

Point of view

Monitoring children after cardiac surgery: a minimalist approach might be maximally effective

Infants after complex heart surgery have arguably the highest rates of adverse events of any major group of children in hospitals of developed countries. Cardiac arrest, cardiopulmonary resuscitation or need for emergency extracorporeal life support, have reported rates varying between 5.9%¹ and 10%² in busy cardiac centres with a one study reporting a one-year survival of 19% in 82 children after postoperative cardiac arrest.¹ Cardiac arrest increases the risk of multi-organ failure, prolongs the requirement for mechanical ventilation and intensive care unit (ICU) stay, causes long term neurological morbidity and carries a huge financial cost.

The descriptions of many adverse events as “sudden” or “unpredictable” reflects our incomplete understanding about risk identification, limitations of routine non-invasive and invasive haemodynamic monitoring and limitations in the evidence published so far. Adverse events are often preceded by a period of inadequate oxygen balance. However, there has been a consistent failure of studies in adults to demonstrate that the use of pulmonary artery catheters help predict patient outcomes or, when applied to guide therapy, improve outcomes.³ Moreover, the risks of invasive haemodynamic monitoring are greater in children than adults.

What we know about predictors of major adverse events

There are several studies of clinical and laboratory risk factors for major adverse events.^{1,2,4-8} One third of cardiac arrests occur in the first 24 hours after ICU admission,¹ so prediction for post-operative planning is best if it can be done around the time of the ICU admission. Table 1 summarises the factors that can be identified at the time of ICU admission which indicate an increased risk of adverse events, including cardiac arrest and death. Comparing studies is difficult as the definitions of predictive variables differ (although young age and low weight could be seen as broadly similar, as could pre-operative congestive cardiac failure and the need for mechanical ventilation). There are major differences in these studies in outcomes of

interest, risk factors examined, case-mix and variations in ICU management. Also, most studies are small, so the risk of a type-two error is high.

However, several risk factors have been identified in two or more studies. These include, young age, durations of cardiopulmonary bypass and aortic cross-clamping, presence and duration of circulatory arrest, higher surgical complexity, delayed closure of the sternum and pre-operative congestive cardiac failure/need for pre-operative mechanical ventilation/pre-operative ICU admission. In the largest study where cardiac arrest was the major adverse outcome, pre-operative need for inotropes and high inotrope requirement at the time of separation from bypass were important predictors.¹ Another study found hypotension on arrival in the ICU one of the earliest predictors of cardiac arrest and other major adverse events.⁸ (Table 1)

In addition to these early clinical indicators of high risk, some biochemical markers may be used to evaluate whether adverse outcomes are more likely, or whether interventions are having a beneficial effect. Biochemical markers that have been shown to predict major adverse events are also markers of inadequate oxygen delivery. Arterial blood lactate levels are the best currently available biochemical predictor of adverse outcomes in infants after cardiac surgery (Table 2).⁸⁻¹³ However, even lactate levels above 4.5 - 6 mmol/L, despite being optimally predictive, have only low to moderate sensitivity. Depending on the threshold used, lactate levels will miss up to two-thirds of children with subsequent adverse events.^{8,14} Moreover, lactate levels above this range have a low positive predictive value (e.g. around 30%).^{8,10,11} The low positive predictive value may be because oxygen debt resulting in lactic acidosis is only one reason for hyperlactataemia after cardiopulmonary bypass. Other reasons include impaired lactate clearance,^{15,16} the age of the child and the type of bypass prime solution used. An upward trend in lactate may be a more specific predictor of major adverse events than the absolute value as a rise in lactate of > 0.75 mmol/L over an hour was found to be more predictive of death after cardiac surgery than a lactate value of 6 mmol/L or more at the time of ICU admission (Table 2).

Knowing the risk factors for adverse events is only useful clinically if it is possible to intervene and improve outcome. Surprisingly there are few controlled trials published comparing various general strategies for monitoring and management of infants after cardiac surgery.

In adults after cardiac surgery, a management protocol aimed at achieving a lactate < 2.2 mmol/L and central venous oxygen saturation (SvO₂) > 70% in the first 8 hours resulted in a shorter length of hospital stay and a lower rate of extra-cardiac organ dysfunction at

Table 1. Early clinical predictors of major adverse events after cardiac surgery in children

Studies	Brown et al, ²	Malviya et al, ⁴	Ben-abraham et al, ⁵	Valsangiacomo et al, ⁶	Suominen, et al ¹	Valvera et al, ⁷	Duke, et al ⁸
Prediction	ICU stay >14 days	Respiratory complications	Death	Arrhythmias	Cardiac arrest	Nosocomial infection	Major adverse events *
Total number of cases/incidence of adverse events (%)	355 (12%)	713 (29.2%)	70 (5.9%)	100 (48%)	1399 (5.8%)	104 (48.1%)	90 (13%)
Risk factors							
Pre-op PGE ₁					Yes		
Pre-op inotropes					Yes		
Pre-op mechanical ventilation	Yes						
Pre-op ICU stay						Yes	
Pre-op CCF			Yes				
Pre-op upper respiratory infection		Yes					
International transfer	Yes						
Low weight	Yes			Yes			
Young age/neonatal			Yes				
Cardiopulmonary bypass time	Yes			Yes	Yes	Yes	Yes
Aortic cross-clamp time					Yes		Yes
Circulatory arrest	Yes				Yes		Yes
Valve prosthesis						Yes	
Surgical complexity	Yes			Yes			
Cyanotic CHD						Yes	
High inotropes on leaving operating room					Yes		
Hypotension on arrival in the ICU							Yes
Delayed closure of chest	Yes					Yes	

ICU = intensive care unit, CCF = congestive cardiac failure, CHD = congenital heart disease

* Major adverse events were cardiac arrest, need for emergency chest opening or extracorporeal support, death or multiple organ failure

the time of hospital discharge. These physiological aims were achieved using fluid volume and dobutamine up to an infusion rate of 15 µg/kg/min.¹³ In a similar trial in adults with sepsis presenting to an emergency department, a protocol that included interventions if the SvO₂ was < 70%, resulted in lower mortality than when management was based only on targets for blood pressure, central venous pressure and urine output.¹⁷

While the comparison with cardiac surgery in infants might seem distant, two messages from these adult studies are relevant: first, simply performed summary measures that reflect the adequacy of oxygen delivery add substantially to information derived from monitoring pressure or flow; and second, protocols improve outcome compared with management left to the discretion of individual clinicians.

Lactate and venous oxygen saturation in infants

Venous oxygen saturation may be a useful adjunct to arterial blood lactate in the routine monitoring of infants after complex cardiac surgery. Lactate and SvO₂ reflect different steps in the pathophysiology of tissue hypoxia and under specific circumstances an abnormality in one measure will be present without an abnormality in the other (Table 3). Changes in cardiac output in the first 8 hours after cardiac surgery are paralleled by changes in SvO₂ (Figure 1). In one study, SvO₂ increased the predictive power of arterial blood lactate levels for mortality after cardiac surgery: in children with a lactate of 7.7 mmol/L, the risk of mortality increased from 10% if the SvO₂ > 60% to 80% if the SvO₂ was < 60%.¹²

Venous oxygen saturation might help clarify the significance of small changes in lactate and might help overcome the low sensitivity and low positive predict-

Table 2. Lactate and prediction of outcome in children after cardiac surgery

Study	Number	Lactate (mmol/L)	When	Prediction	Sensitivity	Specificity	PPV	NPV	Comments
Shime, et al ¹⁴	141	> 3	ICU Ad.	Mortality (7.8%)	64	87			Higher threshold (>4 mmol/L) had lower sensitivity (36%)
Shime, et al ⁹	112	> 2.2	16 hours after Ad.	Mortality (5.4%)	82	72			Failure of lactate to fall to normal levels by day 1 was a sensitive predictor of mortality
Charpie, et al ¹⁰	46 neonates	> 6	ICU Ad.	Poor outcome * (19.6%)	88	65	38	96	Increase in blood lactate by 0.75 mmol/L or more per hour a better predictor than absolute value
Hatherill, et al ¹¹	99	> 6	ICU Ad.	Mortality (9.1%)	78	83	32		Low PPV and much overlap between survivors and non-survivors, authors concluded that lactate not useful in predicting mortality
Duke, et al ⁸	90	> 4.5	ICU Ad.	MAE * (13.3%)	33	91	36	90	Lactate an early predictor of MAE, although sensitivity low

Ad. = admission, ICU = intensive care unit, MAE = major adverse event, PPV = positive predictive value, NPV = negative predictive value

* Poor outcome: death or need for extracorporeal support

** Major adverse events were cardiac arrest, need for emergency chest opening or extracorporeal support, death or multiple organ failure

Table 3. Different combinations of lactate and SvO₂ results may reflect different pathophysiology processes or different stages in the development of inadequate oxygen delivery

	Fall in MvO ₂	Normal MvO ₂
Rise in lactate	Inadequate tissue oxygen delivery with maximal oxygen extraction and anaerobic metabolism	Left-to-right shunt Sepsis Alkalosis Regional ischaemia (e.g. bowel ischaemia) Adequate oxygen delivery but reduced lactate clearance Washout of lactate following adequate restoration of the circulation after global or regional ischaemia Clear bypass prime
Normal lactate	Low tissue oxygen delivery with increased oxygen extraction, but not having reached the state of anaerobic metabolism [Low sensitivity of lactate]	[Low sensitivity of MvO ₂] Adequately functioning circulation [Inadequate sensitivity of both tests]

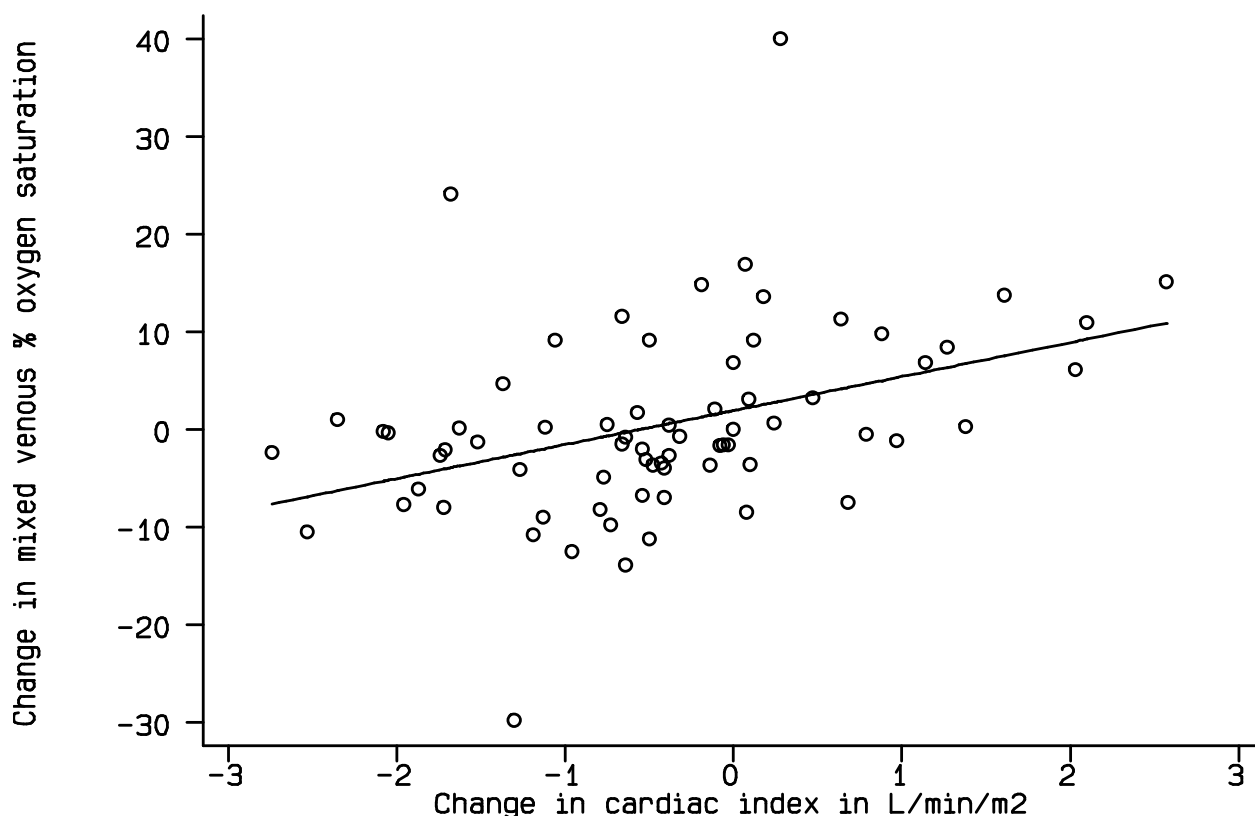


Figure 1. Changes in SvO₂ and cardiac output in the first 8 hours after cardiac surgery in 71 children

ive value of lactate. For example more attention might be appropriately paid to a child with an arterial blood lactate that had risen from 3.0 to 3.8 mmol/L if the SvO₂ had fallen in the same period from 59% to 45%. However, it is important to note that only one study in children after cardiac surgery has established the independent predictive power of SvO₂ and lactate.¹²

In conclusion, it may be possible to be forewarned about most major adverse events occurring after cardiac surgery in children using a framework for risk estimation based on demographic and clinical predictors and biochemical markers of inadequate oxygen delivery. We have developed "Guidelines for monitoring arterial lactate and central venous oxygen saturation after cardiac surgery" in children (Appendix 1) and await the results of a trial that would evaluate the clinical efficacy of such guidelines.

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APPENDIX 1

Guidelines for monitoring arterial blood lactate and central venous oxygen saturation after cardiac surgery

Lactate: a high or rising lactate indicates an increased risk for major adverse events:

- Admission lactate: > 5 mmol/L (30% risk of major adverse events)
- Lactate at 4 hours: > 4 mmol/L (45% risk of major adverse events)

SvO₂: Ideally measured from a pulmonary artery catheter (true mixed venous if there is no left-to-right shunt). If no pulmonary artery catheter, or there is a left-to-right shunt, measure from central venous catheter. Use the same catheter for serial measurements.

- SvO₂ changes in the same direction as cardiac output
- A falling SvO₂ indicates increased tissue oxygen extraction in response to falling cardiac output.

A rising lactate and a falling SvO₂ means inadequate tissue oxygen delivery, despite increased tissue oxygen extraction.

At PICU admission measure arterial lactate and PA or central venous SO₂

If: lactate > 4, and / or SvO₂ < 60% in a fully corrected heart (or the SaO₂-SvO₂ gradient > 35% in uncorrected lesions), and / or mean arterial pressure low.

1. *Correct deficits in oxygen delivery:*

- Maintain adequate circulating volume
- Maintain haemoglobin 120 - 140 g/L
- Do not cease inotropes on first post-operative night.
- Add vasodilators if hypertensive
- Optimise arterial oxygen saturation *

2. *Minimise oxygen consumption*

- Keep well sedated with morphine; use muscle relaxants; \pm fentanyl before tracheal suction.
- If temperature > 37.5°C: cool to normothermia.

3. *Maintain sedation \pm paralysis, and do not wean ventilation on first post-operative night* unless specific reason, e.g. Fontan, Cavopulmonary shunt, and then only with frequent reassessment of perfusion and consultant approval.

4. Commence peritoneal dialysis early if oliguria or oedema develops.

Repeat arterial lactate and SvO₂ at 2-4 hourly intervals on first post-op night:

If lactate rising (OR > 4 mmol/L) or SvO₂ falling at any stage:

- Notify ICU consultant and cardiac surgeon.

Check cardiac rhythm; echocardiograph to examine cardiac function and exclude tamponade; check haemoglobin, optimise intravascular volume, consider: increasing dopamine or dobutamine; adding another inotrope if MAP low; possible detrimental effects of high-dose catecholamine constrictors; need for vasodilators; may benefit from chest opening; may need mechanical support.

* Optimal FiO₂ and SpO₂ will be modified in a Norwood circulation