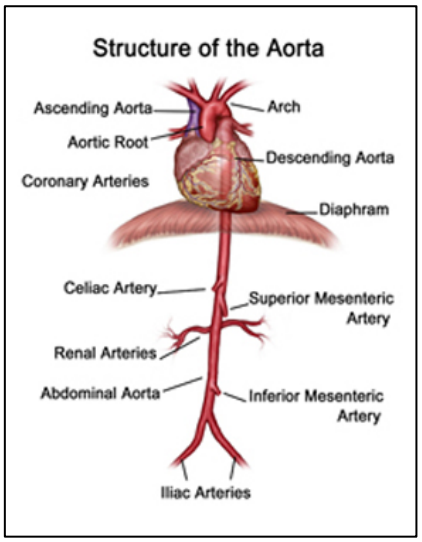
Physiology of Aortic Cross Clamp

Anesthetic Pearls: Anesthetic Implications of the Aortic Cross Clamp



ANATOMY

The descending aorta has 5 major blood vessels originating from it below the level of the left Subclavian artery and above the Iliac bifurcation (Celiac trunk, Superior Mesenteric Artery, Renal Artery x2, Inferior Mesenteric Artery). How the body reacts to cross clamping depends on the location of the clamp to these large vessels.

HEMODYNAMICS

The placement of the clamp, underlying cardiovascular disease, and cardiac function will result in variations of response to the aortic cross-clamp. Clamping above the celiac trunk will result in the highest BP increase while infra-renal cross clamp will have a smaller increase. The cross-clamp will have little effect on blood pressure if the aorta is highly calcified, plaque laden, or pre-clamp flow through this area is low. With the initial clamping of the aorta, the SVR doubles and BP increases. Cardiac output also increases initially as a result of blood "autotransfusion" of vessels returning to the IVC. However, as cross-damp time continues, this blood volume decreases secondary to lack of arterial inflow producing greater anoxic vasodilation and eventually decreasing preload to the right heart. If the clamp is placed above the celiac trunk, the blood pressure and cardiac output may increase dramatically secondary to the redistribution of blood to vessels above the diaphragm and preload returning from the IVC. Infra-renal cross-clamping allows for redistribution of blood flow through the celiac trunk to the splanchnic bed and may actually result in a decrease in BP and CO.

HEART

Cross-clamping puts a profound afterload on the heart by increased SVR. If the heart is otherwise healthy, it does not have difficulty generating the pressures needed to pump against such high pressures. The increased LVEDP (which would normally result in less myocardial blood flow) is compensated mechanically and chemically by increasing the coronary perfusion which avoids heart dysfunction. However, in a heart with CAD or other cardiac pathology, the increased afterload may result in valvular incompetency, CHF, or hypoperfusion of the heart resulting in ischemia or infarction.

METABOLISM

As a result of aortic cross-clamping, total body oxidative metabolism goes down secondary to less tissue being perfused. Areas of hyperperfusion secondary to redistribution will extract less. Mixed venous oxygen saturation will be greater secondary to decreased O_2 extraction. Oxygen saturation and partial pressures will also be higher than normal. Lactic acid increases secondary to anaerobic metabolism below the cross-clamp and therefore produces global metabolic acidosis. Some individuals have recommended continuous bicarbonate infusions to compensate for this acidosis. However, one must remember that secondary to decreased overall tissue perfusion and the conversion to anaerobic metabolism, overall $PaCO_2$ production is decreased. If normocarbia was established prior to clamping and continued throughout the cross clamp time, the compensated respiratory alkalosis will balance the metabolic acidosis after the clamp is placed. Once the clamp is released, a noted increase in CO_2 ($PaCO_2$ & $ETCO_2$) and hypotension will be seen but can be corrected with mild hyperventilation and fluid administration.

ISCHEMIA

Distal aortic perfusion is theoretically maintained secondary to collateral circulation. Overall ischemia may be decreased limiting cross-clamp time, grafting a shunt around the lesion, or cardiopulmonary bypass (especially for upper thoracic aneurysms). Ischemia of the spinal cord with resultant paraplegia is always a concern. Spinal cord ischemia is caused by decreased blood flow to the anterior cord by the Artery Radicularis Magna (Artery of Adamkiewicz) located between T9 –T11 levels. Incidence of paraplegia is reported at ~1% for abdominal aortic aneurysm repairs however it ranges from 7-40% for thoracic aneurysm repairs. Renal ischemia is also concerning. The kidney can tolerate 30-60 minutes of total renal ischemia before irreversible damage. Cross clamping below the renal arteries produces a 75% increase in renal vascular resistance and 38% decrease in renal blood flow that lasts for approximately 1-hour after clamp release. These results are secondary to renal vasoconstriction caused by sympathetic activation as well as activation of the renin-angiotensin system initiated by the cross clamp. Clamping above the renal arteries causes proximal hypertension and distal hypoperfusion with large decreases in GFR, urine output, and renal blood flow.

CROSS-CLAMP RELEASE

Hypotension is secondary to refilling of post clamp organs / vascular beds and hypoxic induced vasodilation which occurs upon release of the clamp. With anticipation, the hemodynamic changes can be blunted by increasing preload, initiation of pressors, and having the surgeons do a controlled / slow unclamping. It is imperative to remember that aortic cross-clamping results in activation of the sympathetic nervous system, clotting cascade, renin-angiotensin systems, and activation of prostaglandins / myocardial depressant factors. Be prepared for cardiac depression, DIC, and severe metabolic acidosis. Patients with heart disease are prone to myocardial ischemia. After release of the clamp, total body oxygen consumption increases (therefore mixed venous oxygenation saturation declines). Metabolic acidosis and increase in $PaCO_2$ production is best managed with increased ventilation and fluids.