African Journal of Emergency Medicine 7 (2017) S20–S26

Contents lists available at ScienceDirect

African Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/afjem

REVIEW ARTICLE

Perspectives on aetiology, pathophysiology and management of shock in African children

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ARTICLE INFO

Keywords:
Shock
Paediatric
Aetiology
Pathophysiology and management

ABSTRACT

Paediatric shock is still a common emergency of public health importance with an estimated 400,000–500,000 reported cases annually. Mortality due to paediatric shock has varied over the years. Data in 1980s show that mortality rates due to septic shock in children were over 50%; but by the end of the year 2000 data indicated that though a marked decline in mortality rates had been achieved, it had stagnated at about 20%. Descriptions of paediatric shock reveal the lack of a common definition and there are important gaps in evidence-based management in different settings. In well-resourced healthcare systems with well-functioning intensive care facilities, the widespread implementation of shock management guidelines based on the Paediatric Advanced Life Support and European Paediatric Advanced Life Support courses have reduced mortality. In resource limited settings with diverse infectious causative agents, the Emergency Triage Assessment and Treatment (ETAT) approach is more pragmatic, but its impact remains circumscribed to centres where ETAT has been implemented and sustained. Advocacy for common management pathways irrespective of underlying cause have been suggested. However, in sub Saharan Africa, the diversity of underlying causative organisms and patient phenotypes may limit a single approach to shock management.

Data from a large fluid trial (the FEAST trial) in East Africa have provided vital insight to shock management. In this trial febrile children with clinical features of impaired perfusion were studied. Rapid infusion of fluid boluses, irrespective of whether the fluid was colloid or crystalloid, when compared to maintenance fluids alone had an increased risk of mortality at 48 h. All study participants were promptly managed for underlying conditions and comorbidity such as malaria, bacteraemia, severe anaemia, meningitis, pneumonia, convulsions, hypoglycaemia and others. The overall low mortality in the trial suggests the potential contribution of ETAT, the improved standard of care and supportive treatment across the subgroups in the trial. Strengthening systems that enable rapid identification of shock, prompt treatment of children with correct antimicrobials and supportive care such as oxygen administration and blood transfusion may contribute to better survival outcomes in resources limited settings.

African relevance

- Shock in African children remain poorly defined.
- Paediatric shock is a leading cause for morbidity and mortality in Africa.
- There are no uniform guidelines for the management of shock in children.

Introduction

Paediatric shock is a common emergency that is responsible for high morbidity and mortality [1,2]. Globally, an estimated 400,000–500,000 reported cases of paediatric septic shock occur annually [3]. In the last three decades, there have been improvements in mortality outcomes in paediatric shock from rates of about 50% to 20% by the year 2000 [3]. However, 20% mortality from a single syndrome cause remains unacceptably high.

Peer review under responsibility of African Federation for Emergency Medicine.
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https://doi.org/10.1016/j.afjem.2017.10.002
Received 20 January 2017; Received in revised form 22 September 2017; Accepted 26 October 2017
Available online 21 November 2017

Table 1
Defining shock.

Shock criterion	Definition of shock
FEAST inclusion criteria [24]	History of fever or temperature ≥ 37.5 °C or < 36.0 °C Plus Impaired consciousness (prostration or coma) and/or respiratory distress (deep breathing or increased work of breathing) Plus ≥ 1 of: CRT > 2 s; lower limb temperature gradient; weak pulse; HR > 180 (< 12 months), > 160 (12 months–5 years), > 140 (> 5 years)
ACCCM clinical practice parameters for hemodynamic support of pediatric & neonatal septic shock (2007 update) [11]	Hypothermia or hyperthermia Plus Clinical signs of inadequate tissue perfusion including any of the following: decreased or altered mental status; CRT > 2 s, diminished pulses, mottled cool extremities (cold shock); flash capillary refill, bounding peripheral pulses, wide pulse pressure (warm shock); urine output < 1 ml/kg/h Hypotension not necessary for clinical diagnosis of septic shock, but its presence in a child with clinical suspicion of infection is confirmatory
Paediatric Advanced Life Support (PALS): 2010 American Heart Association guidelines for cardiopulmonary resuscitation [8]	No single sign confirms the diagnosis Typical signs of compensated shock include: tachycardia; cool and pale distal extremities; CRT > 2 s despite warm ambient temperature; weak peripheral pulses compared with central pulses; normal systolic blood pressure Decompensated shock characterised by signs & symptoms consistent with inadequate delivery of oxygen to tissues (pallor, peripheral cyanosis, tachypnoea, mottling of skin, decreased urine output, metabolic acidosis, depressed mental status); also weak or absent peripheral pulses, weak central pulses, hypotension (systolic BP < 70 mmHg 1–12 months; < 70 mmHg + (2 × age in years) 1–10 yrs; < 90 mmHg ≥ 10 years)
2016 Surviving Sepsis 3 definitions [72]	**Sepsis** is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Organ dysfunction can be identified as an acute change in total SOFA (Sequential [Sepsis-related] Organ Failure Assessment) score ≥ 2 points consequent to the infection. The baseline SOFA score can be assumed to be zero in patients not known to have preexisting organ dysfunction. A SOFA score ≥ 2 reflects an overall mortality risk of approximately10%in a general hospital population with suspected infection. Even patients presenting with modest dysfunction can deteriorate further, emphasizing the seriousness of this condition and the need for prompt and appropriate intervention, if not already being instituted **Septic shock** is a subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality. Patients with septic shock can be identified with a clinical construct of sepsis with persisting hypotension requiring vasopressors to maintain MAP(mean arterial pressure) ≥65 mmHg and having a serum lactate level > 2 mmol/L (18 mg/dL) despite adequate volume resuscitation. With these criteria, hospital mortality is in excess of 40%
European Paediatric Life Support Course (2006) [9]	Compensated circulatory failure: although the blood pressure is normal, poor skin perfusion (CRT > 2 s, mottled, cool peripheries, peripheral cyanosis), weak peripheral pulse, tachycardia: HR > 180 (3 months–2 yrs); > 140 (2–10 yrs); > 100 (> 10 yrs), tachypnoea (RR > 40 < 1 yr; > 34 (1–2 yrs); > 30 (2–5 yrs); > 24 (5–12 yrs) and oliguria are observed Decompensated circulatory failure: hypotension (SBP < 70 mmHg (1–12 months); < 70 + (2 × age in yrs) mmHg (1–10 yrs); < 90 mmHg (> 10 yrs); decreased mental status
WHO/ETAT [73,74]	The presence of cold hands or feet with both capillary refill time > 3 s and weak and fast pulse

Table 2
Describing shock.

Classification of shock	Description
Compensated shock or impaired circulation	The early phase of shock in which the body's compensatory mechanisms (such as increased heart rate, vasoconstriction, increased respiratory rate) are able to maintain adequate perfusion to the brain and vital organs. Typically, the patient is normotensive in compensated shock [75]
Decompensated shock or severely impaired circulation	The late phase of shock in which the body's compensatory mechanisms (such as increased heart rate, vasoconstriction, increased respiratory rate) are unable to maintain adequate perfusion to the brain and vital organs. Typically, the patient is hypotensive in decompensated shock [75]

Shock has been defined in various ways (Table 1), but terms such as compensated shock, decompensated shock, impaired circulation and severely impaired circulation are used and sometimes interchanged. Table 2 describes these terms. The cardinal clinical features and laboratory markers for shock are well documented, but its clinical recognition may be challenging especially in resources-limited settings. Moreover, delay in identifying and promptly treating shock often results in progression to metabolic deregulation especially metabolic acidosis [1,2]. Compensated shock may advance into clinically unredeemable decompensated states with fatal organ dysfunction [4]. While disability especially among neonatal shock survivors is not infrequent [5], it is very rare to have established shock resolve spontaneously without appropriate treatment [6]. In an attempt to improve recognition, treatment and outcomes of paediatric shock in resources-limited settings, training courses such as ETAT (Emergency Triage Assessment and Treatment) [7]; PALS (Paediatric Advanced Life Support) [8]; EPLS (European Paediatric Life Support Course) (2006) [9]; have been developed to equip clinicians with skills in the rapid assessment and treatment modalities of children with shock [10]. The overall impact on child survival where these initiatives have been successfully implemented is tangible [10]. Nevertheless clinicians, especially in resource-limited settings, still find it challenging to diagnose shock and take initial management decisions for a number of reasons. Firstly, while clinical shock is common, there are a confusing number of different definitions (Table 1). Secondly, there are varied practices with a striking lack of consensus on definitive policies and guidelines on the management of paediatric shock. Thirdly, there are conflicting schools of thought about fluids; on one hand, rapid fluid administration, as