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Sunday, Bloody Sunday: Unanticipated Ascending Aortic Dissection and Grafting After Coming Off CPB for CABG

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Et al.

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Unanticipated Ascending Aortic Dissection & Grafting After Coming Off CPB For CABG

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Introduction: Acute ascending aorta dissection as a rare but potentially fatal complication of open heart surgery.

Case Report: A 68 year old male with a past medical history of gastroesophageal reflux, hypertension, and hypothyroidism had a non-ST segment elevation myocardial infarction one week prior to coronary artery bypass graft surgery. The patient was brought to the OR with an intra-aortic balloon pump in place. The patient underwent coronary artery bypass grafts X4 on cardiopulmonary bypass. The cross clamp time was 1.5 hours. Upon its removal the surgeon noted bleeding around the proximal graft sites, progression of hematoma to rupture of the ascending aorta. There was a 10 minute period of permissive hypotention in an effort to decrease the amount of blood loss. The surgeon inserted an arterial cannula in the left femoral artery, CPB was resumed and ascending aorta replaced with a dacron tube graft. Mannitol & solumedrol were administered. The patient underwent circulatory arrest 17 minutes with a total of 2 hours 49 minutes of CPB for this second procedure. The patient was taken to the ICU from the OR and extubated the following day. The patients glasgow coma score was 10 on post operative day 1, and 16 on post operative day 2. His cognition improved daily. He was discharged to cardiac rehabilitation 4 days later. He was seen in cardiac surgery clinic within the following month and was reported to be doing well.

Discussion: Intraoperative aortic dissection during cardiac surgery is reported to have an occurrence of 0.12 to 0.2. Since the first reports in 1976 and 1983 the incidence has not changed. Cannulating the ascending aorta for cardiopulmonary bypass and for the delivery of antegrade cardioplegia can disrupt the three layered aorta through manipulation and flow. Dissections have been reported to originate at the aortic cannulation site, at the cross-clamp site, at the site of the partial-occlusion clamp,at the proximal anastomosis, and as a result of direct injury. Severe atherosclerotic changes in the aortic wall, a thin or dilated ascending aorta, cystic medial necrosis, or collagen vascular disease can predispose the aorta to injury. Dissections characteristically appear as a tense, circumferential dilatation and bluish discoloration of the exposed aorta. Subintimal extravasation of blood produces a false lumen that rapidly bleeds at proximal venous anastomses, at suture lines for aortotomy closures, at cannulation or cardioplegia sites, or when incised. Deep hypothermic circulatory arrest without cerebral perfusion or moderate hypothermic circulatory arrest with antegrade cerebral perfusion have been used for cerebral protection in these emergent events with a median duration 25 min. Circulatory arrest provides a bloodless surgical field without the need for the use of intrusive clamps and cannula. Deep hypothermia significantly decreases brain metabolism and oxygen requirements and thus permits a longer period of interrupted blood perfusion to the brain. Postoperative mortality as been reported at 43% in these patients. The following factors may be associated with aortic dissection during cardia surgery: dilated ascending aorta, known atherosclerosis, previous CABG surgery, older age and high blood pressure at the time of dissection.

Conclusions: Diagnosis of aortic dissection requires a high degree of clinical suspicion. Once the diagnosis is made, immediate repair should be undertaken to limit the extent of dissection. By recognizing those patients at risk and employing gentle operative technique, the surgical team can minimize the risk of dissection. Once dissection has occurred, however, rapid diagnosis and appropriate surgical management are necessary to improve patient outcome. Several medications have been used to improve outcome after DHCA with variable results but the most important factor in outcome is short duration of DHCA.

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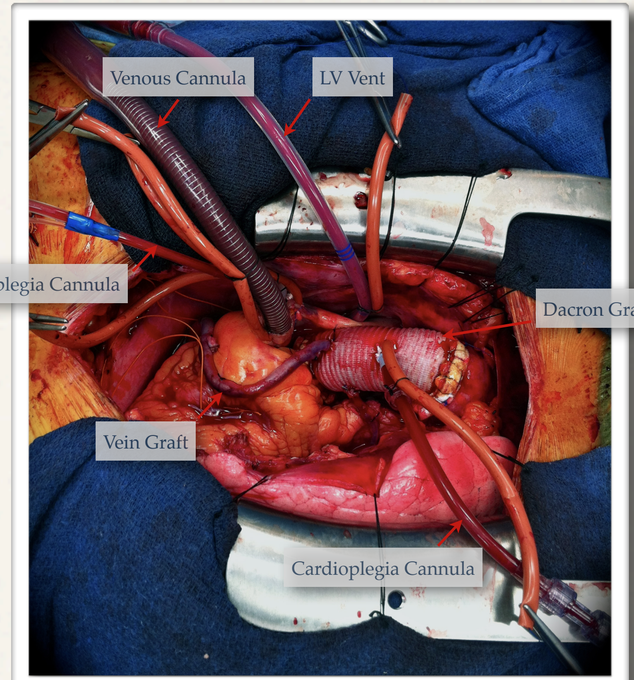

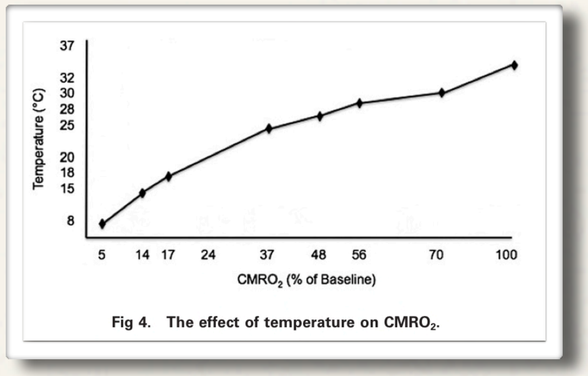

Fig 4. The effect of temperature on $CMRO_2$.

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Table 1. Proposed Mechanisms of Action of Potentially Neuroprotective Pharmacologic Agents

Pharmacologic Agent	Proposed Mechanism
Barbiturates	Reducing $CMRO_2$, CBF, free fatty acids, free radicals, and cerebral edema. Protective in focal ischemia.
Steroids	Decreasing proinflammatory response
Mannitol	Reducing cerebral edema, scavenging free radicals, protecting the kidneys by lowering renal vascular resistance, preserving tubular integrity, and reducing endothelial cell edema
Furosemide	Blocking renal reabsorption of sodium and increasing renal blood flow
Insulin	Controlling hyperglycemia, preventing intracellular acidosis
Calcium channel blockers	Blockade of voltage-sensitive and NMDA-activated neuronal Ca^{2+} channels, decreasing calcium influx into cytoplasm
Lidocaine	Selective blockade of Na+ channels in neuronal membranes, reducing $CMRO_2$
Dexmedetomidine	Inhibition of ischemia-induced norepinephrine release, protective in both focal and global ischemia
Remacemide	Glutamate antagonist
Acadesine	Mitigates the effects of reperfusion injury
β-Blockers	Decreasing inflammatory response

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