Review Article

Anaesthesia for abdominal aortic surgery - a review (Part II)

Anthony J. Cunningham FFARCSI FRCP

Intraoperative anaesthetic management

The objectives of anaesthetic management include an intensive preoperative assessment of risk factors and management of coexisting disease states; the utilization of monitoring techniques to detect promptly signs of myocardial ischaemia and impaired myocardial contractility; maintenance of adequate intravascular volume, optimal cardiac output and tissue oxygenation; avoidance or prompt pharmacological amelioration of untoward haemodynamic or metabolic changes associated with aortic clamping and unclamping; and intensive postoperative care.

Monitoring

Extensive monitoring of patients presenting for aortic vascular surgery is mandatory. Standard monitoring practice should include the patient's exposed hand to check colour, capillary filling and radial pulse palpation; continuous electrocardiographic display — lead II for dysrhythmia detection and a precordial V_5 lead,¹⁸⁰ modified bipolar CM_5 lead¹⁸¹ or oesophageal lead¹⁸² to detect S-T segment changes associated with myocardial ischaemia; oesophageal stethoscope with thermal probe for heart/breath sounds auscultation and temperature monitoring; neuromuscular function monitoring and bladder catheterization for urine output determination. Direct intraarterial pressure monitoring should be standard practice to permit beat to beat recognition of arterial pressure and any adverse haemodynamic response to aortic clamping procedures and for frequent blood gas determinations (Table IV). Occasional complications may be anticipated in patients with severe vascular disease in the cannulated extremity, prolonged low cardiac output states, particu-

late embolization and arterial trauma following cutdown and ligation.¹⁸³

PRELOAD

Pulmonary artery catheterization is indicated in the majority of patients presenting for abdominal aortic surgery to optimize intravenous fluid administration and to detect adverse haemodynamic or ischaemic changes associated with aortic cross-clamping and release. Mangano¹⁸⁴ noted a close and clinically useful correlation between central venous (CVP) and pulmonary capillary wedge pressure (PCWP) changes induced by alterations in preload in patients with normal left ventricular function (ejection fraction >0.5) presenting for coronary artery by-pass surgery. Close correlation between changes in central venous and pulmonary capillary wedge pressure have also been reported following aortic cross-clamp application and release.¹⁸⁵ Although changes in CVP accurately predicted the magnitude and direction of PCWP changes in the majority of patients studied, there existed a group of patients in whom there was no substitute for left-sided filling pressure measurement. Because fewer haemodynamic derangements follow aortic cross-clamping in patients with aorto-occlusive disease and good collateral vascularization compared with abdominal aortic aneurysm patients, central venous pressure may closely reflect the balance between intravascular volume, venous capacitance and left ventricular function in this patient population if no significant coronary artery disease or ventricular dysfunction is apparent.¹⁸⁶

TABLE IV Abdominal aortic surgery — standard monitoring

Clinical
ECG: lead II dysrhythmia
CM_5 ischaemia
Oesophageal stethoscope
Temperature
Urine output
Neuromuscular function
Direct intraarterial pressure
Preload: CVP $\pm$ PCWP

Received from Department of Anaesthesia, Royal College of Surgeons in Ireland.

Address correspondence to: Dr. Anthony J. Cunningham, Department of Anaesthesia, Royal College of Surgeons in Ireland, Professorial Unit, Beaumont Hospital, Dublin 9, Ireland.

Changes in right and left ventricular compliance are common in patients presenting for aortic vascular surgery.¹⁸⁷ Wide variations have been reported in the prediction of a change of PCWP value based on a known change in CVP and preoperative resting left ventricular ejection fraction does not identify a sub-group of patients in whom CVP and PCWP correlate consistently throughout surgery.¹⁸⁸ Recent reports have cited improved left ventricular¹⁸⁹ and renal performance,¹⁵⁶ with lower morbidity and mortality, following pulmonary artery catheterization and appropriate therapy based on the haemodynamic data obtained.

MYOCARDIAL PERFORMANCE

Pulmonary artery catheterization is especially indicated in patients with a history of previous myocardial infarction, angina pectoris or signs of cardiac failure; in patients demonstrating diminished ejection fraction or abnormal ventricular wall motion on preoperative resting or exercise radionuclide or echocardiographic studies and in patients with evidence of redistribution on dipyridamole-thallium imaging. In addition to measurement of pulmonary artery and capillary wedge pressure, pulmonary artery catheterization facilitates calculation of derived cardiac indices (stroke volume, cardiac index, left ventricular stroke work index) systemic and pulmonary vascular resistance and intrapulmonary shunt (Table V). The appearance of an abnormal V wave on the PCWP trace may indicate the onset of myocardial ischaemia before surface ECG S-T segment changes occur.¹⁹⁰ The incorporation of fiberoptic oximetry into pulmonary artery catheters has enabled the perioperative monitoring of mixed venous oxygen saturation (SvO₂). When employed in conjunction with other indicators of tissue oxygenation, SvO₂ may serve as a useful assessment of cardiac output and the response to pharmacological interventions during aortic vascular surgery.¹⁹¹

The incorporation of intraoperative transoesophageal two-dimensional echocardiography into anaesthetic practice has provided a practical means of estimating left ventricular dimensions and myocardial performance in addition to the detection of wall motion abnormalities. Radionuclide cardiography monitoring techniques will evolve from investigative research tools to intraoperative clinical monitoring techniques. Measurement of end-diastolic volume and end-diastolic area allows a more precise definition of left ventricular preload, compared with central venous and pulmonary capillary wedge pressure measurements.¹⁹² Left ventricular ejection fraction changes associated with induction of anaesthesia and critical events during surgery may be monitored and treated.¹⁹³ Favourable initial comparisons between intraoperative cardiac output determination with transoesoph-

TABLE V Abdominal aortic surgery, supplementary intraoperative monitoring techniques

<i>Ischaemia detection</i>
ST segment analysis
PCWP tracing
Regional wall motion abnormalities
<i>Preload</i>
Transoesophageal echocardiography
Nuclear angiography
<i>Left ventricular function</i>
LVEF—echocardiography
LVEF—radionuclide ventriculography
LVSWI—(derived)
<i>Cardiac output</i>
Thermodilution
2D echocardiography
Doppler echocardiography
<i>Afterload</i>
Systemic vascular resistance
End-systolic wall stress—echocardiography
<i>Tissue oxygenation</i>
SVO ₂ —fiberoptic oximetry

ageal echocardiography and thermodilution techniques have been reported.¹⁹⁴ In addition, the development of regional wall motion abnormalities is an earlier and more sensitive method of detecting myocardial ischaemia, compared with surface ECG S-T segment changes.¹⁹⁵ Less reduction in coronary blood flow is required to produce hypokinesia compared with ECG changes and contraction abnormalities appear first in the endocardium, preceding S-T segment changes from epicardial leads.

Anaesthetic technique

No single anaesthetic agent or technique is ideal for all patients presenting for aortic vascular surgery. The anaesthetic agents and techniques chosen should ensure a smooth induction of anaesthesia, a favourable cardiovascular dose-response relationship which preserves the delicate myocardial oxygen supply/demand balance, adequate muscle relaxation with intraoperative analgesia and amnesia. The choice of anaesthetic techniques includes nitrous oxide/oxygen with incremental volatile agent or opiate supplementation; opiate-oxygen or an opiate-oxygen-volatile agent combination with or without regional anaesthesia.

With the development of inert gas and labeled microsphere techniques for measuring coronary blood flow and metabolic indices for detecting myocardial ischaemia,¹⁹⁶ it is now accepted that an anaesthetic technique which produces low-pressure, low-myocardial oxygen demand

provides better preservation of myocardial oxygenation compared with a high-pressure, high-myocardial oxygen demand technique.¹⁹⁷ Only limited data are available in the literature on the effect of anaesthetic technique on cardiac performance during aortic vascular surgery. A "balanced" nitrous oxide-oxygen-opiate-relaxant technique¹⁹⁸ and a nitrous oxide-oxygen-volatile agent-relaxant technique¹⁹⁹ have been associated with depressed left ventricular performance following induction of anaesthesia which persisted following aortic cross-clamp application. The addition of 50 per cent nitrous oxide to anaesthesia with halothane, enflurane or fentanyl in patients with coronary artery disease was associated with depressed left ventricular performance and metabolic evidence of subtle myocardial ischaemia.^{200,201} Hence the use of opioid analgesics as sole anaesthetic agents with oxygen and muscle relaxants for patients undergoing cardiac and major vascular surgery has achieved widespread popularity in recent years. Advantages cited include stable cardiovascular dynamics in patients with ischaemic heart disease,²⁰² with suppression of the humoral and metabolic (catecholamine, cortisol, antidiuretic and growth hormone) responses to anaesthesia and surgery,²⁰³ and prolonged postoperative analgesia. High-dose fentanyl ($100 \mu\text{g} \cdot \text{kg}^{-1}$) oxygen-relaxant anaesthetic technique has been evaluated for elective abdominal aortic surgery. Such an unsupplemented anaesthetic technique may be associated with an unacceptably high incidence of hyperdynamic circulatory responses to surgical stimuli before, during, and after aortic cross-clamping.^{204,205} Moreover, a prolonged elimination half-time was observed, which may be associated with prolonged postoperative respiratory depression, especially in patients with pre-existing pulmonary disease.²⁰⁶

Enflurane or halothane with oxygen consistently depresses myocardial contractility and oxygen demand and early postoperative hypertension has been frequently observed with these techniques.²⁰⁷ A combination of an opiate (fentanyl $30 \mu\text{g} \cdot \text{kg}^{-1}$ induction with maintenance infusion) with a volatile agent halothane²⁰⁸ or enflurane²⁰⁹ in oxygen maintains a reduced myocardial contractility, unchanged systemic vascular resistance, and preserved myocardial oxygenation in patients with good left ventricular function undergoing coronary artery by-pass grafting. The volatile agent may be titrated to prevent autonomic responses to stressful surgical stimuli. Theoretically, isoflurane is the volatile anaesthetic agent of choice for aortic vascular surgery. Despite its direct myocardial depressant effects, isoflurane administration is associated with preserved cardiac output due to increased heart rate and reduced systemic vascular resistance.²¹⁰ In experimental studies performed with intact myocardial tissue and using equipotent concentrations, a lesser direct negative inotropic effect was observed with

isoflurane than with either halothane or enflurane.²¹¹ A low concentration of isoflurane with nitrous oxide has been reported to protect against pacing-induced myocardial ischaemia in patients with documented ischaemic heart disease.²¹² Improved hepatic artery blood flow with comparable renal and cerebral blood flow has been reported with isoflurane compared with halothane.²¹³ Unfortunately, in patients with coronary artery disease, isoflurane administration may be deleterious if a vasodilatory or coronary steal effect is produced. Moffitt *et al.*²¹⁴ studied ten patients with good left ventricular function presenting for coronary artery by-pass surgery following isoflurane-oxygen administration for induction and maintenance of anaesthesia. Despite stable cardiac index and coronary blood flow, coronary vascular resistance decreased by 23 per cent and metabolic indices of global myocardial ischaemia were noted in three of ten patients studied. No such adverse effects were reported by the same authors in similar patient populations given halothane or enflurane.²⁰⁷ A higher incidence of postoperative congestive heart failure and renal insufficiency has been reported in patients following aortic surgery with isoflurane anaesthesia than with a sufentanil-based anaesthetic technique.²¹⁵

The coronary arteriolar vasodilating properties of isoflurane are now well documented in experimental and clinical studies.^{216,217} The clinical implications of isoflurane-induced coronary arteriolar dilatation in patients with coronary artery disease remain controversial.²¹⁸ Comparisons between isoflurane and halothane are difficult because studies have been performed using two markedly different coronary perfusion pressures. The adverse coronary steal effects associated with one per cent end-tidal isoflurane administration have been highlighted by Reiz *et al.*²¹⁹ in patients with coronary artery disease presenting for aortic vascular surgery. With decreased coronary vascular resistance, myocardial oxygen consumption and extraction, ten of 21 patients developed ECG and metabolic changes associated with myocardial ischaemia. In contrast, the same authors noted decreased cardiac index, systemic vascular resistance, coronary sinus blood flow, myocardial oxygen consumption and no myocardial lactate production in aortic vascular surgery patients with coronary artery disease and evidence of heart failure using one per cent end-tidal halothane in 30 per cent oxygen.²²⁰ The authors suggested that unloading of the left ventricle by systemic vasodilatation predominates over the cardiac-depressant action of halothane in this patient population.

MANAGEMENT GUIDELINES

Guidelines for clinical practice based on data from patients with coronary artery disease and good left ventricular function presenting for coronary artery by-

pass grafting and limited information on patients with normal and impaired ventricular function undergoing abdominal aortic surgery would suggest that a combined opiate-oxygen-volatile anaesthetic agent will best ensure a hypodynamic circulation with preservation of myocardial oxygenation. Adequate premedication is required to avoid adverse haemodynamic responses to the placement of monitoring catheters before induction of anaesthesia.²²¹ Induction with fentanyl 30–50 $\mu\text{g} \cdot \text{kg}^{-1}$ followed by maintenance fentanyl infusion in combination with oxygen-enflurane or oxygen-halothane should provide optimal myocardial supply:demand conditions. Based on current information, nitrous oxide and isoflurane should be used with caution in this patient population because of the reported risks of insidious global or regional myocardial ischaemia.

The prophylactic or specific use of vasodilatory agents to prevent haemodynamic changes associated with surgical interventions during aortic vascular surgery remains controversial. Evidence from experimental studies suggests that nitroglycerin infusion during aortic cross-clamping maintains cardiac index by preventing an increased systemic vascular resistance²²² while producing a transmural distribution of coronary blood flow which favours the endocardium.²²³ In a clinical study low-dose (0.25 $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) nitroglycerin infusion was reported to sustain myocardial contractility at pre-clamp levels, while preserving peripheral tissue blood flow. Sodium nitroprusside infusion may maintain normal intrarenal blood flow distribution and may preserve splanchnic blood flow during infrarenal aortic cross-clamping.²²⁴ However, nitroglycerin administration may decrease intestinal blood flow and shunt blood from the distal colon during aortic occlusion.¹⁷² Sodium nitroprusside administration may produce myocardial ischaemia when administered in doses sufficient to reduce mean arterial pressure to 80 mmHg or less, presumably due to decreased coronary perfusion pressure, metabolite-induced toxicity²²⁶ or a direct coronary vasodilatory steal effect.²²⁷ The balance of evidence currently available would support the administration of 1–2 $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ nitroglycerin infusion if hypertension, impaired myocardial contractility and tissue oxygenation or signs of myocardial ischaemia develop following aortic cross-clamp application.²²⁸ This therapy should decrease arterial pressure, systemic vascular resistance and myocardial oxygen consumption. Dobutamine intravenous infusion may also be administered to sustain myocardial contractility if evidence of impaired contractility develops following aortic cross-clamping.²²⁹

Intraoperative fluid and blood transfusion therapy

Patients undergoing abdominal aortic surgery usually experience major functional extracellular fluid and blood

loss. Functional extracellular fluid loss into a non-functional or "third-space" may follow extensive tissue trauma, manipulation, exposure and retraction.²³⁰ Sequestration of fluid within the lumen wall of the intestine and major retroperitoneal oedema account for most of the fluid shift from the circulation. The issue of whether a crystalloid or colloid intravenous fluid regimen better preserves circulatory homeostasis and renal function during major vascular surgery has not been resolved. Restoration of normal intravascular and interstitial fluid volumes is a primary objective in intravenous fluid administration.

Larger volumes of crystalloid solution will be required to restore the intravascular volume compared with colloid solutions. Advocates of a crystalloid regimen claim greater urine output and a reduction in the incidence of oliguric renal failure following abdominal aortic surgery which they attribute to the use of balanced salt solution.¹⁰ Despite the large volumes infused and the lower colloid osmotic pressures, similar extravascular lung water and pulmonary shunt fractions have been reported in studies^{232,233} comparing crystalloid with colloid infusions during major vascular surgery. Conflicting data have been published showing improved postoperative respiratory function following colloid administration.^{234,235} A combination of balanced salt and colloid solutions, guided by appropriate venous pressure monitoring, will ensure adequate intravascular volume, optimal cardiac output, satisfactory renal and end-organ blood flow, and minimal extravascular losses into the pulmonary interstitium and traumatized tissues.

A balanced salt solution, with or without a colloid solution, should be infused in volumes sufficient to maintain a PCWP of 10–15 mmHg during the cross-clamp period and to ensure a urine output greater than 60 $\text{ml} \cdot \text{hr}^{-1}$. If urine output is unsatisfactory, despite PCWP measurement of 15 mmHg or greater, diuretic therapy with mannitol or furosemide should be considered. Low-dose dopamine (2 $\mu\text{g} \cdot \text{kg}^{-1}$) following surgery has been reported to increase renal blood flow, glomerular filtration rate, urine output and sodium excretion.²³⁶ The PCWP should be increased to 3–5 mmHg above the preoperative value before cross-clamp release to prevent declamp hypotension and cardiac output reduction.

The presence of a preoperative coagulation or bleeding diathesis should be established on direct questioning of the patient concerning family history, drug ingestion, the presence of bruising or easy bleeding and the detection of abnormalities on physical examination and laboratory investigation. Partial thromboplastin time (PTT), prothrombin time (PT), thrombin time, platelet count and bleeding time will help determine the nature of any haemostatic defect encountered. Up to 40 per cent of patients presenting for abdominal aortic surgery will have

significant elevation of fibrin split products. Overt disseminated intravascular coagulation will be detected only in a small minority of cases.²³⁷

The greatest blood loss during elective abdominal aortic aneurysm resection usually occurs when the aneurysm is opened and the lumbar arteries are back-bleeding. Extensive blood loss may also arise at proximal and distal graft anastomoses, following graft flushing or secondary to inadequate surgical haemostasis, excess heparinization or bleeding diathesis. Blood transfusion should be commenced when blood loss exceeds 15 per cent of the patient's estimated blood volume. Homologous blood transfusion requirements have ranged from 1.5 to 5.5 units depending on surgical skill, the extent of grafting procedures and the duration of surgery.²³⁸ The potential complications of multiple homologous blood transfusions during elective aortic vascular surgery are well known, especially the risk of transfusion-transmitted diseases such as acquired immune deficiency syndrome, hepatitis and syphilis.²³⁹

The techniques currently available to minimize homologous blood transfusion during elective aortic vascular surgery include multiple pre-deposit autologous collection, storage and retransfusion; immediate preoperative phlebotomy, haemodilution and autologous transfusion; intraoperative blood salvage and reinfusion. Intraoperative autotransfusion may be an economic method of significantly reducing homologous blood transfusions. Initial reports²⁴⁰ suggested a 40–50 per cent avoidance of homologous blood transfusions, which has increased up to 80 per cent in recent studies.²⁴¹ The initial application of autotransfusion was complicated by haemolysis and coagulation disorders; air and fat emboli; platelet and leucocyte microaggregation and sepsis.²⁴² Technological advances have virtually eliminated the problems of air and particulate embolization. No significant haemolysis or coagulopathies have been noted with autotransfusion techniques in recent studies.²⁴¹ Because autotransfused blood is, in essence, a preparation of washed, packed red blood cells suspended in saline solution without platelets or clotting factors, fresh frozen plasma and platelet concentrate transfusion may be necessary. Autotransfusion, by providing fresh warm blood with optimal pH and 2–3 DPG content,²⁴² may prevent some of the adverse cardiovascular effects associated with extensive stored homologous blood therapy.²⁴⁴

Regional anaesthesia

Continuous epidural anaesthesia has been frequently used in association with general anaesthesia and, on occasion, as the primary anaesthetic technique for patients undergoing aortic vascular surgery.²⁴⁵ Proponents of regional anaesthesia claim reduction in volatile anaesthetic and

narcotic requirements and significant alleviation of postoperative pain. Elevation in skin temperature, increased graft blood flow and reduced muscle blood flow have been observed.²⁴⁶ Combined regional and general anaesthetic techniques may attenuate the increased systemic resistance with aortic cross-clamping²⁴⁷ and may produce stable cardiovascular dynamics following cross-clamp release if intravascular volume is maintained.²⁴⁸ Experimental studies suggest that cervico-thoracic epidural blockade may redistribute coronary blood flow, favouring the endocardium, in both the normal and infarcted heart.²⁴⁹ Recent randomized prospective data from mostly major vascular surgical patients comparing general and epidural-general anaesthetic techniques noted fewer postoperative cardiovascular complications in the group receiving regional anaesthesia.²⁵⁰

Despite the well known and accepted advantages of regional anaesthesia alone, or in combination with general anaesthesia and tracheal intubation, these techniques have not been universally adopted, partly due to the lingering controversy surrounding epidural catheters and anticoagulant therapy. Occasional case reports have documented the occurrence of peridural haematomas leading to transient or permanent neurological impairment.^{251,252} The relative safety of anticoagulation following epidural catheter insertion was impressively established by Rao *et al.*²⁵³ in a study involving 3,164 patients. Utilizing proper patient selection, atraumatic techniques and appropriate heparin administration based on activated clotting time monitoring, these investigators noted no peridural haematoma leading to spinal cord compression. Notwithstanding Odoom *et al.*'s²⁵⁴ report of no epidural haematoma formation or spinal cord compression in 1,000 lumbar epidural blocks in vascular surgery patients already anticoagulated, most authorities consider the use of epidural anaesthesia contraindicated in patients already anticoagulated.²⁵⁵ The use of combined general anaesthesia and continuous lumbar epidural anaesthesia for major aortic vascular surgery has increased during the past decade. This technique has been associated with greater total perioperative fluid volume administration and reduced left ventricular function compared with general anaesthesia alone.²⁵⁶ The merits of combined regional and general anaesthetic techniques, compared with conventional general anaesthesia alone for aortic vascular surgery, must await the publication of more extensive clinical investigations.²⁵⁷

Postoperative management

Patients recovering from aortic vascular surgery are at risk of developing cardiac, respiratory and renal failure in the immediate postoperative period. Close monitoring of the patient's intravascular volume status, temperature, respi-

ratory and renal function will be required in addition to assessment of graft patency and lower extremity blood flow. Provision of adequate analgesia is also a priority. This section will focus on the immediate postoperative ventilatory care and the influence of the site of surgical incision on respiratory function; and the problem of hypertension in the immediate postoperative period.

Respiratory care

The postoperative respiratory deficit is primarily restrictive with decreased functional residual capacity and pulmonary compliance. Pre-existing obstructive defects are compounded by altered secretions, impaired cough and mucociliary clearance, atelectasis and postoperative pulmonary infection.²⁵⁸ Early postoperative intermittent mandatory ventilation is normal practice. This patient population is mostly elderly with frequent co-existing cardiac and respiratory disease. The obligatory extended midline incision, abdominal distension following extracellular fluid sequestration, bedrest in the supine position, abdominal pain requiring narcotic administration, large volume blood and electrolyte infusion, and hypothermia following prolonged procedures all preclude early weaning and tracheal extubation. A slow emergence from anaesthesia reduces the likelihood of agitation, shivering and cardiorespiratory instability in the early postoperative period.²⁵⁹

The standard surgical approach for abdominal aortic reconstruction has been a vertical incision with a transperitoneal approach. Reports in the 1960's advocated a transverse flank incision with retroperitoneal approach for high-risk patients with severe cardiac and respiratory compromise.²⁶⁰ Recent comparative studies have highlighted significantly less postoperative morbidity due to atelectasis and pneumonia associated with the retroperitoneal approach. Minimal visceral manipulation, reduced evaporative fluid losses, better temperature control, shorter nasogastric cannulation and intestinal ileus, reduced intraoperative fluid and blood requirements and shorter hospital stay have all been reported with the retroperitoneal approach.²⁶¹ The transabdominal approach is indicated if co-existing intraabdominal pathology is suspected or if extensive suprarenal aneurysm extension is present.²⁶²

Hypertension

Hypertension is a common and potentially serious complication in the immediate postoperative period following abdominal aortic surgery. Postulated mechanisms include overzealous hydration during anaesthesia and exaggerated replacement of blood loss;²³⁴ postoperative hypothermia with compensatory vasoconstriction;²⁶³ rebound hypertension following vasodilator therapy; pre-

existing hypertension and vascular hyper-reactivity.²⁶⁴ Conflicting findings have been reported by different groups studying plasma renin activity during experimental and human aortic vascular surgery. Depending on the extent of intraoperative hydration, plasma renin activity may increase²⁶⁵ or remain unchanged¹⁴⁸ following aortic cross-clamping. Renal renin response could be reduced by volume infusion and blocked by the prior administration of propranolol. A consistent feature of recent studies^{266,267} is the lack of correlation between postoperative hypertension and plasma renin activity or angiotensin II levels in the recovery room. Preoperative propranolol administration inhibited the renin response to anaesthesia, surgery and aortic cross-clamping but postoperative hypertension still remained a problem in this group.²⁶⁸

A positive correlation has been observed between elevated mean preoperative arterial pressure and postoperative hypertension. Adequate preoperative antihypertensive therapy may be the most important prophylaxis against postoperative hypertension, with its attendant risks of myocardial ischaemia and infarction. Antihypertensive medications should be administered until induction of anaesthesia and should be resumed as soon as possible in the postoperative period. An acute hypertensive response may be observed in the recovery room due to pain, apprehension, volume overload, hypoxaemia or hypercapnia. Treatment of postoperative hypertension involves excluding an underlying cause and prompt initiation of anti-hypertensive therapy. Nitroprusside, commencing at $0.5-1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, or nitroglycerin, commencing at $1-2 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, may be chosen because of their prompt onset, titratable dose-response and rapid reversal on discontinuing drug administration. A more delayed onset of 15-30 minutes and frequent tachycardia may follow intravenous hydralazine administration. Incremental doses of $0.5-1 \text{ mg}$ propranolol may effectively control postoperative hypertension, especially if the patient has been taking beta-adrenergic blocking drugs preoperatively.²⁶⁹ Labetalol, a combined alpha- and beta-adrenoreceptor antagonist, may produce the desired blood pressure reduction without compensatory tachycardia.²⁷⁰ The preoperative administration of 200-300 mg clonidine, an α_2 -adrenergic agonist, may prove a useful agent to prevent hyperdynamic responses in the postoperative period.²⁷¹

Summary

Patients presenting for abdominal aortic surgery have a high incidence of vascular disease elsewhere, manifested primarily by hypertension, coronary and cerebrovascular disease, as well as co-existing respiratory, renal and metabolic disorders. Routine clinical assessment, electro-

TABLE VI Abdominal aortic surgery – guidelines for anaesthetic management

1	<i>Preoperative hydration</i> Maintenance fluids IV overnight
2	<i>Premedication</i> Benzodiazepine $\pm$ opiate
3	<i>General anaesthesia</i> Induction – opiate: fentanyl $50 \mu\text{g}\cdot\text{kg}^{-1}$ – relaxant: non-depolarizing Maintenance – O ₂ – Opiate: fentanyl $20\text{--}30 \mu\text{g}\cdot\text{Kg}^{-1}\cdot\text{hr}^{-1}$ – Volatile: incremental enflurane/halothane – Ventilation: controlled normocapnia Nitroglycerin – Myocardial ischaemia – Hypertension $> 20\%$ baseline arterial pressure
4	<i>Intravenous fluid management</i> Crystalloid infusion $\pm$ colloid – PCWP $10\text{--}15$ mmHg – urine output >60 ml/Hr Blood – Loss $> 15\%$ estimated blood volume Mannitol – Urine output $< 60 \text{ ml}\cdot\text{hr}^{-1}$ + PCWP > 15 mmHg
5	<i>Postoperative management</i> Mechanical ventilation – Cardiac and respiratory homeostasis Regional anaesthesia – Postoperative analgesia

cardiogram, chest roentgenograms, resting and exercise radionuclide ventriculography and echocardiography, dipyridamole-thallium scanning are all designed to assess the functional status of the myocardium and to detect the presence of significant coronary artery disease. Patients with no abnormalities on physical examination, routine evaluation and no redistribution on dipyridamole-thallium scanning should proceed to surgery with the expectation of very low perioperative cardiac risk. Patients with evidence of coronary artery disease and significant redistribution on dipyridamole-thallium scan should undergo coronary angiography and possible myocardial revascularization before definitive aortic vascular surgery. For high cardiac risk patients with no bypassable lesions presenting for abdominal aortic aneurysm resection a conservative policy of serial three monthly ultrasound or CT assessment may be adopted, with selective resection for rapid aneurysm expansion or symptom development. A variety of extra-anatomical and angioplastic techniques is available for similar high cardiac risk patients with aortoiliac occlusive disease.

The haemodynamic consequences of aortic cross-clamping, especially in aneurysm patients, include a significant reduction in stroke volume, cardiac index, and myocardial oxygen consumption with an increased systemic vascular resistance. Patients with coronary artery disease may respond to aortic cross-clamping by increasing pulmonary capillary wedge pressure and by demonstrating ECG evidence of myocardial ischaemia. Pulmonary artery catheterization is especially indicated in patients with a history of previous myocardial infarction, angina or signs of cardiac failure and in patients with evidence of diminished ejection fraction, abnormal ventricular wall motion or myocardial redistribution on preoperative scanning. The more widespread application of intraoperative transoesophageal two-dimensional echocardiography and radionuclide cardiography monitoring techniques into anaesthetic practice will enable measurement of left ventricular dimensions, myocardial performance and wall motion. Suggested guidelines for anaesthetic management are presented in Table VI.

A combined opiate-oxygen-volatile anaesthetic agent technique will best ensure a hypodynamic circulation with preservation of myocardial oxygenation. Nitrous oxide and isoflurane should be avoided, if possible, in this patient population because of the risk of insidious global or regional myocardial ischaemia. Nitroglycerin infusion should be commenced if hypertension and signs of impaired myocardial contractility tissue oxygenation or myocardial ischaemia develop following aortic cross-clamp application. Preoperative replacement of extracellular fluid deficits; prompt and aggressive intraoperative hydration and blood loss replacement, guided by appropriate monitoring techniques; a stable cardiac output and optimal surgical techniques are the best prophylactic measures to ensure adequate renal, spinal cord and intestinal blood flow and function during the perioperative period. Adequate preoperative antihypertensive therapy remains the most important prophylaxis against postoperative hypertension, with all its attendant risks of myocardial ischaemia and infarction. The relative safety of anticoagulation following epidural catheter insertion has been recently established. The merits of a combined general and regional anaesthetic technique await further detailed scrutiny.

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References

- 180 Kaplan JA, King SB. The precordial electrocardiographic lead (V5) in patients with coronary-artery disease. *Anesthesiology* 1976; 45: 570-4.
- 181 Froelicher VF, Wolthius R, Keiser N *et al.* A comparison of the bipolar exercise electrocardiographic leads to lead V5. *Chest* 1976; 70: 611-6.
- 182 Kates RD, Zaidan JR, Kaplan JA. Esophageal lead for intraoperative electrocardiographic monitoring. *Anesth Analg* 1982; 61: 781-5.
- 183 Slogoff S, Keats AS, Arlund O. On the safety of radial artery cannulation. *Anesthesiology* 1983; 59: 42-7.
- 184 Mangano DT. Monitoring pulmonary arterial pressure in coronary-artery disease. *Anesthesiology* 1980; 53: 364-70.
- 185 Rice CL, Hobelman CF, John DA. *et al.* Central venous pressure or pulmonary capillary wedge pressure as the determinant of fluid replacement in aortic surgery. *Surgery* 1978; 84: 437-40.
- 186 Cunningham AJ, Boylan J, O'Toole DP. Does peri-aortic collateral vascularisation predict the haemodynamic changes following abdominal aortic cross clamp and release. *Can J Anaesth* 1988; 35: S70.
- 187 Nicholas GG, Martin DE, Osbakken MD. Cardiovascular monitoring during elective aortic surgery. *Arch Surg* 1983; 118: 1256-8.
- 188 Ansley DM, Whalley DG, Bent JM, Derbekyan V. The relationship between central venous pressure and pulmonary capillary wedge pressure during aortic surgery. *Can J Anaesth* 1987; 34: 594-600.
- 189 Cohen JL, Wender R, Maginot A *et al.* Hemodynamic monitoring of patients undergoing abdominal aortic surgery. *Am J Surg* 1983; 146: 174-7.
- 190 Kaplan JA, Wells PH. Early diagnosis of myocardial ischemia using the pulmonary artery catheter. *Anesth Analg* 1981; 60: 789-93.
- 191 Divertie MB, McMichan JC. Continuous monitoring of mixed venous oxygen saturation. *Chest* 1984; 85: 423-8.
- 192 Clements FM, de Bruijn NP. Perioperative evaluation of regional wall motion by transesophageal two-dimensional echocardiography. *Anesth Analg* 1987; 66: 249-61.
- 193 Jorgensen BC, Hoiland-Carlson PF, Marving J, Pedersen JF. Left ventricular performance monitored by radionuclide cardiography during induction of anesthesia. *Anesthesiology* 1985; 62: 278-86.
- 194 Freund PR. Transesophageal Doppler scanning versus thermodilution during general anesthesia. An initial comparison of cardiac output techniques. *Am J Surg* 1987; 153: 490-4.
- 195 Waters DD, Da Luz P, Wyatt HL *et al.* Early changes in regional and global left ventricular function induced by graded reductions in regional coronary perfusion. *Am J Cardiol* 1977; 39: 537-43.
- 196 Sethna DH, Moffitt EA. An appreciation of the coronary circulation. *Anesth Analg* 1986; 65: 294-305.
- 197 Lowenstein E, Hill RD, Rajagopalan B, Schneider RC. Winnie the pooh revisited, or, the more recent adventures of piglet. *Anesthesiology* 1982; 56: 81-3.
- 198 Falk JL, Rackow EC, Blumenberg R, Gelfand M, Fein IA. Hemodynamic and metabolic effects of abdominal aortic cross clamping. *Am J Surg* 1981; 142: 174-7.
- 199 Bush HL, Legerfo FW, Weisel RD *et al.* Assessment of myocardial performance and optimal volume loading during elective abdominal aortic aneurysm resection. *Arch Surg* 1977; 112: 1302-6.
- 200 Moffitt EA, Sethna DH, Gray RJ *et al.* Nitrous oxide added to halothane reduces coronary flow and myocardial oxygen consumption in patients with coronary disease. *Can Anaesth Soc J* 1983; 30: 5-9.
- 201 Moffitt EA, Scovil JE, Barker RA *et al.* The effects of nitrous oxide on myocardial metabolism and hemodynamics during fentanyl or enflurane anesthesia in patients with coronary disease. *Anesth Analg* 1984; 63: 1071-5.
- 202 Bovill JG. Opioid analgesics in anesthesia: with special reference to their use in cardiovascular anesthesia. *Anesthesiology* 61: 731-55, 1984.
- 203 Reier CE, George JM, Kilman JW. Cortisol and growth hormone response to surgical stress during morphine anesthesia. *Anesth Analg* 1973; 52: 1003-9.
- 204 Friesen RM, Thomson IR, Hudson RJ *et al.* Fentanyl oxygen anaesthesia for abdominal aortic surgery. *Can Anaesth Soc J* 1986; 33: 719-22.
- 205 Thomson IR, Putnins CL, Friesen RM. Hyperdynamic cardiovascular responses to anesthetic induction with high-dose fentanyl. *Anesth Analg* 1986; 65: 91-5.
- 206 Hudson RJ, Thomson IR, Cannon JE, Friesen RM, Meatherall RC. Pharmacokinetics of fentanyl in patients undergoing abdominal surgery. *Anesthesiology* 1986; 64: 334-6.
- 207 Moffitt EA, Sethna DH. The coronary circulation and myocardial oxygenation in coronary artery disease. Effects of anesthesia. *Anesth Analg* 1986; 65: 395-410.
- 208 Moffitt EA, McIntyre AJ, Glenn JJ *et al.* Myocardial metabolism and haemodynamic responses with fentanyl-halothane anaesthesia for coronary patients. *Can Anaesth Soc J* 1985; 32: S86.
- 209 Moffitt EA, McIntyre AJ, Barker RA *et al.* Myocardial metabolism and hemodynamic responses with fentanyl-enflurane anesthesia for coronary artery surgery. *Anesth Analg* 1986; 65: 46-52.
- 210 Wade JG, Stevens WC. Isoflurane: anesthetic for the eighties. *Anesth Analg* 1981; 60: 666-82.

- 211 Housmans PR, Murat I. Comparative effects of halothane, enflurane and isoflurane at equipotent anesthetic concentrations on isolated ventricular myocardium of the ferret. 1. Contractility. *Anesthesiology* 1988; 69: 451-63.
- 212 Tarnov J, Marksches-Hornung A, Schulte-Sasse W. Isoflurane improves the tolerance to pacing-induced myocardial ischemia. *Anesthesiology* 1986; 64: 147-56.
- 213 Gelman S, Fowler KC, Smith LR. Regional blood flow during isoflurane and halothane anesthesia. *Anesth Analg* 1984; 63: 557-65.
- 214 Moffitt EA, Barker RA, Glenn JJ *et al.* Myocardial metabolism and hemodynamic responses with isoflurane anesthesia for coronary artery surgery. *Anesth Analg* 1986; 65: 53-64.
- 215 Benefiel DJ, Roizen MF, Lambe GH *et al.* Morbidity after aortic surgery with sufentanil versus isoflurane anesthesia. *Anesthesiology* 1986; 65: A516.
- 216 Buffington CW, Romson JL, Levine A *et al.* Isoflurane induces coronary steal in canine model of chronic coronary occlusion. *Anesthesiology* 1987; 66: 280-92.
- 217 Becker LC. Is isoflurane dangerous for the patient with coronary artery disease? *Anesthesiology* 1987; 66: 259-61.
- 218 Merin RG. Is isoflurane dangerous for the patient with coronary artery disease? Another view. *Anesthesiology* 1987; 67: 284-5.
- 219 Reiz S, Balfors E, Sorensen MB, Ariola S, Friedman A, Truedsson H. Isoflurane - a powerful coronary vasodilator in patients with coronary artery disease. *Anesthesiology* 1983; 59: 91-7.
- 220 Reiz S, Balfors E, Gustavsson B *et al.* Effects of halothane on coronary haemodynamics and myocardial metabolism in patients with ischemic heart disease and heart failure. *Acta Anaesthesiol Scand* 1982; 26: 133-8.
- 221 Waller JL, Zaidan JR, Kaplan JA, Bauman DI. Hemodynamic responses to preoperative vascular cannulation in patients with coronary artery disease. *Anesthesiology* 1982; 56: 219-21.
- 222 Peterson A, Brant D, Kirsh MM. Nitroglycerin infusion during infrarenal aortic cross-clamping in dogs: an experimental study. *Surgery* 1978; 84: 216-23.
- 223 Hummal AW, Raess DH, Gewertz BL *et al.* Effect of nitroglycerin and aortic occlusion on myocardial blood flow. *Surgery* 1982; 91: 159-65.
- 224 Zaidan JR, Guffin AV, Perdue G, Smith R, McNeill DC. Hemodynamics of intravenous nitroglycerin during aortic cross clamping. *Arch Surg* 1982; 117: 1285-8.
- 225 Gelman S, Patel K, Bishop SP, Fowler KL, Smith LR. Renal and splanchnic circulation during infrarenal aortic cross clamping. *Arch Surg* 1984; 119: 1394-9.
- 226 Utsunomiya T, Kravsz MM, Shepro D, Hechtman HB. Induction of myocardial damage with nitroprusside. *J Surg Research* 1981; 31: 195-200.
- 227 Fremes SE, Weisel RD, Baird RJ *et al.* The effects of postoperative hypertension and its treatment. *J Thorac Cardiovasc Surg* 1983; 86: 47-52.
- 228 Gelman S, McDowell H, Varner PD *et al.* The reason for cardiac output reduction after aortic cross-clamping. *Am J Surg* 1988; 155: 578-86.
- 229 Sethna DH, Gray RJ, Moffitt EA *et al.* Dobutamine and cardiac oxygen balance in patients following myocardial revascularization. *Anesth Analg* 1982; 61: 917-20.
- 230 Giesecke AH, Egbert LD. Perioperative fluid therapy - crystalloids. *Anesthesia*. Miller RD (Ed.). Churchill Livingstone 1986; Vol 2 pp. 1313-28.
- 231 Schwartz LB, Bissell MG, Murphy M, Gewertz BL. Renal effects of Dopamine in vascular surgical patients. *J Vasc Surg* 1988; 8: 367-74.
- 232 Shires GT, Peitzman AB, Albert SA *et al.* Response of extravascular lung water to intraoperative fluids. *Ann Surg* 1983; 197: 515-9.
- 233 Virgilio RW, Rice CL, Smith DE *et al.* Crystalloid vs colloid resuscitation, is one better? *Surgery* 1979; 85: 125-8.
- 234 Skillman JJ, Restall S, Salzman EW. Randomized trial of albumin vs electrolyte solutions during abdominal aortic operations. *Surgery* 1975; 78: 291-302.
- 235 Boutros AR, Ruess R, Olson L, Hoyt JL, Baker WH. Comparison of hemodynamic, pulmonary and renal effects of use of three types of fluid after major surgical procedures on the abdominal aorta. *Crit Care Med* 1979; 7: 9-13.
- 236 Schwartz LB, Bissell MG, Murphy M, Gewertz BL. Renal effects of dopamine in vascular surgical patients. *J Vasc Surg* 1988; 8: 367-74.
- 237 Fisher DF, Yawn DH, Crawford ES. Preoperative disseminated intravascular coagulation associated with aortic aneurysms. *Arch Surg* 1983; 118: 1252-5.
- 238 Cutler BS. Avoidance of homologous transfusion in aortic operations: the role of autotransfusion, hemodilution and surgical technique. *Surgery* 1984; 95: 717-22.
- 239 Popovsky MA, Devine PA, Taswell HF. Intraoperative autologous transfusion. *Mayo Clin Proc* 1986; 60: 125-34.
- 240 Brewster DC, Ambrosino JJ, Darling RC *et al.* Intraoperative autotransfusion in major vascular surgery. *Am J Surg* 1979; 137: 507-12.
- 241 Hallett JW, Popovsky M, Ilstrup D. Minimizing blood transfusion during abdominal aortic surgery: recent advances in autotransfusion. *J Vasc Surg* 1987; 5: 601-6.
- 242 Duncan SE, Edwards WH, Dale WA. Caution regarding autotransfusion. *Surgery* 1974; 76: 1024-30.

- 243 O'Hara PJ, Hertzner NR, Santill PH, Beven EG. Intraoperative autotransfusion during abdominal aortic reconstruction. *Am J Surg* 1983; 145: 215–20.
- 244 Weisel RD, Denis RC, Manny J, Mannick JA, Valeri RC, Hechtman HB. Adverse effects of transfusion therapy during abdominal aortic aneurysmectomy. *Surgery* 1978; 83: 682–90.
- 245 Cunningham FO, Egan JM, Inahara T. Continuous epidural anesthesia in abdominal vascular surgery. *Am J Surg* 1980; 139: 624–7.
- 246 Cousins MJ, Wright CJ. Graft, muscle, skin blood flow after epidural block in vascular surgery procedures. *Surg Gynecol Obstet* 1971; 133: 59–65.
- 247 Reiz S, Nath EP, Friedman A, Backlund U, Olsson B, Rais O. Effects of thoracic epidural block and the beta 1 adrenoreceptor agonist prenalterol on the cardiovascular response to infrarenal aortic cross clamping in man. *Acta Anaesth Scand* 1979; 23: 395–403.
- 248 Lunn JK, Dannemiller FJ, Stanley TH. Cardiovascular responses to clamping of the aorta during epidural and general anesthesia. *Anesth Analg* 1979; 58: 372–6.
- 249 Klassen GA, Bramwell RS, Bromage PR, Zborowska-Sluis DT. Effect of acute sympathectomy by epidural anesthesia on the canine coronary circulation. *Anesthesiology* 1980; 52: 8–15.
- 250 Yeager MP, Glass DD, Neff RK, Brinck-Johnsen T. Epidural anesthesia and analgesia in high-risk surgical patients. *Anesthesiology* 1987; 66: 729–36.
- 251 Helperin SW, Cohen DD. Hematoma following epidural anesthesia: report of a case. *Anesthesiology* 1971; 35: 641–4.
- 252 Janis KM. Epidural hematoma following postoperative epidural analgesia: a case report. *Anesthesiology* 1971; 35: 689–92.
- 253 Rao TKL, El-Etr AA. Anticoagulation following placement of epidural and subarachnoid catheters: an evaluation of neurologic sequelae. *Anesthesiology* 1981; 55: 618–20.
- 254 Odom JA, Sih IL. Epidural analgesia and anticoagulant therapy experience with one thousand cases of continuous epidurals. *Anesthesia* 1983; 38: 254–9.
- 255 Bromage PB. Epidural anesthesia. Philadelphia, WB Saunders, 1978, p. 240.
- 256 Bunt TJ, Manczuk M, Varley K. Continuous epidural anesthesia for aortic surgery: thoughts on peer review and safety. *Surgery* 1987; 101: 706–14.
- 257 McPeck B. Inference, generalizability, and a major change in anesthetic practice. *Anesthesiology* 1987; 66: 723–4.
- 258 Smith PK, Fuchs JCA, Sabiston DC. Surgical management of aortic abdominal aneurysms in patients with severe pulmonary insufficiency. *Surg Gynecol Obstet* 1980; 151: 407–11.
- 259 Rodriguez JL, Weissman C, Damask MC, Askanazi J, Hyman AL, Kinney JM. Morphine and postoperative rewarming in critically ill patients. *Circulation* 1983; 68: 1239–46.
- 260 Sharp WV, Donovan DL. Retroperitoneal approach to the abdominal aorta: revisited. *J Cardiovasc Surg* 1987; 28: 270–3.
- 261 Sicard GA, Freeman MB, Vander Woude JC, Anderson CB. Comparison between the transabdominal and retroperitoneal approach for reconstruction of the infrarenal abdominal aorta. *J Vasc Surg* 1987; 5: 19–27.
- 262 Peck JJ, Mc Reynolds DG, Baker DH, Eastman AB. Extraperitoneal approach for aortoiliac reconstruction of the abdominal aorta. *Am J Surg* 1987; 151: 620–3.
- 263 Kaneko Y, Ikeda J, Takeda T, Ueda H. Renin release during acute reduction in arterial pressure in normotensive subjects and patients with renovascular hypertension. *J Clin Invest* 1967; 46: 705–16.
- 264 Haber E. Renin and vascular homeostasis during anesthesia. *Anesthesiology* 1979; 50: 83–4.
- 265 Berkowitz HD, Shetty S. Renin release and renal cortical ischemia following aortic cross clamping. *Arch Surg* 1974; 109: 612–7.
- 266 Gal TJ, Cooperman LH, Berkowitz HD. Plasma renin activity in patients undergoing surgery of the abdominal aorta. *Ann Surg* 1974; 179: 65–9.
- 267 Grindlinger GA, Vegas AM, Williams GH, Mannick JA, Hechtman HB. Independence of renin production and hypertension in abdominal aortic aneurysmectomy. *Am J Surg* 1981; 141: 472–7.
- 268 Grant RP, Jenkins LC. Modification by preoperative beta-blockade of the renin response to infrarenal aortic cross clamping. *Can Anaesth Soc J* 1983; 30: 480–6.
- 269 Feeley TW. The recovery room. In Miller RD (Ed.) *Anesthesia*. New York: Churchill Livingstone 1986, pp. 1921–45.
- 270 Cope DH, Crawford MC. Labetalol in controlled hypotension. *Br J Anaesth* 1979; 51: 359–64.
- 271 Flacke JW, Bloor BC, Flacke WE, Wong D et al. Reduced narcotic requirement by clonidine with improved hemodynamic and adrenergic stability in patients undergoing coronary bypass surgery. *Anesthesiology* 1987; 67: 11–9.