The author reviews the etiology of aortic aneurysm and the anesthetic management of the patient undergoing aortic surgery. She also discusses the complications that may occur and offers steps to counteract them.

Significant progress has been made during recent years in the surgical treatment of aortic aneurysm. Surgical techniques have been developed and improved for successful treatment regardless of the type, location, and extent of the disease, thus, operative mortality has steadily declined.

The management of patients undergoing such procedures represents a major challenge for the anesthetist. The typical victim of aortic disorders is advanced in years and has generalized arteriosclerotic vascular disease including coronary artery and/or cerebral vascular insufficiency, as well as hypertension, diabetes, and renal dysfunction. The surgical procedure can be strenuous for these patients for it may last many hours and there may be significant blood loss, volume shifts and electrolyte and temperature changes. Aortic cross-clamping and declamping are particularly hazardous for the patient. Hypertension, myocardial ischemia, and arrhythmias have occurred after application of the cross clamp, while unclamping often results in severe hypotension with myocardial, cerebral or renal ischemia.

Pathogenesis of aneurysms

The etiology of aneurysms is diverse. Syphilis, once a major cause of arterial disease, has declined in importance but it is the basis of about 5-10% of aneurysms, especially in the ascending aorta. Trauma; Marfan’s syndrome, a hereditary disease characterized by arachnodactyly, deformities of the thoracic cage, dislocated lens, and often thoracic aneurysms; Takayasu’s syndrome, also known as pulseless disease, a bizarre connective tissue disease characterized by stenosis or occlusion of vessels arising from the aortic arch; and other rare degenerative vascular diseases constitute another 10%, especially in younger patients. Congenital aneurysms are rare.

The large “residue” resulting from arterial disease accounts for more morbidity and mortality in the sixth and seventh decades of life than all other diseases combined. This residue refers to arterio- and athero-sclerosis, still thought to be an inevitable accompaniment to aging. Genetic factors are strong determinants of at least three factors predisposing to atherosclerosis: serum lipids, blood pressure, and blood sugar. About 10% of the world population has, or will have, hypertension. Whether essential or secondary to other diseases, notably renovascular disease, hypertension raises the incidence of cerebrovascular accidents (CVA) and myocardial infarction (MI). Twenty percent of vascular patients have diabetes mellitus, and arteriosclerosis is the most common complication of diabetes.
Cigarette smoking has been positively linked to Buerger's disease. No direct relationship between consumption of moderate amounts of alcohol and arterial disease has been shown. The essential factor in the pathogenesis of aneurysms is damage to the medial elastic coat of the vessel, leaving only the outer adventitia to withstand the repeated force of systole. Once this has occurred, progressive dilatation begins at the weakened area and pressure is exerted on surrounding tissue. Compensation may take two forms. One is development of fibrous tissue secondary to chronic irritative hyperplasia on perianeurysmal tissues. The other involves deposition of laminated thrombi on the inner surface of the aneurysm in an effort to maintain normal size of the lumen. Perhaps the greatest challenge for the anesthetist is the management of the hemodynamic changes produced by aortic cross-clamping without shunts. Complications do not always depend on the position of the clamp. They occur when the aorta is clamped and the systemic circulation is suddenly reduced, leading to increased system arterial pressure (SAP), pulmonary artery pressure (PAP), and peripheral vascular resistance (PVR). The PVR and SAP drop considerably when the clamp is released.

Using mongrel dogs and direct measurement techniques, Brusoni demonstrated the following changes during 20 minutes of thoracic aortic cross-clamping: (1) coronary artery blood flow (CABF) increased 65%; (2) cardiac work increased 77%; (3) left ventricular systolic pressure increased over 62%; (4) mean arterial pressure increased 55%; (5) cardiac work increased; (6) increased dp/dt; and, (7) myocardial O2 consumption increased. All of these values decreased to lower than baseline after declamping. Contractility indexes slowly decreased, continuing after declamping. Mean coronary resistance showed no significant change during clamping, but decreased below baseline upon clamp removal. Surgical experiments with 13 patients for resection of abdominal aortic aneurysms indicated that cardiac reserve is the major factor in determining operative survival. Cardiac index falls following induction of anesthesia while mixing time rises, indicating compromised left ventricular function. Cardiac index drops further with clamp application and mixing time again rises. The fall in stroke volume is also significant and continues with declamping, as does the decrease in cardiac index. Mixing time increases to the highest level following removal of the second clamp. Most reports of aortic operations find 50% of postoperative deaths to be of cardiac origin—congestive heart failure (CHF), MI, and arrhythmias. The cardiovascular complications almost always occur at the time of clamp application. The relationship between myocardial oxygen and available subendocardial blood supply is disrupted by left ventricular outlet obstruction. The increase in total peripheral resistance (TPR) from absence of collateral vessels and the subsequent increase in afterload adversely affect the myocardial O2 supply-demand ratio. Thus, contractility worsens.

Another study involving 12 patients showed that immediately after two minutes of aortic occlusion, there are significant increases in SAP and systemic vascular resistance. The maximum changes take place after eight minutes of clamping, with SAP increasing by 13% and vascular resistance by 37%. Right atrial pressure and heart rate do not differ significantly from controls. Obviously, this great increase in afterload adversely affects cardiac output. However, there are physiological compensatory mechanisms which can maintain stroke volume in healthy hearts in spite of this load. The first is the Starling mechanism, in which increases in afterload cause the amount of blood remaining in the ventricle at the end of systole to increase. A larger end diastolic volume remains, resulting in fiber elongation. The consequent increase in contractile force maintains stroke volume.

The second mechanism was demonstrated by Anrep and is secondary to the enhancement of

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contractility induced by increased myocardial activity. Ventricular work is augmented and ventricular pressure is increased. This autoregulation depends on the level of circulating catecholamines. When catechol levels are high, the heart can maintain stroke volume against increased afterload without increasing end diastolic pressure. But in a diseased myocardium, the lack of an inotropic response precipitates failure when afterload increases. 

It is known that anesthesia, old age, and hypertension modify the circulatory reflexes. The hemodynamic modification following aortic cross-clamping can be explained in part by inadequate reflexes, either at the level of the baroreceptors, medulla, afferent pathways or effectors. 

Clinically, one may expect no significant change in mean arterial pressure (MAP), while systolic blood pressure (BP) increases and diastolic BP stay the same, as does rate pressure product (RPP) if vasodilators are used. Heart rate, central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) do not change greatly with clamp application or removal. Upon declamping, systolic BP, MAP, diastolic BP, RPP, and TPR fall. The same work is performed, a better cardiac output is obtained, and myocardial O2 cost decreases.

Obviously, without very careful management and oftentimes even with it, significant hypotension follows release of the aortic clamp. Hypotension is potentially fatal in patients with occlusive arterial disease of the coronary or cerebral arteries. Though the exact cause of the hypotension is not known, several explanations have been offered.

Following release of aortic occlusion, blood returning from previously ischemic limbs as well as blood in the systemic circulation is more acid than normal. Reactive hyperemia intensifies the systemic effect by "washing out" acid metabolites due to poor tissue perfusion and ischemia. There is a tendency toward excess lactate production during occlusion and a further increase with declamping. Arterial and venous pH tend to fall with release of aortic occlusion. Metabolic acidosis decreases the responsiveness of the vasculature and heart to sympathomimetic amines. An acid load may decrease vascular tone, causing pooling of blood and decreased cardiac output, possibly by direct effect. Sequestration of blood in the lower extremities results in decreased venous return, decreased cardiac output, and decreased BP. Ischemic tissue distal to occlusion may produce an unknown vasodepressor substance with local and systemic effects that cause hypotension.

Metabolically, myocardial O2 consumption increases during clamping and decreases with clamp removal. Arterial and coronary venous lactate increase slowly during clamping and continues to rise after declamping, with myocardial lactate consumption increasing during clamping. Arterial pyruvate decreases during clamping but rapidly returns to normal. Oxygenation shows continuous improvement throughout.

Added complications may occur as shock, acidosis and blood stasis, leading to intravascular coagulation. A syndrome of multiple pulmonary microemboli has been described with pO2 levels as low as 20 torr. Inadequate oxygenation further aggravates declamping acidosis and impairs the buffering mechanisms that compensate for it. Transfusion of large amounts of bank blood, because of low (6.4-7.1) pH and lactate content (1.1-2.9 mEq) complicates the picture.

In conjunction with metabolic and cardiovascular changes, cross-clamping of the aorta has frequently been associated with renal failure. Incidences of up to 47% decreased renal function, 37% oliguria, and 11% mortality have been reported. Modern techniques of anesthesia and surgery have decreased the occurrence of renal failure to 5-5%; however, the mortality of that complication, should it present, approaches 25% despite therapy. The etiology is obscure. While early experiments demonstrated acute tubular necrosis, subsequent findings concluded that aortic cross-clamping has no detrimental renal effect. Normal function requires adequate extracellular fluid replacement sufficient to prevent hypotension and maintain normal cardiac output, renal blood flow, and urine output. Previous reports of decreased renal blood flow, creatinine clearance, and urine output generally occurred when insufficient saline was administered.

Anesthetic management

In light of the above, successful anesthetic management of patients undergoing aortic surgery requires careful monitoring, prevention, and treatment of the changes noted. Some hospitals regard vascular surgery as a prime indication for regional anesthesia because a vasospastic element involving arteriosclerotic vessels is relieved by sympathetic blockade. Sympathetic blockade increases skin temperature, pulsation, and skin and muscle blood flows, and decreases PVR and increases oxygenation of venous blood. These circulatory changes are beneficial in the absence of hypotension.

Studies demonstrate that there is little difference between general and epidural techniques.
though declamping hypotension without volume loading may be more likely with epidural anesthesia. The discomfort for the patient of lying prone for many hours can be a difficult problem. The anxiety of patients with ischemic heart disease and angina may override any advantage of regional anesthesia.\(^1\)

Whether a regional or general technique is chosen, the time spent at the preoperative visit to gain the patient's confidence and to educate him as to what to expect goes a long way in decreasing anxiety. There are many drugs and combinations of drugs available to alleviate anxiety in the elective surgical patient. Morphine sulfate and scopolamine may be given safely in poor risk cardiac patients to produce sedation and some amnesia. Narcotics, tranquilizers, sedatives and anticholinergics are used routinely unless there are specific contraindications.

Baseline arterial blood gases should be drawn prior to surgery and metabolic acidosis needs to be treated if present (total dose mEq NaHCO\(_3\)=\(\frac{1}{2}\) body weight [kg] \(\times\) base excess).\(^7\)

The extent of monitoring is dependent upon the nature of the operation and the patient's medical condition. All patients should have at least one, and preferably two, large-bore IV catheters as well as ECG, temperature, BP, and urine output monitoring. Indwelling arterial catheterization via radial, ulnar, brachial or axillary artery allows continuous direct BP monitoring as well as readily available samples of arterial blood gases. A central venous catheter should be used for measuring CVP and for administration of cardiotoxic drugs. In patients with previous cardiac failure, unstable angina, or recent MI, a pulmonary artery catheter is required as a guide to left ventricular function and volume.

Patients with significant pulmonary and cardiac disease for major vascular operations are best managed with general endotracheal anesthesia and careful monitoring. Although the technique of balanced anesthesia with \(\text{N}_2\text{O}\), narcotics, relaxants and barbiturates provides excellent operative conditions, it frequently requires the addition of either an inhalation agent or IV vasodilator to manage hypertension that develops with clamping.

Regardless of the choice of technique, induction must be managed to minimize the sympathetic activity, hypertension and tachycardia produced by laryngoscopy and intubation. After pre-oxygenation, adequate depth must be achieved and the trachea sprayed with 2-3 ml 4% lidocaine (Xylocaine\(^8\)). Struggling, coughing, and straining may cause hypoxia and hypertension, possibly resulting in aortic dissection or rupture. Infusion of trimethaphan (Arfonad\(^8\)) 1 mg/ml or sodium nitroprusside (Nipride\(^8\)) during induction helps control the BP response. A full stomach presents even greater hazard and risk. “Crash” induction with the Sellick maneuver should be utilized to prevent regurgitation and aspiration.\(^11\)

Equally important is the avoidance of hypertension from myocardial depressants or peripheral vasodilators. A useful approach in elective patients is slow titration of narcotics as part of induction such as morphine 0.5-1 mg/kg, thus avoiding myocardial depression and hypotension.

Muscle relaxants should be chosen according to the circulatory effect desired or best avoided. Because of its ability to decrease BP, d-Tubocurarine may be preferred. Pancuronium (Pavulon\(^\circledR\)) and gallamine are known producers of tachycardia and should be titrated slowly or avoided completely. Metocurine is useful because its cardiovascular effects are minimal.\(^12\)

### Intubation

Patients with aneurysms who undergo left thoracotomy in the right lateral decubitus position benefit from use of a double lumen endobronchial tube. The advantages are: (1) surgical exposure is facilitated by collapse of the left lung, and a quieter surgical field is presented; (2) trauma to lung tissue is diminished; and (3) the dependent right lung is protected from contamination from the left lung.

Intrapulmonary bleeding is caused by: (1) surgical separation of the lung from the aneurysm; (2) contusion of lung by retractors; (3) forceful ventilation against retractors; and (4) obstruction of pulmonary veins. Hemorrhage tends to continue after heparinization if cardiopulmonary bypass is utilized.

\(\text{PO}_2\) during ventilation of one lung tends to decrease with time, with shunt increasing with duration of atelectasis. After collapse of greater than one hour, an \(\text{FIO}_2\) of less than 100 may prove hazardous. \(\text{CO}_2\) elimination may pose a problem in patients who are hypercarbic before the operation.

With one lung partially or totally atelectatic, unoxygenated venous blood flow through the perfused but non-ventilated left lung mixes with blood returning from the ventilated right lung where uptake of \(\text{O}_2\) and anesthetics takes place. This admixture results in dilution of arterial anesthetic concentration with lightening of depth when inhalation agents are used. Positive end-expiratory pressure (PEEP) will prevent complete expiratory atelectasis of the dependent lung.
Use of the Carlens™ double lumen or the Robert Shaw™ left sided endobronchial tubes is recommended. The position of either must be verified, with the patient in both the supine and right lateral positions, by breath sounds after inflation of each separate lumen.

While the use of endobronchial tubes is considered to be a significant contribution to resection of thoracic aneurysms, their use in long-term intubation is not practical. Small tube diameters increase resistance to ventilation and create an unusual nursing care demand for suctioning, which may cause obstruction of the lumen of the tube. Rubber composition and the use of high pressure/low volume cuffs further limit clinical application for prolonged use.

This rise in afterload mentioned earlier which occurs with clamp application may be managed by judicious titration of peripheral vasodilators such as sodium nitroprusside, trimethaphan or nitroglycerin. Halothane, which decreases peripheral vascular resistance and increases skin and muscle blood flow, has proven useful in this situation.

As a decrease in renal cortical blood flow is associated with aortic cross-clamping, mannitol has been advocated to maintain urine output. Patients not diuresing adequately in the preclamping period with adequate hydration should be given 12.5-25.0 gm of mannitol. Mannitol also has a direct vasodilating effect on the coronary vasculature. Decreasing endothelial swelling may result in increased capillary patency. Increased blood flow allows a more rapid washout of accumulated metabolic end products and replenishes the intracellular bicarbonate buffer system.

Mannitol is an osmotic diuretic completely excreted by the kidney together with a volume of obligatory water of about 3 ml/gm. Because it requires water for excretion, mannitol should not produce diuresis in dehydrated patients. As it increases the osmotic pressure of extracellular fluid, it draws water from the intracellular compartment, especially from red blood cells. The fall in hematocrit without change in hemoglobin or red blood cell count produces a reduction in viscosity, a decrease in renal vascular resistance, and an increase in renal blood flow.

If mannitol is ineffective, 10-20 mg of furosemide (Lasix®) usually brings about a continued adequate urine output. Potassium supplementation may be necessary during diuresis, as determined by frequent electrolyte checks.

Dextran prevents erythrocyte aggregation and sludging in small blood vessels because it is a volume expander. Dextran 40 prevents early thrombosis by coating the red blood cell membrane, conferring a negative charge and repelling red blood cells from the negatively charged endothelium. Even when given according to specifications, allergies and oozing are the most frequent side effects of dextran.

Heparinization is beneficial to graft placement and function. An IV dose of heparin 1 mg/kg (100 units/mg) prolongs clotting time fivefold. The effect of heparinization lasts one to three hours. Its inactivation is achieved by neutralization of the strong electronegative charges by basic compounds such as protamine. However, if excessive amounts of protamine are injected rapidly IV, protamine itself can act as an anticoagulant and can cause hypotension by direct myocardial depression.

Probably the most important aspect of management, aside from careful monitoring, is the administration of adequate volume. Lactated Ringer's solution should be administered in doses of 750-1000 ml/hr with additional blood and colloid solutions given as needed. Prevention of hypotension with clamp removal is thus achieved.

The anesthetist should be notified at least five minutes prior to clamp removal. This will allow use of a vasodilator to increase venous capacitance and to permit more volume to be infused before clamping. When volume is administered to maintain PCWP between 10-20 torr, hypotension is insignificant when the cross-clamp is released.

While hypovolemia must be avoided, pulmonary edema is equally dangerous. In patients with decreased cardiac reserve, the transition from just enough to too much fluid may occur rapidly. PCWP is an excellent indicator of left atrial pressure; the CVP has been shown to vary greatly from PCWP in patients with abnormal cardiopulmonary function. This is because changes occur more rapidly and to a greater degree on the left than on the right. CVP is accurate in 90% of patients, but there is a small group in which there is no substitute for PWCP monitoring.

Note: To minimize a fall in body temperature, blood and fluids should be warmed and the operating room should be at least 75° F.

In treating emergency aneurysms, the main priorities are control of hemorrhage and volume replacement. Mannitol is administered early to preserve renal function. Anesthesia is minimal: ketamine, diazepam, droperidol, scopolamine, and N₂O are administered as hemodynamics permit. The patient with a leaking aneurysm should be brought to the operating room and IV and intra-arterial lines inserted as time permits. Preoxygenation and defasciculation should follow prior to

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induction while the patient is being prepared and draped. The anesthetic drugs may be titrated.\textsuperscript{18}

Emergence

Anesthetic emergence should proceed gradually, accompanied by controlled ventilation. Slow emergence decreases the likelihood of excitement, shivering, and postoperative instability. Muscle relaxants are allowed to wear off spontaneously. Transport is best managed with ventilatory support and an assurance that the patient is well anesthetized. Portable monitors should follow cardiovascular function during transport. Ventilation, circulation, renal function, coagulation, electrolyte balance, and level of consciousness are important parameters to be observed postoperatively.\textsuperscript{12}

REFERENCES

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Donna Lee Hinton, CRNA, BSN, received her BSN from the University of Mississippi in 1977. After working for two years in an Intensive Care Unit, she entered the University of Mississippi Medical Center School of Nurse Anesthesia. She graduated in May, 1981 with a second Bachelor's degree in Anesthesia, receiving the school's award for academic achievement. She is currently a staff nurse anesthetist at Woman's Hospital in Jackson, Mississippi. This article was written while Mrs. Hinton was a senior nurse anesthesia student.