change in ventricular pressure generated per unit of time. As noted previously, cardiac contractility is affected by preload due to the Frank-Starling relationship. Cardiac contractility is also influenced, however, by the autonomic nervous system. Specifically, increased sympathetic activity results in increased cardiac contractility, whereas increased parasympathetic activity decreases contractility. Stimuli that increase cardiac contractility are said to have a positive inotropic effect, and those that decrease contractility are said to be negative inotropes. Sympathetic stimulation increases contractility by increasing calcium release during contractions and by increasing the sensitivity of myofilaments to calcium. The negative inotropic effect of parasympathetic activity likely primarily results from loss of normal tonic sympathetic activity. Unfortunately, contractility is a difficult variable to measure in clinical practice. One option for assessing contractile function is to measure ejection fraction by echocardiography.

The final variable that impacts cardiac output is heart rate. Changes in heart rate primarily reflect changes in autonomic nervous activity, with sympathetic stimulation increasing heart rate (i.e., positive chronotrope) and parasympathetic stimulation decreasing heart rate (i.e., negative chronotrope). Heart rate is also affected by intrinsic mechanisms, however. For instance, stretch of the right atrial wall during increases in venous return causes an increase in the heart rate by as much as 10–30%. Increases in heart rate generally correlate with increases in cardiac output, but beyond critical levels, further changes in heart rate may have the opposite effect on cardiac output. As an example, at very high rates above a critical level, stroke volume decreases, thereby limiting cardiac output. Decreased stroke volume at high heart rates results from limited

availability of metabolic substrates to support myocardial contraction and a decreased ventricular preload. Conversely, a low heart rate is usually associated with an increase in stroke volume due to increased ventricular preload.

Cardiac output is affected by the above-noted variables in patients of all ages, but neonates are somewhat unique in that they have a limited ability to increase stroke volume. The difference reflects a relatively lower compliance of the neonatal myocardium, thereby limiting increases in cardiac output associated with increases in preload. Also, the neonatal myocardial contractility is less responsive to sympathetic stimulation due to differences in calcium transits. Therefore, the neonate is much more dependent upon changes in heart rate to increase cardiac output in times of need.

Anatomy and Physiology of the Circulation

The body has two circulatory systems: the pulmonary circulation and the systemic circulation. Normally, the two circulations are separate. Structurally, both circulations are similar in that they start with a single large vessel that, through sequential branchings, distributes blood to arteries of decreasing diameter but increasing number. Ultimately the small arteries (arterioles) empty blood into a series of capillaries, which are the primary site of exchange of solutes between the intravascular and extravascular compartments. From the capillaries, blood enters a large number of small veins (venules), which, through a series of junctions with other venous structures of similar calibre, coalesce into several large venous structures that return blood to the atria.

An important concept to remember is that blood always flows down a pressure gradient and will always take the path of least resistance. As a result, points of increased resistance in the circulatory system will result in an increase in pressure proximal to the point of the obstruction until either the pressure is adequate to overcome the cause of the resistance or blood flow is diverted through an alternate pathway. The pulmonary and systemic circulations differ in that the pulmonary circulation is a high-flow, low-resistance, and thus low-pressure system, whereas the systemic circulation has much higher overall resistance and as a result has higher intraluminal pressures. Reflecting these differences in pressures, the relative stiffness and thickness of the arteries are greater in the systemic circulation than in the pulmonary circulation. However, in patients with abnormal connections between the pulmonary and systemic circulations (e.g., patent ductus arteriosus), the pulmonary arteries will ultimately hypertrophy in response to the higher pressures experienced by the pulmonary vessels. The following section primarily addresses the mechanisms that control pressure and blood flow within the systemic circulation.

Control of pressure and blood flow in the systemic circulation occurs both globally and locally. Table 4.1 lists ranges for average blood pressure based upon age, with the mean pressure $\pm 20\%$ at the 95% confidence limit. Values for females are approximately 5% lower than for males. These paediatric blood pressure references may help guide diagnosis and management during times when the patient is demonstrating signs of shock.

The central nervous system (CNS) and the kidneys are the primary organs responsible for global blood pressure control. The CNS affects systemic blood pressure both directly via the autonomic nervous system and indirectly by inducing release of humoral factors. Increased sympathetic nervous system (SNS) activity increases blood pressure by increasing cardiac contractility (as described previously) and by causing vasoconstriction through release of norepinephrine from nerve endings that innervate the vasculature. The latter effect increases arterial resistance, thus increasing proximal pressures, and also increases venous return to the heart—and thus cardiac output—by increasing venous tone and decreasing the capacitance of the venous system. SNS activation also increases blood pressure by stimulating release of the vasoconstrictors, epinephrine, and norepinephrine from

Table 4.1: Age and average blood pressures.

Age	Average blood pressure (mm Hg)
Premature	Systolic 40–60
Full term	75/50
1–6 months	80/50
6–12 months	90/65
12–24 months	95/65
2–6 years	100/60
6–12 years	110/60
12–16 years	110/65
16–18 years	120/65
Adult	125/75

Source: Modified from Zuckerberg AL, Wetzel RC, Shock, fluid resuscitation, and coagulation disorders. In Nichols DG, Yaster M, Lappe DG, Buck JR (eds). *Golden Hour: The Handbook of Advanced Pediatric Life Support*. Mosby-Year Book, 1991.

the adrenal medullae. Finally, the CNS also affects blood pressure over the long term by releasing vasopressin (antidiuretic hormone) from the posterior pituitary, which primarily has an effect by increasing water reabsorption by the renal tubules and increasing intravascular volume. Vasopressin is also a potent vasoconstrictor; however, under normal conditions, the circulating concentration of this hormone is too low to have a direct effect on vascular control.

Given the potent effects of the CNS on blood pressure regulation, it is not surprising that there are multiple mechanisms for controlling the vasomotor centres in the brain. These vasomotor centres are located in the medulla and pons and include both a vasoconstrictor area and a vasodilator area. The vasoconstrictor area causes excitation of vasoconstrictor neurons in the SNS, whereas the vasodilator centre primarily functions to cause inhibition of neurons in the vasoconstrictor area. The activities of these two vasomotor centres are affected by afferent impulses (1) from stretch receptors (baroreceptors) located in the carotid sinus and the wall of the aortic arch, which respond to short-term changes in pressure in these arteries; (2) from low pressure receptors in the atria and pulmonary arteries that reflect changes in blood volume; and (3) from higher brain centres that respond to stressful stimuli (e.g., pain, alarm) and CNS ischaemia. Under normal conditions, the vasoconstrictor centre of the brain stem is continuously active, causing partial contraction of the blood vessels and maintaining baseline vasomotor tone. This explains why rapid loss of SNS activity (such as following a cervical spinal cord injury) often results in hypotension.

The other organ that has important global effects on blood pressure is the kidney. In the kidney, the juxtaglomerular cells located in the proximal arterioles release renin into the bloodstream in response to a decrease in perfusion. Renin is an enzyme that cleaves circulating plasma angiotensinogen, resulting in release of angiotensin I. Angiotensin I is subsequently metabolised to angiotensin II by converting enzyme, which is primarily located in the walls of small vessels in the lung. Angiotensin II has several effects, including vasoconstriction of both arterioles and veins, resulting in an increase in vascular resistance and venous return. It also decreases salt and water loss by the kidney (both by a direct effect on the kidney and by stimulating secretion of aldosterone by the adrenal cortex), resulting in expansion of the circulating blood volume. Ultimately, these effects cause an increase in blood pressure and renal perfusion, resulting in a negative feedback on renin release.

The global mechanisms for affecting blood flow are primarily involved in maintaining adequate central systemic blood pressure

and thus assuring adequate perfusion to organs with high metabolic needs, including the heart and CNS. The vasoconstricting effects of sympathetic nervous activity and circulating humoral agents result in a decrease in vascular compliance (the same as an increase in vascular resistance). The cumulative effect of increasing vascular tone contributes to the body's total vascular resistance. The total vascular resistance is one factor that affects pulse pressure, or the difference between systolic and diastolic blood pressures. The other factor is the stroke volume output of the heart. Increases in vascular resistance primarily are reflected by increases in diastolic pressure, whereas increases in cardiac output typically result in an increase in both the systolic and the diastolic pressure. Typically, the diastolic pressure is two-thirds to three-fourths of the systolic pressure. Changes in pulse pressure can be a valuable indicator of circulatory derangements.

Whereas the globally active mechanisms primarily affect systemic blood pressure, local control mechanisms are primarily involved in controlling blood flow to individual organs and tissues. Both metabolic and myogenic mechanisms may be involved in local control of blood flow. Myogenic control reflects the ability of the vascular smooth muscle to constrict in response to increased wall stretch. The myogenic mechanism allows local autoregulation of blood flow that is somewhat independent of upstream pressures. The physiologic importance of the myogenic response is debatable, but it may provide a means for preventing local hyperperfusion and tissue oedema during periods of elevated systemic blood pressure. Likely, the more important control mechanism is metabolic control, which enables the local vasculature to respond to changes in local tissue demand.

Two theories have been proposed to explain how increases in tissue metabolic demand can affect blood flow. The first theory is that a vasodilator substance (e.g., adenosine, carbon dioxide, histamine, or similar) is produced by tissues in response to local decreases in the availability of oxygen or another metabolite. Of the proposed substances, adenosine is a likely candidate. Once released, the vasodilator agent is believed to diffuse locally and induce dilatation of upstream arterioles. The resultant increase in local blood flow would increase the local supply of oxygen and other metabolites to the tissues, thus creating a negative feedback mechanism. The other theory is that local decreases in oxygen tension are directly responsible for causing local vasodilation. This response is based upon the requirement of vascular smooth muscle for oxygen to maintain active contraction. Thus, in response to local decreases in oxygen tension, the vascular smooth muscle of the local upstream arterioles would relax, resulting in an increase in blood flow and oxygen delivery to the tissues in need.

Of course, local vasodilation of downstream blood vessels is of no use if perfusion is limited due to vasoconstriction of more proximal arteries. Local activation of vasodilator responses cannot affect the tone of proximal arterioles. However, as downstream vessels dilate, blood flow velocity in the upstream vessels is increased. The endothelial cells lining arterioles have the ability to sense increases in flow velocity as shear stress. As shear stress increases, endothelial cells release vasodilator substances locally, thereby resulting in relaxation of the adjacent vascular smooth muscle. The most important of these vasodilator agents is the endothelial-derived relaxing factor, nitric oxide. Thus, in response to increases in tissue metabolic need, both local and upstream vessels dilate, resulting in increasing blood flow to meet metabolic demands.

Shock and Clinical Implications in the Paediatric Surgical Patient

Shock is defined as a severe pathophysiological alteration in the normal homeostatic processes of oxygen delivery and cellular metabolism that, if untreated and prolonged, can lead to major changes in these processes and cellular death. The traditional classifications of shock in the paediatric population include: hypovolaemic, septic, cardiogenic,

and neurogenic. Each of these forms of shock can be present in the paediatric perioperative surgical patient, and it is imperative that each type of shock be adequately treated prior to an elective procedure. One may encounter those occasions when the shock may not be completely resolved, however, and surgery becomes unavoidable or emergency surgery is required. During these types of patient presentations, the understanding of each form of shock needs to be understood and, if possible, treated before surgery ensues and further complications arise.

Hypovolaemic Shock

Hypovolaemic shock is the most common form of shock in the paediatric population and results primarily from a decreased intravascular volume, causing a diminishing venous return and consequently, a decreased preload. Neonates and young infants have a relatively set stroke volume due to the immaturity of their cardiac muscle, which results in the compensation mechanism of an increased heart rate when the preload decreases.

In the typical African clinic, it would be common to see a paediatric patient who has a 2- or 3-day history of diarrhoea or vomiting and presents in hypovolaemic shock with cool, pale extremities; decreased peripheral perfusion (>4 seconds); and decreased urine output. In general, the blood pressure decrease seen in adult patients who have lost 15–25% of their intravascular volume does not occur in the paediatric population, and blood pressure alone is an insensitive indicator of dehydration in children due to their ability to increase their heart rate (Table 4.2). The SNS discharge attempts to compensate for the loss in intravascular volume, but when the acidosis persists and overcomes the vasoconstriction, capillary leak may occur as well.

The paediatric patient may develop tachypnea in an effort to decrease the acidosis that is produced due to the low tissue perfusion occurring during the shock phase. Lethargy and decreased responsiveness to pain occur secondary to decreased cerebral perfusion and low oxygen delivery. These findings associated with a drop in heart rate and blood pressure are ominous signs, and immediate action needs to be quickly pursued. The aetiology of the shock needs to be determined. The most common causes of hypovolaemic paediatric shock include trauma, burns, peritonitis, severe vomiting, and diarrhoea, and in some cases, hyperthermia with decreased intake, which is common with malaria.

Table 4.2: Clinical effects of dehydration based upon percentage of body weight decrease.

Dehydration (% body weight)	Clinical observation
5%	<ul style="list-style-type: none"> • Increase in heart rate (10–15% above baseline) • Dry mucous membranes • Concentration of the urine • Poor tear formation
10%	<ul style="list-style-type: none"> • Decrease in skin turgor • Oliguria • Soft, sunken eyes • Sunken anterior fontanelle
15%	<ul style="list-style-type: none"> • Decrease in blood pressure, tachycardia, tachypnoea • Poor tissue perfusion and acidosis • Delayed capillary refill

Source: Modified from Zuckerberg AL, Wetzel, RC. Shock, fluid resuscitation, and coagulation disorders. In Nichols DG, Yaster M, Lappe DG, Buck JR (eds). Golden Hour: The Handbook of Advanced Pediatric Life Support. Mosby-Year Book, 1991.

Initial management would include the management of the airway, and every patient in shock should receive 100% oxygen by a face mask until the shock resolves. If the airway needs a more definitive measure, then endotracheal intubation needs to be performed because the combination of shock and respiratory problems has a very high mortality rate.

It is always important to remember that shock is a very dynamic process, and changes occur rapidly—this is especially true in the paediatric population, which requires adjustments that are ongoing in the management plan.

Fluid resuscitation in the hypovolaemic patient with a large-bore intravenous cannulae is required, and locations such as the saphenous, femoral, external jugular, and intraosseous may need to be used. The goal is to replace the intravascular volume as quickly as possible with a crystalloid solution, such as normal saline and not dextrose in water. Normal saline is readily available in most areas of Africa. The expansion effect in the extracellular compartment is greatest, and the cost of the fluid is inexpensive compared to other fluids. At Kijabe Hospital in Kenya, we do not use Ringer's lactate with paediatric patients due to the presence of potassium in Ringer's lactate and its effects on a patient with potentially poor renal function. Figure 4.1 presents an algorithm for treatment of hypovolaemic shock in children. Although this algorithm may need to be adjusted for each specific clinical dilemma, the figure will provide a guide for taking the necessary steps needed to prepare the paediatric shock patient for emergency surgery.

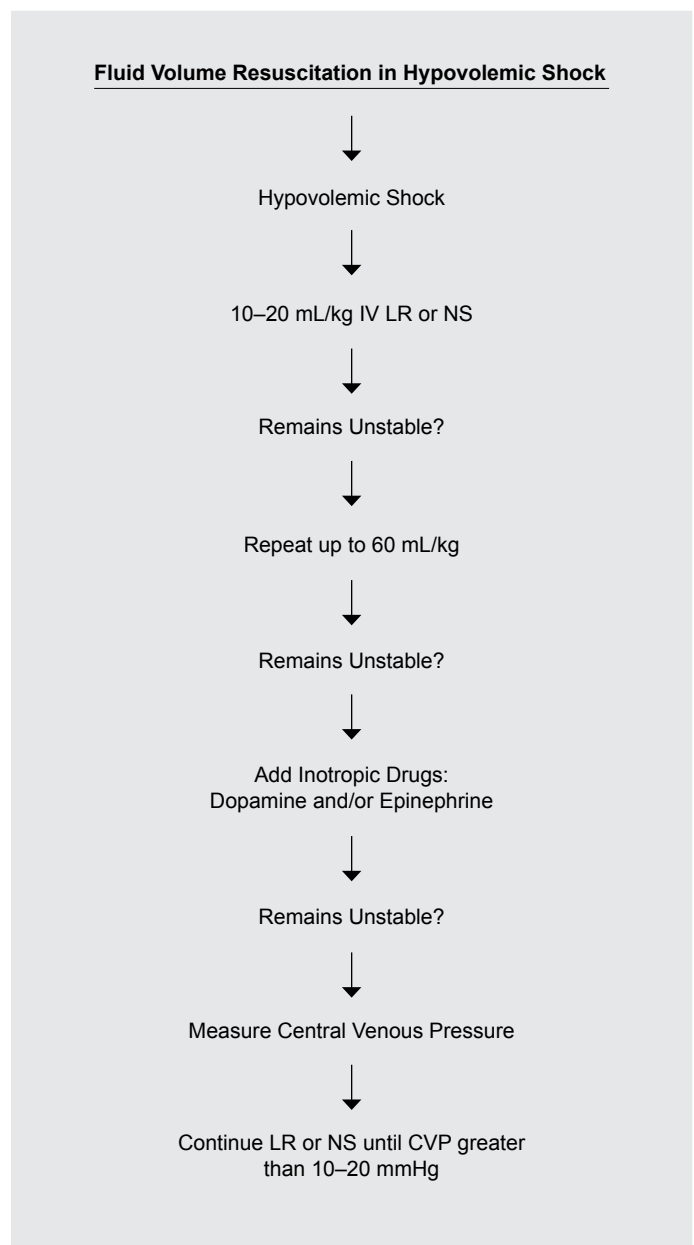
In situations whereby the hypovolaemic shock is due to acute blood loss, the resuscitation team needs to be prepared to infuse appropriate volumes of blood in an attempt to maximise the oxygen-carrying capacity of the intravascular volume. The patient's blood pressure, heart rate, respiratory rate, urine output, and mental status need to be monitored to help determine the appropriate volume to be infused. Most who work in Africa will not have access to central venous monitoring devices; therefore, these indirect measurements of intravascular volume need to act as guides for adequacy of replacement. O-negative or merely type-specific blood can be infused rapidly in the paediatric patient who needs blood urgently to survive due to the shock. If the patient fails to respond to the fluid resuscitation measures, before considering an inotropic agent such as dopamine, look for an additional cause of bleeding or decreased cardiac output, such as tension pneumothorax.

Septic Shock

Septic shock is associated with microorganisms in the blood and the effects of toxic products with an associated inadequate delivery of oxygen to the tissues. Initially, the oxygen delivery can be high, with warm and well-perfused tissues, but this can change if lactic acidosis overcomes the compensatory mechanisms of the paediatric patient due to excessive demand for oxygen. Although gram-negative and gram-positive organisms are a common cause of sepsis, tuberculosis, herpes, and malaria are forms of sepsis seen more often in the African environment. In an environment where the patients arrive late in their course of distress, septic shock can be severe and the mortality very high in the paediatric population.

The factors that indicate septic shock syndrome are as follows:

- clinical suspicion or evidence of infection;
- temperature instability (fever or hypothermia);
- tachycardia/tachypnea; and
- impaired organ system function:
 - peripheral hypoperfusion;
 - altered level of consciousness;
 - oliguria;
 - hypoxaemia;
 - acidosis; and/or
 - pulmonary oedema.



Source: Modified from Litman, RS. Pediatric critical care. In: Litman RS. Pediatric Anesthesia: The Requisites in Anesthesiology. Mosby, Inc., 2004, P 331.

Figure 4.1: Algorithm for treatment of hypovolaemic shock.

The cardiovascular effects that implicate septic shock include lower systemic vascular resistance, increased capillary leak, and increased venous capacitance, which will directly decrease preload and therefore cardiac output. In patients for whom direct myocardial contractility is affected, the inability to provide sufficient oxygen supply for the high demand results in rapid deterioration. The patient who presents early with “warm shock” will demonstrate a significantly different picture than the delayed presenter who is hypovolaemic with “cold shock”.

The management of septic shock is similar to hypovolaemic shock in relation to the need for oxygen and fluids, but these patients need to have the aetiology of the septic shock discovered rapidly so that the toxic effect can be diminished and eventually removed from the system. Antibiotics, antituberculosis, or antimalarial drugs need to be administered early and in appropriate doses so that the cause of the sepsis can be resolved. Disseminated intravascular coagulation (DIC), renal failure, acute respiratory failure, and even liver failure can be caused by sepsis.

Cardiogenic Shock

Cardiogenic shock is defined as shock due to cardiac failure, which can be due to infections, trauma, drug overdose, cardiomyopathies, and congenital heart disease. Although cardiac failure may be present in other forms of shock, this form is directly due to the cardiac function. In the newborn period, cardiogenic shock can be caused by a hypoplastic left heart, which is difficult to manage in any environment.

The management of cardiogenic shock depends on the aetiology of the hypotension, but the use of vasopressors such as dopamine and epinephrine with the addition of 5–10 ml/kg boluses of fluids while monitoring cardiac volume indirectly may help temporarily. Arrhythmias may occur more commonly in this form of shock, and the identification of the type of electrocardiogram (ECG) abnormality will help as treatment options are considered.

Cardiogenic shock carries a high mortality rate, and invasive monitoring with mechanical circulatory assistance is sometimes difficult to obtain in resource-poor settings. Without surgical correction of the correctable cardiac paediatric lesions, at times only palliative care can be provided for these patients.

Neurogenic Shock

Cervical spinal cord injury is associated with dysfunction of the sympathetic nervous system, resulting in such cardiovascular changes as severe bradycardia, asystole, and loss of peripheral vascular tone. Cardiovascular problems known to arise from SNS dysfunction include low resting blood pressure, orthostatic hypotension, autonomic dysreflexia, reflex bradycardia, cardiac arrest, limited cardiovascular response to exercise, and alterations in skin microcirculation.

Patients in neurogenic shock initially have warm extremities and low diastolic pressure, which may eventually develop into a situation of acidosis and a decrease in perfusion pressure. With the sudden loss of sympathetic tone, especially if the lesion is above T6, the patient may demonstrate signs of bradycardia and other arrhythmias due to the effect of the cardioaccelerator fibers. Pulmonary oedema may develop due to fluid resuscitation when the loss of sympathetic tone results in peripheral vasodilatation. The management of neurogenic shock depends upon the level of injury and the involvement of the levels for ventilation. If the level is below C8, then the diaphragm is intact and providing the necessary muscles of inspiration needed to maintain oxygenation. Fluid resuscitation and monitoring for bradycardia may prompt the use of intravenous atropine and even vasopressors to maintain the appropriate blood pressure.

Management of Shock

All forms of shock—hypovolaemic, septic, cardiogenic, and neurogenic—can have similar effects on the paediatric patient, and therefore

have similar management plans. The foundation of oxygen delivery to compensate for oxygen utilisation allows medical care providers a target to aim toward as they seek to resolve the hypovolaemia, identify the organism in sepsis, search for the cardiac resolution of the shock, or treat the acute spinal cord injury and associated implications of the physiological implications of no sympathetic nervous system. Table 4.3 lists some common cardiovascular medications used in shock management.

Clinical Correlations

The following scenarios illustrate the clinical impact of alterations in cardiovascular function and provide recommendations for management.

Case Scenario #1

Presentation

You are planning a posterior sagittal anorectoplasty (PSARP) on an 8-month-old male with high imperforate anus and unrepaired tetralogy of Fallot (TOF). The patient had a colostomy at one month of age and since that time has had approximately two episodes of central cyanosis, which resolve spontaneously per day. The patient is not on any medications except for iron supplement, and the room air oxygen saturation is 90%. His preoperative haemoglobin level is 8.1. He is small for his age, at 5.1 kg, and has no known respiratory issues.

1. What is the likely aetiology of his cyanotic episodes?
2. How should this patient be managed intraoperatively?

Treatment

This patient has documented tetralogy of Fallot, a cardiac anomaly characterised by right ventricular outflow obstruction associated with a ventricular septal defect (VSD), overriding aorta, and right ventricular hypertrophy. Due to the obstruction of right ventricular outflow, blood flow through the pulmonary circulation in most patients occurs through persistence of the foetal connection between pulmonary and systemic circulations, the ductus arteriosus. Therefore, in these patients, oxygenated blood returning from the lungs and unoxygenated blood returning from the peripheral tissues are mixed in the ventricles through the VSD. The percentage of cardiac output passing through the pulmonary circulation determines the severity of cyanosis.

Patients with TOF frequently experience episodes of worsening cyanosis (“tet” spells) associated with decreased pulmonary perfusion in response to stimuli that increase pulmonary outflow obstruction or decrease systemic vascular resistance. Options for treating cyanotic episodes include IV fluid boluses, pressure on the abdominal aorta, liver compression, morphine 0.1 mg/kg IV, or intravenous sodium bicarbonate. Oxygen is seldom helpful during a “tet” spell due to decreased pulmonary perfusion. During an anaesthetic, it is important to avoid a drop in systemic blood pressure, as this will worsen right-

Table 4.3: Common cardiovascular medications.

Drug	Paediatric dosing	Uses	Classification	Mechanism of action
Dopamine	5–20 mcg/kg per min IV	Shock	Inotropes/ vasopressors	Alpha- and beta1- agonist; stimulates dopaminergic receptors
Epinephrine (Adrenalin)	0.01 mg/kg IV q 3–5 min prn for arrhythmia; SC/IM q 20 min–4 hr for anaphylaxis or asthma	Asystole, VF, pulseless VT, bradycardia, asthma, anaphylaxis	Inotropes/ vasopressors; anti-arrhythmics; anaphylaxis	Sympathomimetic stimulation of alpha- and beta- adrenergic receptors
Phenylephrine (Neo-Synephrine)	5–20 mcg/kg IV bolus, then 0.1–0.5 mcg/kg/min IV; or 0.1 mg/kg SC/IM q 1–2 hr for mild hypotension; or 5–10 mcg/kg IV x 1 for paroxysmal supraventricular tachycardia (PSVT) conversion	Shock, hypotension, PSVT conversion	Inotropes/ Vasopressors	Smooth muscle alpha-agonist (vasoconstrictor)
Ephedrine	10- 50 mg IV (adults) prn hypotension titrated to effect	Hypotension	Inotropes/ Vasopressors; Decongestants	Smooth muscle alpha-agonist (vasoconstrictor)

to-left shunting. Patients may respond to an IV fluid bolus, decreasing inhalation anaesthetic, and vasoconstrictor drugs such as phenylephrine (alpha agonist) to increase systemic vascular resistance. Most cyanotic patients are polycythemic, which improves oxygen delivery. Therefore, consideration should be given to transfusing this patient prior to surgery. Ketamine is a good choice for induction because it tends to maintain systemic blood pressure. Narcotics and low-dose halothane are good choices for this particular surgery, which must be completed without muscle relaxants to allow nerve stimulation during surgery. The goal should be to extubate the patient in the immediate postoperative period to minimise airway stimulation, which can induce a “tet” spell.

Case Scenario #2

Presentation

You are called to see an 8-year-old, previously healthy boy with a 2-day history of abdominal pain and vomiting. On examination, the patient is moderately distended and has diffuse abdominal tenderness with involuntary guarding. The patient seems somewhat anxious, he is tachypneic, his temperature is 39.5°C, his heart rate is 140, and his blood pressure is 90 over 45. His extremities are cool to the touch.

1. What is the likely aetiology of this patient’s altered vital signs?
2. What should you do to prepare this patient for surgery?

Treatment

This patient presents with an acute abdomen of two days duration. Based upon the findings on clinical examination, he has diffuse perito-

nitis. His tachycardia, low blood pressure, anxiety, and poor peripheral perfusion are consistent with circulatory shock. He is febrile and has a wide pulse pressure, which would suggest that shock may be due to sepsis. However, patients with peritonitis lose a large amount of intravascular volume due to transudative and exudative losses into the peritoneal cavity. Therefore, this patient likely also has a component of hypovolaemia contributing to his shock state.

The first and most important step in the management of this patient is to recognise that he is in shock. Due to the cardiodepressive effects of most anaesthetic agents, worsening hypotension and organ dysfunction would likely result if this patient were taken directly to the operating room without prior resuscitation. Therefore, an effort should be made to optimise his haemodynamics prior to the induction of anaesthesia. Because both septic shock and hypovolaemic shock respond initially to expansion of the intravascular blood volume, a large-bore IV should be started and the patient should receive one or more boluses of a crystalloid solution. During the period of preoperative resuscitation, vital signs should be monitored frequently, and a bladder catheter should be inserted to monitor urine output as a measure of adequacy of end organ (renal) perfusion. In addition, because sepsis is suspected, the patient should be started on a broad-spectrum antibiotic. It is likely that the ultimate treatment for the cause of this patient’s shock will require surgical intervention; therefore, resuscitation should occur as expeditiously as possible.

Key Summary Points

1. Alterations in venous return (preload), vascular resistance (afterload), heart rate, and contractility all impact cardiovascular function.
2. In the healthy patient, compensatory mechanisms allow maintenance of adequate cardiac output and organ blood flow in the face of limited changes in these variables.
3. Neonates have a limited ability to increase cardiac output by increasing contractility and stroke volume, and thus are dependent upon heart rate to maintain cardiac output.
4. Shock is the result when pathologic conditions severely alter one or more factors and overwhelm compensatory responses, resulting in cellular ischaemia due to inadequate cardiac output or a maldistribution of blood flow.
5. Recognition and treatment of the cause of shock is central to optimising patient outcome.

Suggested Reading

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