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PEDIATRIC ORIGINAL

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The lactate:pyruvate ratio following open cardiac surgery in children

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Abstract *Objective:* To explore the relationship between lactate:pyruvate ratio, hyperlactataemia, metabolic acidosis, and morbidity. *Design and setting:* Prospective observational study in the paediatric intensive care unit (PICU) of a university hospital. *Patients:* Ninety-seven children after open cardiac surgery. Most children (94%) fell into low-moderate operative risk categories; observed PICU mortality was 1%. *Interventions:* Blood was sampled on admission for acid-base analysis, lactate, and pyruvate. Metabolic acidosis was defined as standard bicarbonate lower than 22 mmol/l, raised lactate as higher than 2 mmol/l, and raised lactate:pyruvate ratio as higher than 20. *Measurements and results:* Median cardiopulmonary bypass and aortic cross-clamp times were 80 and 46 min. Metabolic acidosis occurred in 74%, hyperlactataemia in 42%, and raised lactate:pyruvate ratio in 45% of children. In multivariate analysis lactate:pyruvate ratio increased by 6.4 in children receiving epinephrine infusion and by 0.4 per 10 min of aortic cross-clamp. Duration of inotropic support increased by 0.29 days, ventilatory support by 0.27 days, and PICU stay by 0.42 days, for each 1 mmol/l increase in lactate. Neither standard bicarbonate nor lactate:pyruvate ratio were independently associated with prolongation of PICU support. *Conclusions:* Elevated lactate:pyruvate ratio was common in children with mild metabolic acidosis and low PICU mortality. Hyperlactataemia, but not elevated lactate:pyruvate ratio or metabolic acidosis, was associated with prolongation of PICU support. Routine measurement of lactate:pyruvate ratio is not warranted for children in low-moderate operative risk categories.

Keywords Lactate · Pyruvate · Acidosis · Child · Cardiac surgery

Introduction

Post-operative blood lactate level is correlated with outcome after cardiac surgery in children and is associated with the rate of post-operative adverse events and paediatric intensive care unit (PICU) mortality [1–4]. Based on these prognostic data, serial lactate levels have been incorporated into goal-directed therapeutic algorithms in the post-operative period [5–7]. Lactate is generated by the reaction of pyruvate with NADH, as a consequence of anaerobic glycolytic breakdown of glucose to pyruvate during oxygen-limited energy depletion (dysoxia) [8]. Since lactate requires either oxygen for re-conversion into pyruvate, or energy for conversion to glucose, excess lactate accumulates under anaerobic conditions [8]. However, lactate may also accumulate during accelerated aerobic glycolysis driven by epinephrine, or if renal and hepatic lactate clearance is reduced [8, 9]. Under these conditions even transient increases in exogenous lactate delivery via red blood cell transfusions or cardiopulmonary bypass pump prime may result in hyperlactataemia and trigger escalation of haemodynamic support [5, 7, 10–12]. It follows that

mild lactic acidosis does not always signify regional tissue hypoxia or intraoperative oxygen debt [9, 13, 14].

Associations between cardiac surgical mortality, adverse events, and post-operative lactic acidosis were documented a decade ago among children with mortality rates of up to 17% [1–4]. Pediatric cardiac surgical mortality has since fallen to 5% or less in some centres [5–7]. Admission lactate measurement may no longer provide accurate prognostic data, particularly among patients with low mortality rates, and alternative markers of tissue dysoxia and low cardiac output syndrome should be sought [15]. Unfortunately, measures of the magnitude of metabolic acidosis such as standard bicarbonate have limited value, due to the frequency of non-lactic metabolic acidosis caused by unmeasured anions or hyperchloraemia, as we have described previously [3, 15–17].

We speculated that the lactate:pyruvate ratio would be a more discriminating marker of occult tissue dysoxia and morbidity following cardiopulmonary bypass than the lactate level alone. The lactate:pyruvate ratio is a marker of cytosolic redox potential which is elevated in both cardiogenic and septic shock and is associated with early mortality [18–20]. The combination of elevated lactate and lactate:pyruvate ratio is also associated with increased mortality in critically ill adults [21]. However, these findings contrast with the early work of Weil and Afifi [22] who found that considering the lactate:puyruvate ratio adds no value to the lactate level in predicting survival from circulatory shock. This study was designed to elucidate the relationships between the lactate:pyruvate ratio, lactate levels, metabolic acidosis, and morbidity amongst children undergoing open cardiac surgery at this referral centre in Cape Town, South Africa.

Materials and methods

Setting

The study was performed in the PICU of a university children's hospital over a 1-year period from February 2003 to March 2004. This regional PICU is staffed for 18–22 beds, admits approx. 1,200 children per year, and has full-time paediatric intensivist cover. The children's hospital functions as the paediatric cardiology and cardiothoracic surgical referral centre for the Western Cape Province, which has a population of approx. 4 million. Between 250 and 300 paediatric cardiac operations are performed per annum, of which approx. 170 are open cases using cardiopulmonary bypass. However, neither surgical palliation of hypoplastic left heart syndrome nor extracorporeal membrane oxygenation were offered at this centre during the study period.

Patients

Children were eligible for enrolment following surgical correction of congenital or acquired heart defects if they had undergone open cardiac surgery on cardiopulmonary bypass and required haemodynamic support in the form of either inotrope infusion or correction of intravascular volume depletion at the time of admission to PICU. Eligible children were enrolled with the informed consent of a parent or guardian during the duty periods of the principal investigator (M.H.), and were not pre-selected on the basis of diagnosis or surgical complexity. The study was approved by the university research ethics committee.

During the study period 278 paediatric cardiac surgical procedures were performed, 169 of which were performed on cardiopulmonary bypass. The study group in the present examination consisted of 97 of these children, whose demographic, clinical, and biochemical data are summarised in Table 1. Inotropic support was being received by 95 children on admission to PICU, 11 of whom 11 received epinephrine by continuous infusion. The group included 91 patients (94%) who fell into Risk Adjustment in Congenital Heart Surgery (RACHS-1) categories 2 or 3. Median predicted mortality was 2%, and there was only a single death; observed mortality in the study group did not differ significantly from that among cardiopulmonary bypass patients overall (2.4%).

Routine management

The fluid used to prime the cardiopulmonary bypass circuit (the 'pump prime') was a mixture of blood and stabilised human serum (SHS), a colloid prepared by the regional blood transfusion service (Western Province Blood Transfusion Service, Parow, South Africa). In the case of severe pre-operative polycythaemia SHS alone was used. Blood and SHS were the fluids of choice for intra-operative volume resuscitation, in preference to 0.9% saline. Lactate-containing solutions such as Ringer's lactate were not used for intra-operative fluid resuscitation. Dopamine or dobutamine were the first line intra-operative inotropic agents of choice and epinephrine might be added for haemodynamically unstable patients with suspected low cardiac output. Post-operative PICU management might include the use of Ringer's lactate and alternative inotropic agents, such as the phosphodiesterase inhibitor milrinone, but these changes were made after admission blood sampling. The administration of sodium bicarbonate for correction of metabolic acidosis, whether due to hyperchloraemia or to hyperlactataemia, was not recommended PICU practice at the time of the study.

Table 1 Biochemical and clinical data are median, interquartile range (*IQR*), range

Parameter	Median	IQR	Range
Age (months)	56	19–95	0–166
Weight (kg)	14	9.1–22.6	2.1–50
Bypass time (min)	80	60–115	17–232
Aortic cross-clamp time (min)	46	27–65	0–149
Risk of mortality (PIM)	0.02	0.02–0.06	0.01–0.59
pH	7.38	7.32–7.430	7.17–7.61
Standard bicarbonate (mEq/l)	20.1	18.4–22	10.6–28.8
Lactate (mmol/l)	1.8	1.3–2.5	0.7–9.1
Pyruvate (mmol/l)	0.10	0.07–0.15	0.04–0.39
Lactate:pyruvate ratio	19	14.6–22.5	5.4–38
Sodium (mmol/l)	138	136–140	129–146
Chloride (mmol/l)	111	109–114	97–121
Albumin (mmol/l)	30	27–33	16–44
SIG (mEq/l)	0.7	–2.7 to 3.6	–13.7 to 14.8
Duration of mechanical ventilation (days)	2	2–3	1–16
Duration of inotropic support (days)	3	2–3	0–10
Duration of PICU stay (days)	4	3–4	2–20

Blood sampling and data collection

Immediately on admission to PICU a single sample of arterial blood was obtained from the indwelling cannula for arterial blood gas analysis and measurement of routine electrolytes, lactate, and pyruvate. Arterial pH, pCO_2, standard bicarbonate, and standard base excess were measured and derived using a Radiometer ABL 520 blood gas analyser (Copenhagen, Denmark). Lactate was measured by the enzymatic method using a Beckman CX5 analyser (Berlin, Germany) with a within-assay coefficient of variation of 3% and total coefficient of variation of 4.3% (National Health Laboratory Service, Cape Town, South Africa). Strong ion difference and strong ion gap (SIG) were calculated from the electrolyte and acid-base data, as previously described [17]. From the same arterial blood specimen 1 ml was immediately placed in a perchloric acid medium, placed in a freezer and stored at –70,3 °C. Pyruvate samples were subsequently analysed in batches using HPLC with fluorescein detection, with a coefficient of variation less than 15% (Ampath Laboratories, Pretoria, South Africa).

Metabolic acidosis was defined as standard bicarbonate less than 22 mmol/l. Thresholds for clinically significant biochemical derangements were defined a priori as lactate above 2 mmol/l, pyruvate above 0.1 mmol/l, and lactate:pyruvate ratio greater than 20 [15, 16, 23]. The cause of metabolic acidosis was assigned to hyperlactataemia (lactate > 2 mmol/l), hyperchloraemia (corrected chloride > 110 mmol/l), SIG (> 2 mmol/l), or in the case of mixed acidosis any combination thereof [17]. We recorded cardiac diagnoses, surgical procedures, duration of cardiopulmonary bypass, duration of aortic cross-clamp, RACHS-1 categories, predicted risk of mortality (using Paediatric Index of Mortality 1), duration of mechanical ventilation, duration of inotropic support, and duration of PICU stay (expressed as calendar days, or part thereof), and observed PICU mortality [24, 25].

Data are reported as median (interquartile range, IQR) with range, absolute frequencies with percentages, and 95% confidence intervals (CI). Non-parametric continuous data were analysed by the Mann-Whitney and Kruskal-Wallis tests, and categorical data by Fisher's exact test or the χ^2 test for trend as appropriate, using Analyse-It statistical software (Analyse-It, UK). In the multivariate analysis the explanatory variables aortic cross-clamp time, cardiopulmonary time, cyanotic heart disease, and epinephrine infusion were entered, with lactate:pyruvate ratio as the outcome variable. Forward stepwise multiple linear regression was performed for the outcome variables duration of mechanical ventilation, inotropic support, and PICU stay, and the risk factors standard bicarbonate, lactate, and lactate:pyruvate ratio, using Stata version 8.2 statistical software (Statacorp, Texas, USA).

Results

Operative risk categories

Cardiac surgical procedures are stratified according to modified RACHS-1 categories in Table 2. Children in higher categories of operative risk had higher admission levels of both lactate ($p = 0.007$) and pyruvate ($p = 0.04$). There was no significant increase in lactate:pyruvate ratio, nor was there a significant difference in admission pH ($p = 0.89$) or standard bicarbonate ($p = 0.41$) with increased operative risk category.

Metabolic acidosis

On admission to PICU 72 children (74%) demonstrated a metabolic acidosis, for which hyperlactataemia was the single primary cause in only one child. The most common primary causes of metabolic acidosis were hyperchlo-

Table 2 Cardiac surgical procedures stratified by RACHS-1 risk categories; data are medians (*parentheses* interquartile range)

Risk category	1 ($n=3$)	2 ($n=46$)	3 ($n=45$)	4–6 ($n=3$)	p
Cardiopulmonary bypass time (min)	34 (26–73)	66 (50–82)	101 (80–134)	131 (114–182)	0.0001
Aortic cross-clamp time (min)	17 (13–35)	33 (20–51)	62 (44–87)	63 (51–76)	0.0002
pH	7.4 (7.35–7.41)	7.39 (7.34–7.43)	7.38 (7.32–7.44)	7.42 (7.35–7.46)	0.89
Standard bicarbonate (mmol/l)	20 (20.9–21.2)	20.3 (19.5–22.3)	19.9 (18.3–21.6)	17.2 (16.4–21.3)	0.41
Lactate (mmol/l)	0.9 (0.9–1.1)	1.8 (1.2–2.3)	2.0 (1.4–2.6)	5.1 (4.3–5.3)	0.007
Pyruvate (mmol/l)	0.05 (0.05–0.07)	0.10 (0.07–0.14)	0.10 (0.07–0.15)	0.15 (0.15–0.27)	0.04
Lactate:pyruvate ratio	18.0 (15.7–18.0)	18.5 (14.7–22.0)	20.0 (15.0–23.0)	22.7 (18.4–28.3)	0.55

raemia in 24 children (25%) and elevated SIG in 9 children (9%) [17]. In the 38 children with mixed metabolic acidosis (39%) hyperlactataemia constituted one of the causative factors in 28 children (29%), compared to hyperchloraemia in 29 children (30%; Fig. 1) [17].

Hyperlactataemia

Hyperlactataemia occurred in 41 children (42%) and raised lactate:pyruvate ratio in 44 (45%). The incidence of raised lactate:pyruvate ratio among children with hyperlactataemia (56%) was similar to that in children with normal lactate (38%, $p=0.11$; Fig. 1). Raised admission lactate was associated with longer cardiopulmonary bypass time, median 98 min (IQR 20–232) vs. 75 min (IQR 17–211, $p=0.009$), and an increased requirement for epinephrine infusion (24% vs. 2%, $p=0.0006$).

Raised lactate:pyruvate ratio

Children with and without raised lactate:pyruvate ratio are compared in Table 3. Raised lactate:pyruvate ratio was associated with longer aortic cross-clamp times ($p=0.01$), and these children were more likely to receive epinephrine by infusion (21% vs. 4%, $p=0.02$). In the bivariate analysis, lactate:pyruvate ratio increased on average by 6.1 (95% CI 5.4–6.9) with each 1 mmol/l increase in lactate, and by 1.3 (95% CI 1.1–1.4) with each 0.01 mmol/l decrease in pyruvate. In the multivariate explanatory model lactate:pyruvate ratio increased on average by 6.4 (95% CI 2.2–10.5) in children receiving epinephrine and by 0.4 (95% CI 0.07–0.8) for every 10 min of aortic cross-clamp time, but there was no significant relationship between lactate:pyruvate ratio and duration of cardiopulmonary bypass ($p=0.82$) or pre-existing cyanotic heart disease ($p=0.46$).

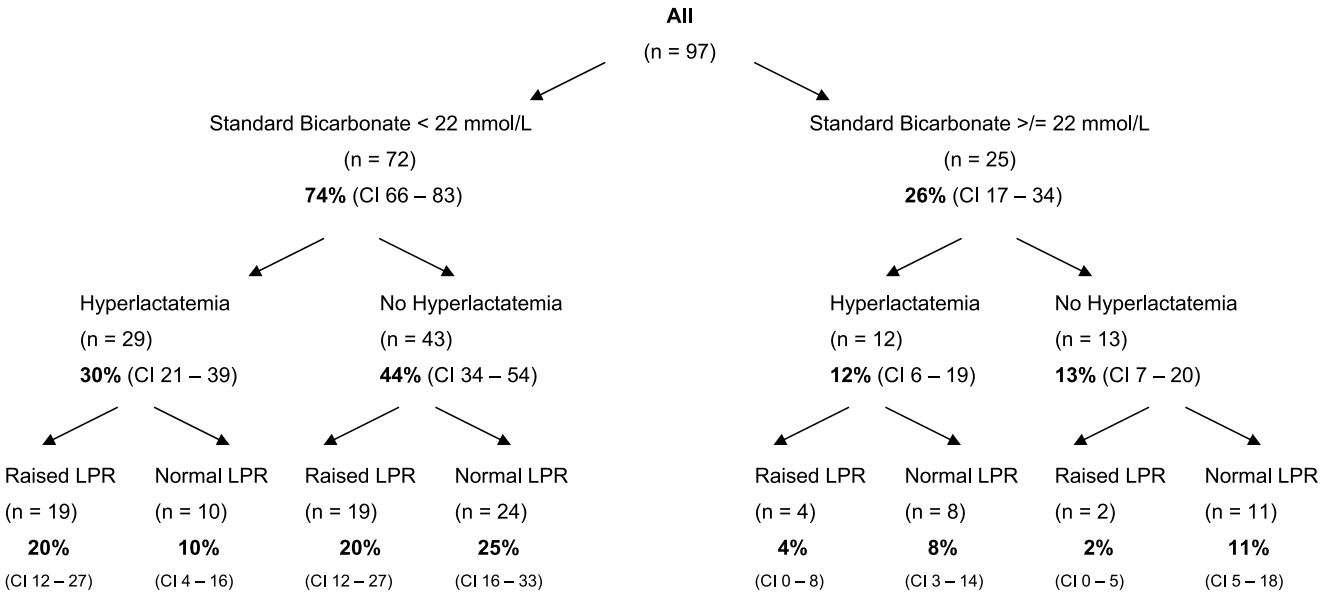

Fig. 1 Children with metabolic acidosis, hyperlactataemia, and raised lactate:pyruvate ratio (*LPR*). *CI* 95% confidence interval

Table 3 Comparison of children with and without raised lactate:pyruvate ratio (*LPR*) in the immediate post-operative period (*IQR* interquartile range)

	LPR > 20 ($n = 44$)		LPR ≤ 20 ($n = 53$)		p
	Median	IQR	Median	IQR	
Cardiopulmonary bypass time (min)	94	65–117	80	49–109	0.23
Aortic cross-clamp time (min)	53	34–77	38	19–53	0.01
pH	7.36	7.31–7.41	7.39	7.35–7.45	0.03
Standard bicarbonate (mmol/l)	19.9	18.2–21	20.6	18.8–23.10	0.02
Lactate (mmol/l)	2.1	1.4–3.2	1.8	1.2–2.2	0.07
Pyruvate (mmol/l)	0.08	0.06–0.14	0.12	0.09–0.15	0.004
LPR	27.8	21.7–26.4	15.0	13.3–17.5	N/A
Duration of mechanical ventilation (days)	2	2–3	2	2–3	0.25
Duration of inotropic support (days)	3	2–4	3	2–3	0.20
Duration of PICU stay (days)	4	3–5	3	3–4	0.22

Table 4 Children with hyperlactataemia plus metabolic acidosis ($n = 29$), hyperlactataemia alone ($n = 12$), metabolic acidosis alone ($n = 43$), and without either hyperlactataemia or metabolic acidosis ($n = 13$); data are medians (*parentheses* interquartile range) (*SB* standard bicarbonate)

Parameter	↑ Lactate ↓ SB	↑ Lactate only	↓ SB only	N lactate N/↑ SB	p
Aortic cross-clamp time (min)	53 (27–80)	50 (36–68)	45 (20–62)	34 (30–39)	0.32
Cardiopulmonary bypass time (min)	98 (77–135)	90 (68–140)	79 (48–110)	66 (50–84)	0.054
pH	7.38 (7.29–7.42)	7.47 (7.44–7.51)	7.36 (7.32–7.39)	7.44 (7.40–7.50)	–
pCO_2 (kPa)	4.6 (3.5–5.1)	4.1 (3.4–4.1)	4.5 (4.1–5.3)	4.2 (3.2–4.9)	0.039
SB (mmol/l)	18.8 (18.3–20.1)	23.7 (23.1–25.1)	19.8 (17.7–20.3)	23.1 (22.3–26.0)	–
Lactate (mmol/l)	2.7 (2.3–3.8)	2.9 (2.3–4.3)	1.3 (1.2–1.7)	1.2 (0.9–1.6)	–
Pyruvate (mmol/l)	0.15 (0.11–0.19)	0.16 (0.15–0.27)	0.08 (0.06–0.10)	0.09 (0.06–0.13)	< 0.001
Lactate:pyruvate ratio	22 (18–26)	17 (14–24)	19 (15–22)	15 (13–18)	0.0025
Sodium (mmol/l)	138 (136–139)	139 (135–143)	138 (137–140)	139 (137–140)	0.77
Chloride (mmol/l)	109 (108–112)	110 (107–114)	112 (110–115)	110 (107–113)	0.03
Albumin (mmol/l)	28 (25–31)	30 (29–35)	30 (27–34)	31 (27–34)	0.067
SIG (mEq/l)	2.1 (–1.2 to 6.1)	–2.9 (–5.1 to 0.2)	1.1 (–1.7 to 4.5)	–1.6 (–4.2 to 1.8)	0.023
Mechanical ventilation (days)	2 (23)	2 (2–4)	2 (2–3)	2 (2–3)	0.66
Inotropic support (days)	3 (2–4)	3 (2–4)	3 (2–3)	2 (2–3)	0.54
PICU stay (days)	4 (3–6)	3 (3–5)	3 (3–4)	3 (3–4)	0.05

Raised lactate:pyruvate ratio in the presence of hyperlactataemia and metabolic acidosis

Lactate:pyruvate ratio and other biochemical parameters are compared in children with and without hyperlactataemia and metabolic acidosis in Table 4. Lactate:pyruvate ratio and SIG were highest ($p = 0.0025$ and $p = 0.023$, respectively) and chloride lowest ($p = 0.03$) in those children with both hyperlactataemia and metabolic acidosis.

PICU morbidity

Using simple linear regression, the admission lactate was associated with duration of mechanical ventilation ($p = 0.0497$), duration of inotropic support ($p = 0.015$), and duration of PICU stay ($p = 0.01$). None of these outcome variables was associated with either standard bicarbonate or lactate:pyruvate ratio (data not shown). Using forward stepwise multiple linear regression, of the risk factors standard bicarbonate, lactate, and lactate:pryruvate ratio, only lactate was independently associated with prolongation of PICU support. On average the duration of inotropic support increased by 0.29 days (95% CI 0.06–0.52), duration of mechanical ventilation increased by 0.27 days (95% CI 0.0004–0.56) and duration of PICU stay increased by 0.42 days (95% CI 0.10–0.74) for each 1 mmol/l increase in admission lactate.

Discussion

We describe the relationship between admission lactate:pyruvate ratio, lactate, metabolic acidosis, and morbidity, in a group of children characterised by low-moderate RACHS-1 operative risk categories and predicted PICU mortality of less than 5% [24, 25]. Since observed post-operative mortality was negligible, surrogate markers of operative morbidity, such as duration of mechanical ventilation, inotropic support, and PICU stay, were selected for evaluation. The patient group was characterised by mild-moderate metabolic acidosis and hyperlactataemia at the time of admission to PICU, yet

derangement of the lactate:pyruvate ratio was frequent. Almost one-half of the children had clinically significant hyperlactataemia or elevated lactate:pyruvate ratio, and almost one-quarter had elevation of both lactate and lactate:pyruvate ratio simultaneously. Similarly, three-quarters of children demonstrated a metabolic acidosis in the immediate post-operative period (see Fig. 1) and, as might be expected, a higher proportion of children with metabolic acidosis had raised lactate levels. Although hyperlactataemia was an infrequent primary cause of metabolic acidosis, since most primary metabolic acidosis was due either to hyperchloraemia or elevated SIG, a large proportion of mixed metabolic acidosis was due in part to hyperlactataemia [17].

It is striking that many children with elevation in lactate did not have a metabolic acidosis, which we attribute to co-existing alkalotic forces such as hypoalbuminaemia [17, 26]. The presence of respiratory alkalosis may also have contributed directly to hyperlactataemia due to inhibition of lactate clearance [27]. It should also be noted that many children with elevated lactate:pyruvate ratio did not have hyperlactataemia since elevation in the lactate:pyruvate ratio was due not only to increased lactate but also partly to lower pyruvate. The clinical significance of this finding is not clear, although abnormal lactate:pyruvate ratios with normal lactate levels, suggesting pyruvate utilisation, have been noted previously in children with septic shock [28]. This discordance between elevation in lactate and the lactate:pyruvate ratio may arise from epinephrine-driven aerobic glycolysis and anaerobic glycolysis due to cellular dysoxia occurring at different rates in different tissues [8, 9].

Hyperlactataemia was associated with longer operative cardiopulmonary bypass time, elevated lactate:pyruvate ratio with longer aortic cross-clamp time, and both hyperlactataemia and elevated lactate:pyruvate ratio with use of epinephrine infusion. These findings are not entirely surprising, since epinephrine has previously been shown to increase both lactate and lactate:pyruvate ratio [18]. We might have expected that both admission lactate and lactate:pyruvate ratio would increase with higher RACHS-1 categories (see Table 3), but this was not the case [24]. In the multivariate analysis hyperlactataemia was associated with longer duration of inotropic support, mechanical ventilation, and PICU stay, even among children with relatively low operative risk and low post-operative mortality. It appears that the admission lactate level remains a useful prognostic marker of morbidity following open cardiac surgery in children with relatively mild metabolic acidosis. Metabolic acidosis per se was not a useful marker of post-operative outcome [3].

We had speculated that the lactate:pyruvate ratio would distinguish patients with occult tissue dysoxia from those with hyperlactataemia due to aerobic glycolysis, on the basis of markers of PICU morbidity [10–12]. However, raised lactate:pyruvate ratio was associated not only with elevated lactate but with lower pyruvate levels, and elevation in the lactate:pyruvate ratio was not associated with prolongation of PICU stay, mechanical ventilation, or inotropic support. This finding is in agreement with Weil and Aififi [22] who demonstrated that elevated lactate:pyruvate ratio is a poor prognostic indicator in human circulatory shock. The lactate:pyruvate ratio may be a poor marker of tissue hypoxia if the anaerobic threshold is variable in different organ systems, and both oxidative and anaerobic glycolytic energy production proceed in separate independent compartments [8, 9]. It is also likely that the timing of blood sampling confounds interpretation of the lactate:pyruvate ratio. Weil and Afifi [22] demonstrated in an animal model of haemorrhagic shock that pyruvate rises promptly, whereas the rise in lactate is slower but sustained, leading to delayed elevation in the lactate:pyruvate ratio. Determination of serial intra- and post-operative lactate:pyruvate ratios, in conjunction with mixed venous oxygen saturation, would be required to clarify this question. It is a limitation of this study that we are unable to present mixed venous oxygen saturation and cardiac output data in conjunction with the acid-base parameters.

The principal limitations of this study arise from the relatively low pre-operative risk and low post-operative mortality rate in the study group. Since patients were not pre-selected on the basis of surgical procedure or severity of illness, and because the mortality rate was similar to that of all children who underwent cardiopulmonary bypass, we believe the study group accurately reflects children undergoing open cardiac surgery at this centre, where higher risk procedures (RACHS-1 categories 4–6) are performed less frequently due to resource limitations [24]. It follows that these findings should not be extrapolated directly to high volume cardiac surgical centres where procedures in high operative risk categories are performed routinely, since the incidence of severe hyperlactataemia and metabolic acidosis, and the PICU mortality rate, may be greater [29]. Future research should examine the relationship between serial lactate:pyruvate ratios and objective measures of tissue hypoxia or hypoperfusion in selected high-risk surgical categories. Nevertheless, these data provide an indication that measurement of the lactate:pyruvate ratio in children with mild-moderate metabolic acidosis would not add useful prognostic information to that derived from routine operative risk data, including the post-operative lactate level alone. Given that pyruvate analysis requires specialised sample processing (perchloric acid medium) and laboratory techniques, routine measurement of pyruvate would not be warranted.

In summary, elevation in the lactate:pyruvate ratio was common in this group of children with mild-moderate metabolic acidosis and low mortality following open cardiac surgery. Raised lactate:pyruvate ratio was more frequent in children with metabolic acidosis, yet it occurred commonly in children without hyperlactataemia,

since elevation in the lactate:pyruvate ratio was due partly to lower pyruvate. Children with raised lactate:pyruvate ratio had longer aortic cross-clamp times and were more likely to receive epinephrine by infusion. However, it was hyperlactataemia rather than elevation in the lactate:pyruvate ratio or metabolic acidosis that was associated with prolongation of inotropic support, mechanical ventilation, and PICU stay. Analysis of pyruvate and calculation of lactate:pyruvate ratios did not add useful prognostic information in this clinical setting. These findings should be tested in centres with a larger proportion of cardiac surgical procedures in high operative risk categories [29].

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