Patterns of Lactate Values after Congenital Heart Surgery and Timing of Cardiopulmonary Support

Robert L. Hannan, MD, Marion A. Ybarra, BS, Jeffrey A. White, MS, Jorge W. Ojito, CCP, Anthony F. Rossi, MD, and Redmond P. Burke, MD

Congenital Heart Institute, Miami Children's Hospital, Miami, and Arnold Palmer Hospital, Orlando, Florida

Background. We sought to determine if postoperative serial lactate determinations follow predictable patterns that could be useful in directing management, especially the initiation of postoperative mechanical cardiopulmonary support (CPS).

Methods. Eight patients undergoing CPS in a 2-year period and 147 patients not requiring postoperative CPS in 6 months of that period were stratified into 6 categories based on short-term risk for mortality (1 being the lowest risk). Lactate values for the first 48 hours postoperatively were retrospectively analyzed.

Results. Survivors not requiring CPS in category 6 (n = 16) followed a distinct pattern different from those of categories 1 through 4 (n = 128). Review of postoperative CPS survivors (n = 4) indicated that CPS was initiated electively without cardiac arrest in all 4, and lactate values showed a downward trend within 12 hours of initiation in all cases (mean lactate, 10.12 ± 1.88 mmol/L; range, 1.4 to 16 mmol/L; mean initiation time, 16.5 hours postoperatively). Three fourths of the CPS nonsurvivors suffered cardiac arrest before CPS and showed rising lactate values despite support (mean lactate, 11.95 ± 1.37 mmol/L; range, 1.6 to 18.6 mmol/L; mean initiation time, 21.25 hours postoperatively). Indications for initiation of CPS in patients with elevated lactate values were reviewed. Two thirds of patients who died without CPS had preterminal cardiac arrest.

Conclusions. We have defined the normal pattern of postoperative lactate values in our institution. These data suggest that an abnormal lactate pattern may be useful in determining the timing of CPS initiation in hemodynamically stable patients with high or rising lactate values, before cardiac arrest or end organ damage.

The postoperative care of children after congenital heart surgery remains challenging. Children undergoing palliative operations have, by definition, abnormal physiology, and optimizing hemodynamic parameters may be difficult. Children with complete two-ventricle repairs may have diminished cardiac output and systemic oxygen delivery despite stable hemodynamic indicators. Diminished cardiac output and systemic oxygen delivery may persist despite stable blood pressure and heart rate [1]. Indicators of diminished cardiac output and oxygen delivery are an important tool in the intensive care unit, and may lead to therapeutic interventions before unstable hemodynamic indicators are manifested. Such indicators might assist in minimizing morbidity and mortality and improving overall patient outcomes.

We adapted an algorithm of care in our cardiac intensive care unit based on serial lactate levels. Goal-directed therapy with lactate level as the endpoint, initiated in early 2001, has apparently contributed to decreased mortality in our program compared with historical controls [2] (Fig 1). The expected pattern of serial lactate determinations is, however, unknown [3–5]. We hypothesized that a retrospective evaluation of lactate values obtained from risk stratified patients after congenital heart surgery would allow the recognition of abnormal lactate patterns. Recognition of abnormal patterns might allow earlier and more effective therapeutic interventions, specifically cardiopulmonary support (CPS), in selected patients.

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Address reprint requests to Dr Hannan, Division of Cardiovascular Surgery, Miami Children’s Hospital, 3200 SW 62nd Court, Miami, FL 33155; e-mail rhannan001@aol.com.

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Fig 1. Cumulative summary mortality 1995 to 2004. A graphical depiction of the trend in mortality indicating a change from 4% mortality to 2% mortality.
Patients and Methods

For this retrospective study, we obtained an exemption from the Institutional Review Board. Serum lactate determinations were ascertained using the hand held i-STAT 1 Clinical Analyzer (Abbott; Abbott Park, IL). These levels were followed in the intensive care unit (ICU) by a previously described protocol [2]. Medical intervention was based on the exact clinical situation, but included blood transfusion to increase oxygen delivery, escalation of inotropic support, addition of inotropes, increasing or adding afterload reducing agents, changes in ventilator settings, increasing sedation or initiating paralysis, and the initiation of CPS.

Serial serum lactate levels were made available to caregivers in multiple formats. These included directly from the i-STAT unit or from a conventional lab results screen on the computerized care computer in the ICU (Eclipsys; Eclipsys Technologies, Boca Raton, FL). In addition, a graphical real-time presentation of serial lactate levels was available at the bedside or on the Internet (i-Rounds; Teges Corp, Coral Gables, FL; Fig 2).

All patients undergoing postoperative CPS (8 patients) in a 2-year period and 147 consecutive patients not requiring postoperative CPS in 6 months of that period were obtained from two separate databases (Lab Access and Cardioaccess) and analyzed retrospectively. Patients in whom CPS was initiated preoperatively, or in the OR, were excluded. The patients were divided into categories based on the Risk Adjustment in Congenital Heart Surgery (RACHS-1) method, which categorizes patients based on the complexity of their condition. The RACHS-1 method was created as a consensus based method of stratification of cases to compare hospital mortality rates. The RACHS-1 method groups procedures based on similar expected short-term mortality rates from category 1, which has the lowest risk for death, to category 6, which has the highest risk for death [17]. The conditions that qualify as category 5 are rare, and there were no category 5 patients treated in the included time frames (Table 1). As a result, category 5 was excluded from this study. Patients in each category were then divided into subcategories: survivors, nonsurvivors, CPS survivors, and CPS nonsurvivors (Table 2).

Fig 2. Sample laboratory results feed page from i-Rounds Web-based charting. Page can be customized by the user to view up to six different laboratory results simultaneously from any location with Web access, including the patient’s bedside. (Max = maximum; Min = minimum; Sat = saturation; WBC = white blood cell count.)
The lactate values obtained for each patient during the first 48 hours after surgery were plotted over time on a scatter plot. The means and standard deviations were calculated for each category, plotted, and fitted using polynomial regression. Statistical significance was determined using the SigmaStat 3.1 Advisory Statistics for Scientists Software (Systat Software, Richmond, CA).

Patients who underwent CPS were reviewed to determine indications for CPS, including cardiopulmonary arrest or hemodynamic instability, lactate patterns, acid-base status, urine output, and level of pressor support. Data were obtained from CardioAcess, Web-based charting (i-Rounds, Teges Corp) as well as supplemental perfusion databases and chart reviews.

**Results**

Category 1 through 4 patients who survived without CPS (n = 128) showed remarkably similar lactate patterns (Fig 3). These patterns, when combined, can be represented by the polynomial y = 4E-05x^3 - 0.0018x^2 - 0.0196x + 2.5667 (Fig 4). Gradually decreasing serial lactate values postoperatively are indicators of favorable outcomes. Category 6 patients who survived without CPS (n = 16; Fig 4) showed a very different lactate trend, with an initial moderate rise in lactate values and a longer plateau. The category 6 pattern is represented by the polynomial y = 0.0002x^3 - 0.0176x^2 + 0.3418x + 3.0448.

The lactate trends of patients in whom CPS was initiated postoperatively and who survived are shown in Figure 5. All 4 had CPS initiated before cardiopulmonary arrest. Declining urine output or new metabolic acidosis in the setting of rising or high lactates despite escalating medical therapy was the indication for CPS in the CPS survivors group. Lactate levels generally declined after the initiation of CPS. The overall trend in all patients initiated on CPS in the cardiac intensive care unit who survived (n = 4) showed an average initiation time of 16.5 hours postoperatively and, in general, a declining lactate pattern after initiation (Fig 5). Lactates in this group ranged from values as low as 1.4 mmol/L to 16 mmol/L, with a mean value of 10.12 ± 1.88 mmol/L at initiation.

Cardiopulmonary support nonsurvivors are shown in Figure 6. Three of 4 had initiation after cardiopulmonary arrest, and 2 showed continued elevation of lactate levels despite CPS. All had other indications including metabolic acidosis and anurea. Indications for CPS are shown in Table 3. The mean lactate value at initiation in this group was 11.95 ± 1.37 mmol/L within an overall range of 1.6 mmol/L to 18.6 mmol/L. (The difference between the mean lactate at initiation of the CPS survivors and CPS nonsurvivors was not statistically significant.)

The remaining 3 nonsurviving patients did not go on CPS. Two out of these 3 had preterminal cardiac arrest (Fig 7).

**Comment**

Adequate oxygen delivery to vital organs is an immediate postoperative goal in critically ill children after heart surgery. Oxygen delivery may be assessed by direct measurement of mixed venous oxygen saturation, utiliz-

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**Table 1. Risk Adjustment in Congenital Heart Surgery (Abridged Table)**

<table>
<thead>
<tr>
<th>Category 1</th>
<th>Category 2</th>
<th>Category 3</th>
<th>Category 4</th>
<th>Category 5</th>
<th>Category 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASD surgery</td>
<td>VSD</td>
<td>Fontan</td>
<td>Konno procedure</td>
<td>Ebstein anomaly</td>
<td>HLHS stage I</td>
</tr>
<tr>
<td>PAPVC repair</td>
<td>Subaortic stenosis</td>
<td>Arterial switch</td>
<td>TAPVC repair</td>
<td>Truncus arteriosus</td>
<td>Damus Kaye Stansel</td>
</tr>
<tr>
<td></td>
<td>resection</td>
<td></td>
<td></td>
<td>with interrupted arch</td>
<td>Stage I repair of</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>nonhypoplastic left</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>heart syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>conditions</td>
</tr>
</tbody>
</table>

ASD = atrial septal defect; HLHS = hypoplastic left heart syndrome; PAPVC = partial anomalous pulmonary venous connection; TAPVC = total anomalous pulmonary venous connection; VSD = ventricular septal defect.

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**Table 2. Patient Demographics**

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
<th>Median Age (Days)</th>
<th>Median Weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category 1 survivors</td>
<td>19</td>
<td>17</td>
<td>569</td>
<td>10.5</td>
</tr>
<tr>
<td>Category 2 survivors</td>
<td>21</td>
<td>15</td>
<td>167</td>
<td>6.2</td>
</tr>
<tr>
<td>Category 3 survivors</td>
<td>16</td>
<td>22</td>
<td>177.5</td>
<td>6.15</td>
</tr>
<tr>
<td>Category 4 survivors</td>
<td>12</td>
<td>6</td>
<td>194</td>
<td>6.05</td>
</tr>
<tr>
<td>Category 6 survivors</td>
<td>10</td>
<td>6</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Nonsurvivors</td>
<td>0</td>
<td>3</td>
<td>5</td>
<td>2.6</td>
</tr>
<tr>
<td>CPS survivors</td>
<td>4</td>
<td>0</td>
<td>65.5</td>
<td>3.65</td>
</tr>
<tr>
<td>CPS nonsurvivors</td>
<td>2</td>
<td>2</td>
<td>3.5</td>
<td>2.65</td>
</tr>
</tbody>
</table>

CPS = cardiopulmonary support.
ing an indwelling catheter. In the absence of a catheter, surrogates have been proposed to estimate effectiveness of tissue oxygen delivery. These surrogates include the measurement of arterial serum lactate concentrations as well as various parameters such as DCO₂, cardiac index, base deficit, and arterial pressure as possible indicators of adverse events [1]. Recent studies indicate that elevated serum lactate has been linked to an increase in the risk of tissue oxygen deficiency, morbidity, or mortality [6–10]. More recently, serum lactate levels have been shown to be predictive of outcomes after congenital heart surgery [8, 11–13]; however, the utility of these values is not as well established.

Our philosophy in the cardiac intensive care unit is for early and rapid intervention, by maximization of oxygen delivery and cardiac output, before the onset of organ damage or hemodynamic deterioration. Rising or elevated lactate levels were assumed to be secondary to diminished oxygen delivery. The management of such critically ill patients is focused on achieving low lactate levels as well as the more traditional goals of stable hemodynamics, adequate urine output, and normalized acid-base status. This goal-directed therapy, utilizing serum lactate level as the immediate endpoint of treatment, has been facilitated by point-of-care testing. Point-of-care testing can be described as the rapid availability of laboratory results at the bedside to increase the tempo of the therapeutic decision making and intervention feedback loop. Point-of-care testing enhances the efficiency of goal-directed therapy in which the aim is intervention before hemodynamic compromise or critical organ system compromise. Response to increasing lactate levels is rapid with appropriate medical therapeutic intervention or with the initiation of mechanical cardiopulmonary support.

The precise role of mechanical cardiopulmonary support in children after congenital heart surgery is in a state of evolution. The actual technique of mechanical cardiopulmonary support and expected outcome after support
may influence the willingness to initiate support. Indications for the initiation of support, short of cardiopulmonary arrest, have not been defined. Our institution utilizes a novel mechanical CPS system, previously described[14], which we believe offers significant advantages over the conventional extracorporeal membrane oxygenation circuits. Recent reports include a series of patients after stage 1 palliation for hypoplastic left heart syndrome, all of whom were electively supported postoperatively. Other reports point to suboptimal outcomes utilizing conventional extracorporeal oxygenation techniques[15, 16].

The heterogeneous nature of congenital heart disease and the wide range of expected outcomes have been addressed by stratifying patients by diagnosis into groups. The RACHS-1 method allows the categorization of patients with different anatomic diagnosis into similar risk groups[17]. Doing so allows analysis based on risk-stratified results.

Lactate patterns in children after open heart surgery followed definable trends, with different trends for RACHS categories 1 through 4 and category 6. We have used these trends to identify patients for whom medical therapy should be escalated and for whom the elective initiation of CPS should be considered. We consider a steadily rising lactate pattern ominous, and escalate medical support in an effort to reverse the trend. If maximal medical therapy results in continued rising lactate patterns, or a stable elevated lactate, CPS is considered as the next intervention. In our experience, relative indicators such as new metabolic acidosis, decline in urine output, or very high levels of pressor support are indicators that support should be considered before hemodynamic compromise or collapse. Persistent elevated or rising lactate levels after initiation of CPS were invariably indicative of fatal outcomes. Patients who suffered cardiac arrest before the initiation of CPS had worse outcomes than patients who had support initiated before arrest.

The precise role of obtaining serial lactate levels has become clearer in our cardiac intensive care unit. Deviation from the expected lactate pattern in each risk category leads to escalating medical therapy and consideration of the early initiation of CPS. Our goal-directed therapy, using lactate level as an endpoint, now recognizes that the initiation of CPS before hemodynamic collapse may lead to improved outcomes.

### Table 3. Indications

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Diagnosis</th>
<th>Hours to Initiation</th>
<th>Lactate at Initiation (mmol/L)</th>
<th>Urine Output (cc/kg/h)</th>
<th>Hemody namics</th>
<th>Epinephrine (µg/kg/min)</th>
<th>Base Excess or Deficit</th>
<th>Cardiac Arrest</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>TAPVC/single ventricle</td>
<td>16</td>
<td>11.8</td>
<td>1</td>
<td>Stable</td>
<td>0.2</td>
<td>&gt; -2</td>
<td>No</td>
<td>Survived</td>
</tr>
<tr>
<td>2</td>
<td>HLHS</td>
<td>10</td>
<td>6.5</td>
<td>1</td>
<td>Volume requirement</td>
<td>&gt; 0.2</td>
<td>&lt; -2</td>
<td>No</td>
<td>Survived</td>
</tr>
<tr>
<td>3</td>
<td>HLHS</td>
<td>18</td>
<td>12.6</td>
<td>1</td>
<td>Unstable</td>
<td>0.2</td>
<td>&gt; -2</td>
<td>No</td>
<td>Survived</td>
</tr>
<tr>
<td>4</td>
<td>HLHS</td>
<td>22</td>
<td>9.6</td>
<td>1</td>
<td>Stable</td>
<td>&gt; 0.2</td>
<td>&lt; -1</td>
<td>No</td>
<td>Survived</td>
</tr>
<tr>
<td>5</td>
<td>TGA/VSD single coronary</td>
<td>26</td>
<td>8.2</td>
<td>1</td>
<td>Stable</td>
<td>&gt; 0.2</td>
<td>&lt; -2</td>
<td>No</td>
<td>Deceased</td>
</tr>
<tr>
<td>6</td>
<td>TAPVC/single ventricle/preoperative shock</td>
<td>38</td>
<td>12.6</td>
<td>1</td>
<td>Unstable</td>
<td>&gt; 0.2</td>
<td>&lt; 2</td>
<td>yes</td>
<td>Deceased</td>
</tr>
<tr>
<td>7</td>
<td>TAPVC/single ventricle</td>
<td>10</td>
<td>10.1</td>
<td>1</td>
<td>Unstable</td>
<td>&gt; 0.2</td>
<td>&lt; -2</td>
<td>Yes</td>
<td>Deceased</td>
</tr>
<tr>
<td>8</td>
<td>HLHS</td>
<td>11</td>
<td>16.9</td>
<td>1</td>
<td>Unstable</td>
<td>&gt; 0.2</td>
<td>&lt; -2</td>
<td>Yes</td>
<td>Deceased</td>
</tr>
</tbody>
</table>

HLHS = hypoplastic left heart syndrome; TAPVC = total anomalous pulmonary venous connection; TGA = transposition of the great arteries; VSD = ventricular septal defect.

### References


DISCUSSION

DR ERLE H. AUSTIN III (Louisville, KY): Just looking at your slides, the good news is that there weren’t many cases requiring mechanical support. But looking at the individual patients, I get the impression that it was more the rate of rise of lactate than anything else that seemed to indicate the patients who were going to require support.

DR HANNAN: I can talk to you a little bit about that without having to sign a confidentiality agreement. And clearly one of the things we didn’t talk about was who we put on support in the operating room. And as of today, we, the surgeons, are extremely reluctant to leave the operating room with a rapidly rising lactate. And in fact, we have our perfusion students take a pen and paper and plot the second derivative and the first derivative of the lactate. When we see that second derivative go to zero, we start thinking about leaving the operating room; and when we plateau, we will leave the operating room.

DR CARL L. BACKER (Chicago, IL): I have several questions. I believe you also have a poster at this meeting on your cardiopulmonary support system?

DR HANNAN: Yes, sir.

DR BACKER: First, I wonder if you could talk to us a little about your ECMO strategy and some of the points from that poster regarding the ability to do rapid initiation of ECMO support. The other questions relate to the paper this morning from [name] regarding the ability to do rapid initiation of ECMO support.

DR HANNAN: To answer the second question first, these babies received steroids in the operating room. Only if we see some direct evidence that there may be a problem with their steroid levels or their thyroid levels, or whatever, would we address that in a routine fashion.

The rapid CPS circuit was designed by George Ojito, our chief perfusionist, who is in the back, specifically to eliminate what he and the team perceived to be the deficiencies of a conventional ECMO circuit: heparinization requirement, large volume prime requirement, blood prime requirement, and extreme difficulty in transport. So what we have now is a circuit that is designed by him that is Carmeda-coated, heparin-bound coating, from cannula tip to cannula tip, including the oxygenator. The circuit that we use for babies has about a 200-cc prime, and we will routinely go on with an asanguinous prime. It takes them about 90 seconds to build a circuit. So typically they have a circuit built while you’re still getting your headlight adjusted in the ICU. We have, again, extreme confidence in this because of the low rate of complications and because our perfusionists run it.

The portability is a major issue. We can go to the cardiac catheterization laboratory very easily, and we do quite commonly. We can go out into the field. We’ve flown this circuit in Lear 100s, Lear 200s, three or four different brands of helicopters, a couple of different ambulances. So in terms of portability, rapid setup, and the other advantages, we believe this is a superior system.


[Further discussion on lactate values and mortality after congenital heart surgery]
DR HANNAN: That's a great question. We initially thought when we started doing point-of-care testing, and over a thousand patients into it, that we wouldn't see those things without a rise in lactate. In the past 6 months, we have seen 2 patients, both neonates, both very small, who had those events occur somewhat simultaneously at a low lactate level. What I can tell you is that there was a cardiac surgeon and the chief of the ICU at the bedside of both of those patients. Previously I had thought that we would always see an elevated lactate first. Both of those patients were—1 was 1.6-kg, heterotaxy total veins, single-ventricle child, and the other was an equally complex 1.8-kg child. Their lactates spiked from 3 to 18 in a matter of minutes. It may simply be a sampling error. We found, quite surprisingly, that simply, for instance, having a baby who is unrepaired get cold, that baby can have an extraordinary rise in lactate in 20 or 30 minutes, going from a normal lactate to a lactate of 20 unoperated on.

So I would agree with you that when the hemodynamic signs are stable, the babies are making urine, they're not acidic, lactate is very important. We have seen now a very low number of patients who can just up and get very sick. Neither one of those patients arrested before they were on support, but that's one of those things.

DR JACOBS: And my second question, I think, reveals my naivete about this type of scenario, or perhaps I missed part of your discussion, but was there anything about this type of continual metabolic monitoring that suggested to you which patients in the setting of resuscitation with mechanical circulatory support were going to need to have a revision of their plumbing and which had been the result of a transient lapse in supportive care and could be expected to recover with a period of adequate mechanically supported circulation?

DR HANNAN: In terms of technical issues, we’re loath to leave the operating room with unresolved technical issues. So for almost every baby, we have a TE in the operating room. If there are issues in the operating room, we do the appropriate on-table drawing of saturations or whatever. For the babies who we don't have a TE in, specifically baby status post–stage 1 palliation, if there appears to be a technical issue the operating room, we will go directly to the catheterization laboratory and catheterize them there. I operated on a baby with mixed total veins and a hypoplastic left heart syndrome, and we went directly from the operating room to the catheterization laboratory on CPS for that baby.

DR JACOBS: During the period of acquisition of this series, did you have any patients who were put on circulatory support in the ICU and proved to require a change in their plumbing, a revised shunt or a revision of pulmonary venous anastomosis, or anything of that sort?

DR HANNAN: The first patient in the series in 2002 required a revision of an LPA.