Perfusion Treatment Algorithm: Methods of Improving the Quality of Perfusion

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Abstract: The pathophysiological consequence associated with cardiopulmonary bypass (CPB) has generated a movement away from this technology in the treatment of heart disease. The negative outcomes are multifactorial in origin and may be associated both with the conduct of CPB and the instrumentation of extracorporeal flow. The purpose of this study was twofold. First, to develop a bedside patient risk assessment to aid in the development of a perfusion care plan. Second, to identify the controllable variables used during CPB that contribute to overall morbidity. Controllable perfusion-related variables that were positively linked to improved patient outcomes were identified from randomized, peer-reviewed human studies. Such variables as hematocrit, mean arterial pressure, thermic perfusion, blood lactate, colloid osmotic pressure, pulsatile perfusion, acid base homeostasis, oxygenation, and coated circuitry were included. Patient risk assessment was developed using the Society of Thoracic Surgeon database, where 61 variables affecting postoperative morbidity were identified. These variables were used to develop a bedside tool, Mortality Assessment Perfusion Score (MAPS), to guide the perfusion patient care plan. The MAPS generates a specific value that may predict patient morbidity and mortality based on past mortalities. In conclusion, the improvement in patient outcome may be associated with both the change in conduct of CPB and the quantitative assessment of patient risk stratification and a patient treatment algorithm. Keywords: risk, risk stratification, patient care plan, outcomes. JECT. 2003;00:000–000

Coronary artery bypass graft (CABG) surgery has developed over the past three decades with a standard operative technique consisting of a median sternotomy, with surgical revascularization with or without cardiopulmonary bypass (CPB). This provides the surgeon with an unobstructed means of access to the coronary arteries and, with chemically induced cardioplegia, creates a flaccid heart. Positive outcomes with coronary surgical revascularization have made this technique the procedure of choice for many suffering from ischemic heart disease (1).

Bypassing of blocked coronary arteries has been shown to relieve such symptoms as angina and dysrhythmia and to prolong life expectancy (2). Methods of treating patients who are at high risk for cardiac surgery because of left heart dysfunction or with impaired ejection fraction have included such procedures as port access bypass grafting (2). The most appropriate treatment mode will depend on the individual’s disease category and associated risk factors. The outcomes associated with the treatment will directly affect quality of life. The climate of health care reform encourages scrutiny of traditional and new forms of medical and surgical therapy. With the emergence of technology and innovative cardiac procedures, advocates must weigh cost versus patient outcomes (3). It has been suggested, based on large cohorts of patients stratified by clinical risk, that the cost of operation can be predicted from models of clinical risk because length of stay (LOS) is highly correlated (4).

For much of the latter half of the 20th century, there was a great deal of interest in establishing and using indices to assess perioperative risk. One of the first and well-known attempts was Dripps’s use of the American Society
of Anesthesiologist (ASA) Physical Status Classification of surgical mortality (5). Beginning in 1977, with the development of the Cardiac Risk index by Goldman et al. there has been a plethora of such indices related to cardiovascular disease in individuals undergoing noncardiac surgery (5). Subsequently, the development and use of risk indices has taken on an increasing importance in the arena of cardiac surgery. The underlying assumption in the development of a risk index is that specific factors (disease history, physical findings, laboratory data, nature of surgery) are unmodified with respect to their influence on outcome (5). Although considerable information has been published on preoperative predictors of operative death at cardiac surgery, few studies have assessed such predictors of major complications after cardiac surgery (6). The lack of standardized criteria for comparing outcome in relation to preoperative condition limits comparisons between institutions. Previous methods could not adequately explain whether conflicting results from separate institutions were attributable to differences in patient severity or quality of care. The Society of Thoracic Surgeons (STS) National Cardiac Surgery Database allows for more accurate modeling, and groups patients’ risks categories whereby individual hospitals and surgeons can compare their results with larger, statistically valid patient populations (7).

CPB has pathophysiologic sequelae that may be more severe in high-risk subsets (8). Minimally invasive direct coronary bypass and off-pump (OPCAB) have eliminated one major component of aberrant physiology intrinsic to on-pump coronary bypass procedures. Coronary bypass, without the use of CPB, has been shown to reduce the overall systemic inflammatory response, including cytokine-mediated responses (8). Therefore, with the appearance of off-pump procedures, there is considerable debate on the safeness of the traditional CPB method. The question remains if the difference in outcomes is based on the traditional CPB method or if the perfusionist could alter treatment to enhance outcomes better. Through a review of articles on preoperative predictors, high-risk categories have been identified. With enhanced knowledge of the high-risk patient population, the perfusionist can then make clinical decisions on how to enhance treatment on pump. Despite the advancement of surgical techniques (robotics, minimally invasive surgery), few developments in the conduct of CPB have been made.

No adult perfusion algorithm exists to date. The lack of an adult treatment algorithm could reflect negatively on advancement of CPB. The purpose of this study was to formulate a perfusion treatment algorithm to enhance the outcomes of our patients and to improve the CPB system.

MATERIALS AND METHODS

Database and Literature
An extensive literature search was conducted pertaining to the following: hematocrit, mean arterial pressure, hypothermic versus normothermic, lactate levels before procedure, colloid osmotic pressure, Pulsatile versus nonpulsatile perfusion, pH-stat versus alpha-stat, FiO2, and heparin-coated circuits. No animal studies were present in the literature search.

Society of Thoracic Surgery
The Society of Thoracic Surgeons (STS) maintains an extensive database that represents the most current patient data (9). Over 500 institutions report annually to the STS. The resultant risk stratification is used to illustrate the various outcomes as they pertain to individual procedures. Because of the extensive nature of the STS database, it was used for this study. Figure 1 represents the cumulative number of procedures performed per the STS database. Figure 2 represents the cumulative number of isolated CAB procedures performed.

The eight different procedures were listed as follows: coronary artery bypass, coronary artery bypass plus aortic valve replacement, coronary artery bypass plus mitral valve replacement, aortic valve replacement, mitral valve replacement, mitral valve repair, coronary artery bypass plus mitral valve repair, and aortic valve repair plus mitral valve repair.

The STS database represents numerous comorbidities (9). The focus on comorbidities is based on their effect on the patient’s status pre-, peri-, and postoperative. For the purpose of this study, it was necessary to limit the comorbidities represented by selecting those with a relative risk of 1.0, or above, in six out of eight procedures listed. This would represent a 75% rate of relative risk of 1.0 per procedure. After selection, 61 comorbidities were represented. The mortality for each comorbidity was listed. The average mortality of each comorbidity per procedure was listed in Table 1. This represents a number score that illustrates the average past morbidity for each parameter for CPB.

![Cumulative Number of Procedures Performed](image)

Figure 1. Cumulative number of procedures performed per the STS database.
Mortality Assessment Perfusion Score

The Mortality Assessment Perfusion Score (MAPS) chart is to be used to perform a bedside assessment of the patient before CPB. The information in Table 1 was placed into five categories: general, cardiac, renal/pulmonary, vascular, and miscellaneous/medications. The placement of the information into categories was to organize better and ensure simplicity of the chart. As noted in Table 2, the score of each individual section is totaled at the bottom of the chart. The score represents the overall average mortality score indicative of past patients with the same co-morbidities. This score is used to predict the possible risks associated with the present patient as it pertains to the past mortalities of the comorbidities listed. With enhanced awareness, the perfusionist can make clinical choices based on literature-based findings to improve patient care.

Perfusion Parameters

To define parameters that could be altered during CPB to affect patient outcome, a review of literature on CPB was conducted. Papers that identified variables in prospective studies were used to identify what treatments would best benefit patient outcome.

Hematocrit

Hematocrit < 23% trends toward death (10). The mortality of patients with hematocrit < 19% is twice as high as patients with a hematocrit of 25%. Patients with a hematocrit < 19%, 7.5% return to bypass (10). Hematocrit is an independent predictor of mortality. A hematocrit of 14% is a statistically significant cut-off point for postoperative mortality (11). Minimum hematocrit did not correlate with stroke or coma (12). Postoperative hematocrit predicts early pulmonary dysfunction without regard to number of red blood cell (RBC) units transfused during surgery (13).

Mean Arterial Pressure

Mean arterial pressure (MAP) of ≥ 90 mmHg is a predictor of postoperative pulmonary dysfunction (13). Table 3 relates MAP 50–60 mmHg with MAP 80–100 mmHg in accordance with cardiac and neurologic complications, total mortality, stroke rate, and cardiac complications (12,14).

Hypothermic versus Normothermic

Patients undergoing normothermic CPB required less transfusion of blood and platelets in the postoperative period (15).

Perfusion temperature does not influence renal function during CPB (16).

Active cooling confers no benefit in terms of cerebral protection (17). There is a risk of stroke when actively rewarming to maintain bladder temperature between 35 and 37°C (18). Mclean et al. suggest that there is no evidence that actively cooling to 28 from 30°C confers no additional benefit in terms of cerebral protection. Because 2–3°C temp decrease confers significant protection in the setting of transient ischemia, it would seem reasonable practice to allow the temperature to “drift” during CPB (19).

Lactate Levels

Pediatrics A change 3 mmol/L during CPB illustrates an optimal sensitivity of 82%, specificity of 80% for mortality (20). A change of 0.75 mmol/L/h was associated with a poor outcome and 89% sensitivity and 100% predictive value (21). Lactate levels measured early in preoperative period and duration of CPB were the best indicators of major adverse events (22).

Adults An admission lactate level of greater than 4.2 mmol/L has a positive predictive value of 100% and a negative predictive value of 97% for postoperative death (23). A lactate level of 4.0 mmol/L or higher during CPB is associated with postoperative mortality (24).

Colloid Osmotic Pressure and Albumin

The addition of 75 g of albumin, 50 g of mannitol followed by 50 g at commencement of rewarming or both of the above to prime solution greatly reduce the need for crystalloid fluid addition (25). Low albumin level (2.5 g/dL) was independently associated with increased mortality after CPB (26). Albumin levels of < 3.5g/dL is indicated as the most powerful indicator of postoperative renal dysfunction (27).

Every standard deviation decrease in albumin indicates odds of dying to be 1.24 (28). Most authorities regard 35 g/L as the lower limit of normal. Edema does not appear until the concentration drops below 20g/L (29).

COP 15 mmHg or less is associated with a 50% survival rate (30).

Serum albumin < 2.5g/dL have a fourfold increase in complications and sixfold increase in mortality (31).

Pulsatile versus Nonpulsatile perfusion

In the nonpulsatile group, there were significantly higher rates of mortality, myocardial infarction (MI), and
intra-aortic balloon pump (IAPB) use than in the pulsatile group (32). Pulsatile flow resulted in lower incidence of MI, mortality associated with cardiogenic shock, cardiovascular complications, and postoperative circulatory support (32). The pulsatile group had greater incidence of spontaneous cardiac conversion and better renal function (33). During CPB, it was necessary to perform higher pump flows in the nonpulsatile group to keep the MAP at the appropriate level (34).

PH Management

Alpha-stat was supported by 44% of the literature as opposed to 33% that supported pH-stat. Twenty-two percent of the literature cited no difference in method (32,35–41).

PaO2

Delay in recovery in patients treated with 100% oxygen with an increased of pro-inflammatory cytokines. 
FiO2 of 0.5 showed a quicker recovery postoperatively (42). Venting with 100% oxygen leads to slower recovery when compared to venting with 21% room air. One hundred percent O2 during CPB leads to increases of extravascular lung water and intrapulmonary shunting (43). In contrast, pediatric patients managed with pH-stat strategy and hyperoxia had the lowest level of acidosis production during DHCA (44).

Heparin-Coated Circuits

Heparin-coated circuits result in improved clinical outcomes, including a lower incidence and magnitude of homologous transfusion, incidence of postoperative MI, postoperative inotropic support, respiratory complications, postoperative atrial fibrillation, and overall incidence of any postoperative complications (47).

DISCUSSION

Results obtained after cardiac surgical procedures, especially coronary artery bypass grafting (CABG), are subjected to closer scrutiny than those of any other form of treatment in any field of medicine. The reasons for this intense scrutiny are multifactorial. Most importantly, more health care dollars are spent on the diagnosis and treatment of coronary artery disease than on any other illness, and this expenditure will only grow as the population ages better and longer. In addition, cardiac surgery is a high-profile specialty (48). Because of the high profile nature of cardiac surgery, more attention is paid, by the patient population, to changes and perceived advances in cardiac surgery techniques. With the greater interest in off-pump coronary artery bypass (OPCAB), Minimally invasive procedures and robotics, a high state of concern exists within the perfusion community. In an era of widespread concerns about variations in the quality of care and use of health care resources, methods to assess the risks of cardiac surgery are of increasing importance. If any risk model is to be easily used by clinicians, then there will likely be a tradeoff between the simplicity of a model and its statistical precision (49).

Table 1. STS analysis of selected comorbidities per procedure.

<table>
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<tr>
<th>Variable</th>
<th>Count</th>
<th>CAB</th>
<th>CAB+AVR</th>
<th>CAB+MVR</th>
<th>AVR</th>
<th>MVR</th>
<th>MVP</th>
<th>CAB+MVP</th>
<th>AV R+MVR</th>
<th>Mean/Points</th>
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<td>15.19</td>
<td>4.09</td>
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<td>5.67</td>
<td>10.07</td>
<td>8.44</td>
<td>7.7</td>
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<td>13.99</td>
<td>4.23</td>
<td>5.94</td>
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<td>11.61</td>
<td>9.98</td>
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<td>9.8</td>
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<td>7.09</td>
<td>20.45</td>
<td>4.18</td>
<td>6.59</td>
<td>6.59</td>
<td>11.46</td>
<td>8.45</td>
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<td>14.47</td>
<td>18.03</td>
<td>18.03</td>
<td>13.71</td>
<td>25.56</td>
<td>17.5</td>
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<td>36.36</td>
<td>18.79</td>
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<td>21.95</td>
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<td>6.44</td>
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<td>8.7</td>
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<td>13.03</td>
<td>4.93</td>
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<td>6.59</td>
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<td>9.80</td>
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<td>4.82</td>
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<td>5.36</td>
<td>8.09</td>
<td>17.61</td>
<td>5.8</td>
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<td>8.63</td>
<td>16.46</td>
<td>15.15</td>
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<td>Prior MI 6 hrs–24 hrs no MI</td>
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<td>44.1</td>
<td>54.17</td>
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<td>3.88</td>
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<td>7.69</td>
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<td>13.96</td>
<td>0</td>
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<td>Prior MI 7–21 days vs no MI</td>
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<td>7.55</td>
<td>15.79</td>
<td>5.26</td>
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<td>23.08</td>
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Table 2. Mortality assessment perfusion score (MAPS).

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<tr>
<td>Noncaucasian</td>
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<tr>
<td>Age 61–65 vs age &lt;60</td>
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<tr>
<td>Age 66–70 vs age &lt;60</td>
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<tr>
<td>Age 71–80 vs age &lt;60</td>
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</tr>
<tr>
<td>Age &gt;80 vs age &lt;60</td>
<td>12.6</td>
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</tr>
<tr>
<td>Diabetes</td>
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</tr>
<tr>
<td>Morbid obesity</td>
<td>Over 1.5 ideal times</td>
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</tr>
<tr>
<td>Hypertension</td>
<td>Over 140/90, hx of H</td>
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<td>Left main disease</td>
<td>Stenosis &gt;50%</td>
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<td>Preop IAPB</td>
<td>Present at time of su</td>
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<td>Prior CAB</td>
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<td>Prior valve</td>
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<tr>
<td>Prior MI other vs no MI</td>
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</tr>
<tr>
<td>Prior MI 1–7 days vs no MI</td>
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<td>Prior MI &gt;21 days vs no MI</td>
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<td>E.F. 35%–44% vs E.F. = 55%</td>
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<td>Urgent vs elective</td>
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**Cardiac**

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<th>Risk Factor</th>
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<td>Cardiomegaly</td>
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<td>LV/EDP = 15</td>
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<td>PA wedge = 15</td>
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<td>Aortic valve disease</td>
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<tr>
<td><strong>TOTAL</strong></td>
<td></td>
<td><strong>51.5</strong></td>
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**Renal/pulmonary**

- Renal failure Daily rise in serum creati >2 mg/dL/day | 17.5
- RF with dialysis Mean pressure >30 | 24.7
- Pulm HTN | 7.7

**Vascular**

- Periph Vas Dz | 10.2
- Cereb Vas Dz | 7.9
- CVA | 8.7

**Misc/medication**

- Immuno RX | 9.2
- Resuscitation | 22
- Ca antagonists | 5.9
- Nitrates-PD | 9
- Nitrates-IV | 12.8
- Antihypertensics | 8
- Antiplatelets | 6.7
- Anticoagulants | 7
- Diuretics | 7.5
- Inotropes | 18.8
- Steroids | 8.4
- CCS Class IV vs 1 | 11.4
- NYHA Class III vs 1 | 3.4
- NYHA Class IV vs 1 | 10.9

**TOTAL**

Levaraut, et al. state that initial hyperlactemia is associated with a high mortality but an elevated anion gap is a poor indicator of hyperlactemia. A patient whose blood lactate does not rise above 2.5 mmol/L will maintain a normal anion gap, if it is initially smaller than 14 mEq/L (50). Preoperative lactate levels were identified as reliable indicators of hypoperfusion before CPB. An admission lactate level of greater than 4.2 mmol/L has a positive predictive value of 100% and a negative predictive value of 97% for postoperative death (23). Once identified, consult the treatment staff and assist in trying to normalize before CPB.

The overall goal is to provide a bedside risk assessment that is unique to patients scheduled for CABG. No perfusion-oriented risk assessment exists as of yet. The STS National Cardiac Surgery Database model is the most widely used model in the United States and is an unparalleled effort in terms of its size and comprehensiveness (49). The analysis of the STS is provided with the results noted on Table 1. The mortality assessment perfusion score (MAPS) chart, as noted in Table 2, is used as the first step in the perfusionist's preoperative assessment of their patient. The perfusion treatment algorithm, as noted in Figure 3, illustrates the steps suggested to the perfusionist for preoperative evaluation as well as the utilization of the MAPS to identify risk levels. The preoperative patient assessment will include the following: early lactate levels, PaO2 on room air, hematocrit, and albumin levels.

The patients' preoperative PaO2 levels will indicate if they are used to high levels of CO2 in their systems. Watching the MAP higher will improve outcomes for the patient. This may key the perfusionist to the length the patient has been acidotic, and steps can be taken to avoid reperfusion injuries (i.e., to ease into higher PaO2 on pump).

The patient's preoperative lactate will indicate if blood needs to be ordered or if you need to add blood to the pump prime. The total number of transfusions is important to note. If the patient has had many transfusions, the pathology associated needs to be examined.

The patient's initial albumin levels need to be checked. Low albumin level (2.5 g/dL) was independently associated with increased mortality after CPB as well as a powerful indicator of postoperative renal dysfunction (26,27).

Any indications of previous admission for congestive heart failure (CHF) and/or peripheral edema, needs to be noted. The MAPS will indicate the patient's risk status: 20–30 low risk, 30–50 moderate risk, >50 high risk. If the patient scores moderate to high, then considerations need to be considered on CPB. Mean arterial pressures between 80 to 100 mmHg illustrated 4.8% lower complication for cardiac and neurologic complications (11). Keeping the MAP higher will improve outcomes for the patient.

Temperature regulation is dictated by the local proto-
Perfusion Treatment Algorithm

Mortality Assessment Perfusion Score (MAPS)
- 20-30 Low Risk
- 30-50 Moderate Risk
- >50 High Risk

Pre-operative Lactate levels
An admission lactate level of greater than 4.2 mmol/L? Consult staff to treat prior to CPB
Keep value below 4.0 mmol/L during CPB.

Patient’s pH, Acidosis? ? Patient who received reperfusion injury via CPB
Patient’s PaO2 on room air? ? patient used to low PaO2?

Pre-operative Hematocrit?
<40%? ? Note protocol is blood to be ordered
Any pre-operative blood transfusions? How many?

Note patient’s Prior Medical History (Pmhtx) for any associated bleeding disorders

Pre-operative Albumin levels
3.5 g/dl? ? Pulmonary edema? Peripheral edema? ? Note Albumin prime protocol,
consider adjustment to increase CPB dose, consult with staff to increase value closer to normal
prior to CPB.

Mortality Assessment Perfusion Score (MAPS)
Moderate to High Risk?
1. Manipulation of perfusion pressure and flow rates.
2. Level of hypothermia and duration of cooling and rewarming.
3. Alteration in the circuit prime to ensure optimal Hct value and oncotic pressure.
4. Ultrafiltration during rewarming or after separation from CPB.
5. Use of agents to specifically modify systemic inflammatory response.

Figure 3. Perfusion treatment algorithm.

col. Mclean, et al. suggest there is no evidence that actively cooling to 28 from 30°C confers no additional benefit in terms of cerebral protection. Because 2 to 3°C decrease confers significant protection in the setting of transient ischemia, it would seem reasonable practice to allow the temperature to “drift” during CPB (19). Local protocol will dictate pH-management and is also regulated by the temperature management.

A circuit that serves to lower the inflammatory response will assist to improve the patient’s outcomes. Heparin-coated circuits are suggested.

Munoz, et al. suggest the following possible interventions before CPB to improve patient outcomes.

1. manipulation of perfusion pressure and flow rates
2. level of hypothermia and duration of cooling and rewarming
3. alteration in the circuit prime to ensure optimal Hct value and oncotic pressure
4. ultrafiltration during rewarming or after separation from CPB
5. use of agents specifically to modify systemic inflammatory response

These concepts need to be used for all patients on CPB as well as all high-risk patients.

REFERENCES
PERFUSION TREATMENT ALGORITHM


