CHAPTER 12
INTENSIVE CARE

Andrew Gustaf Nyman
Alison Shefler

Introduction

Injury or illness is defined as critical when one or more organ systems are either in danger of failing or have begun to fail. In this situation, the possibility of incomplete recovery or death exists. Critical care comprises the monitoring, support, treatment, and interventions for the organ systems in failure. Paediatric critical care not only encompasses bedside management of children with severe, potentially life-threatening medical or surgical illness, but also extends to providing support to the child's family or caregivers. The challenge lies in the complex balance of providing support of single or multiple organ systems in failure while at the same time minimising adverse consequences of treatment. This level of care is usually, but not always, provided in a dedicated paediatric intensive care environment with the capacity to offer sophisticated monitoring, diagnostic and therapeutic interventions, as well as advanced technological support for the critically ill child. When the outcome is poor or death ensues, the critical care focus shifts to palliative and, if necessary, bereavement support. The spectrum of disease in children differs from that of the adult population, as does the paediatric response to illness, surgery, or injury. Congenital abnormalities, genetic syndromes, inborn errors of metabolism, and toxins, as well as trauma, including birth-related and nonaccidental injury, all influence the differential diagnosis of an acutely unwell child. Regardless of the aetiology, basic principles of initial management and stabilisation should be applied in all situations.[1,2]

Approach to the Acutely Unwell Child

Respiratory failure is a common manifestation of critical illness and generally requires early recognition and intervention to prevent progression to full cardiopulmonary arrest, which carries a grave prognosis.[3–9] This section of the chapter therefore begins by outlining the systematic approach that underpins all paediatric life support and intensive care management of the acutely unwell child, namely, addressing the child's airway, breathing, and circulation.[10]

Airway

The goals of airway management are to overcome obstruction, promote adequate gas exchange, and prevent aspiration.

The first priority in the assessment of a critically ill or injured child is to ensure a patent airway. Any compromise to airway patency, either structural or functional, is a potential medical emergency, and it is important to recognise it because failure to establish or maintain the airway can result in or worsen respiratory compromise. Respiratory failure may, in turn, progress to cardiopulmonary arrest; thus, every effort should be made to secure airway stabilisation in a timely manner.

The paediatric airway is more susceptible to airway compromise than that of adults for a number of reasons.[11–15]

- A child's proportionally larger head and prominent occiput result in neck flexion, with the potential for exacerbating upper airway obstruction when lying supine.
- The tongue is relatively large and its muscle tone is reduced.
- The epiglottis is shorter, narrower, and more horizontally positioned than in an adult.
- The larynx is in a more anterior and cephalad position than in an adult.
- The trachea is smaller and narrower.
- The airway is funnel shaped, with the narrowest portion at the level of the cricoid cartilage.

Functional airway compromise results in children with decreased muscle tone in the head and neck. It may be secondary to a decreased level of consciousness and/or the effects of anaesthesia or analgesic or sedative drugs. An inability to maintain a patent airway, even in the absence of a structural abnormality, may present as great a risk as the presence of anatomical obstruction.

Airway compromise may be due to or exacerbated by congenital anomalies, the presence of foreign bodies, or extrinsic compression by structures outside the airway. The most significant difference in the paediatric airway compared to that of adults, and therefore a major contribution to the vulnerability of the airway, is its size and diameter.[15] According to the Hagen-Poiseuille law, which relates to the flow of gas, a change in the radius of the airway has the greatest effect on air flow. As a result, any oedema of the paediatric airway will significantly reduce the calibre of the airway, resulting in a dramatic increase in resistance to air flow and, consequently, the work of breathing. This is particularly important in infants, who are obligatory nasal breathers. Nasal breathing, without any additional obstruction, doubles resistance to flow. The nares in infants and children are significantly smaller than in adults and can account for up to 50% of total airway resistance. With this is mind, it is important to note that simply removing secretions from the nares may result in a dramatic decrease in the work of breathing.[15]

When intervention is required to establish airway patency, a stepwise approach is essential. If basic manoeuvres, such as positioning the head, chin, or jaw, are insufficient, one may have to use airway adjuncts such as a Guedel oropharyngeal airway or a nasopharyngeal airway. For all children who have a potential cervical spine injury, the spine should be adequately immobilised, and unnecessary manipulation should be avoided. Should previous efforts to establish an airway be unsuccessful, endotracheal intubation, laryngeal mask airway, or—rarely—surgical intervention in the form of a tracheostomy may be required, both to establish airway patency and to maintain adequate gas exchange.

Breathing

Acute respiratory failure is a major cause of paediatric morbidity and mortality. It accounts for approximately 30–50% of admissions to paediatric intensive care facilities.[7,16–21] Numerous clinical situations have the potential for progression to respiratory failure, reflecting the complex involvement of the respiratory system with other organ systems. Diagnosis and management of respiratory failure require an understanding of normal respiratory physiology as well the pathophysiological processes occurring in acute medical or surgical disease.

Respiratory failure is inadequate exchange of oxygen and carbon dioxide resulting in failure to meet metabolic demands.

Anatomically, the respiratory system structure comprises the lungs and respiratory pump. The lungs include the airways, alveoli, and pulmonary circulation. Failure in any of these elements may result in abnormal gas exchange, which is manifested by hypoxia; this condition is termed hypoxic respiratory failure. The respiratory pump refers to the thorax, respiratory muscles, and nervous innervations. The inability to effectively pump air into and out of the lungs results in hypoventilation and thus hypercarbia; this condition is termed hypercarbic respiratory failure. Although the two systems can be described separately, the two interact significantly with each other. Failure in one of these systems often results in failure of the other.

To achieve adequate gas exchange, several conditions must be met:

- Adequate gas must reach the alveoli.
- Inspired gas in the alveoli must match the blood distribution within the pulmonary capillaries.
- The alveolar-capillary membrane must permit gas exchange.

A child with a decreased level of consciousness due to any cause—including the postoperative patient under the influence of anaesthetic, analgesic, or sedating drugs—may have inadequate respiratory drive, resulting in an inadequate respiratory rate (see Table 12.1). Acute respiratory distress may result from disease in the large or small airways, the lung parenchyma, the pleural space, or a combination of all of these. Disease in other organ systems, such as cardiac failure or metabolic acidosis associated with diabetic ketoacidosis or toxin ingestion, may give rise to increased respiratory effort. Should any of these disease processes result in inadequate gas exchange, respiratory failure ensues.

Table 12.1: Normal respiratory rate in children.

Age	Breaths per minute
Birth–1 year	20–30
2–5 years	20–25
>5 years	16–20

In addition to the above factors, gas exchange may be affected by systemic processes such as the systemic inflammatory response syndrome (SIRS) seen in sepsis and following cardiac bypass, as well as nonpulmonary factors, including acute blood loss, poor cardiac output (CO), increased oxygen demand, and chronic anaemia. Nutritional deficiencies may contribute to an inability to meet the demands of acute medical or surgical illness.

The most serious manifestation of respiratory insufficiency is hypoxia. Initial compensatory hyperventilation may cause an early drop in $PaCO_2$; however, as these compensatory mechanisms fail, hypercapnia ensues.

Oxygen should be administered to all critically ill or injured children in the highest possible concentration until the assessment of cardiorespiratory status is complete.

The early goal of administering the highest possible oxygen concentration to the acutely unwell patient remains the highest priority, as oxygen delivery to the tissues may be suboptimal in the child with decreased circulating volume or abnormalities in microcirculation, such as may be seen in sepsis, hypovolaemia, or haemorrhage.

Respiratory insufficiency is generally recognised by an early increase in respiratory rate, which may be followed by a decrease in respiratory rate as the child's clinical condition worsens.[22] Apnoea in the small infant is worrying and requires immediate intervention. Cyanosis and tachycardia are early findings, and as hypoxia worsens, progression to bradycardia and cardiac arrest may occur.[23]

Supplemental oxygen can be delivered via many different delivery devices. The choice of device will be dictated by clinical situation and local availability as well as by which device is best tolerated by the child.

Nonrebreathing face mask

A nonrebreathing face mask is the most effective way of delivering oxygen by face mask. It consists of a face mask connected by a unidirectional valve to an oxygen reservoir bag. The unidirectional valve delivers all inhaled gas from the oxygen reservoir and prevents exhaled air from entering the reservoir. The mask must fit snugly over the nose, and the fresh gas flow rate must be maintained to ensure the reservoir remains distended by at least half its volume at all times. In ideal conditions, these masks can provide 100% oxygen; however, it is often slightly less than this in practice.

Venturi masks

The Venturi mask works on Bernoulli's principle, which states that as the velocity of gas increases the pressure surrounding that gas decreases. Oxygen is introduced through a tapered inlet into the device. As the oxygen flows through the narrowed inlet the velocity increases and a resultant decrease in pressure surrounding the stream of gas causes room air to be entrained into the device through side ports in the device. The concentration of oxygen delivered with these devices remains relatively constant. These masks can deliver 24–40% oxygen.

Nasal cannulae

Nasal cannulae consist of two protruding prongs that are placed into the child's nares. The delivered concentration of oxygen depends on the flow rate as well as the child's minute ventilation and the volume of the nasopharynx as these determine the amount of entrained room air. Generally, children accept flow rates of up to 2 litres per minute; flow rates in excess of this are uncomfortable and poorly tolerated. Correctly fitted and at appropriate flow rates, nasal cannulae are often better tolerated than face masks in most children, but they are less suitable when oxygen needs are high.

Oxygen hood, tent, and head box

Oxygen hoods, tents, or head boxes are clear plastic systems that enclose either the head, upper body, or entire body. The child breathes fresh gas supplied into the enclosure. The concentration within the enclosure can be monitored by using a gas analyser. High oxygen delivery is difficult to maintain with this system because gas is lost through leakage; this system may thus be most suitable for small infants. If this system is used, a minimum fresh gas flow of 2–3 l/kg per minute should be used to prevent carbon dioxide retention.

Bag-mask ventilation

Some children require positive pressure ventilation, either to overcome a degree of upper airway obstruction or to provide breathing support. Effective bag-mask ventilation requires a good seal between the mask and face to provide adequate inflation pressures as well as the ability to compress the gas-containing bag in a coordinated manner, which is sometimes better achieved by two health care providers. It is often necessary to gently move the child's head and neck to determine the optimum position to provide effective ventilation. Excessive flexion or extension of the head and neck should be avoided, however, as this often results in airway obstruction. As mentioned previously, for all children who have a potential cervical spine injury, the spine should be adequately immobilised and unnecessary manipulation should be avoided.

The two bags commonly used in bag-mask valve ventilation include the self-inflating bag and the standard anaesthetic circuit. The self-inflating bag consists of a bag, oxygen inlet, connector for the face mask or tracheal tube, pressure relief valve, and a reservoir. The self-inflating bag is relatively easy to use and more available, and when used with the reservoir, it can provide near 100% oxygen. It can provide emergency ventilation without a fresh gas source because the gas movement generated by bag inflation will inflate the chest with room air, even without an external gas source. Because the valve mechanism opens only in response to manual bag inflation, the self-inflating bag is not appropriate to deliver oxygen or continuous positive pressure in the spontaneously breathing child. The bag in a standard anaesthetic circuit, however, requires a constant supply of fresh gas in order for it to fill. The bag must therefore be connected to a fresh gas supply to inflate the lungs via either a face mask or tracheal tube. The advantage of the standard bag over the self-inflating bag is the ability to deliver fresh gas and continuous positive pressure to the spontaneously breathing child and to control the pressures administered with each breath. The system can be difficult to use, however, in all but experienced anaesthetic hands.

Mechanical ventilation

Mechanical ventilation provides a way of supporting the respiratory system while waiting for the natural history of the pathological process to improve or for specific treatment to be effective. The goals of mechanical ventilation are to ensure adequate oxygen delivery, decrease the work of breathing, and ensure adequate elimination of carbon dioxide.[24] Mechanical ventilation is generally provided by using positive pressure-ventilated breaths superimposed on a background of positive end expiratory pressure (PEEP) to maintain alveolar patency during expiration. PEEP can stabilise alveoli, decrease ventilation-to-perfusion (V:Q) mismatch, and reduce the alveolar shear injury incurred through repetitive inflation with positive pressure—the so-called "ventilator-induced lung injury".[25] Excessive PEEP, however, may result in overdistention of recruited alveoli and have a negative impact on cardiac output.

The goals of mechanical ventilation are to optimise alveolar ventilation, maximise V:Q matching, decrease the work of breathing, and minimise the risk of ventilator-associated injury.

The usual starting point when calculating the initial inflation pressure requirement is a simple visual assessment of the amount of pressure required to move the chest. Adjustments can then be made according to the clinical situation and oxygen requirement, and, if available, as dictated by blood gas analysis. If the bedside ventilator system enables tidal volume calculation, the ventilator pressure should be adjusted with the aim of delivering a tidal volume of 5–10 ml/kg. This will vary with chest compliance, but it often requires peak inspiratory pressures in the range of 20–25 cm H_2O, with a PEEP of 3–5 cm H_2O.[26] Higher PEEP may be required to achieve adequate oxygenation when extensive airspace disease or pulmonary oedema is present. Wherever possible, avoid excessive inflation pressures to avoid ventilator-associated lung disease, which has been associated with high inspiratory pressures.[27] Tidal volumes of 6 ml/kg and limiting the peak inspiratory pressure to less than 32 cm H_2O has demonstrated a significant reduction in mortality.[27] In setting the rate, both the inspiratory and expiratory times—that is, the proportion of the respiratory cycle occupied by inspiration and expiration, respectively—can be adjusted. The inspiratory time is usually decided based on the age, size, and disease process of the patient. As a guide, inspiratory times increase from approximately 0.5 seconds in a neonate to 1 second in children older than 5 years of age. A useful starting point is to simply start with a ventilator rate of 20 breaths per minute and adjust as necessary. Small infants and neonates may require a higher starting rate, in the range of 40–60 breaths per minute.

When making adjustments to the mode and frequency of mechanical ventilation, effective oxygenation is determined by manipulations of inspired oxygen (FiO_2) and mean airway pressure (MAP). The factors that most reliably influence MAP are the amount of set PEEP and the inspiratory time. To optimise ventilation and thus carbon dioxide clearance, minute volume should be increased by increasing the peak inspiratory pressure as well as the ventilator rate. Note, however, that changes in mechanical ventilation to achieve improvements in oxygenation and ventilation have an impact on each other as well as on other organ systems, most notably the cardiovascular system. Increasing mean airway pressures, for example, may potentially impede venous return and thus negatively affect cardiac output. With any adjustment, the clinician should establish whether a positive change has been effected with the fewest possible negative clinical consequences.

The inability to achieve effective oxygenation and ventilation by using conventional positive pressure ventilation may suggest the need for nonconventional modalities of ventilator support, such as high-frequency oscillatory ventilation, high-frequency jet ventilation, or extracorporeal membrane oxygenation (ECMO). Generally, these therapies are available mainly as rescue therapy in intensive care centres.

Circulation

Many children with severe disease require cardiovascular monitoring and support. Circulatory compromise frequently accompanies critical illness and may be either a primary cause or be secondary to the presence of untreated respiratory failure and hypoxia. Early recognition and intervention is therefore essential to prevent further progression to circulatory collapse and death. When the cardiovascular system is unable to provide adequate perfusion of end organs to supply adequate oxygen and nutrients to cells, the situation is referred to as shock. Table 12.2 shows a common scheme for the classification of shock.

Table 12.2: Classification of shock.

Shock classification	Aetiology
Hypovolaemic	Haemorrhage Diarrhoea and vomiting Burns Peritonitis
Distributive	Sepsis Anaphylaxis Vasodilating drugs Spinal cord injuries
Cardiogenic	Arrhythmias Cardiomyopathy Myocardial infarction or contusion Congenital structural heart disease Cardiac tamponade
Obstructive	Tension haemo/pneumothorax Flail chest Pulmonary embolism
Dissociative	Anaemia Carbon monoxide poisoning Methaemoglobinaemia

In evaluating a child with signs of shock, the earliest and most sensitive—but not exclusively reliable—sign is tachycardia. This may also be caused by pain, anxiety, fever, or medications, but these causes are often easily excluded. The presence of additional significant signs consistent with inadequate blood supply to end organs, such as altered mental state, poor peripheral skin perfusion due to vasoconstriction, thready rapid pulses that may be difficult to palpate, and decreased urine output as a result of poor organ perfusion, help to establish a diagnosis of shock. Vasodilatation as a sign of shock is less common in children as compared to adults.[28] Metabolic acidosis commonly accompanies suboptimal perfusion due to tissue anaerobic metabolism,

and the tachypnoea that results may be a useful clinical marker.[29]

It is important to remember that children have robust compensatory mechanisms, and blood pressure may be preserved even in moderate circulatory insufficiency. A fall in blood pressure is a late ominous sign and defines decompensated shock. Normal cardiovascular parameters in children are given in Table 12.3.

Table 12.3: Normal paediatric cardiovascular parameters.

Age (years)	Heart rate (beats per minute)	Mean blood pressure (mm Hg)
Neonate	100–180	40–60
1	100–200	50–100
2	80–160	50–100
5	80–150	60–90
10	60–120	60–90
15	50–120	65–95

Cardiac output depends on the following factors:

- *Preload:* Affected by circulating blood volume and effective delivery to the heart
- *Afterload:* Systolic blood pressure and vascular tone
- *Inotropic state:* Cardiac contractility
- *Chronotropy:* Heart rate as well as rhythm

In supporting circulation, the goal is to optimise cardiac output. The most common clinical situation resulting in shock is hypovolaemia secondary to dehydration or blood loss.

In surgical pre- and postoperative patients, hypovolaemia may be exacerbated by distributive shock due to losses into third spaces as a result of increased capillary permeability. It is therefore of the utmost importance to restore adequate intravascular volume promptly by using isotonic crystalloid or colloid. If there has been significant haemorrhage, packed red blood cells should be considered. It is essential to rule out a cardiogenic cause of shock to avoid excessive fluid administration and overloading a compromised heart. This is usually distinguishable by clinical examination to rule out signs of heart failure, such as cardiomegaly or hepatomegaly, as well as with electrocardiogram (ECG) monitoring and chest x-ray, which may demonstrate congestive heart failure or pulmonary oedema and an increased heart shadow. In children, distention of neck veins is a less reliable sign.

Fluid resuscitation in an attempt to restore intravascular volume is best initiated by using boluses of 20 ml/kg crystalloid or colloid solutions, titrated to clinical markers of cardiac output (heart rate, urine output, capillary refill, and level of consciousness). Numerous clinical trials have attempted to determine the optimal fluid in paediatric resuscitation. The choice of fluid is controversial, but because no clear benefit has been demonstrated for crystalloid over colloid, the choice must be dictated by availability as well as local policy in the acute situation.[30–34] The femoral vein is frequently used to provide central venous access, enabling administration of vasoactive drugs, parenteral nutrition and drugs in higher concentration than would be tolerated in a peripheral venous cannula.

Large fluid deficits typically exist in sepsis, and initial volume resuscitation usually requires 40–60 ml/kg, but may be as much as 200 ml/kg. It is important to note that at the same time as providing ongoing fluid resuscitation, respiratory support in the form of intubation and ventilation may be required. Airway and respiratory support, if needed, should be initiated simultaneously, and neither should delay the other. In patients with suspected or known cardiomyopathy, clinicians must be cautious with aggressive fluid resuscitation because overdistention of a poorly functioning myocardium is likely to cause the patient to deteriorate. In these patients, it is often advisable to give smaller fluid boluses and assess their effects on an ongoing basis.

Patients with poor physiological reserve as well as those who do not respond to fluid resuscitation may benefit from invasive haemodynamic monitoring to more accurately titrate their fluid and inotropic therapies.[28,29,35–44]

In evaluating the cardiovascular system, it is important to confirm sinus rhythm on an ECG by establishing that every QRS complex is preceded by a P wave and that every P wave is followed by a QRS complex. More definitive assessment with cardiac ultrasound is helpful but not often available in the acute situation. Arrhythmias are uncommon in critically ill children without structural heart disease, and sinus tachycardia is commonly present. In the presence of tachycardia that persists when measures such as fever, hypovolaemia, anxiety, and pain are controlled. Tachyarrhythmias such as supraventricular tachycardia (SVT), atrial fibrillation, and ventricular tachycardia must be considered.[45]

When measures such as fluid therapy and maintaining sinus rhythm do not produce an adequate cardiac output, the circulation may be supported further by using vasoactive drugs to increase cardiac inotropy and chronotropy and/or vasoconstrict the peripheral vascular system. Invasive pressure monitoring of central venous or arterial pressure, where available, may provide cardiovascular information to further guide volume and pharmacological support. First-line vasoactive drugs commonly used in the intensive care environment include dopamine and adrenaline, both of which increase heart rate and contractility as well as increasing systemic vascular resistance to varying degrees that generally favour increased cardiac output. Noradrenaline is primarily a vasoconstrictor, and its use is limited to the less common situations in which vasodilatation is present, such as in the older child with sepsis or following cardiac surgery when an increase in systemic vascular resistance is desirable. Milrinone, a phosphodiesterase inhibitor, is increasingly used for inotropic support, especially following cardiac surgery when diastolic function in addition to systolic function may be impaired. Most patients will respond to a simple approach using a single inotropic agent. Detailed and repeated clinical examination, haemodynamic monitoring and—if available—cardiac imaging enables vasoactive support with one or more agents tailored according to the patient's individual requirements.[46–49]

Specific Organ System Dysfunction

Neurology

Acute neurological abnormality may result from a primary disorder of the nervous system or from the consequences of severe illness in other organ systems. Children with respiratory or cardiovascular compromise are often hypoxic with varying degrees of impairment of their levels of consciousness. This may range from irritability and lethargy to seizures or coma. Seriously ill children are often hypotonic but may exhibit more worrying signs of abnormal posturing and eventually respiratory arrest. Primary neurological disease, such as acute meningitis or encephalitis, trauma, a mass lesion, or intoxication may present with a similar spectrum of nonspecific abnormalities. In the early stages of assessment, it is important to stabilise airway, breathing, and circulation, as previously noted, to ensure optimal oxygenation of the brain, regardless of aetiology.

Causes of decreased level of consciousness include hypoxia; hypotension or shock; infection; metabolic disturbances (e.g., hypoglycaemia); toxins or drugs; trauma, especially head injury, including nonaccidental injury; and intracerebral haemorrhage.

Once oxygenation and appropriate circulation are restored, the diagnosis may be clarified with a more detailed history and physical examination to elicit specific signs. Radiological imaging, such as computed tomography

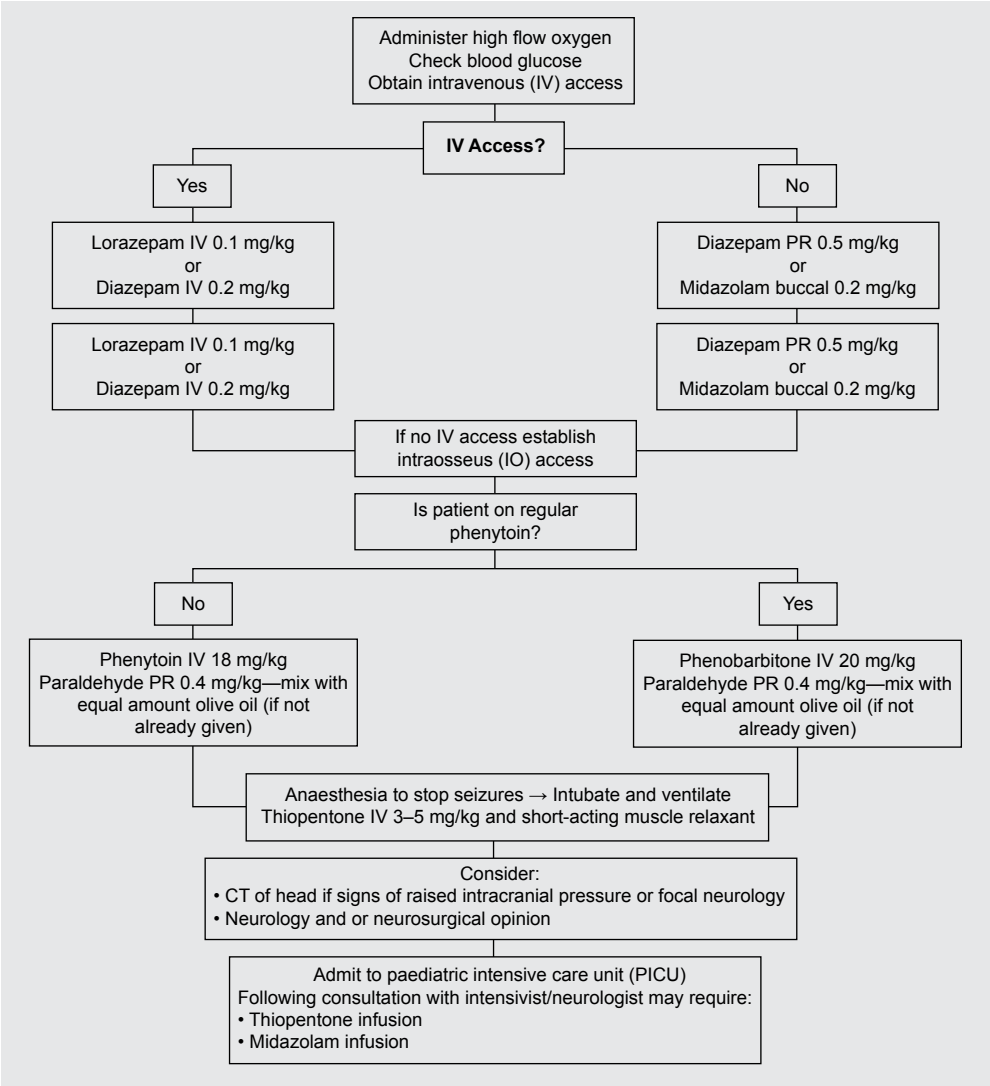

Figure 12.1: Management of status epilepticus in a child.

(CT) of the head, may be required. The diagnosis of central nervous system (CNS) infection generally requires lumbar puncture to assess cell count, protein, and glucose content.

Lumbar puncture should be performed in a controlled manner, provided there are no contraindications,[50] such as focal neurological signs, raised intracranial pressure (ICP), cardiovascular instability, coagulopathy, local skin infection over the proposed puncture site, suspected spinal cord mass or intracranial mass lesion, or spinal column deformities.

Always defer lumbar puncture in unstable patients, but never delay antibiotic or antiviral treatment, if indicated.

The use of hypotonic fluid is contraindicated when signs of raised intracranial pressure are evident. Hypotonic fluid should generally be avoided in all children with acute neurological disease. This is due to the risk of sudden fluid shifts resulting from changes in plasma osmolality and the potential for rapid increases in intracranial pressure.[51] Not enough data exist to support routine fluid restriction.[52–53] In the setting of acute neurological deterioration with signs of rising intracranial pressure and possible brain stem herniation, the use of hypertonic saline or intravenous mannitol may be warranted.[54]

Seizures are a common feature of both primary and secondary CNS abnormality. It is important to address the underlying cause, which mirrors the differential diagnosis of decreased level of consciousness, and establish oxygenation and circulation as detailed above. If appropriate, specific therapy should be instituted as soon as possible (e.g., the treatment of hypoglycaemia with a dextrose infusion of 5–10 ml/kg of 10% dextrose solution). The treatment for a prolonged seizure or recurrent seizures is indicated in Figure 12.1.[55–57]

Although the paediatric CNS may be relatively immature, children do still have appropriate response to pain. It is therefore important to remember that critically ill children who require invasive diagnostic or therapeutic procedures, or intubation and mechanical ventilation, require appropriate analgesia and sedation. In addition to the obvious benefits of analgesia and sedation, there is evidence to support decreased morbidity and mortality following cardiac surgery in infants treated with effective analgesia.[58–60] In the intensive-care setting, opioids, such as morphine and fentanyl, are commonly used for analgesia, whereas sedation is commonly achieved with benzodiazepines, such as midazolam or diazepam.

Infection

Bacterial, viral, fungal, and protozoal infections are responsible for more than 60% of deaths worldwide. Acute respiratory infections are among the leading cause of childhood mortality and account for almost two million childhood deaths annually. Approximately 70% of these deaths occur in Africa. The most important causes of death in the developing world continue to be malaria, pneumonia, malnutrition, and HIV-related illnesses.[61–63]

In evaluating a child with severe infection, the priority remains to ensure adequate airway, breathing, and circulation. If severe shock is present in the setting of infection, it is important to aim to reverse the shock with aggressive fluid resuscitation and inotropic support, if required, with the ultimate goal of achieving age-appropriate heart rates, normal blood pressure, and capillary refill time.[64] As in the treatment of hypovolaemia, boluses of crystalloid or colloid totalling up to 200 ml/kg may be required, and inotropic support should be considered early in patients unresponsive to two or three boluses of 20 ml/kg. Specific infections may dictate variations in management after initial stability is established. The site of infection as well as likely pathogens will guide the choice of antimicrobial therapy.

Postoperative Care

Following major elective or emergency surgery, the child probably will be haemodynamically unstable and therefore require support for one or more organ systems. Other factors that influence this instability include the age, underlying nutritional status, and premorbid health of the patient; the length of the operation; intraoperative cardiovascular status; and fluid losses. Intensive care may be required from the immediate postoperative period, but delayed deterioration must always be anticipated in high-risk cases and these patients should be monitored accordingly.

In caring for high-risk patients, the degree of support will be dictated by the specific set of clinical circumstances as well as the local resources. Attention must be paid to ensure that adequate oxygen is delivered to the recovering patient. This involves also ensuring adequate ventilation and circulation and appropriate monitoring of the vital signs of the patient. Fluid balance is important in the perioperative period, and careful attention to and monitoring of intake and output are required. Many abdominal surgical procedures result in patients requiring a prolonged period of nutritional support; if available, total parenteral nutrition should be considered in patients who are unable to adequately meet their nutritional demands enterally. Other challenges include the need for total immobility following complex reconstructive surgery, such as repair of an oesophageal atresia. These patients will require intubation and ventilation over several days in order to maintain adequate sedation, analgesia, and muscle relaxation. Finally, it is important that each child be regularly and adequately assessed for pain and distress so that appropriate analgesia and/or sedation may be administered.

Trauma

Traumatic injuries are a cause of significant childhood morbidity and mortality worldwide. Children are particularly vulnerable to traumatic brain as well as major organ injury due to their relatively immature and elastic skeleton and their inability to withstand large blunt forces.[65–67] It is paramount that the initial resuscitation and stabilisation of a critically injured child be both methodical and thorough in order to recognise and institute appropriate therapy in a timely manner. In children who die immediately or soon after an injury, the predominant causes of death are airway compromise resulting in hypoxia, hypovolaemic shock, and injury to the brain and cervical spine. Children are extremely vulnerable to cerebral hypoxia; it is therefore understandable that airway management and attention to respiratory function are the most critically important aspects of resuscitation and ongoing management of the seriously injured child.[10] Attention to these areas of care enable optimal oxygenation and ventilation and minimise secondary injury to the brain and other vital organs. The cervical spine should be immobilised by using either manual in-line stabilisation or on a firm surface with the neck placed in a hard collar of the appropriate size, with lateral support using firm blocks and straps. These cervical spine precautions should remain until such injuries have been excluded clinically, radiologically, or both. With attention to the cervical spine, the airway may be secured by simple airway manoeuvres, or require more specialised equipment or intervention in the form of oral endotracheal intubation.[68]

Thoracic injury may be potentially life-threatening, requiring prompt recognition and treatment.[69] The adequacy of ventilation should be assessed clinically by looking at respiratory rate, pattern, and symmetry of chest movement and breath sounds on auscultation. Adequate oxygenation can be confirmed if the patient appears pink or more definitively with pulse oximetry. Once the airway is definitely secure, if ventilation and oxygenation continue to be inadequate, lung injury should be suspected. Pulmonary contusion may result in respiratory distress and may be confirmed radiologically, although it may be difficult to distinguish from acute aspiration or atelectasis. The presence of unilaterally decreased breath sounds, hypoxia, and cardiovascular compromise should raise the suspicion of pneumothorax or haemothorax. These conditions should be treated urgently by needle or drain thoracocentesis. Mediastinal injuries with the potential for large vessel or major airway trauma are surgical emergencies and require prompt imaging and surgical intervention.[70]

Children are capable of losing up to 40% of their intravascular volume while maintaining a relatively normal blood pressure. The signs and symptoms of early shock due to blood loss may be subtle; it is therefore important to begin fluid resuscitation early, before a seriously injured child becomes haemodynamically unstable. Direct pressure should be applied to any visible external bleeding site in an attempt to stop the bleeding. Secure intravenous access should be sought; common veins used include antecubital, saphenous, and femoral. In the absence of obvious lower limb fracture, an intraosseous (IO) needle into the anterior tibia is a reliable and potentially life-saving form of intravenous (IV) access. Initial fluid resuscitation should consist of two 10-ml/kg boluses of isotonic crystalloid solution, which may be followed by a further two 10-ml/kg boluses of fluid. If signs of shock with evidence of ongoing blood loss persist, consider the use of either O-negative or type-specific packed red blood cells for further fluid boluses.

Once the child is physiologically stable, perform a complete systemic examination. This would normally include various radiological investigations of the cervical spine, chest, and pelvis, and, if intrabdominal injury is suspected, ultrasound or CT. The specific investigations are guided by the clinical scenario and availability of specialised imaging. Many intrathoracic and intraabdominal injuries may be managed conservatively; however, some surgical intervention may be required. Bowel injury may present in a delayed fashion, requiring surgery some time after the initial injury. Detailed surgical management of specific injuries is beyond the scope of this chapter, and is addressed in relevant chapters of this book.

Challenges of Paediatric Critical Care

The biggest challenges for health care providers in the developing world include improving primary care and public health efforts, such as immunisation and sanitation, as well as ensuring universal access to health care.[71]

The provision of critical care and the limited means available to run a dedicated critical care environment suitable for children from infancy to adolescence present an ethical dilemma when multiple health care demands coexist. In an attempt to address such dilemmas, certain units have developed intensive care admission criteria that would not exist in the developed world. Examples of these criteria include refusing admission to an augmented or critical care environment in patients known to be infected with human immunodeficiency virus (HIV) as well as refusing admission to a critical care environment in patients who present acutely to a health care centre and would require transfer to the regional paediatric intensive care unit (PICU). This is particularly evident in sub-Saharan Africa, where there is a huge burden on health as a result of the HIV epidemic.[61–62]

Approximately two million children worldwide have HIV; of these, 90% live in sub-Saharan Africa. Although the rate of annual HIV-related deaths in children is gradually decreasing, approximately

300,000 children continue to die of AIDS-related illnesses worldwide every year.[63]

Paediatric critical care in developing countries is necessarily highly centralised. Most developing countries have large centres that provide varying degrees of tertiary medical care, including paediatric critical care. However, the resources available to these tertiary centres are extremely inconsistent and unpredictable; as a result, the medical capabilities differ widely across centres and countries.[72]

In an attempt to provide critical care for children, hospitals must use existing resources to the best of their ability, including, for example, using existing theatre facilities as an environment to care for critically ill children in the short term or combining adult, paediatric, and neonatal critical care.

In view of the limited availability of paediatric critical care in developing countries, it is important to define priorities and recognise those children who might benefit from being transferred and admitted to a dedicated unit. The additional costs incurred by safe and effective transport of the most vulnerable paediatric patients are a major consideration in the decision-making process and undoubtedly will influence the allocation of limited resources. Determining those admission criteria will depend on local as well as wider factors within a defined geographical area.

Unfortunately, data available on the provision of paediatric critical care in the developing world are lacking. Only the most sophisticated and developed units with dedicated resources for expensive diagnostic and therapeutic drugs and equipment tend to publish data, thereby creating a publication bias.[1] As a result, published data may not reflect the true spectrum of the clinical workload.

Conclusion

Many complex factors affect the ability to provide dedicated paediatric critical care units. Much of the modern infrastructure may be out of reach to units due to cost. Caring for paediatric patients during acute critical illness or injury as well as following major surgery can be both challenging and rewarding. Attention and priority should be at maintaining a secure airway, followed by providing adequate respiratory and cardiovascular support. Detailed clinical history, examination, and, where possible, further investigations will provide clearer diagnostic information in the aim of providing definitive care. Paediatric critical care not only provides the management of children with severe medical or surgical illness, but frequently goes beyond cure to encompass holistic care of the patient and family.

Evidence-Based Research

Tables 12.4 and 12.5 present evidence-based reviews of ventilation strategies and sepsis management, respectively.

Table 12.4: Evidence-based research.

Title	Review of paediatric intensive care ventilation practice
Authors	Turner DA, Arnold JH
Institution	Harvard Medical School and Department of Anesthesia, Division of Critical Care Medicine, Children's Hospital, Boston, Massachusetts, USA
Reference	Curr Opin Crit Care 2007; 13(1):57–63
Problem	Review current paediatric ventilation strategies and evidence.
Outcome/ effect	Mechanical ventilation with pressure limitation by using low tidal volumes has become the main form of ventilation in paediatric intensive care units.
Historical significance/ comments	Various ventilator strategies such as high-frequency oscillatory ventilation, airway pressure release ventilation, and adjuncts such as surfactant, need further evaluation.

Table 12.5: Evidence-based research.

Title	Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update from the American College of Critical Care Medicine
Authors	Brierley J, Carcillo JA, Choong K, Cornell T, DeCaen A, Deymann A, et al.
Institution	American College of Critical Care Medicine, Mount Prospect, Illinois, USA
Reference	Crit Care Med 2009; 37(2):666–688
Problem	Clinical guidelines required to promote best practices and improve patient outcomes in paediatric and neonatal septic shock.
Intervention	Extensive literature search with experts in field grading evidence.
Comparison/ control (quality of evidence)	Compares centres that implemented previous guidelines.
Outcome/ effect	Early use of paediatric and neonatal sepsis guidelines was associated with improved outcome.
Historical significance/ comments	Continue to support the early use of age-specific therapies to attain time-sensitive goals. Compared to adults, children require proportionally larger quantities of fluid in resuscitation for sepsis. Early use of inotropic support is recommended.

Key Summary Points

1. Respiratory failure is a major cause of paediatric morbidity and mortality worldwide, and early intervention is essential to prevent progression to cardiopulmonary arrest.
2. Oxygen should be administered to all critically ill or injured children in the highest possible concentration until the assessment of cardiorespiratory status is complete.
3. International consensus guidelines on the management of paediatric and neonatal septic shock provide a clear treatment framework, demonstrating that aggressive fluid management of hypovolemic and septic shock has a positive impact on outcome.
4. Whenever possible, efforts should be made to minimise inflation pressures in positive pressure ventilation to protect the patient from lung injury.
5. The degree of intensive care support is dictated by the specific set of clinical circumstances as well as the local resources.

References

1. Wheeler DS, Wong HR, Shanley TP. Pediatric Critical Care Medicine. Basic Science and Clinical Evidence. Springer-Verlag London Limited, 2007.
2. Macnab A, Macrae D, Henning R. Care of the Critically Ill Child, 1st ed. Churchill Livingstone, 15 September 1999.
3. Hickey RW, Cohen DM, Strausbaugh S, Dietrich AM. Pediatric patients requiring CPR in the prehospital setting. Ann Emerg Med 1995; 25:495–501.
4. Innes PA, Summers CA, Boyd IM, Molyneaux EM. Audit of pediatric cardiopulmonary resuscitation. Arch Dis Child 1993; 68:487–491.
5. Thompson JE, Bonner B, Lower GM. Pediatric cardiopulmonary arrests in rural populations. Pediatrics 1990; 86:302–306.
6. Teach SJ, Moore PE, Fleischer GR. Death and resuscitation in the pediatric emergency department. Ann Emerg Med 1995; 25:799–803.
7. Peters MJ, Tasker RC, Kiff KM, Yates R, Hatch DJ. Acute hypoxic respiratory failure in children: case mix and utility of respiratory indices. Intensive Care Med 1998; 24:699–705.
8. Sirbaugh PE, Pepe PE, Shook JE, et al. A prospective, population-base study of the demographics, epidemiology, management, and outcome of out-of-hospital pediatric cardiopulmonary arrest. Ann Emerg Med 1999; 33:174–184.
9. Young KD, Seidel JS. Pediatric cardiopulmonary resuscitation: a collective review. Ann Emerg Med 1999; 33:195–205.
10. Advanced Life Support Group (ALSG). Advanced Paediatric Life Support. The Practical Approach, 4th ed. BMJ Books, 4 February 2005.
11. Brown OE. Structure and function of the upper airway. In: Westmore RF, Muntz HR, McGill TJI, eds. Pediatric Otolaryngology. Principles and Practice Pathways. Thieme Publishers; 2000: 679–688.
12. Eckenhoff J. Some anatomic considerations of the infant larynx influencing endotracheal anesthesia. Anesthesiology 1951; 12:401–410.
13. Cote CJ, Ryan JF, Todres ID, Groudsouzian NG, eds. A Practice of Anesthesia for Infants and Children, 2nd ed. WB Saunders, 1993.
14. McNiece WL, Dierdorf SF. The pediatric airway. Semin Pediatr Surg 2004; 13:152–165.
15. Dickinson AE. The normal and abnormal pediatric upper airway. Recognition and management of obstruction. Clin Chest Med 1987; 8:583–596.
16. Rotta AT, Wiryawan B. Respiratory emergencies in children. Respir Care 2003; 48(3):248–258; discussion 258–260.
17. Flori HR, Glidden DV, Rutherford GW, Matthay MA. Pediatric acute lung injury: prospective evaluation of risk factors associated with mortality. Am J Respir Crit Care Med 2005; 171(9):995–1001. Epub 23 December 2004.
18. DeBruin W, Notterman D, Magid M, Godwin T, Johnston S. Acute hypoxic respiratory failure in infants and children: clinical and pathological characteristics. Crit Care Med 1992; 24:699–705.
19. Timmons OD, Havens PL, Fackler JC. Predicting death in pediatric patients with acute respiratory failure. Pediatric Critical Care Study Group, Extracorporeal Life Support Organization. Chest 1995; 108(3):789–797.
20. PICANet National Report 2006–2008. Available at: http://www.picanet.org.uk/Documents/General/Annual 20report_2009/PICANet 20National 20Report 202006 20-202008_new.pdf.
21. Hoyert DL, Kung H-C, Smith BL. Deaths: preliminary data for 2003. Natl Vital Statistics Rep 2005; 53(15):1–48.
22. Taussig LM, Landau LI. Pediatric Respiratory Medicine. Mosby, 1999.
23. Zaritsky A. Cardiopulmonary resuscitation in children. Clin Chest Med 1987; 8:561–571.
24. Wheeler DS, Wong HR, Shanley TP. Pediatric Critical Care Medicine. Basic Science and Clinical Evidence. Springer-Verlag London Limited, 2007, Pp 299 –307.
25. Gattinoni L, Carlesso E, Cadringher P, Valenza F, Vagginelli F, Chiumello D. Physical and biological triggers of ventilator-induced lung injury and its prevention. Eur Respir J Suppl 2003; 47:15s–25s.
26. Tobin MJ. Principles and Practice of Mechanical Ventilation. McGraw-Hill, 1994.
27. Network TA. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med 2000; 342:1301–1308.
28. Ceneviva G, Paschall JA, Maffei F, Carcillo JA. Hemodynamic support in fluid-refractory pediatric septic shock. Pediatrics 1998; 102(2):e19.
29. Carcillo JA, Pollack MM, Ruttimann UE, Fields AI. Sequential physiologic interactions in pediatric cardiogenic and septic shock. Crit Care Med 1989; 17(1):12–16.
30. Cochrane Injuries Group Albumin Reviewers. Human albumin administration in critically ill patients: systematic review of randomised controlled trials. BMJ 1998; 317(7153):235–240.
31. Finfer S, Bellomo R, Boyce N, French J, Myburgh J, Norton R. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. N Engl J Med 2004; 350(22):2247–2256.
32. Upadhyay M, Singhi S, Murlidharan J, Kaur N, Majumdar S. Randomized evaluation of fluid resuscitation with crystalloid (saline) and colloid (polymer from degraded gelatin in saline) in pediatric septic shock. Indian Pediatr 2005; 42(3):223–231.
33. Schroth M, Plank C, Meissner U, Eberle KP, Weyand M, Cesnjevar R, et al. Hypertonic-hyperoncotic solutions improve cardiac function in children after open-heart surgery. Pediatrics 2006; 118(1):e76–e84.
34. Ngo NT, Cao XT, Kneen R, Wills B, Nguyen VM, Nguyen TQ, et al. Acute management of dengue shock syndrome: a randomized double-blind comparison of 4 intravenous fluid regimens in the first hour. Clin Infect Dis 2001; 32(2):204–213.
35. Nhan NT, Phuong CXT, Kneen R, et al. Acute management of dengue shock syndrome: A randomized double-blind comparison of 4 intravenous fluid regimens in the first hour. Clin Infect Dis 2001; 32:204–212.
36. Carcillo JA, Davis AI, Zaritsky A. Role of early fluid resuscitation in pediatric septic shock. JAMA 1991; 266:1242–1245.
37. Lee PK, Deringer JR, Kreiswirth BN, et al. Fluid replacement protection of rabbits challenged subcutaneous with toxic shock syndrome toxins. Infect Immun 1991; 59:879–884.
38. Ottoson J, Dawidson I, Brandberg A, et al. Cardiac output and organ blood flow in experimental septic shock and treatment with antibiotics, corticosteroids, and fluid infusion. Circ Shock 1991; 35:14–24.
39. Wilson MA, Choe MC, Spain DA. Fluid resuscitation attenuates early cytokine mRNA expression after peritonitis. J Trauma 1996; 41:622–627.
40. Boldt J, Muller M, Heesen M. Influence of different volume therapies and entoxifylline infusion on circulating adhesion molecules in critically ill patients. Crit Care Med 1998; 24:385–391.
41. Zadrobilek E, Hackl W, Sporn P, et al. Effect of large volume replacement with balanced electrolyte solutions on extravascular lung water in surgical patients with sepsis syndrome. Intensive Care Med 1989; 15:505–510.

42. Carcillo JA, Tasker RC. Fluid resuscitation of hypovolemic shock: acute medicine's great triumph for children. Intensive Care Med 2006; 32(7):958–961.
43. Pladys P, Wodey E, Betremieux P. Effects of volume expansion on cardiac output in the preterm infant. Acta Paediatr 1997; 86:1241–1245.
44. Ranjit S, Kissoon N, Jayakumar I. Aggressive management of dengue shock syndrome may decrease mortality rate: a suggested protocol. Pediatr Crit Care Med 2005; 6(4):412–419.
45. Reinelt P, Karth GD, Geppert A, Heinz G. Incidence and type of cardiac arrhythmias in critically ill patients: a single center experience in a medical-cardiological ICU. Intensive Care Med 2001; 27(9):1466–1473.
46. Coffin LH Jr, Ankeney JL, Beheler EM. Experimental study and clinical use of epinephrine for treatment of low cardiac output syndrome. Circulation 1966; 33(4 Suppl):I78–I85.
47. Richard C, Ricome JL, Rimailho A, Bottineau G, Auzepy P. Combined hemodynamic effects of dopamine and dobutamine in cardiogenic shock. Circulation 1983; 67(3):620–626.
48. Stopfkuchen H, Racké K, Schwörer H, Queisser-Luft A, Vogel K. Effects of dopamine infusion on plasma catecholamines in preterm and term newborn infants. Eur J Pediatr 1991; 150(7):503–506.
49. Duggal B, Pratap U, Slavik Z, Kaplanova J, Macrae D. Milrinone and low cardiac output following cardiac surgery in infants: is there a direct myocardial effect? Pediatr Cardiol 2005; 26(5):642–645.
50. Ward E, Gushurst CA. Uses and technique of pediatric lumbar puncture. Am J Dis Child 1992; 146(10):1160–1165.
51. Pollock AS, Arieff AI. Abnormalities of cell volume regulation and their functional consequences. Am J Physiol 1980; 239(3):F195–F205.
52. Duke T. Fluid management of bacterial meningitis in developing countries. Arch Dis Child 1998; 79:181–185.
53. Singhi SC, Singhi PD, Srinivas B, et al. Fluid restriction does not improve outcome of acute meningitis. Pediatr Infect Dis J 1995; 14:495–503.
54. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents. Chapter 11, Use of hyperosmolar therapy in the management of severe pediatric traumatic brain injury. Pediatr Crit Care Med 2003; 4(3 Suppl):S40–S44.
55. Morrison G, Gibbons E, Whitehouse WP. High-dose midazolam therapy for refractory status epilepticus in children. Intensive Care Med 2006; 32(12):2070–2076.
56. Qureshi A, Wassmer E, Davies P, Berry K, Whitehouse WP. Comparative audit of intravenous lorazepam and diazepam in the emergency treatment of convulsive status epilepticus in children. Seizure 2002; 11(3):141–144.
57. Appleton R, Choonara I, Martland T, Phillips B, Scott R, Whitehouse W. The treatment of convulsive status epilepticus in children. Members of the Status Epilepticus Working Party. Arch Dis Child 2000; 83(5):415–419.
58. Anand KJ, Hansen DD, Hickey PR. Hormonal-metabolic stress responses in neonates undergoing cardiac surgery. Anesthesiology 1990; 73(4):661–670.
59. Anand KJ, Hickey PR. Halothane-morphine compared with high-dose sufentanil for anesthesia and postoperative analgesia in neonatal cardiac surgery. N Engl J Med 1992; 326(1):1–9.
60. Anand KJ, Hickey PR. Pain and its effects in the human neonate and fetus. N Engl J Med 1987; 317(21):1321–1329.
61. Jeena PM, McNally LM, Stobie M, et al. Challenges in the provision of ICU services to HIV infected children in resource poor settings: a South African case study. J Med Ethics 2005; 31:226–230.
62. Frey B, Argent A. Safe paediatric intensive care. Intensive Care Med 2004; 30:1041–1046.
63. 2008 Report on the global AIDS epidemic. Available at: http://www.unaids.org/en/KnowledgeCentre/HIVData/GlobalReport/2008/2008_Global_report.asp.
64. Brierley J, Carcillo JA, Choong K, et al. Clinical practice parameters for the hemodynamic support of pediatric and neonatal septic shock: 2007 update from the American College of Critical Care Medicine. Crit Care Med 2009; 37(2):666–688.
65. Walker ML, Storrs BB, Mayer T. Factors affecting outcome in the pediatric patient with multiple trauma. Further experience with the modified injury severity scale. Childs Brain 1984; 11(6):387–397.
66. Mayer T, Walker ML, Johnson DG, Matlak ME. Causes of morbidity and mortality in severe pediatric trauma. JAMA 1981; 245(7):719–721.
67. Luerssen TG, Klauber MR, Marshall LF. Outcome from head injury related to patient's age. A longitudinal prospective study of adult and pediatric head injury. J Neurosurg 1988; 68(3):409–416.
68. Chiaretti A, De Benedictis R, Della Corte F, Piastra M, Viola L, Polidori G, Di Rocco C. The impact of initial management on the outcome of children with severe head injury. Childs Nerv Syst 2002; 18(1–2):54–60. Epub 18 December 2001.
69. Sartorelli KH, Vane DW. The diagnosis and management of children with blunt injury of the chest. Semin Pediatr Surg 2003; 13:98–105.
70. American College of Surgeons. Advanced Trauma Life Support, 7th ed. American College of Surgeons, 2002.
71. Sachdeva RC. Intensive care—a cost effective option for developing countries? Indian J Pediatr 2001; 68:339–342.
72. Mathivhal LR. ICU's worldwide: an overview of critical care medicine in South Africa. Crit Care Med 2001; 2:108–112.