Problems in Family Practice

Shock in Infants and Children

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Shock is a descriptive term indicating generalized inadequacy of organ perfusion. In the critically ill patient, the common denominator is insufficient microcirculatory flow. All patients have excessive sympathetic tone with arteriolar and venular vasoconstriction. Two phases may be clinically recognized. Phase 1 is usually reversible and is characterized by ischemic hypoxia. Phase 2 is often irreversible and is characterized by stagnant hypoxia, cell disruption, autolysis, and diffuse intravascular coagulation. Although the presentation of shock may vary with age, one usually detects evidence of inadequate tissue perfusion, hypotension, and poor urine output. Treatment consists of early recognition, establishment and maintenance of intravascular volume, and cardiorespiratory assistance with mechanical ventilation and various inotropic and vasoactive drugs. Pediatric mortality remains high despite new and sophisticated treatment methods.

The concept of shock as a syndrome characterized by protracted prostration and hypotension is well established. The recognition and management, however, continue to offer important challenges to all physicians caring for infants and children. Although voluminous literature exists detailing the pathophysiology in experimental animals, some confusion continues regarding extension of these concepts from the laboratory to the hospital intensive care unit and the critically ill patient. In addition, little attention has been paid to the recognition and management in infants and children,[1-3] who have usually been regarded as "small adults." This paper reviews the basic pathophysiology and hemodynamics of shock and presents a stepwise approach to the recognition and management of this syndrome in infants and children, with special emphasis on problems peculiar to this age group. Burn therapy and infectious disease aspects will not be discussed.

Definition and Etiology

Shock is a descriptive term which in the critically ill patient indicates generalized inadequacy of organ perfusion (ischemia). Ischemia of one vital organ is easily recognized as stroke, acute tubular necrosis, or myocardial infarction. In contrast, shock is present when there is generalized ischemia to several major organ systems. Shock includes a constellation of signs and symptoms and therefore is a syndrome. It is not a specific disease or single entity. It is always an acute process because all patients will quickly die without rapid and appropriate intervention. Lastly, shock always includes circulatory dysfunction of varying degrees with generalized inadequacy of microcirculatory flow.

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Table 1. Etiologies of Shock

Common
- Newborn
  - Intra -or extrauterine sepsis
  - Metabolic, eg, hypoglycemia, severe acidosis, hypoxia
- Infants and children
  - Trauma, eg, hemorrhage, burns
  - Diarrhea and dehydration, sepsis

Less Common
- Cardiogenic
  - Cardiomyopathy
  - Myocarditis
  - Arrhythmia, eg, persistent paroxysmal atrial tachycardia (PAT)
  - Post surgery for congenital heart disease
- Neurogenic
  - Spinal cord injury
  - Drug overdosage, eg, barbiturates

Table 1 lists the most common etiologies of shock in infants and children. In the neonate, shock usually results from sepsis acquired from the intra or extrauterine environment. Postnatal etiologies include meningitis, necrotizing enterocolitis, omphalitis, and urinary tract infection.[4] Less commonly, neonatal shock may result from fetoplacental hemorrhage, ante or postpartum hypoxia, hypoglycemia, or severe metabolic acidosis.[5] In older infants and children, the usual causes are trauma resulting in fluid (blood) loss, burns, infection, or diarrhea and dehydration. Less common etiologies include cardiogenic shock from cardiomyopathy, myocarditis, arrhythmias, or following surgery for congenital heart disease. Lastly, shock occasionally presents in children secondary to spinal cord injury of drug overdosage.

Pathophysiology

Several mechanisms are basic to all forms of shock.[6-9] All patients have excessive sympathetic tone, resulting in arteriolar and venular vasoconstriction. Ischemia and hypoxia are present to variable degrees, with increased capillary permeability and lysosomal cell death.[6] Early, there is hypovolemia with normal or increased cardiac pump performance. In later stages, the patient may be hypo-, normo-, or hypervolemic with depressed cardiac pump performance.[7]

Two phases may be clinically recognized.[2,7,9] Phase 1 is usually reversible and characterized by ischemic hypoxia. Hypovolemia from whatever cause results in a decrease in blood volume relative to vascular capacity. Venous return to the heart decreases, resulting in a lowering of central venous pressure (CVP). The diminished venous return is reflected by a decrease in cardiac output (CO), which results in a decrease in regional blood flow to splanchnic, renal, pulmonary, and cutaneous beds.[7] Reduction in cardiac output results in reflex tachycardia and an increase in total peripheral resistance due to a sympathoadrenal response initiated by aortic and carotid sinus baroreceptors. These receptors in turn stimulate the hypothalamus, which instructs the adrenal glands to increase production of the catecholamines epinephrine and norepinephrine. Increased circulating catecholamines result in intense vasoconstriction of pre- and post-capillary sphincters as a compensatory mechanism to maintain perfusion (alpha effect). This vasoconstriction is intensified early to endotoxin, and later reversed.[10] The heart rate and force of contraction increase (beta effect) as compensatory mechanisms to maintain cardiac output. Small vessels in the kidney, skin, and gut contract, and blood is redirected preferentially to the brain and heart. The arteriolar and venular vasoconstriction produce ischemic hypoxia and an obligatory decrease in capillary hydrostatic pressure. Early, plasma oncotic pressure remains normal resulting in a net influx of extravascular fluid into the intravascular space. This represents a vital early compensatory mechanism to combat shock. Usually all that is required to reverse this phase is replacement of intravascular volume and elimination of the inciting cause.

In marked contrast, Phase 2 is usually irreversible and is characterized by stagnant hypoxia. With prolonged ischemic hypoxia, there is a shift from aerobic to anaerobic glycolysis. Pyruvate is metabolized predominantly to lactic acid and very little high energy phosphate (ATP) is produced (Figure 1). Post-capillary sphincters remain constricted while pre-capillary sphincters relax. In endotoxic shock, this relaxation may be due to the effect of large amounts of endotoxin and catecholamines which in experimental animals results in vasodilation.[10] There is a net influx of fluid into the capillary beds, resulting in an increase in hydrostatic pressure. Stagnant hypoxia now replaces is-

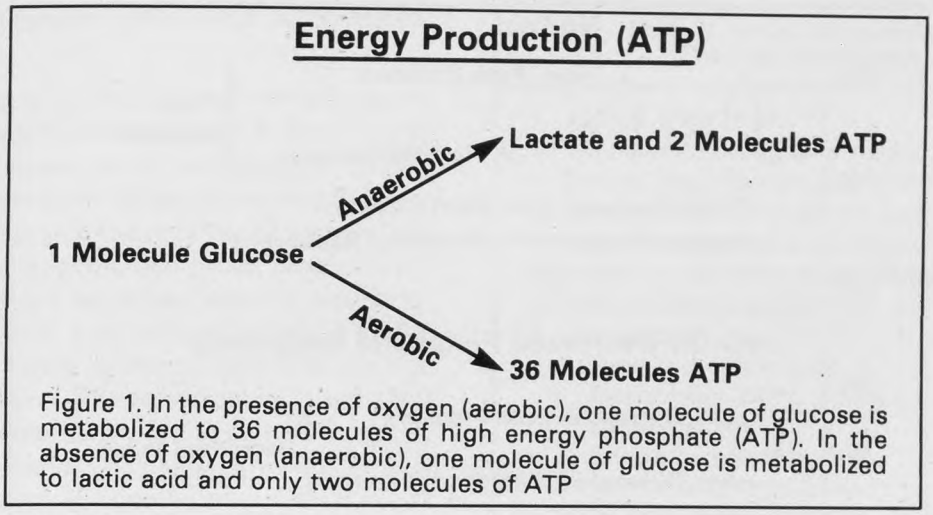

Figure 1. In the presence of oxygen (aerobic), one molecule of glucose is metabolized to 36 molecules of high energy phosphate (ATP). In the absence of oxygen (anaerobic), one molecule of glucose is metabolized to lactic acid and only two molecules of ATP

chemic hypoxia. In the absence of oxygen and high energy phosphate, there is cellular swelling and destruction, increased capillary permeability, and autolysis.[6] Intravascular oncotic pressure diminishes while hydrostatic pressure increases, resulting in pooling of fluid in the extravascular spaces, especially within the hepatic, splanchnic, and pulmonary beds. This phase is often signaled by pulmonary or gastrointestinal hemorrhage, respiratory and metabolic acidosis, and severe oliguria or frank renal failure. The stagnant microcirculation results in platelet aggregation and activation of clotting factors, leading to microthrombi formation and consumption coagulopathy (DIC).[10-12]

Endotoxic Shock

The initiation of Phase 1 and its progression to stagnant hypoxia are readily apparent in the case of traumatic fluid losses. The initiating factor in Gram negative septicemia, however, is endotoxin,[10] a lipopolysaccharide moiety which directly and in combination with complement and other leukocytic components forms vasoactive substances, which in turn cause intense vasoconstriction in the presence of epinephrine and norepinephrine.[13] Many Gram positive organisms release exotoxin, which may act directly on the microvasculature.[14] Shock may also be produced by certain viruses and fungi. The main differences between hemorrhagic (traumatic) and septic shock are: (1) the time to produce stagnant hypoxia, and (2) the mortality rate. With *traumatic shock,* stagnant hypoxia may require *several hours* to appear. In *septic shock,* it may appear within *an hour or less*.[9] The mortality rate with neonatal or infantile septicemia ranges from 13 percent to greater than 50 percent.[4,15] Of important clinical note in early septic shock is the transient finding of decreased total peripheral resistance with normal or increased cardiac output. The patient is warm and appears well perfused (warm shock). Later, total peripheral resistance increases, cardiac output decreases, and the extremities become cold and poorly perfused (cold shock).[15]

Table 2. Clinical Features of Shock

1. Low systemic arterial pressures with peak pressures of:
   a. Neonate: <50 mmHg
   b. Infant: <60 to 70 mmHg
   c. Child: <70 to 80 mmHg
2. Narrow pulse pressure (systolic minus diastolic)
3. Respiratory *alkalosis* early: $P_aCO_2 \leq 35$ mmHg
4. Respiratory and metabolic *acidosis* late: $P_aCO_2 \geq 45$ mmHg, pH<7.3
5. Oliguria (<1 to 2 ml/kg/hr)
6. Altered sensorium and vital signs:
   a. Neonate: bradycardia, apnea, emesis, poor feeding, right-to-left shunting
   b. Infant or child: tachycardia, lethargy, poor feeding, somnolence
7. Reduced tissue perfusion
   a. Mottling
   b. Cold extremities
   c. Poor capillary filling

Recognition of Shock

Table 2 lists the clinical findings indicative of shock for all ages. The first and most easily recognized findings are hypotension and evidence for reduced skin perfusion. Peak arterial blood pressure less than 50 mmHg in the neonate, 60 to 70 mmHg in the infant, and 80 mmHg in the child

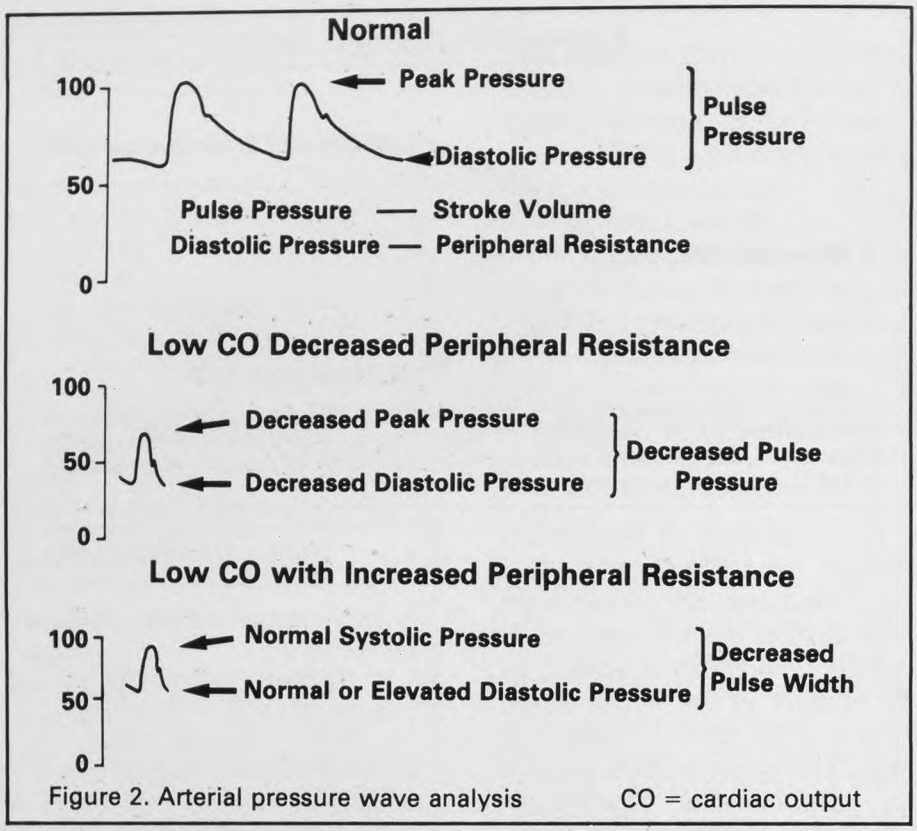

Figure 2. Arterial pressure wave analysis CO = cardiac output

should arouse suspicion of impending or frank shock. When shock is suspected, a standard blood pressure cuff should not be used. Hypotension, low cardiac output, and vasoconstriction may make Korotkoff sounds impossible to hear and systolic pressures are often underestimated.[16] Use a Doppler or other sonic type blood pressure measuring device if available.[17,18] The best method for monitoring arterial pressure as well as serum electrolytes and arterial blood gases is the indwelling arterial line. A 20- or 22-gauge, 1.5 inch cannula may be easily passed into the radial artery either percutaneously or by cutdown while the wrist is fixed to an arm board in hyperextension. A narrow pulse pressure (difference between systolic and diastolic pressures) (Figure 2) is an indirect indication of decreased cardiac output, especially in the presence of tachycardia. The pulse pressure reflects stroke volume; diastolic pressure reflects peripheral resistance to flow. Flow is more important than pressure, but total flow (cardiac output) is not the best measure of perfusion. The brain and kidneys are the best indicators of perfusion adequacy as reflected by mental status and activity level, and urine flow. If the patient is awake, active, and vigorous, and urine flow is of adequate volume and osmolarity, shock is not present regardless of the levels of blood pressure or cardiac output.

Early in Phase 1 the respiratory rate is increased, resulting in respiratory alkalosis (P_aCO_2 < 35 mmHg). This is in response to trauma and stress or results from a direct endotoxic effect on the central nervous system.[15] As ischemic hypoxia continues, respiratory and metabolic acidosis supervene. These result directly from pulmonary capillary damage (shock lung) and lactic acid production (anaerobiosis).[8] Hypovolemia from whatever source will result in oliguria. In the neonate or young child, urine flow of <1-2 ml/kg/hr should arouse suspicion of inadequate renal perfusion. Such a decrease in renal perfusion (prerenal azotemia) is usually reflected by an elevation in blood urea nitrogen (BUN), a decrease in urine sodium to <20 mEq/liter, and a urine to serum osmolarity ratio ≥2:1. Lastly, there is an alteration in the sensorium and vital signs. The older infant or child may present with lethargy, somnolence, and tachycardia. The neonate, in contrast, often manifests bradycardia, apnea, poor feeding, and right-to-left shunting at the ductus arteriosus or foramen ovale.[15,19] Lastly, the neonate may be hypo- or hyperthermic in the presence of sepsis.[4,19]

Initial Resuscitation in the First 30 Minutes (Table 3)

Assess the general activity and consciousness levels and consult with the nurse as to their course over time. Measure rectal temperature. If the patient has a low core temperature, shock is severe. Measure the skin temperature of a neonate in an isolette. The hypothermic infant demands excessive calories and consumes more oxygen in attempts to maintain body temperature.[19] For the neonate in a radiant warmer, note the average power consumption. The servocontrol system will maintain skin temperature in the neutral range; if, however, the unit is constantly drawing power, this may be a sign of hypothermia. Check blood pressure by Doppler, not by cuff, and begin preparation to establish an indwelling arterial line. Ventilate if breathing is slow or irregular. Be prepared to intubate immediately when there is evidence of impending respiratory failure. This may be heralded by a subtle change in arterial P_aCO_2 from respiratory alkalosis to normal or respiratory acidosis in the presence of continued hyperventilation. Establish one or more routes for venous infusion, including a central venous line placed at the superior vena cava-right atrial junction. The venous line can best be accomplished by cutdown over the greater saphenous vein at the ankle.[20] The CVP line is best introduced through a brachial vein by a cutdown slightly medial and about 1 to 2 cm cephalad to the antecubital fossa.[21] In the newborn, the umbilical vein may be used to establish a central venous line. Care must be taken to ensure its tip is advanced through the ductus venosus into the right atrium, since the pressure in the portal sinus and ductus venosus may be higher than the central venous pressure.[22] Assure adequate intravascular volume by giving 400 ml/m² (dose per meter of body surface), or 20 ml/kg crystalloid as normal saline or Ringer's lactate. In the neonate, infuse this volume slowly over approximately 45 minutes to one hour. In the older infant or child, this volume may be infused more rapidly over 15 to 30 minutes. Draw blood for type and crossmatch, and tests for serum electrolytes, blood-urea nitrogen (BUN), glucose, complete blood count, serum lactate, and serum osmolarity. Obtain samples for blood cultures and arterial blood gas determinations. Insert a transurethral catheter for monitoring urine output. Give antibiotics as indicated. Maintain a neutral thermal environment to minimize oxygen consumption. Order the recording of vital signs every 15 minutes, and central venous pressure, urine output, temperature, and general level of activity and pupil size every hour.

Table 3. Initial Resuscitation: The First 30 Minutes

1. Assure open airway
2. Ventilate if breathing is shallow or irregular
3. Start at least two venous cutdowns— one to be a central venous pressure line
4. Ensure adequate intravascular volume with 400 ml/m² or 20 ml/kg as crystalloid (normal saline or Ringer's lactate)
5. Draw blood:
   a. Type and crossmatch
   b. Electrolytes, BUN, glucose, CBC
   c. Serum lactate
   d. Serum osmolarity
   e. Arterial and venous blood gases
   f. Blood cultures
6. Insert transurethral catheter
7. Record vital signs every 15 minutes; urine output, temperature, and activity levels every hour
8. Antibiotics if indicated
9. Neutral thermal environment

Maintenance of Intravascular Volume and Subsequent Management

Fluids should be administered first, drugs last. If the systolic pressure rises to >50 mmHg with a central venus pressure of 6 to 8 mmHg in the neonate, or >70 to 80 mmHg with a CVP of 12 to 15 mmHg in the infant or child, volume replacement is adequate. Measure of pressure in mmHg or cm of H_2O often leads to confusion. Remember that 1.0 mmHg is equivalent to 1.34 cm H_2O. It should also be remembered that vascular expansion with crystalloid will require repeat volume infusions after adequate expansion has been achieved.[23,24] This is because crystalloids distribute approximately 70 to 80 percent in the extravascular, and 20 to 30 percent in the intravascular space after several hours, necessitating continued volume replacement. In traumatic shock, crystalloids are satisfactory for initial resuscitation, but one should follow with properly crossmatched whole blood (colloid) in order to maintain intravascular volume and oncotic pressure. With septic shock, crystalloids probably should be used in

preference to colloids for initial and subsequent resuscitation.

The issue of crystalloid vs colloid resuscitation in the clinical setting is still controversial. Recent studies in the baboon and in the sheep have demonstrated increased interstitial lung water and poorer survival in septic shock when Plasmanate was used for resuscitation rather than Ringer's lactate.[25,26] These findings occurred in the absence of any demonstrable increase in capillary permeability, which did not occur for four to six hours after the onset of shock. Despite these findings, one is cautioned to remember that rapid volume infusions which result in a marked increase in capillary hydrostatic pressure relative to oncotic pressure may result in pulmonary edema especially in Phase 2 shock. Stein et al[27] demonstrated a margin of safety when albumin was infused rather than normal saline in hypovolemic adult patients. When initial left ventricular end-diastolic pressures were similar, they found that large infusions of crystalloid resulted in pulmonary edema more often than when colloid was infused. Infants and children in Phase 2 shock with reduced myocardial pump performance, elevated left ventricular end-diastolic pressures, and damaged pulmonary capillary endothelium are probably also at risk for development of pulmonary edema. From available data, crystalloid may be superior to colloid for initial resuscitation, but regardless which type of resuscitation fluid is used, one should constantly look for the development of pulmonary edema and be ready to provide ventilatory support together with positive end-expiratory pressure (PEEP). If the hematocrit is less than 30 percent, the red cell volume should be augmented to at least 40 percent with packed cells to increase oxygen carrying capacity.

Fluid Challenge

If the central venous and arterial pressures remain low and evidence of poor peripheral perfusion continues, give a fluid challenge as follows: infuse a bolus through the CVP line (neonates 5 ml, older infants and children 20 to 30 ml colloid or balanced salt solution) over a two- to four-minute period and watch for a rise in mean central venous pressure. If the pressure rises above 5 mmHg and remains elevated after ten minutes with inadequate arterial pressure, cardiac dysfunction exists. Stop the infusion and begin inotropic and/or vasoactive drugs (see Cardiotonic and Vasoactive Drugs). If the central venous pressure rises ≤5 mmHg and returns to within 2 mmHg of the original pressure without achievement of adequate arterial pressure, repeat venous infusions every 10 to 20 minutes until the mean central venous pressure reaches 6 to 8 mmHg (8 to 11 cm H_2O) in the neonate or 13 to 15 mmHg (17 to 20 cm H_2O) in the older infant or child. Remember the neonate has a less well-developed Frank-Starling cardiac stretch mechanism, and lower peak stroke volumes are achieved at lower end-diastolic pressures than in the older infant or child.[28,29]

Cardiotonic and Vasoactive Drugs

If, in the presence of an adequate central venous pressure, the arterial pressure, tissue perfusion, and urine flow remain inadequate, then cardiac pump performance is depressed and inotropic and/or vasoactive drugs are indicated. Vasopressors such as norepinephrine, phenylephrine, metaraminol, methoxamine, or high dose epinephrine are rarely indicated. Shock patients have more circulating catecholamines than they need, and such vasopressors may accentuate maldistribution of flow, vasoconstriction, and inadequate oxygen transport.[30] They may also increase cardiac oxygen demands out of proportion to available supply and thereby further compromise pump performance.[31] If pressure is raised with a vasopressor, one may forget that vascular volume is low. The goals of drug therapy should be to improve myocardial performance and reduce systemic vascular resistance.

Although controversy exists as to whether digoxin is effective for increasing contractility in the newborn heart,[32,33] there is little question as to its efficacy in improving the low output which may accompany congenital heart disease in the infant and child. A major drawback, however, concerns its excretion in the presence of reduced renal function, since digoxin is eliminated primarily by the kidney.[34] In addition, digoxin has a relatively long half-life,[34] and its toxic effects can not be rapidly reversed. Since other rapidly acting inotropic agents are available which can be readily stopped, shock patients should probably not be digitalized.

Intropin (dopamine) may be the best currently available cardiotonic agent for the treatment of shock in infants and children.[35,36] Stroke volume is increased more than heart rate, and renal and splanchnic arterioles are selectively dilated while

striated muscle arterioles are constricted or not affected. It has a rapid onset of action and termination and is conveniently administered intravenously. It should not be mixed with bicarbonate. With doses $\geq$10 μg/kg/min, alpha effects may become manifest. A similar beta adrenergic agonist, dobutamine, has not yet been adequately tested in conditions of cardiac dysfunction in infants and children,[37] and until more studies are completed, this drug should not be used. Isoproterenol (Isuprel) is a powerful beta adrenergic stimulator with predominant chronotropic (rate) effects. It does not selectively dilate renal or splanchnic arterioles but instead dilates arterioles in striated muscle. It should be reserved for use only when there is sustained bradycardia (<100 beats/min in the neonate, <85 to 90 beats/min in the older infant, and <80 beats/min in the child).

Alpha blockers such as phentolamine (Regitine) or direct smooth muscle dilators such as sodium nitroprusside (Nipride) may be helpful as afterload reducers, ie, to reduce systemic vascular resistance where peripheral arterial pressure is adequate but tissue perfusion (cardiac output) remains poor.[38] This situation suggests excessive systemic resistance. The reduction in systemic vascular resistance by alpha blockade or by direct vasodilation may improve cardiac output and increase arterial pulse pressure (stroke volume) with little or no drop in peak or mean arterial pressure. Both phentolamine and sodium nitroprusside[39] have been used successfully in infants and children following open heart surgery for congenital heart disease when, in the presence of adequate ventricular filling pressures, systemic vascular resistance was moderately elevated (>30 units) and tissue perfusion (cardiac output) remained poor.

The use of a positive inotropic agent in combination with afterload reduction should be considered for those patients in whom ventricular filling pressures are adequate but tissue perfusion (cardiac output) remains inadequate in spite of the presence of adequate doses of a positive inotropic agent. To date, the combination of dopamine plus nitroprusside or phentolamine has not been reported in infants and children. Benzing et al,[40] however, have studied the effects of low dose epinephrine plus nitroprusside treatment of low cardiac output in children after open heart surgery. They found that epinephrine in doses insufficient to increase systemic vascular resistance (ie, doses which produce predominant beta effects without concomitant alpha effects) increased cardiac output where afterload reduction alone failed. All their patients had moderately or greatly elevated systemic vascular resistance and adequate ventricular filling pressures. Table 4 lists the cardiotonic and vasoactive drugs and dosages currently used to treat the pediatric shock syndrome.

Steroids

Steroids should probably be given in septic shock, since evidence in adults suggests they may help stabilize cellular membranes if given early.[41] They should not be used in place of specific inotropic or vasodilating agents.

How Much Bicarbonate Is Enough?

Withhold bicarbonate therapy for pH $\geq$7.25 or a serum $HCO_3 \geq 10$ mEq/liter. Use 0.3 ml x kg x base excess diluted with an equal volume of 5 percent dextrose in water. Attempts to normalize pH will result in alkalosis overshoot when lactate is metabolized to bicarbonate.[42] Mild acidosis shifts the oxyhemoglobin dissociation curve to the right, resulting in a decrease in hemoglobin affinity for oxygen and a greater release of oxygen to the tissues; alkalosis does the reverse.[8] Severe acidosis (pH$\leq$7.20) may result in cardiac pump dysfunction[42,43] and should be treated with bicarbonate.

Should a Pulmonary Artery Catheter Be Used?

A thermodilution flow-directed balloon catheter may be passed to the pulmonary artery at the bedside without the aid of fluoroscopy. It can be used to measure cardiac output, central venous pressure, left ventricular filling pressure (pulmonary artery diastolic pressure), and systemic vascular resistance (mean arterial pressure divided by cardiac output) as guides to fluid and drug therapy. Placement and use of such a catheter unfortunately require technical skills usually beyond those of the average family physician or pediatrician. In the absence of a thermodilution catheter, changes in cardiac output may be estimated by one of several methods. One may measure the oxygen content (percent oxygen saturation x gm of hemoglobin x 1.34 + PO_2 in mmHg x .003) difference between the central venous pressure line and the arterial line (normal = 3 to 5 volume percent). The smaller the difference, the higher the cardiac output. Another simpler method is to measure central venous PO_2 from the central venous pressure line

Table 4. Cardiotonic and Vasodilating Agents Used in the Treatment of Shock in the Pediatric Age Group

Drug	Dosage and Route	Frequency	Indication
Dopamine (Intropin)	1 to 15 μg/kg/min IV Do not mix with bicarbonate	Continuous drip	Normal heart rate and adequate central venous pressure; inadequate arterial pressure and/or arterial perfusion
Epinephrine	0.1 to 0.45 μg/kg/min IV	Continuous drip	Normal heart rate and central venous pressure; inadequate arterial pressure and/or tissue perfusion
Isoproterenol (Isuprel)	0.05 to 0.2 μg/kg/min IV	Continuous drip Titrate to desired heart rate	Sustained bradycardia with or without cardiac dysfunction: neonate<100 beats/min infant<80 to 90 beats/min older child<80 beats/min
Phentolamine (Regitine)	1 to 30 μg/kg/min IV	Continuous drip	Adequate central venous pressure (or left atrial pressure at least 14 to 16 mmHg if pulmonary artery catheter used); adequate systemic arterial pressure but with poor peripheral perfusion (ie, high systemic vascular resistance)
Sodium Nitroprusside (Nipride)	0.5 to 8.0 μg/kg/min IV Protect from light	Continuous drip	Adequate central venous pressure (or left atrial pressure at least 14 to 16 mmHg if pulmonary artery catheter used); adequate systemic arterial pressure but with poor peripheral perfusion (ie, high systemic vascular resistance)
Steroids, eg, Hydrocortisone or equivalent	50 mg/kg IV	Initial dose and every 6 hours × 48 hours, then stop	Give early to stabilize cell membranes

(P_vO_2).[44] Arterial PO_2 reflects the adequacy of the lungs to oxygenate blood destined for use by body tissues. The central venous PO_2 reflects the volume of blood flow (cardiac output) through the tissues and the degree of oxygen extraction which increases as cardiac output decreases. A third method of estimating adequacy of tissue perfusion is to frequently measure serum lactate (normal <2 mg/100 ml). With improving cardiac output and tissue perfusion, the degree of anaerobiosis will diminish and will be reflected by a steadily decreasing serum lactate level.[45] With continuous or frequent monitoring of these parameters, changes in cardiac output can be estimated and appropriate interventions made. Total cardiac output ≤2 liter/min/m², a P_vO_2 of ≤30 mmHg, or a persistently elevated serum lactate are very poor prognostic signs.[45,46]

Conclusion

The patient in shock is critically ill and requires constant attention in a critical care environment. Each patient needs one personal physician who knows him or her well and coordinates total care. The central figure is the nurse who must be at the bedside constantly. The best computerized monitoring system cannot entirely replace the physician's hand, and the patient's prognosis depends in part on the frequency and duration of hands-on evaluation. The management of shock in

infants and children is difficult at best. Without a rational stepwise approach, it becomes chaos.

In this paper, the basic pathophysiology, diagnosis, and a stepwise treatment plan have been presented. Special emphasis has been placed on repletion of intravascular volume prior to use of cardiotonic or vasodilating agents. It is hoped that through this guide, the treatment of this complex syndrome in infants and children will be simplified and the mortality decreased.

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