Recognition of Respiratory Failure and Shock: Anticipating Cardiopulmonary Arrest

Chapter 1

Cardiopulmonary arrest in infants and children is not usually a sudden event. Instead, it is often the end-result of a progressive deterioration in respiratory and circulatory function, the final common pathway of which is cardiopulmonary failure, regardless of the underlying disease (Figure 1.1). An arrest can often be prevented if the clinician recognizes symptoms of respiratory failure and/or shock and promptly initiates therapy. This chapter presents guidelines for anticipating cardiopulmonary arrest in infants and children and for establishing priorities in care.

Respiratory failure is a clinical state characterized by inadequate elimination of carbon dioxide and/or inadequate oxygenation of the blood. This may occur because of intrinsic lung or airway disease or because of inadequate respiratory effort (e.g., the patient with apnea or shallow respirations from intracranial pathology). *Shock* is a clinical state characterized by inadequate delivery of oxygen and metabolic substrates to meet the metabolic demands of tissues; note that this definition fails to mention blood pressure. Shock may occur with a normal, increased, or most frequently, decreased cardiac output. Shock may be further classified into compensated and decompensated conditions. In compensated shock, blood pressure is normal. Decompensated shock is characterized by hypotension and, often, by a low cardiac output.

As with shock, respiratory failure is often preceded by a "compensated" state in which the patient is able to maintain adequate gas exchange at the expense of an increase in the work of breathing. This compensated state is characterized by respiratory distress, the use of accessory muscles of respiration, inspiratory retractions, tachypnea, and tachycardia.

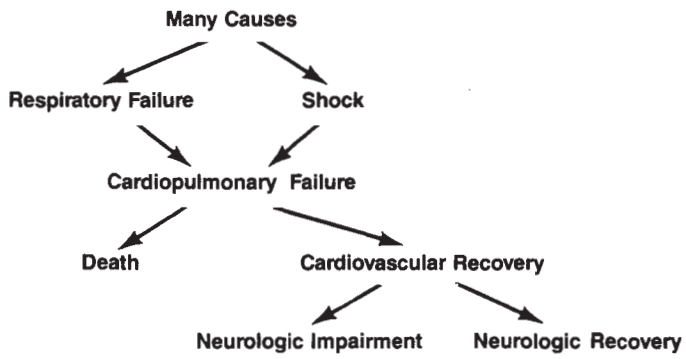

Figure 1.1. Path of various disease states leading to cardiopulmonary failure in infants and children.

Although respiratory failure and shock may begin as clinically distinct syndromes, they progress to an indistinguishable state of cardiopulmonary failure in the final moments before arrest. Their common clinical features are caused by insufficient oxygen delivery to tissues and reduced clearance of metabolites. In shock associated with a low cardiac output state, blood, even though it may be well oxygenated, is delivered too slowly to meet tissue metabolic demand. In advanced respiratory failure, poorly oxygenated blood may be delivered at a normal or elevated flow rate to the tissues. In both cases tissue hypoxia is present. In shock, tissue metabolic demand exceeds oxygen delivery and lactic acid is produced; in respiratory failure, blood carbon dioxide tension increases because pulmonary gas exchange is impaired. Both cases result in acidemia (low blood pH) and tissue acidosis.

Clinical signs of shock and respiratory failure result from end-organ dysfunction caused by tissue hypoxia and acidosis. These include altered levels of consciousness, hypotonia, tachycardia, and weak central (proximal) pulses with absent peripheral pulses. Bradycardia, hypotension, and irregular respirations are late, ominous signs. Cardiopulmonary failure should be suspected in any infant or child who has respiratory distress, severe multiple or blunt trauma, a reduced level of consciousness, cyanosis, or pallor (Table 1.1).

I. Evaluation of Respiratory Performance

Normal ventilation is accomplished with minimal work. The normal respiratory rate decreases with age; it is less

Table 1.1. Selected Conditions Requiring a Rapid Cardiopulmonary Assessment

Any of the following:	
___ Respiratory rate	> 60
___ Heart rate	>180 or <80 (under 5 years)
	>160 (over 5 years)
___ Respiratory distress	
___ Trauma	
___ Burns	
___ Cyanosis	
___ Failure to recognize parents	
___ Diminished level of consciousness	
___ Seizures	
___ Fever with petechiae	
___ Admission to an ICU	

than 40 breaths/min in the newborn, approximately 24 breaths/min in the 1-year-old child, and approximately 18 breaths/min in the 18 year old. These rates may vary with excitement, anxiety, or fever. Tidal volume (the volume of each breath) per kg of body weight, however, remains fairly constant throughout life. Tidal volume is assessed clinically by auscultation over the lungs, noting the quality of air movement and the adequacy of chest wall excursion.

Abnormal respirations are classified as too fast (tachypnea), too slow (bradypnea), absent (apnea), and/or associated with an increased work of breathing. Minute ventilation, the product of tidal volume and respiratory rate, may be low (hypoventilation) because each breath is shallow or because too few breaths are taken each minute. Acute respiratory failure results from any pulmonary or neuromuscular disease that impairs the elimination of carbon dioxide (ventilation) and/or oxygen exchange (oxygenation) at the alveolar–capillary membrane.[1] The resultant hypercapnia and/or hypoxemia reflects the severity of respiratory failure.

Possible respiratory arrest should be anticipated in infants and children who have 1) an increased respiratory rate, an increased respiratory effort, or diminished breath sounds, 2) a diminished level of consciousness or response to pain, 3) poor skeletal muscle tone, or 4) cyanosis.

The assessment of respiratory function is based on careful evaluation of respiratory rate, respiratory mechanics, and skin and mucous membrane color as outlined below.

A. Respiratory Rate

Tachypnea may be the first manifestation of respiratory distress in infants. Tachypnea without respiratory distress ("quiet tachypnea") commonly results from nonpulmonary diseases, especially metabolic acidosis associated with shock, diabetic ketoacidosis, inborn errors of metabolism, salicylism, severe diarrhea, and chronic renal insufficiency. Thus, quiet tachypnea is often an attempt to maintain a normal pH by increasing minute ventilation and thereby cause a compensatory respiratory alkalosis.

A slow respiratory rate in an *acutely ill* infant or child is an ominous sign. Causes include hypothermia, fatigue, and central nervous system depression. Fatigue needs to be especially emphasized. An infant breathing at a rate of 80 breaths/min will likely tire; a decreasing respiratory rate is not necessarily a sign of improvement!

B. Respiratory Mechanics

Increased work of breathing is evidenced by nasal flaring and intercostal, subcostal, and suprasternal inspiratory retractions; it may be seen in children with respiratory problems, including airway obstruction and alveolar diseases. As work of breathing increases, a greater proportion of the cardiac output must be delivered to the respiratory muscles, which in turn produce more carbon dioxide. Respiratory acidosis is followed by metabolic acidosis when the work of breathing exceeds the ability to provide adequate tissue oxygenation.

Head bobbing, stridor, prolonged expiration, and grunting are important signs of altered respiratory mechanics. A bobbing of the head with each breath is often an indication of impending respiratory failure. Extreme inspiratory efforts draw the chest in while thrusting the abdomen out, causing "see–saw" or rocky respirations.

Stridor (an inspiratory, high-pitched sound) is a sign of upper airway obstruction occurring between the supraglottic space and the lower trachea. Causes of upper airway obstruction include 1) congenital abnormalities (e.g., vocal cord paralysis, airway tumor, or cyst), 2) infections (e.g., epiglottitis or croup), and 3) aspiration of a foreign body.

Prolonged expiration, usually accompanied by wheezing, is a sign of bronchial and bronchiolar obstruction and is often caused by bronchiolitis or asthma.

Grunting is produced by premature glottic closure accompanying active chest wall contraction during early expiration. Infants and children grunt to increase airway pressure, thereby preserving or increasing functional residual capacity in diseases that cause accumulation of interstitial or alveolar fluid. Grunting is heard in patients whose disease causes alveolar collapse and loss of lung volume, including patients with pulmonary edema, pneumonia, atelectasis, and adult respiratory distress syndrome.

C. Cyanosis

Cyanosis is a fairly late and inconsistent sign of respiratory failure and is best seen in the mucous membranes of the mouth and nail beds; cyanosis of the extremities alone (peripheral cyanosis) is more likely due to circulatory failure (shock) than to pulmonary failure. Arterial blood gases should be measured whenever a question of serious respiratory impairment exists, even in the absence of cyanosis.

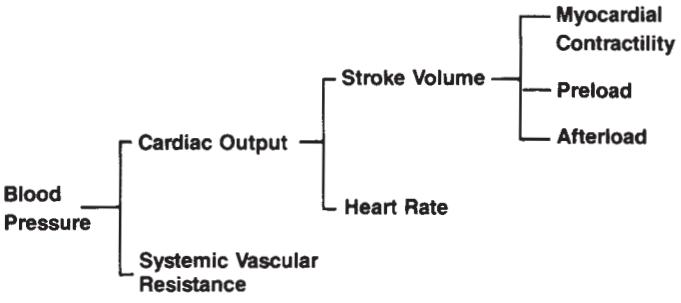

Figure 1.2. Hemodynamic relationships.

II. Evaluation of Cardiovascular Performance

Shock is the failure of the cardiovascular system to adequately perfuse vital organs.[2–4] Failure to deliver critical substrates and remove metabolites leads to anaerobic metabolism, accumulation of acid, and irreversible cellular damage. Death due to cardiovascular collapse may follow shortly; death due to multiple organ system failure resulting from cellular damage may be delayed.

Cardiac output is the volume of blood pumped by the heart each minute (heart rate × stroke volume), while stroke volume is the volume pumped with each beat. Blood pressure is the product of cardiac output and peripheral resistance (Figure 1.2). Organ perfusion is determined by cardiac output and perfusion pressure. Of the variables in Figure 1.2, heart rate and blood pressure can be measured easily; stroke volume and peripheral vascular resistance must be qualitatively assessed by examining pulses and evaluating tissue perfusion.

Although shock is often associated with a low cardiac output, septic and anaphylactic shock may be characterized by an increase in cardiac output and a normal blood pressure.[5] When cardiac output is increased, vascular resistance is low and the patient may appear well perfused, with bounding pulses and a wide pulse pressure. Despite appearing well-perfused, the patient's metabolic demand exceeds the increased oxygen supply, and there is often a mismatch between tissue blood flow and metabolic demand ("distributive shock"). This form of shock may be quite subtle, and a high index of suspicion is required for its recognition. Attention to the assessment signs discussed below and analysis of arterial blood gases should lead to the correct diagnosis.

A. Heart Rate

Normal heart rates in infants and children are given in Chapter 6. Sinus tachycardia (see Chapter 6) is a common response to many types of stress (e.g., anxiety, fever, hypoxia, hypercapnia, or hypovolemia). The presence of tachycardia therefore mandates further evaluation to determine the underlying cause. *Newborns* increase their cardiac output principally by increasing heart rate and, to a lesser extent, by increasing contractility and altering preload and afterload. The primary response of the neonatal heart to hypoxemia often is bradycardia; in older children, tachycardia is the first response. When tachycardia fails to compensate adequately, tissue hypoxia and hypercapnia with acidosis develop and bradycardia ensues. Bradycardia in a distressed child is an ominous sign of impending cardiac arrest.

B. Blood Pressure

As noted previously, blood pressure is determined by cardiac output and peripheral vascular resistance. Normal blood pressure can be maintained as long as the circulation compensates adequately with vasoconstriction, tachycardia, and increased cardiac contractility. When compensation fails, hypotension occurs. Tachycardia persists until cardiac reserve is depleted. Figure 1.3 shows a model for the cardiovascular response to hemorrhagic shock. Although cardiac output falls in an almost linear fashion as blood volume is depleted, blood pressure remains initially unchanged because of increased vascular resistance. Hypotension is a *late and often sudden sign* of cardiovascular decompensation. Even mild hypotension must be taken seriously and treated quickly and vigorously since cardiopulmonary arrest is often close at hand.

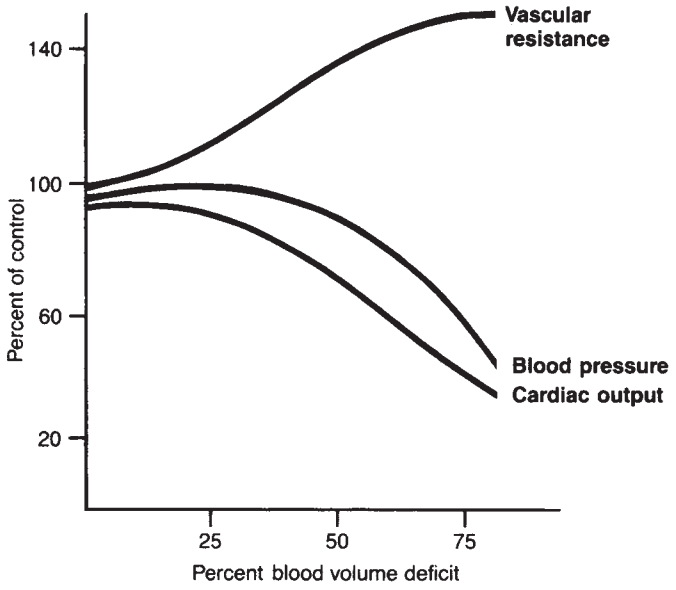

Figure 1.3. Model for cardiovascular response to hypovolemia from hemorrhage.

Normal blood pressure values for age are given in Figure 1.4. A formula that has been used to approximate a typical systolic blood pressure in children over the age of 2 years is 90 + (2 × age in years). The *lower limit* of systolic blood pressure has been approximated by the formula 70 + (2 × age in years). An observed fall of 10 mmHg in systolic pressure should prompt careful serial evaluations for other signs of shock.

C. Peripheral Circulation

Since sinus tachycardia is a nonspecific sign of shock and hypotension is a late sign, the diagnosis of early shock depends on the clinical assessment of stroke volume and peripheral vascular resistance. This is best accomplished by evaluating the presence and volume of peripheral pulses and the adequacy of end-organ perfusion.

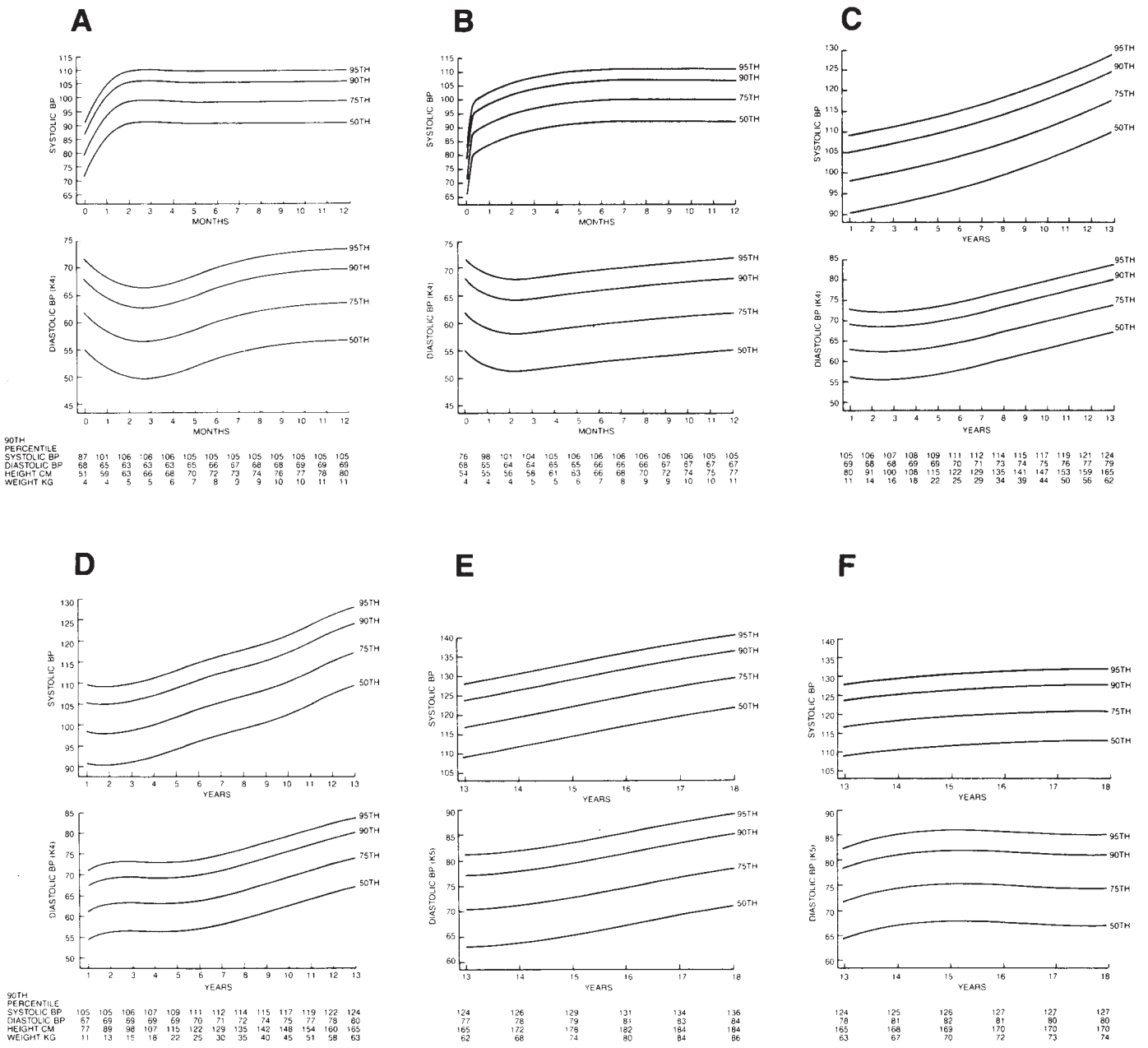

Figure 1.4. Normal blood pressures in infants and children. Age-specific percentiles of blood pressure measurements in A) boys from birth to 12 months, B) girls from birth to 12 months, C) boys from 1 to 13 years, D) girls from 1 to 13 years, E) boys from 13 to 18 years, F) girls from 13 to 18 years. Korotkoff Phase IV used for diastolic blood pressures in A–D; Phase V, in E and F. (From Report of the Second Task Force on Blood Pressure — 1987. *Pediatrics* 79:1, 1987. Reproduced by permission of *Pediatrics*.)

The carotid, axillary, brachial, radial, femoral, dorsalis pedis, and posterior tibial pulses are readily palpable in healthy infants and children. A discrepancy in volume between central and peripheral pulses may be due to vasoconstriction associated with hypothermia, or it may be an early sign of a diminished stroke volume. Pulse volume is related to pulse pressure (difference between systolic and diastolic pressures). As shock progresses, pulse pressure narrows, making the pulse "thready" and, finally, impalpable. Loss of central pulses is a premorbid sign. Early septic shock is a high output state and, in contrast to low-output shock, is often characterized by a wide pulse pressure and, therefore, bounding pulses.

D. End-Organ Perfusion

1. The Skin

End-organ perfusion is evaluated best in the skin, brain, and kidneys. Decreased perfusion of the skin is an *early* sign of shock. Normally, the hands and feet are warm, dry, and pink to the distal phalanx. As cardiac output decreases, the line of demarcation between warmth and coolness ascends toward the trunk.[6] Slow capillary refilling (> 2 seconds) after blanching is evidence of low cardiac output or hypothermia. In testing for capillary refill, the extremity should be lifted slightly above the level of the heart.

Mottling, pallor, poor capillary refill, and peripheral cyanosis indicate poor skin perfusion; acrocyanosis may be normal in the newborn. Severe vasoconstriction causes a grey or ashen color in newborns and pallor in older age groups. The pattern of mottling changes with time and represents variable cutaneous perfusion.

2. The Brain

The signs of brain hypoperfusion exhibited depends on its severity and duration.[7] In children with sudden onset of brain ischemia, few signs of neurologic compromise precede loss of consciousness. Muscular tone is lost, and generalized convulsions and pupillary dilation may occur.

In subacute hypoperfusion, such as shock, symptoms are more insidious. Alteration of consciousness occurs, with confusion and lethargy. An alternating picture of agitation and lethargy is often seen, with the child quiet when undisturbed and combative when procedures are attempted. Infants may be irritable and have a fretful look, weak cry, and wrinkled brow. After 2 months of age, an infant should normally focus on his or her parents' faces. Failure to recognize parents may be an early, ominous sign of cortical hypoperfusion or cerebral dysfunction. This may be obvious only to the parents, who may not be able to describe it any better than to say, "There's something wrong."

More profound degrees of hypoperfusion produce greater changes in the level of consciousness. In decreasing order of levels of consciousness, the child may be:

1. awake,
2. responsive to voice,
3. responsive to pain,
4. unresponsive.

Deep tendon reflexes may be depressed, pupils may be small but reactive, and a crescendo–decrescendo breathing pattern (Cheyne–Stokes) may be apparent.

Hypotonia and intermittent flexor or extensor posturing may occur with prolonged cerebral hypoperfusion or extreme hypoxemia (arterial pO_2 < 30 mmHg).

3. The Kidneys

Urinary output is directly proportional to glomerular filtration rate and renal blood flow. The rate of urinary flow is a good indicator of therapeutic progress, but it is not very helpful in the initial evaluation since the history of urine production in the recent past is often vague and imprecise. Normal urine output is 1–2 mL/kg/hr; a urine flow of < 1 mL/kg/hr in the absence of renal disease, is a sign of poor perfusion. An indwelling urinary catheter facilitates accurate urine flow determination.

III. Rapid Cardiopulmonary Assessment

Recognition of the physiologically unstable infant or child is a critical clinical challenge that can be made by physical examination alone. Laboratory tests are useful adjuncts in evaluating the severity but are not essential in making the diagnosis. Every clinician working with sick children should be able to diagnose pulmonary and circulatory failure and impending cardiopulmonary arrest based on a *rapid cardiopulmonary assessment* (Table 1.2). The rapid cardiopulmonary assessment is a survey whose main goal is to answer the question, "Does this child have pulmonary or circulatory failure that may lead to arrest?" It takes less than half a minute to complete and, by integrating important physical findings, is designed to evaluate pulmonary and cardiovascular integrity through effects on target organs.

Table 1.2. Rapid Cardiopulmonary Assessment

Respiratory Assessment	Cardiovascular Assessment
A. AIRWAY PATENCY	C. CIRCULATION
B. BREATHING	Heart Rate
Rate	Blood Pressure
Air Entry	Peripheral Pulses
Chest rise	Present/absent
Breath sounds	Volume
Stridor	
Wheezing	Skin Perfusion
	Capillary refill time
Mechanics	Temperature
Retractions	Color
Grunting	Mottling
Accessory muscle use	
Nasal flaring	CNS Perfusion
	Recognition of parents
Color	Reaction to pain
	Muscle tone
	Pupil size

The assessment uses the A–B–C approach of cardiopulmonary resuscitation (the "ABC's of CPR").

A. **A**irway: The airway is determined to be either 1) *patent,* 2) *maintainable* with head positioning, suctioning, or adjuncts, or 3) *unmaintainable* and requiring interventions such as removal of a foreign body, intubation, or cricothyrotomy.
B. **B**reathing: Evaluation of breathing focuses on the presence of inspiratory breath sounds, an evaluation of the work of breathing, the adequacy of the tidal volume and resultant chest expansion, and the adequacy of minute ventilation to sustain heart rate and circulation.
C. **C**irculation: An examination of the peripheral circulation qualitatively reveals, the adequacy of cardiac output. The presence and quality of peripheral pulses, capillary refill time, and end-organ perfusion are assessed using criteria discussed previously.

Examination of the ill infant or child does not end when the rapid cardiopulmonary assessment is completed. These patients are often in a dynamic state, and *repeated* assessments are necessary. Repeat evaluations determine whether the patient's clinical condition has deteriorated or is improving in response to therapy.

IV. Priorities in Management

On the basis of the Rapid Cardiopulmonary Assessment, the child is categorized as 1) stable, 2) in questionable respiratory failure or shock, 3) in definite respiratory failure or shock, or 4) in cardiopulmonary failure.

When pulmonary or circulatory failure is suspected but not definitely present, sequential assessments should be performed. Supplemental laboratory studies, especially arterial blood gas analysis and chest x-ray, may be useful. When poor ventilation, oxygenation, or perfusion threatens cardiopulmonary stability, supportive interventions and assessments should be performed promptly and continued until stability is established before attempting a definitive diagnosis.

The child with respiratory distress or well-compensated shock should be approached in a prompt and efficient, yet thoughtful and gentle, manner that minimizes stress and oxygen demand. Supplemental oxygen should be given in a nonthreatening manner whenever possible; several methods may need to be tried. In patients with upper airway obstruction, the child's position of maximal comfort should be respected in order to minimize the work of breathing and optimize airway patency. Normal body temperature should be maintained, and feeding should be withheld.

In patients with respiratory failure, the airway should be secured and adequate ventilation assured with maximum supplemental oxygen (see Chapter 3). In shock, vascular access should be established rapidly (see Chapter 4) and volume expansion and medications given, as necessary (see Chapter 5).

In cardiopulmonary failure, initial priority is given to ventilation and oxygenation. If circulation and perfusion fail to improve rapidly, therapy for shock is given (see Chapter 5).

V. Special Conditions Predisposing to Cardiopulmonary Arrest

Certain conditions that may place infants and children at risk of cardiopulmonary failure deserve special attention. More comprehensive discussions of the pathophysiology and management of these conditions can be found in texts relating to pediatric trauma, emergency, and critical care medicine.

A. Positive-Pressure Ventilation

Cardiovascular collapse due to hypoxemia may occur despite tracheal intubation and positive-pressure ventilation if 1) the endotracheal tube is obstructed, 2) the tube is displaced into the esophagus or main-stem bronchus, 3) a tension pneumothorax is present, or 4) the mechanical ventilation device fails. See Chapter 3 for recognition and management of these problems.

B. Epiglottitis

Acute epiglottitis usually presents before 5 years of age with an acute onset of fever, sore throat, drooling, muffled voice, and inspiratory stridor. The child prefers to lean forward while sitting and holds his or her head in a sniffing position. This preference should be respected and supported; supplemental oxygen can be administered while the child is sitting in the parent's lap. Respiratory arrest can occur from total airway obstruction or a combination of partial airway obstruction and fatigue. If epiglottitis is strongly suspected, the patient should proceed *directly* to the operating room. Such patients deserve definitive airway management by the physician most skillful in managing the difficult airway, since the inflamed, swollen surpraglottis makes intubation difficult. If respiratory arrest occurs, bag-mask ventilation with 100% oxygen should precede any attempt to intubate the patient or to perform emergency tracheotomy or needle cricothyrotomy.

C. Tracheostomy

An infant with a tracheostomy is at high risk for respiratory arrest if a mucous plug obstructs the artificial airway (see Chapter 3). The infant's small tracheal diameter and inability to cry for help compound the possibility of obstruction. Tracheal obstruction should be considered the cause of an arrest in a tracheostomy patient until proven otherwise. If a brief suctioning attempt fails to relieve the obstruction, the tracheostomy tube should be removed immediately. The infant often has enough

reserve to breathe spontaneously before the stoma is suctioned and a new tube is inserted. Otherwise, the stoma can be manually occluded and the infant ventilated with a bag and mask prior to orotracheal intubation.

D. Burns

Children with major burns require careful and frequent assessments for shock and respiratory failure. Patients with severe burns of the head and neck may need prompt endotracheal intubation, in order to protect the airway, before edema distorts the anatomy. Carbon monoxide poisoning should be suspected and treated with maximal supplemental oxygen.

E. Trauma

Respiratory failure in children with trauma may occur as a result of central nervous system depression, upper airway obstruction, pneumothorax, pulmonary contusion, or flail chest. Suspected cervical fractures should be treated by neck immobilization until that diagnosis has been excluded. Hemorrhagic shock is most often due to hepatic or splenic injury, but hemothorax, a fractured femur with vascular laceration, scalp lacerations, and intracranial hemorrhage (in the newborn or infant) may be other sites of major blood loss.[8,9]

F. Gastroenteritis

Hypovolemia due to fluid loss secondary to a gastrointestinal infection may cause shock in infants and children. Signs of dehydration precede those of shock and include lethargy, an absence of tearing, dry mucous membranes, and a dry, sometimes doughy, consistency of the skin. Rapid volume expansion is indicated to prevent cardiovascular collapse.

G. Seizures

Seizures may cause respiratory depression, or upper airway obstruction by secretions or prolapse of soft tissues into the hypopharynx. Apnea may result from some medications used to treat the seizures.

H. Critically Ill Patients

All infants and children who require intensive care, including those in an intensive care unit, in an operating room, in an emergency room, or who are in transit within or between hospitals, are at high risk for cardiopulmonary arrest.[10] Cardiopulmonary arrest may result from:

1. the natural history of a life-threatening disease;
2. a complication of therapy;
3. premature withdrawal of support, including mechanical ventilation, supplemental oxygen, or vasoactive drugs; or
4. the accidental withdrawal of such support.

In patients who have recently been resuscitated, cardiovascular instability and arrest may recur because:

1. catecholamines administered during resuscitation have been metabolized and not replaced by a continuous infusion;
2. the inciting event recurs; or
3. hypoxic–ischemic myocardial, pulmonary, or cerebral damage has occurred.

I. Coma

In a comatose infant or child, intubation is required to treat hypoventilation and to provide airway protection. Intracranial hypertension, which may be present in some comatose patients, may be aggravated by intubation by an inexperienced operator. In such situations bag–valve ventilation should be used and tracheal intubation should be postponed until it can be safely performed using muscle relaxants and an anesthetic regimen for neuroinduction. Posturing after head trauma must be considered a sign of increased intracranial pressure. Cushing's triad (bradycardia, hypertension, and apnea) is a very late manifestation, and its absence in children does not exclude the presence of increased intracranial pressure.

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