Emergency management of collapsed infants

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INTRODUCTION

Infants have a limited repertoire of clinical signs in severe illness. Lethargy, respiratory distress, apnoea and poor perfusion can result from a wide spectrum of disorders. Fortunately for the emergency physician, the resuscitation of infants with respiratory and/or circulatory collapse remains universal for all aetiologies (*Table 1*). Investigations can be initiated during the stabilization of the child that will narrow the differential diagnosis. This article will discuss the resuscitation of collapsed infants, the causative disorders in the first few weeks of life, their early investigation and management.

CAUSES OF COLLAPSE IN INFANTS

Sepsis

Infants (children <1 year of age) account for 48% of paediatric cases of severe sepsis in the United States and the non-neonatal, infant group (1–12 months of age) have a significantly higher mortality rate (13.5% *vs* 10.3%) (Watson et al, 2003). Infants are susceptible to pathogens commonly found in older children (*Haemophilus influenzae*, *Streptococcus pneumoniae*, *Neisseria meningitides*) but neonates may also present with late onset sepsis caused by organisms associated with the genitourinary tract: group B streptococcus, *Escherichia coli*, Klebsiella or *Listeria monocytogenes*. In the weeks before vaccination, infants are vulnerable to pathogens like *Bordetella pertussis*. Viral infections are also implicated in infant sepsis, including those caused by respiratory syncytial virus, adenovirus, influenza, herpes simplex virus and rotavirus.

Infection should be suspected in those with a history of fever, rash, coryzal symptoms, diarrhoea or irritability. Infants with respiratory infections may present with tachypnoea, grunting and recession or simply with recurrent apnoea. Infants may be shocked with poor perfusion demonstrated by prolonged capillary refill time (>2 seconds), tachycardia, altered mental status and poor urine output. The shock state is classified as 'warm' shock if there is a widened pulse pressure and vasodilatation, or 'cold shock' if vasoconstriction and cool peripheries predominate. Infants with CNS infections can present with altered consciousness, seizures or bulging fontanelle, indicative of raised intracranial pressure.

Immediate management: Early reversal of shock in paediatric and neonatal sepsis is associated with improved outcome (Han et al, 2003). The American College of Critical Care Medicine and Paediatric Advanced Life Support (ACCM-PALS) guidelines provide parameters for haemodynamic support (*Figure 1*) (Carcillo and Fields, 2002). Shock is aggressively treated with fluid resuscitation while carefully monitoring for signs of fluid overload: pulmonary oedema, hepatomegaly or gallop rhythm. Persistent shock in children after fluid resuscitation is associated with a low cardiac output and high systemic vascular resistance. Inotropic support is indicated and titrated according to response. Dopamine is commenced first at 10 μg/kg/min and thereafter adrenaline is added in cold shock or noradrenaline is added in warm shock.

Early stabilization should include the administration of empiric, broad spectrum, intravenous antimicrobial therapy that penetrates the blood–brain barrier. A combination of a third generation cephalosporin, a macrolide and aciclovir is recommended. Antibiotic administration should not be delayed by culture collection.

Cardiac disease

Duct-dependent congenital cardiac disease: Serious congenital cardiovascular malformations are not excluded by a normal neonatal or 6-week examination (Wren et al, 1999; Richmond and Wren, 2001). Duct-dependent abnormalities, particularly malformations causing left heart obstruction, are the main causes of infant deaths (Richmond

TABLE 1.
European Paediatric Life Support rapid cardiopulmonary assessment for the identification and treatment of seriously ill infants and children

Rapid ABC assessment and management priorities:	
A	Open and maintain patent airway May require intubation
B	Assess respiratory rate, air entry, work of breathing and oxygen saturation Administer 100% oxygen Ventilate if breathing is inadequate
C	Assess for poor perfusion: capillary refill, heart rate, pulse volumes, mental status, urine output, blood pressure Assess electrocardiogram Cardiopulmonary resuscitation algorithm if pulseless or heart rate <60 beats/min (infant) Gain intravenous access Administer 20 ml/kg volume for poor perfusion
D	Assess mental status: awake, responds to voice, responds to pain, unresponsive Check pupillary response Check fontanelle if appropriate
G	Check blood glucose Bolus of 10% dextrose 5 ml/kg if glucose low, followed by an infusion
Reassess from A again	

From Resuscitation Council UK and European Resuscitation Council (2004)

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and Wren, 2001). Hypoplastic left heart and interrupted aortic arch present earlier, but critical aortic stenosis and coarctation of the aorta can present late. A review showed that only 54% had presented by 6 weeks (Richmond and Wren, 2001). Poor femoral pulses are characteristic of coarctation of the aorta, but this finding is common in the low cardiac output state of sepsis. Infants with duct-dependent pulmonary blood flow will be cyanosed with evidence of right heart failure.

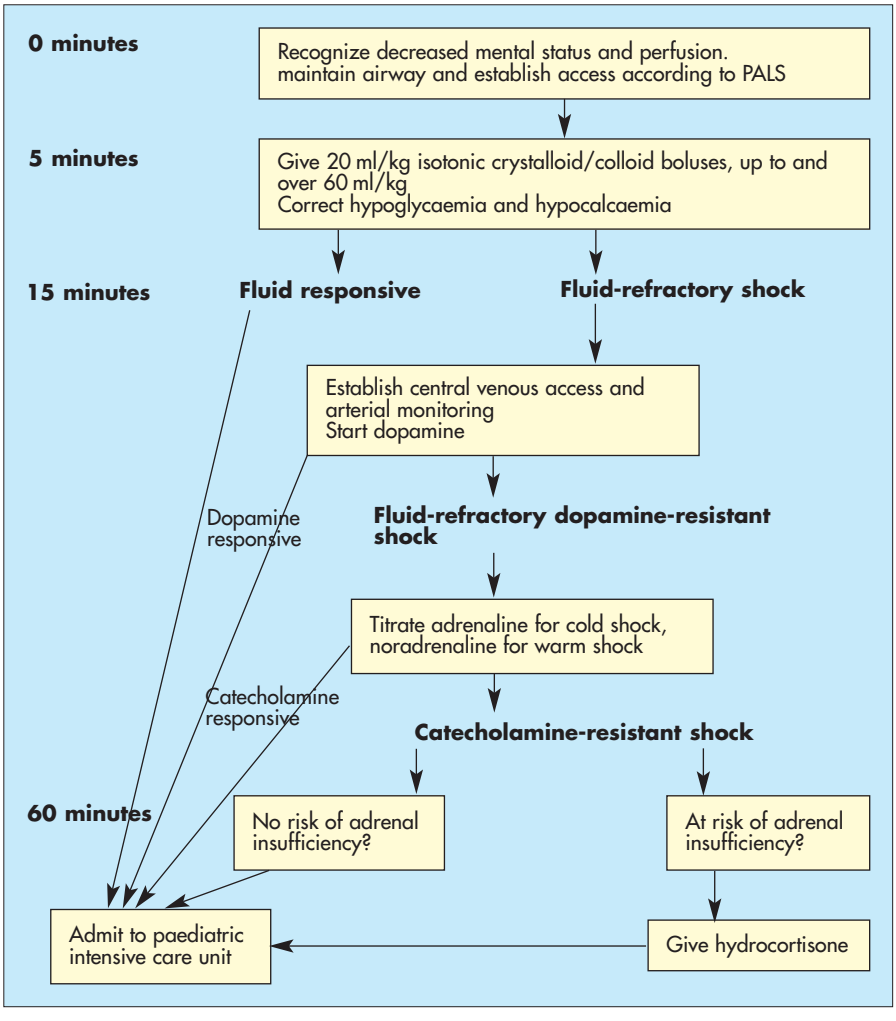
Immediate management: Prostaglandin E_1 infusion maintains the patency of the ductus arteriosus. There are no congenital cardiac lesions in which it is contraindicated, therefore an infusion should be started in all cases of suspected duct-dependent flow (Penny and Shekerdemian, 2001). Prostaglandin E_1 can cause apnoea, therefore intubation and ventilation should be considered.

Pump failure: Pulmonary vascular resistance decreases over the first few weeks of life. Left to right shunts subsequently increase as the pressure differential between systemic and pulmonary circulation increases. Infants with large left to right shunts (ventricular septal defect, complete atrioventicular defect) can present in congestive cardiac failure (Richmond and Wren, 2001). Viral myocarditis can occur and presents with cardiac failure or arrhythmias. Cardiomyopathy, arrhythmias and sudden death can rarely be the presenting feature of long chain fatty acid disorders (Leonard and Morris, 2000).

Immediate management: Fluid should be judiciously administered during resuscitation in order to optimize preload. Infants should be carefully monitored for signs of fluid overload. Inotropic support with dopamine or low dose adrenaline may be considered, noradrenaline should be avoided as it increases afterload. Intubation and ventilation will reduce the cardiac workload. Diuresis can reduce afterload, provided the child is fluid resuscitated. Paediatric cardiologists should be involved early in the stabilization period.

Arrhythmia: Arrhythmias are rare causes of infant death (Wren, 2002). Infants with narrow complex tachycardia (supraventricular tachycardia) can present in shock and should be treated according to the European Paediatric Life Support (EPLS) guidelines (Resuscitation Council UK and European Resuscitation Council, 2004) (*Figure 2*). Early advice should be sought from paediatric cardiologists in cases of symptomatic bradycardia. Treatment with isoprenaline or transfer for urgent pacing may be necessary.

Figure 1. American College of Critical Care Medicine and Paediatric Advanced Life Support (PALS) clinical practice parameters for haemodynamic support of paediatric and neonatal patients in septic shock. Management algorithm of therapeutic interventions. Adapted from Carcillo and Fields (2002).



Metabolic disease

Inborn errors of metabolism can present in the first few weeks with a combination of signs: encephalopathy, metabolic acidosis, hyperammonaemia or hypoglycaemia (Burton, 1998; Leonard and Morris, 2000; Ellaway et al, 2002). The clinical picture is similar to that of sepsis and cardiac disease. Metabolic disease should be considered if there is parental consanguinity, family history of other affected individuals or previous sudden infant death.

The urea cycle defects, organic acidaemias, fatty acid oxidation defects and some amino acid disorders are most likely to underlie life-threatening episodes in infants (Ellaway et al, 2002). The baseline investigations that may aid diagnosis are listed in *Table 2*.

Immediate management: Once resuscitated, ongoing supportive therapy is required to regain and maintain homeostasis. This may require intensive care admission. Continuing acidosis despite normal tissue perfusion will require bicarbonate infusion. Enteral feeds must be stopped and intravenous dextrose infused at a high rate to minimize catabolism. Hyperammonaemia will require urgent dialysis to minimize neurological

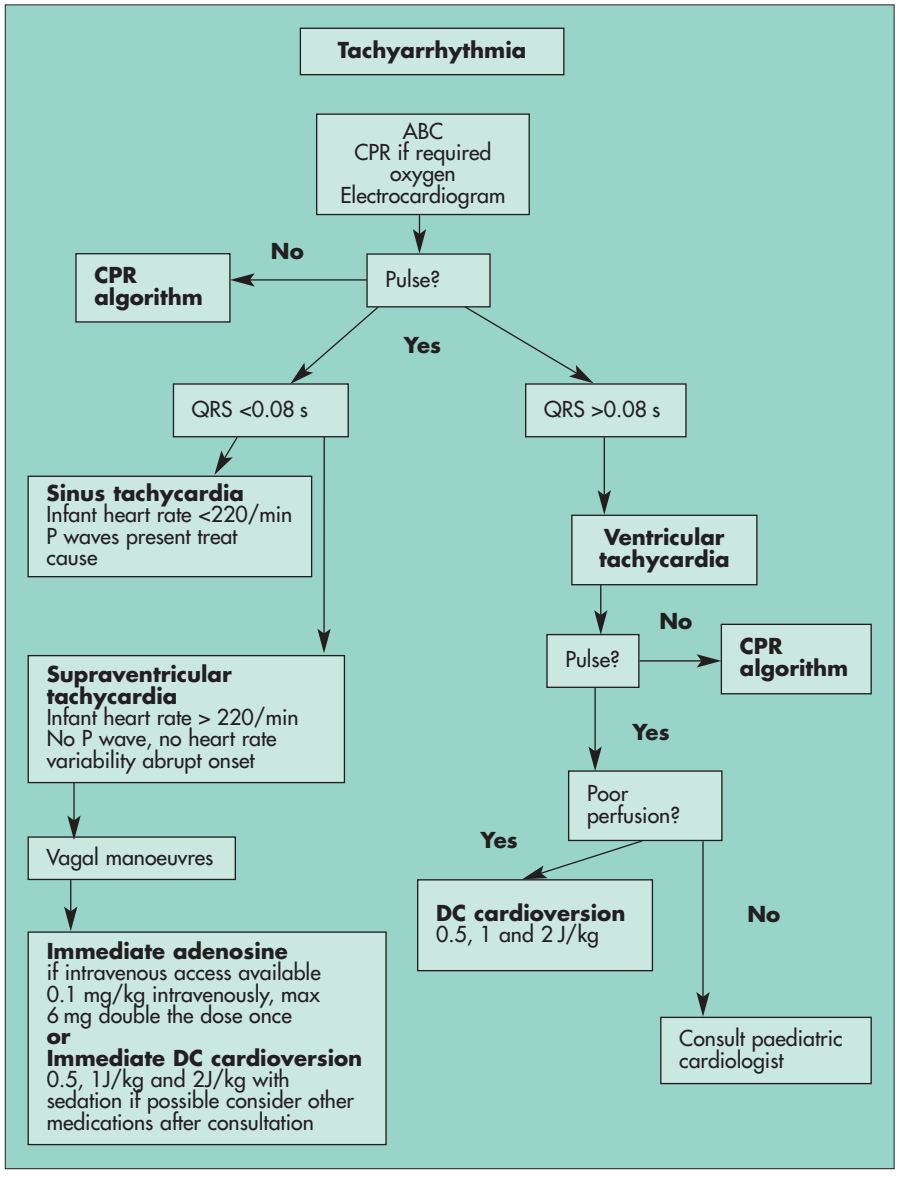
sequelae and therefore transfer to the paediatric intensive care unit should be arranged (Burton, 1998; Leonard and Morris, 2000; Ellaway et al, 2002).

Endocrine disease

Adrenal insufficiency can manifest as circulatory collapse in the neonate. The secretion of all adrenal steroids is reduced in congenital adrenal hypoplasia. Congenital adrenal hyperplasia is a group of disorders resulting from deficiencies in the enzymes required for the synthesis of cortisol (Speiser and White, 2003). Accumulation of precursors can result in virilization of affected females. The syndrome can be missed in male infants and they present with circulatory collapse. The clinical features are predominantly related to mineralocorticoid deficiency and salt wasting: hypovolaemia, hyponatraemia, hyperkalaemia, acidosis and hypoglycaemia.

Immediate management: These patients require fluid resuscitation and correction of hypoglycaemia. Baseline bloods should include serum electrolytes, anion gap, cortisol and saved serum. Urine should be sent for steroid profiling. These patients require replacement therapy with hydrocortisone and fludrocortisone.

Figure 2. European Paediatric Life Support algorithm for the stepwise management of children with tachyarrhythmia (Resuscitation Council UK and European Resuscitation Council, 2004). ABC = airways, breathing, circulation; CPR = cardiopulmonary resuscitation.



Cerebrovascular disease

Intracranial haemorrhage should be suspected in infants with a reduced level of consciousness, bulging fontanelle and low haemoglobin level. The pupillary size, equality and reaction to light should be checked as a matter of urgency. Specific history should be sought with regard to the vitamin K status in a neonate, any recent trauma, irritability or seizures. Late onset vitamin K deficiency bleeding is associated with oral vitamin K administration after birth and exclusive breast feeding (D'Souza and Rao, 2003). Intracranial haemorrhage may be a manifestation of severe meningoencephalitis, e.g. caused by herpes simplex virus. Intracranial haemorrhage may be a feature of non-accidental injury and the child should be examined for evidence of other injuries (Duhaime et al, 1998).

Immediate management: These infants will all require an urgent computed tomography (CT) scan. Intubation, ventilation and sedation are recommended to minimize intracranial pressure. Therapy should be aimed at the maintenance of a normal blood pressure in order to improve cerebral perfusion. Inotropes may be required. Blood transfusion may be necessary if the haematocrit is low. Bradycardia, hypertension and unequal pupils herald imminent cerebral herniation. The airway must be secured, the patient should be sedated, hyperventilated for a short period (<2 minutes), and 20% mannitol 0.5 g/kg administered. A paediatric neurosurgical centre should be contacted as a matter of urgency.

Gastrointestinal disease

Abdominal surgical emergencies can present with circulatory collapse. Bilious vomiting, bloody stools, abdominal distension and tenderness are important clues in the history. Possible diagnoses include malrotation with midgut volvulus or Hirschsprung's colitis as well as intussusception in the slightly older infant (McCollough and Sharieff, 2003).

Immediate management: Following resuscitation, a nasogastric tube should be placed to deflate the stomach, the child must be kept nil by mouth and an

abdominal X-ray should be obtained. Broad spectrum antibiotics, including metronidazole, should be administered. Seek surgical advice at the earliest opportunity.

PRIORITIES OF CLINICAL MANAGEMENT

Resuscitation

Resuscitation should follow the EPLS and Resuscitation Council UK guidelines outlined in *Table 1*.

The EPLS ABC algorithm ensures that the critical signs of imminent cardiorespiratory failure are sought, treated if present and, most importantly, the effect of therapy is repeatedly assessed.

Many of these infants have been unwell for a significant period and have little remaining cardiopulmonary reserve. Early intubation and ventilation is beneficial in this population as it improves oxygenation, reduces cardiac workload and allows procedures under sedation without causing more stress, e.g. intraosseous needle and other line placement, or CT scanning. It can be difficult to gain intravenous access in a shocked infant. The intraosseous route is recommended after two failed attempts at peripheral cannulation. An initial fluid bolus is recommended for all shocked patients. Patients with severe cardiac failure will benefit from preload optimization but they must be carefully monitored. Dehydrated or septic patients may require large volumes of fluid to restore normovolaemia.

Stabilization: early investigations and therapy

Once the ABCs are treated and maintained, a period of stabilization is required to achieve homeostasis: normalization of oxygenation, ventilation tissue perfusion, correction of metabolic acidosis and electrolyte abnormalities. All infants should receive empiric antimicrobial treatment, including metronidazole if a gastrointestinal cause is suspected. A prostaglandin E_1 infusion should be started if there is any suspicion of a duct-dependent lesion. A CT scan of the brain should be performed in cases of suspected raised intracranial pressure or encephalopathy. The initial investigations listed in *Table 2* will aid diagnosis and dictate ongoing management and investigation plans.

Specialist centres should be contacted early to coordinate ongoing management and possible transfer to an intensive care unit. HM

TABLE 2.
Initial investigations in the emergency room to aid the differential diagnosis and ongoing management of a collapsed infant

Blood glucose
Blood gas and pH
Full blood count
Electrolytes and anion gap
Coagulation screen
Blood and urine cultures
Lactate
Ammonia
Liver function test
Save serum and urine for later investigations, e.g. cortisol, amino acids, acyl carnitine, urine organic acids
Chest X-ray
Urine dipstix for blood, pH and ketones

KEY POINTS

- Cardiopulmonary failure in infants can result from a wide variety of disorders.
- The resuscitation management is universal.
- The commoner causes of infant collapse are sepsis, congenital cardiac disease and inborn errors of metabolism.
- All infants should be empirically treated with broad spectrum antimicrobial cover.
- Prostaglandin E_1 can be safely used to maintain duct patency and should be considered if a duct-dependent cardiac lesion cannot be excluded.
- Paediatric intensivists and other specialists should be contacted early.

Burton BK (1998) Inborn errors of metabolism in infancy: a guide to diagnosis. *Pediatrics* **102**(6): E69

Carcillo JA, Fields AI (2002) Clinical practice parameters for haemodynamic support of pediatric and neonatal patients in septic shock. *Crit Care Med* **30**(6): 1365–78

D'Souza IE, Rao SD (2003) Late hemorrhagic disease of newborn. *Indian Pediatr* **40**(3): 226–9

Duhaime AC, Christian CW, Rorke LB, Zimmerman RA (1998) Nonaccidental head injury in infants--the 'shaken-baby syndrome'. *N Engl J Med* **338**(25): 1822–9

Ellaway CJ, Wilcken B, Christodoulou J (2002) Clinical approach to inborn errors of metabolism presenting in the newborn period. *J Paediatr Child Health* **38**(5): 511–17

Han YY, Carcillo JA, Dragotta MA, Bills DM, Watson RS, Westerman ME, Orr RA (2003) Early reversal of pediatric-neonatal septic shock by community physicians is associated with improved outcome. *Pediatrics* **112**(4): 793–9

Leonard JV, Morris AA (2000) Inborn errors of metabolism around time of birth. *Lancet* **356**: 583–7

McCollough M, Sharieff GQ (2003) Abdominal surgical emergencies in infants and young children. *Emerg Med Clin North Am* **21**(4): 909–35

Penny DJ, Shekerdemian LS (2001) Management of the neonate with symptomatic congenital heart disease. *Arch Dis Child Fetal Neonatal Ed* **84**(3): F141–F145

Resuscitation Council UK and European Resuscitation Council (2004) *European Paediatric Life Support Provider Manual.* 1st edn. European Resuscitation Council, Antwerp, Belgium

Richmond S, Wren C (2001) Early diagnosis of congenital heart disease. *Semin Neonatol* **6**(1): 27–35

Speiser PW, White PC (2003) Congenital adrenal hyperplasia. *N Engl J Med* **349:** 776–88

Watson RS, Carcillo JA, Linde-Zwirble WT, Clermont G, Lidicker J, Angus DC (2003) The epidemiology of severe sepsis in children in the United States. *Am J Respir Crit Care Med* **167**(5): 695–701

Wren C (2002) Sudden death in children and adolescents. *Heart* **88:** 426–31

Wren C, Richmond S, Donaldson L (1999) Presentation of congenital heart disease in infancy: implications for routine examination. *Arch Dis Child Fetal Neonatal Ed* **80**(1): F49–F53