

Fluid therapy in the emergency unit

This article focuses on the fluid management of shock in the emergency department.

R J Diedericks, MB ChB, FCPaed (SA)

Senior Specialist Paediatrician, Department of Ambulatory and Emergency Paediatrics, Red Cross War Memorial Children's Hospital, Cape Town

Dr Diedericks is currently head of the Paediatric Emergency Service at Red Cross Hospital. He has previous experience in private paediatric practice, held a consultant position at the NHS in the UK, and a level 2 paediatrics position in Worcester, Cape. He is a generalist with a special interest in respiratory paediatrics.

Correspondence to: R J Diedericks (Ralph.Diedericks@uct.ac.za)

Many children entering emergency units require immediate vascular access and intravenous fluid administration. Choosing the correct fluid and administering effective and safe amounts of fluid for resuscitation are important determinants of morbidity and mortality outcomes.

The American College of Critical Care Medicine (ACCM) Clinical Practice Parameters for Hemodynamic Support of Pediatric and Neonatal Shock first published in 2002, and revised in 2007, were aimed at implementation of the best clinical practices associated with improved outcomes in previously healthy and chronically ill children with septic shock.¹ The use of goal-driven guidelines is widely endorsed by paediatric life-support training programmes.

Venous access

Advanced Paediatric Life Support (APLS) guidelines have regularly taught that intravenous access should not be delayed unnecessarily in emergency situations and that intra-osseous (IO) line placement is a safe and an effective route for delivering fluid and drugs for resuscitation.

It is important that staff in emergency departments become skilled in the placement of IO lines in children. Large amounts of fluid can be given rapidly through IO lines. Specially designed needles are available; however, 18-gauge spinal needles can be safely used for infants and children.

Which fluid?

The urgent need for fluid administration is determined by the presence of shock, which is defined as impaired tissue perfusion.

There are many different forms of shock, which may be the result of:

- fluid depletion/losses (hypovolaemic)
- poor cardiac action (cardiogenic)
- defective vasculature (distributive)
- restriction to flow (obstructive)
- inadequate oxygen-carrying/-releasing capacity of the blood (dissociative).

Recognition of shock

Early recognition of shock is essential. The clinical signs of poor tissue perfusion are prolonged capillary refill time (CRT) (>2 s), cool peripheral temperatures with a differential between central and peripheral assessments, and impaired level of consciousness. The ACCM's haemodynamic definitions of shock further differentiates between:

- cold shock – prolonged CRT, mottled cool extremities, reduced pulses; and
- warm shock – flash capillary refill and bounding pulses.

Shocked patients may present with tachypnoea and are sometimes erroneously labelled as having respiratory distress.

The blood pressure (BP) may be normal initially in the compensated phase of shock when vital organ perfusion is maintained. Hypotension signifies uncompensated shock with progression to anaerobic metabolism and lactate production. This is a late sign. Elevated lactate levels above 2.5 mmol/l are associated with increased mortality.²

In many instances, when shock has not been appreciated early, re-examination of the observation records identifies a rapid tachycardia >160/min as a sign of a compromised circulation. In these patients it is likely that the signs of delayed CRT and temperature changes are present if specifically assessed.

Shocked patients may present with tachypnoea and are sometimes erroneously labelled as having respiratory distress. It may then be useful to check for signs of increased respiratory effort. If these are absent, other mechanisms may be responsible for the tachypnoea. Patients with life-threatening infection often present with fever and severe, persistent tachypnoea.

Monitoring signs of shock

Shock can be assessed and monitored effectively without high-tech facilities by observing the following:

- pulse rate (tachycardia initially as heart rate increases in an attempt to maintain cardiac output;³ bradycardia as a sign of poor tissue perfusion occurs late)
- capillary refill
- peripheral temperature
- coma score
- urine output
- BP
- oxygen saturation.

Airway and breathing

It is extremely important to consider the ABC approach in the initial management of shock, with emphasis on the airway, oxygen administration, and treatment of hypoglycaemia and hypocalcaemia.

Airway and breathing should be carefully monitored. The progression of metabolic

Fluid therapy

changes after septic shock may lead to hypoxaemia and metabolic acidosis with a high risk of respiratory acidosis, especially in small infants who may become exhausted. This is more likely if there is concomitant lung disease. The decision to intubate and ventilate is a clinical one and there should not be delays while waiting for laboratory test results. Intubation and subsequent adequate ventilation can improve shock. Ketamine 1 - 2 mg/kg is an effective induction agent to facilitate intubation. Etomidate is no longer recommended for this indication in septic shock because of the risk of adrenal suppression.

Crystalloid v. colloid

Several randomised trials have shown that for children in shock who received fluid resuscitation the survival was significantly increased regardless of the fluid composition used.

The ideal fluid required to restore the intravascular volume may depend on the cause of shock.

The ideal fluid required to restore the intravascular volume may depend on the cause of shock. There is evidence of improved outcome with 4% albumin in septic shock due to malaria and meningococcal sepsis (SAFE study).⁴

Ringer's lactate is cheap and readily available and is a physiological, isotonic fluid. Normal saline as an alternative isotonic fluid has been shown to be as effective as IV albumin. However, large volumes of normal saline can cause hyperchloraemic acidosis. This should be appreciated when evaluating the acid base status after resuscitation, especially in diarrhoeal disease.

Fluids that should not be used in septic shock are 5% dextrose or hypotonic solutions, e.g. 0.2% saline.

How much fluid?

When shock is diagnosed fluid resuscitation should commence with a rapid (over 5 - 10 min)

bolus infusion of 20 ml/kg isotonic crystalloid, e.g. Ringer's lactate. During the period of rapid bolus fluid therapy check for signs of fluid overload (laboured respiration, chest crackles, gallop rhythm or hepatomegaly).

If signs of shock are still present the bolus should be repeated up to a volume of 60 ml/kg. Heart rate, CRT, urine output, and level of consciousness should be monitored and reassessed after each bolus infusion.

Children commonly require 40 - 60 ml/kg in the first hour. It may be necessary to continue bolus therapy provided the patient continues to improve and there are no signs of fluid overload. These patients require careful clinical and haemodynamic monitoring.

Some studies in Africa – especially in children with malaria or low haemoglobin levels – have shown that smaller fluid volumes given more slowly may have a better outcome. It is recommended that smaller volumes of fluid be used as bolus infusions, starting with 15 ml/kg Ringer's lactate and using subsequent boluses of 10 ml/kg if improvement is noted and there are no signs of fluid overload. Caution is stressed and the same targets of 60 ml/kg in the first hour should not always be used in all patients. It is important to evaluate the clinical context and consider the risks of volume overload where co-morbidities exist.

The ETAT guidelines⁵ advise even slower rates, starting with 15 ml/kg over an hour and repeating 15 ml/kg over the next hour if improvement in the pulse and respiratory rate occurs. The guidelines also advise switching to oral or nasogastric fluids early in the management, as this is much less likely to lead to fluid overload.

During this time check for hypoglycaemia and treat rapidly with 3 - 5 ml/kg of 10% dextrose. The clinical endpoints for resuscitation are CRT <3 s, normal pulse rates, warm limbs, urine output 1 ml/kg/h and normal mental status.⁶

Use of inotropes

Inotropes should be considered for the fluid refractory patient. When considering the use

of these vasoactive drugs it is important to recognise that they may have varying effects on systemic vascular resistance, heart rate and contractility. Drug pharmacodynamics may be altered by the effect of shock on renal and liver function, with higher levels than anticipated.

The inotrope can be delivered through a peripheral line or via the IO route while establishing a central venous line. The dosage should be delivered as a dilute solution (with normal saline) with a syringe pump when given via a peripheral line. Dobutamine or mid-dosage dopamine (5 - 9 µg/kg/min) can be used as the first line of inotropic support to increase cardiac contractility in patients with normal BP.

Antibiotics

IV antibiotics should be given early within the first hour in the management of septic shock. Cefotaxime 100 mg/kg is an appropriate broad-spectrum antibiotic with good CSF penetration.

Special problems Severe malnutrition

The fluid management of children with severe malnutrition is more difficult than

of those with normal nutritional status. Severe malnutrition may be complicated by hypoalbuminaemia and sepsis, which both lead to poor retention of intravascular fluids and complications related to extravascular leakage. Poor cardiac contractility may result in inability to cope with large fluid volumes. In addition, hepatomegaly due to fatty infiltration renders it impossible to use the liver size as a guide to fluid overload.

Recent important research

Publication of the FEAST study in the *NEJM* in June 2011, evaluating the effect of fluid resuscitation in resource-poor settings, has raised the issue of safety of bolus fluids in children with febrile illness and impaired perfusion. In the study, fluid boluses significantly increased the 48-hour mortality in critically ill children with evidence of impaired perfusion.⁷ The results challenge the conventional established intervention of bolus resuscitation, which is currently widely used throughout the developed world in well-resourced settings. The reasons for this unexpected study outcome are not clear. Assisted ventilation was not available to the study population, except for short-term bag and mask support.

Raised intracranial pressure

Patients with uncompensated shock may have signs of impaired levels of consciousness and progress to coma as a result of poor cerebral perfusion. In these situations the most urgent therapy is to address the management of shock as described. However, the abnormal CNS status may be due to the primary cause of encephalopathy, with more complex implications for fluid therapy. The issue of intracranial pressure monitoring and maintenance of cerebral perfusion pressure with regard to traumatic brain injury was described by Figaji in the March 2010 edition of *CME*.⁸ It is likely that continued research in this field will have implications for fluid management of cerebral oedema in the acute setting. It is important to check for hyponatraemia in these patients and to avoid using large intravenous volumes of hypotonic fluid.

Hypernatraemic dehydration

Severe dehydration in infants with gastroenteritis and higher water than sodium loss results in hypernatraemia, defined as a serum sodium above 151 mmol/l. Infants presenting with severe hypernatraemia characteristically are well nourished and have metabolic acidosis. There may be intravascular volume depletion and evidence of hypovolaemic shock.

The initial fluid management to correct shock requires the goal-orientated approach already described with bolus therapy using isotonic Ringer's lactate. The sodium content of this fluid (131 mmol/l) does not contribute to the sodium load and possibly reduces the risk of a rapid fall in serum sodium level with fluid volume correction.

Following the correction of shock the dehydration should be corrected slowly over 24 - 48 hours to avoid a rapid decrease in serum sodium levels. The fall in serum sodium should not be allowed to exceed 1 mmol/h.

Conclusion

There is clearly a need for ongoing research in this area of management in under-resourced settings. Young infants with sepsis and evidence of shock should be carefully evaluated and monitored during resuscitation. Paediatric intensive care unit (PICU) management may

be needed to reduce mortality, bringing into focus the lack of availability of PICU facilities in the country.

IV antibiotics should be given early within the first hour in the management of septic shock.

Circulatory failure (shock) is an important cause of morbidity and mortality in children and results from fluid or blood loss, or fluid maldistribution within the circulatory system due to common problems such as gastroenteritis, sepsis, burns and trauma. Delayed or inadequate fluid resuscitation leading to prolonged ischaemia massively reduces the chances of intact neurological survival.

While many questions still remain with regard to resuscitation of children in low-resource settings with a high incidence of sepsis, by following existing guidelines aimed at effective, early stabilisation of the cardiovascular status in pre-hospital and emergency department settings, the outcome for in-hospital mortality will be reduced.

References available at www.cmej.org.za

IN A NUTSHELL

- Inadequate tissue perfusion (shock) should be recognised early in young children presenting with acute illness.
- Vascular access should not be delayed and insertion of intra-osseous lines may be life-saving techniques.
- Goal-directed therapy aimed at correcting shock within the first hour has been shown to reduce mortality.
- Isotonic crystalloid solutions are effective in the treatment of intravascular volume depletion due to fluid losses.
- Patients who are refractory to fluid management may require inotrope support, which can be given via the peripheral vascular route.
- Early administration of antibiotics reduces mortality in septic shock.
- Caution is advised when administering intravenous fluid to malnourished children. When shock is present IV fluids should be given at slower rates than those advised for the developed world.

References

1. Brierley J, Carcillo JA, Choong K, et al. Clinical practice parameters for 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.
2. Shapiro N. Serum lactate as a predictor of mortality in emergency department patients. *Ann Emerg Med* 2005;45(5):524-528.
3. Han YY, Carcillo JA, Dragotta MA, et al. Early reversal of pediatric-neonatal septic shock by community physicians is associated with improved outcome. *Pediatrics* 2003;112(4):793-799.
4. Myburgh J, Cooper DJ, Finfer S, et al., SAFE Study Investigators; Australian and New Zealand Intensive Care Society Clinical Trials Group; Australian Red Cross Blood Service; George Institute for International Health. Saline or albumin for fluid resuscitation in patients with traumatic brain injury. *New Engl J Med* 2007;357:874-884.
5. WHO. ETAT Manual for Participants. Geneva: WHO, 2005.
6. Parker M, Hazelnet JA, Carcillo J. Pediatric considerations. *Crit Care Med* 2004;32(11):S591-S594.
7. Maitland K, Kiguli S, Opoka RO, et al., for the FEAST Trial Group. Mortality after fluid bolus in African children with severe infection. *New Engl J Med* 2011;364(26):2483-2495.
8. Figaji AA. Targeted treatment of severe head injury. *CME* 2010;28(3):104-107.