Nonheparinized Partial Cardiopulmonary Bypass for Repair of Traumatic Aortic Rupture

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Abstract: Traumatic aortic rupture requires rapid, definitive repair for optimal outcome, particularly with respect to distal neurologic function. Over the past 10 years, the R Adams Cowley Shock Trauma Center of the University of Maryland has used partial cardiopulmonary bypass without systemic heparinization exclusively for all repairs of the descending aorta. A heparin-bonded circuit allows for controlled distal perfusion and obviates the need for heparinization. Excellent outcome with minimal morbidity has been achieved using this technique. Our protocol and perfusion apparatus are discussed within.

Keywords: aortic injury, cardiopulmonary bypass, partial; trauma, blunt chest; heparin-bonded circuit.

Dissection or rupture of the descending aorta (Figure 1) may follow severe blunt chest trauma, particularly if rapid deceleration injury is involved (e.g., high-speed motor vehicle accidents or falls) (1). Although such injuries carry high mortality in the field—exceeding 50% in some series (2)—survival is substantially improved with rapid transport, diagnosis, and definitive repair at an appropriate trauma facility. Although so-called “clamp & go” (3) or passive shunting (4) techniques have historically been used during periods of high aortic cross-clamping, significant distal neurological (paraparesis, paraplegia) or other injury may follow otherwise successful repair of traumatic aortic rupture (TAR) from inadequate perfusion (5). Conversely, use of routine cardiopulmonary bypass (CPB) to supplement distal perfusion requires systemic heparinization—quite undesirable in most patients with significant other (e.g., orthopedic, neurological, or gastrointestinal) injuries (6–9). Length of cross-clamp time, however, seems to be the single most important predictor of poor neurological outcome in most studies (4–10).

The University of Maryland R Adams Cowley Shock Trauma Center is a large tertiary facility devoted entirely to the care of patients with traumatic injuries throughout the mid-Atlantic region. Since 1992, we have used a partial CPB apparatus without systemic heparinization (hereafter, NHP-CPB) during all TAR repairs with good success and minimal morbidity. Approximately 200 patients have been perfused using this technique over this 10-year period.

DESCRIPTION

Circuit Preparation

A heparin-bonded cardiopulmonary support (CPS) circuit (Medtronic Corp., Carmeda® CB2506, Minneapolis, MN) is used for distal circulatory support during TAR repair. The circuit is composed of 3/8" tubing, an oxygenator (Medtronic Corp., Maxima PLUS® PRF®) (The Maxima PLUS® has been discontinued, the Affinity® oxygenator is now used.) and a centrifugal pump (Medtronic Corp., Biomedicus® BP-80). This closed circuit is primed with one liter of balanced electrolyte solution (Baxter, Plasmalyte-A®, Deerfield, IL) and allowed to recirculate until the lines are needed before cannulation.

Cannulation

After the induction of general anesthesia and single-lung ventilation (using an endobronchial tube or blocker), an appropriately sized femoral venous heparin-bonded
cannula (Medtronic Corp., Carmeda® CB96535, 17-23 Fr sized) is placed percutaneously, de-aired, and flushed with normal saline. Routine transesophageal echocardiography is performed to confirm appropriate positioning of the venous cannula at the level of the right atrial/inferior vena canal confluence. Following venous cannulation, the patient is placed in a right lateral decubitus position, and a left thoracotomy is performed. Following adequate surgical exposure of the descending aorta distal to the injury site, aortic cannulation with a standard 6.5-mm angled cannula (Sarns, Terumo® 15495, Ann Arbor, MI) is performed (Figure 2). Arterial and venous lines are attached sterilely from the pre-primed NHP-CPB apparatus to the implanted cannulae and de-aired. Heparin is neither given intravenously nor through the perfusion system; activated clotting time is not routinely measured intraoperatively.

**Conduct of Perfusion**

Standard parameters that measure perfusion adequacy are monitored during TAR repair. Proximal and distal systemic arterial pressures are measured from appropriately located catheters (i.e., right radial and femoral arterial, respectively). Femoral artery pressures of >60 mmHg are targeted and are associated with decreased paraplegia (10); proximal artery pressures ± 30% of normal range are targeted. Pulmonary and central venous filling pressures are monitored from an inserted pulmonary artery catheter. Perfusion flow rates of 2–3.5 L/min can be achieved typically. Adequacy of end-organ perfusion; that is, urine output and acid/base status, is monitored. Meticulous attention must be paid to the radial artery line pressures, as adjustments in flow can have profound affects on the atrial preload and, thus, affect perfusion proximal to the proximal aortic clamp.

When determining specific FiO₂ and sweep flow rates, consideration must be made for the concurrent oxygenation/ventilation via the anesthetic circuit. Arterial and venous blood gases from the perfusion circuit and radial artery blood gases are performed 5 minutes after bypass initiation and every 30 minutes thereafter, while on bypass. A PO₂ of 250–280 mmHg and PCO₂ of 35–45 mmHg are targeted; all other measured blood gas parameters are maintained in normal therapeutic ranges. Most trauma patients with TAR present with low core temperatures, often <32–33°C. Rewarming is performed slowly while on bypass to 37–38°C to improve peripheral circulation and prevent worsening of coagulopathy; however, frank hyperthermia is avoided for its potential enhancement of distal neurological injury. The choice of volume replacement versus vasoconstriction depends on several criteria: decreasing filling pressures or flow rates, or venous chattering suggests insufficient intravascular volume; arterial hypotension in the setting of optimal perfusion and filling pressures suggests inadequate vascular tone. Such agents as phenylephrine HCl or metaraminol bitartrate are frequently chosen as vasoconstrictors in these patients.
Weaning

Termination from bypass is done rapidly following completion of the aortic repair. Residual perfusate is flushed from the perfusion circuit with additional balanced electrolyte solution, after which arterial decannulation occurs. Removal of the venous cannula occurs after completion of thoracotomy closure and supination of the patient.

DISCUSSION

Multitrauma injuries, such as long-bone/pelvic fractures, abdominal injuries (e.g., spleen, liver, or mesentery) or central nervous system (e.g., subdural or intracerebral hematomas) may complicate TAR repairs. These concurrent injuries make heparinization relatively contraindicated and often dangerous (6–10). These patients present for repair in a hypocoagulable state due to hemorrhagic shock, consumption of clotting factors at injury sites, volume resuscitation, disseminated intravascular coagulopathy, and hypothermia (8).

Because NHP-CPB does not require systemic heparinization, we have found decreased operative bleeding and better hemostasis at concurrent injury sites. Furthermore, without heparin, protamine—and the inherent risks of heparin reversal—is not needed, and minimal coagulation testing is performed intraoperatively. The described perfusion circuit is similar to that employed for cardiopulmonary support and adult extracorporeal membrane oxygenation, is easily portable, compact, assessable, and can rapidly infuse volume.

The so-called “clamp & go” method of surgical repair is less desirable theoretically because it expectedly increases left ventricular strain—and, therefore, the potential for arrhythmias, acute heart failure, and pulmonary edema—and central venous and cerebrospinal pressures (6). Active perfusion during the TAR repair decreases spinal cord, visceral and kidney ischemia (5,6) and the negative attributes of reperfusion or “declamping shock” (6) in high-risk multisystem-injured patients (7). Use of an oxygenator improves hemodynamic stability, especially during one-lung ventilation or when significant lung injury exists (e.g., pulmonary contusion or aspiration). Furthermore, use of an oxygenator affords improved distal oxygenation.

Although a heparin-bonded circuit obviates systemic anticoagulation, the potential does exist for clotting to occur in susceptible patients (i.e., pre-existing hypercoagulability or low flow states). In only one patient at our institution receiving NHP-CPB did femoral arterial thrombosis occur, necessitating amputation; until that time, we had used routinely direct femoral artery cannulation. Since then, all distal cannulations were performed in the intrathoracic aorta without similar occurrence. In addition, the possibilities exist for false-lumen cannulation or difficulty in cannulation due to injury or limited exposure. We have not noted any of these problems.

OUTCOME

Since our routine institution of NHP-CPB, we have not noted new onset of paraplegia despite use in approximately 200 cases (7,8). Morbidity and mortality have not been related directly to TAR repair, but associated with other traumatic complications. It would be quite difficult today for us to directly compare outcomes between TAR with and without use of NPH-CPB, although comparison historically to patients with TAR before its use suggests that NPH-CPB is superior in terms of safety, consistency, and preference by its providers.

REFERENCES