

What's new in paediatric trauma resuscitation?

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Paediatric cardiac arrest

The most recent modifications to the American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care were published in 2006.¹ In this publication the critical role that high-quality and continuous chest compressions play in successful resuscitation has been emphasised. Compressions should be done hard and fast, at a rate of about 100 compressions per minute, allowing time for the chest to fully recoil. Continuity while giving compressions is vital and every effort should be made to minimise interruptions. A compression to ventilation ratio of 30:2 should be used in all patients and each breath should be given over 1 second, producing a visible chest rise. In paediatric patients this ratio should be adjusted to 15:2 for 2-person CPR. Defibrillation should be followed by 5 cycles or 2 minutes of chest compressions before checking for a rhythm.

While a respiratory cause of cardiac arrest carries with it a better prognosis, arrest following blunt trauma has a particularly poor outcome, with survival rates of 1 - 6%, and survivors may have severe neurological deficits.² Despite sustaining significant injury, the long-term quality of life in patients with head injury who do not undergo cardiac arrest is high.³ The use of hypothermic therapy following cardiac arrest in adult populations has shown improved neurological outcomes. Due to differences in the mechanisms of cardiac arrest between adult and paediatric patients therapeutic hypothermia has not been shown to be as effective, and further study in this field has been suggested.⁴ In addition, the applicability of this modality of treatment to a trauma population where patients often present with severe hypothermia is uncertain.

Airway management and ventilation

Intubation remains the definitive method of airway management in the trauma patient but presents a greater challenge in the paediatric population than in adults.

Table I. LEMON mnemonic for the evaluation of a difficult airway

L-ook	For trauma, large incisors, large tongue
E-valuate	Mouth opening, inter-incisor distance
M-allampati	Perform a Mallampati score
O-bstruction	Identify obstructions in the airway
N-eck	Evaluate neck flexion, extension and thickness

Prehospital intubation performed by health care providers with little paediatric airway experience has led to unacceptably high complication rates.⁵ In these cases airway management should be achieved by means of bag-valve-mask-ventilation. When the practitioner has specific paediatric airway skills, prehospital intubation can be performed safely and successfully.⁶

Fluid resuscitation should be targeted at the restoration of organ perfusion and to this end lactate, base deficit and central venous oxygenation may have clinical utility, particularly in the first 24 hours following injury.

Intubations should be performed using a rapid sequence induction technique and tracheal intubation confirmed with an exhaled CO₂ detector in both the pre- and intra-hospital setting.⁷ The use of the mnemonic LEMON has been suggested as a tool for pre-intubation airway evaluation (Table I).³ Although useful in drawing attention to the potential difficult airway, the utility of the (e)valuation and (m)allampati components of the mnemonic in the paediatric trauma patient is doubtful. When faced with a difficult airway the gum elastic bougie and the laryngeal mask airway (LMA) may be useful. The use of cuffed endotracheal tubes in the paediatric population is increasing. They provide additional airway protection together with reductions in air leaks and if appropriate sizes are used and cuff pressures are monitored (<20 cm H₂O), are not associated with an increase in tracheal stenosis.⁸

Over-ventilation results in alkalosis, increased intrathoracic pressures with subsequent impairment of venous return and reduces the chance of a successful resuscitation. Once the patient is intubated, 8 - 12 breaths should be delivered per minute, which can be increased to between 12 and 20

per minute once a perfusing rhythm has been established.

Fluid and venous access

Hypovolaemic shock in paediatric patients may be difficult to appreciate. Together with a tachycardia the signs of peripheral vasoconstriction – cold, mottled extremities and delayed capillary refill time – assume greater importance than in adults.⁹ Normal saline, Ringer's lactate or low substitution hydroxyethyl starches should be used as first-line agents in resuscitation and should be administered in boluses of 10 - 20 ml/kg of crystalloid or 7 - 10 ml/kg of colloids.¹⁰ Care should be taken to avoid over-resuscitation with crystalloids as this may lead to the development of multiple systemic complications, including abdominal compartment syndrome.¹¹ The use of hypertonic saline may have a role to play in the management of the patient with head trauma but is currently not recommended except within the context of clinical trials. In the hospital setting paediatric patients are at significant risk of developing hyponatraemia due to their retention of water during physiological stress. The use of hypotonic fluids such as half Darrow's dextrose (DD), half normal saline or 5% dextrose in the paediatric trauma population is strongly discouraged.¹²

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Fluid resuscitation should be targeted at the restoration of organ perfusion and to this end lactate, base deficit and central venous oxygenation may have clinical utility, particularly in the first 24 hours following injury.¹³ When haemorrhage is ongoing fluid administration should be limited and a balance struck between maintaining organ perfusion and avoiding exsanguination, even if this means allowing a lower than normal blood pressure in the patient. Cognisance

should be taken that the normal paediatric blood volume is 70 - 80 ml/kg and thus blood loss may often be underestimated. Care should be taken to avoid hypotension in patients who have sustained blunt trauma with associated head injury.³

Venous access remains a challenge in the paediatric population. After 3 unsuccessful attempts at percutaneous line placements an intraosseous tibial, femoral or central venous line should be placed immediately. These techniques have largely replaced the use of the saphenous cutdown, which should now be seen as a technique of last resort.¹⁴

Temperature management

Temperature management is often omitted during paediatric trauma management.¹⁵ The relatively large body surface area of children, ineffective vasoconstriction and cold fluid infusion may combine to rapidly cause hypothermia. Care should be taken to avoid unnecessary exposure, to keep the patient dry and to provide a warm environment during resuscitation. Forced air warmers and in-line fluid warmers are essential in trauma patient management.

References available at www.cmej.org.za

Duration of antimicrobial therapy

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The appropriate duration of therapy is often open to question. Extended antimicrobial therapy is associated with selection of resistant organisms, adverse events, expense and poor patient compliance. Evidence suggests that a short duration of treatment is as effective as a longer course of treatment for certain common community-acquired infections, such as acute otitis media, acute bacterial sinusitis, infectious exacerbations of chronic bronchitis, community-acquired pneumonia, and acute pyelonephritis.¹

Acute otitis media

In a meta-analysis (MA) comparing short-duration with long-duration therapy, treatment of fewer than 7 days was compared with treatment of more than 7 days. No statistical difference was found between the 5-day and 8 - 10-day regimens, with primary outcome defined as treatment failure (lack of clinical resolution, relapse or recurrence).²

Acute bacterial sinusitis

In an MA involving adult patients with radiologically confirmed sinusitis no difference in clinical outcome was found between a 3 - 7-day course compared with a 6 - 10-day course of treatment. Comparing a 5-day with a 10-day regimen revealed fewer adverse events with short-course treatment.³

Chronic bronchitis

Falagas *et al.*⁴ studied 7 randomised controlled trials (RCTs) of patients with acute exacerbations of chronic bronchitis, comparing short-duration (5 days) with long-duration (7 or 10 days) antimicrobial treatment. No difference was found between the two groups with regard to treatment success.

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Community-acquired pneumonia

A short-treatment arm of 3 - 7 days was compared with a long-treatment arm of 7 - 10 days in an MA of 7 RCTs. No difference was found with regard to clinical success at the end of therapy, microbiological success, relapses, mortality or adverse events.⁵

Acute pyelonephritis

No difference was found between short-duration treatment (7 - 14 days) compared with long-duration treatment (14 - 42 days) in terms of clinical success, relapse, recurrence or bacteriological efficacy in an MA of 4 RCTs.⁶

Cystitis

Three days of treatment was compared with 5 - 10 days of treatment in women with cystitis. Although there was no difference in symptomatic failure between the two groups, bacteriological cure rates were improved with longer duration of treatment, both at 2 weeks and at 8 weeks of follow-up.⁷ However, in another MA of elderly women with cystitis no difference was found with regard to persistence of the infection.⁸

For certain community-acquired infections duration of therapy can be shortened, with a resultant reduction in cost, better patient compliance, less selection pressure for resistant organisms and fewer adverse effects. On the other hand, infections such as endocarditis, osteitis, and septic arthritis, to name a few, still require extended duration of antimicrobial therapy.

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Cardiovascular protection in type 2 diabetes mellitus

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The cardiovascular (CV) event rate in patients with type 2 diabetes mellitus (DM) is 2 - 4 times higher than the population average, making macrovascular atherosclerotic disease the commonest cause of death in patients with type 2 DM. CV events are not only more frequent in patients with type 2 DM, but are also associated with higher morbidity and mortality.¹ Coronary artery disease, for example, tends to be more extensive and diffuse in diabetic patients and outcomes are worse after myocardial infarction or revascularisation. Type 2 DM is therefore best regarded as a cardiometabolic disorder (high CV risk state with elevated blood glucose) rather than a pure metabolic disorder in which the elevated blood glucose is the primary abnormality and focus of therapy. Type 2 DM keeps 'bad company': hypertension and dyslipidaemia are highly prevalent and combine with dysglycaemia to damage the vascular system. Single risk factor control is therefore inadequate for CV event prevention - removing only one gang member from the street does not solve the crime problem. Trials that simultaneously address multiple risk factors have reduced the CV event rate most, but even in these trials the residual (unprevented) risk remains unacceptably high.² Finding new strategies and treatments to reduce residual risk is high on the research agenda.

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CV protection in type 2 DM therefore requires multifactorial intervention. Lifestyle advice remains central to CV risk reduction. Smoking cessation must be pursued aggressively, regular exercise (within the patient's limitations) should be encouraged and dietary advice may help with lipid, glycaemic and blood pressure (BP) control. BP and lipid control are the 'low

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hanging fruit' of CV protection – they are often easier to achieve than tight glycaemic control and the numbers needed to treat (NNT) are lower.³

NNT for antihypertensive therapy (10/5 mmHg BP reduction over 10 years) to prevent a cardiac event is 26 and 49 for stroke, basing calculations on the event rates found in the United Kingdom Prospective Diabetes Study (UKPDS) control group.³ The target BP is 130/80 mmHg. The antihypertensive regimen should be based on either an angiotensin-converting enzyme (ACE) inhibitor or an angiotensin-receptor blocker (ARB). Diuretics, calcium-channel blockers and other agents may be added to achieve BP control. Most patients require at least two drugs for BP control.

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Reducing cholesterol by 1 mmol/l for 10 years will prevent one cardiac event for every 25 patients treated and one stroke for every 118 patients treated.³ The primary lipid target is low-density lipoprotein cholesterol (LDLC). The LDLC target for patients clinically free of CV disease is <2.5 mmol/l, while a target of <1.8 mmol/l is desirable for those with CV disease if health care resources are adequate.

All patients with CV disease should receive a statin, irrespective of baseline lipid levels. Patients older than 40 years with one other CV risk factor or younger patients with multiple risk factors also need treatment. In clinical practice almost all patients with type 2 DM require a statin. The question is not which patients to treat but whether not treating a particular patient can be justified. Moderate hypertriglyceridaemia and low high-density lipoprotein cholesterol (HDL) are common in diabetes and treatment of these abnormalities is a promising avenue to reduce residual risk. In a recent study the routine addition of a fibrate to statin therapy did not lower risk, except in the subgroup with the lowest HDL and highest triglycerides.⁴ Combination lipid-lowering therapy is therefore not routinely indicated and should only be initiated at specialist level.

In epidemiological studies there is a clear and consistent link between glycaemia and CV outcomes. In practice it has been difficult to prove that tight glycaemic control reduces not only microvascular complications (which has been conclusively proven) but also macrovascular events. The extensive and often controversial literature on the subject may perhaps be summarised as follows: Tight glycaemic control early in the disease course lowers CV events but the benefits are only seen after many years of follow-up. In older patients with established CV disease and other co-morbidities tight glycaemic control (HbA_{1c} <7.0%) often requires multiple drugs and complex insulin regimens and may be associated with harm.^{1,3} The NNT for glycaemic control (HbA_{1c} reduced by 1%) is

also higher than that for lipid or BP control, and is estimated to be 41 for coronary heart disease and 400 for stroke.³ It is therefore important to aim for tight glucose control early in the disease course when patients are still free of co-morbidities and glycaemic control usually requires less complex therapy. These patients are likely to benefit from their 'banked good glycaemic control years' when they are older. Aiming for very tight glucose control in older and sicker patients is likely to cause more harm than benefit.

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Unless contraindicated, all patients with clinically overt CV disease should take aspirin or clopidogrel (if allergic to aspirin). For the benefits of aspirin to outweigh its risks, prescription for primary prevention should be limited to those with an estimated 10-year risk of >10%. This risk level is generally found in men over 50 years and women over 60 years with at least one other major CV risk factor.⁵

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