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Intraoperative Anesthetic Management for Vascular and Endovascular Abdominal Aortic Surgery

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1. Introduction

Management of patients undergoing vascular surgery concerns elderly patient, high incidence of coexisting disease as coronary artery disease, hypertension, and diabetes mellitus, acute changes in arterial pressure or metabolic stress associated with arterial ischemia or arterial cross-clamping. As a result, the ischemic insults to vital organs (heart, kidneys, and spinal cord) cause more frequent perioperative morbidity and mortality than other surgical procedures. Cardiovascular instability is more common and marked in patients with cardiovascular disease or those receiving vasoactive medications and undergoing cardiovascular surgical procedures.

The care must focus on preservation of vital organ, specially on the heart, which is the most important cause of morbidity after vascular surgery.

Major vascular surgery is particularly challenging to the anaesthesiologist because these are high-risk operations in a patient population with a high prevalence of either overt or occult coronary artery disease, which is the leading cause of perioperative and long-term mortality after vascular surgery. The last decade the multidisciplinary field of endovascular surgery has provided less invasive approaches or alternatives to conventional vascular reconstruction. These less invasive procedures, initially offered to patients traditionally considered unfit for open surgery, are being widely applied to the larger cohort of patients undergoing vascular surgery.

2. Preoperative improvement

Perioperative risk is multifactorial and depends on the interaction of anaesthesia, surgery and patient-specific factors. The fundamental purposes of preoperative evaluation are firstly, the identification of those patients for whom the perioperative period may constitute an increased risk of morbidity and mortality, aside from the risks associated with the underlying disease through the medical history information and, secondly, to design perioperative strategies for optimizing the patient's condition in case of presence of risk factors and reducing additional perioperative risks. Age per se, however, seems to be

responsible for only a small increase in the risk of complications; greater risks are associated with urgency and significant cardiac, pulmonary, and renal disease.

An interesting question of who should – at a certain point in the pre-operative trajectory – in close cooperation with the patient - decide whether or not the surgical intervention should be performed. The decision is not only a surgical one and should be based on knowledge of risk but also on the close knowledge of the individual patient, the planned procedure and also of alternative procedures. The increasing burden that expensive technical investigations put on social health care systems will oblige us to consider this aspect of the preoperative management of the patients in the near future.

Cardiac complications are a major cause of perioperative morbidity and mortality. Perioperative cardiac complications can occur in patients with documented or asymptomatic ischaemic heart disease, ventricular dysfunction and valvular heart disease. It has been estimated that in non-cardiac surgery major perioperative cardiac events may occur in up to 4% of cardiac patients, and 1.4% of an unselected patient population. In particular, vascular surgery patients are at increased risk with reported mortality rates of 1.5-2% for endovascular and 3-4% for open procedures. Mortality is mainly caused by perioperative myocardial infarction (accounting for 10-40% of postoperative deaths), in addition non-fatal perioperative myocardial infarction is associated with an increased risk of late mortality. Because of the systemic nature of atherosclerotic disease, vascular patients frequently have arterial disease affecting multiple vascular territories. It is not clear whether any specific category of vascular disease is associated with a greater likelihood of coexisting CAD. Perioperative cardiac complications are either caused by myocardial ischemia resulting from an increase in myocardial oxygen demand (tachycardia, hypertension, pain) or decreased supply (hypotension, vasospasm, tachycardia, hypoxia, anaemia) or, by acute coronary plaque rupture caused by factors that increase intra-coronary wall stress and the presence of a hypercoagulable state, leukocyte activation, and activation of the inflammatory response may contribute.

In 2009, the European Society of Cardiology published guidelines for pre-operative cardiac risk assessment and perioperative cardiac management in non-cardiac surgery, which were endorsed by the European Society of Anaesthesiology (www.escardio.org/guidelines). It shows the present evidence (and lack of evidence) in this field that is so important for the specialty. The European guidelines should not overrule the national ones but should be seen as a help to create harmonization of practice. Not all of these can be covered by recommendations. In addition, evidence on many issues is scarce and of low quality. Therefore, where possible, recommendations will be provided based on the best available evidence and when this is not possible, the recent available evidence will be summarized.

As surgical techniques become increasingly complex, the physical fitness required of the patients as well as the surgical impact on perioperative risk increases. Depending on duration of procedure, estimated blood-loss, estimated fluid shifts and anatomical region, the risk of surgery may vary tremendously. Surgical risk for cardiac events has been described by a 3 part classification that distinguishes between low, intermediate and high risk procedures according to the AHA/ACC-guideline and the guideline of the European Society of Cardiology (ESC). Therefore, in order to stratify overall perioperative risk, it is essential to consider the nature and duration of a surgical procedure. The cardiac risk can also influence the type of operation and guide the choice to less invasive interventions, such as peripheral arterial angioplasty instead of infra-inguinal bypass, or extraanatomic reconstruction instead of an aortic procedure, even when these may yield less favourable results in the long term.

A key component in the preoperative assessment is the evaluation of the presence of active or unstable cardiac conditions (table 1), the surgical risk factors (table 2), the functional capacity of the patient (< or > 4 METs), and the presence of cardiac risk factors (table 3). Decision for further testing and possible treatment should be performed in close cooperation with the cardiologist.

2.1 ESC Guidelines

The ESC guidelines propose a step-wise approach for perioperative cardiac assessment and management of cardiac patients scheduled for non-cardiac surgery.

1. The first step determines the urgency of the operation. The necessity for immediate surgery is such that no time is left for further cardiac assessment and/or treatment. Adequate measures for perioperative surveillance and treatment should be taken. Further risk stratification and risk factor management will be planned during the postoperative period.
2. The second step if there is no need for emergency surgery is to screen the patients for the presence of active cardiac conditions (Table 1). If one of these conditions is present, they should be evaluated and treated. For all these conditions, the potential benefits of delaying surgery to optimize the effects of treatment must be weighed against the risk of delaying the surgical procedure. With respect to a previous recent myocardial infarction, it is recommended to wait 4-6 weeks before performing elective surgery, even if there are no adequate clinical trials on the subject.
3. The third step if no active cardiac conditions are present is to assess the risk of surgery (Table 2). Many surgical procedures are associated with a low risk of perioperative complications even in high-risk patients. In such cases it is recommended to proceed with planned surgery.
4. The fourth step in the case of intermediate or high risk surgery evaluates whether the patient can sustain a functional capacity equal or greater than 4 metabolic equivalents (METs) without symptoms. If so, the recommendation is to proceed with surgery. In patients with coronary artery disease or risk factors, statin therapy and a titrated low-dose beta-blocker regimen can be initiated prior to surgery.
5. The fifth step in patients with a poor functional capacity or it is unknown consider the risk of the surgical procedure. When patients are scheduled for intermediate risk surgery statin therapy and a titrated low-dose beta-blocker regimen appears appropriate prior to surgery. In patients with systolic left ventricular (LV) dysfunction (ejection fraction <40%) ACE-inhibitors are recommended prior to surgery.
6. The sixth step in patients undergoing high risk surgery clinical risk factors (Table 3) are noted. In patients with up to two clinical risk factors, statin therapy and a titrated low-dose b-blocker regimen are recommended prior to surgery. In patients with systolic LV dysfunction ACE inhibitors (or ARBs in patients intolerant of ACE inhibitors) are recommended before surgery.
7. The step seventh consider non-invasive testing in patients with ≥ 3 clinical risk factors (Table 3). Patients without stress-induced ischemia, or mild to moderate ischemia suggestive of one- or two-vessel disease, can proceed with the planned surgical procedure.

It is recommended that statin therapy and a titrated low dose b-blocker regimen be initiated. In patients with extensive stress-induced ischemia, as assessed by non-invasive testing, an individualized perioperative management is recommended considering the potential benefit of the proposed surgical procedure compared with the predicted adverse outcome, and the

effect of medical therapy and/or coronary revascularization not only for immediate postoperative outcome, but also for long-term follow-up. In patients referred for percutaneous coronary artery intervention, the initiation and duration of antiplatelet therapy will interfere with the planned surgical procedure.

In patients referred for angioplasty, non-cardiac surgery can be performed within 2 weeks after intervention with continuation of aspirin treatment.

In patients with bare metal stent placement, non-cardiac surgery can be performed after 6 weeks to 3 months following intervention. Dual antiplatelet therapy should be continued for at least 6 weeks, preferably for up to 3 months. After this period, at least aspirin therapy should be continued.

In patients with recent Drug-eluting stent placement, non-cardiac surgery can be performed after 12 months following intervention, before which time dual antiplatelet therapy is recommended. After this period, at least aspirin therapy should be continued.

If applicable, discuss the continuation of chronic aspirin therapy. Discontinuation of aspirin therapy should be considered only in those patients in which haemostasis is difficult to control during surgery (intracranial neurosurgery, medular surgery, posterior chamber of the eye) that not concerns vascular surgery.

2.2 Recommendations

1. If active cardiac disease is suspected in a patient scheduled, the patient should be referred to a cardiologist for assessment and possible treatment (grade of recommendation: **D**).
2. In patients currently under b-blocking or statin therapy:
   a. should not be stopped preoperatively (grade of recommendation: **A**);
   b. is recommended to high risk patients;
   c. low and intermediate risk patients should not routinely be subjected to beta-blockade.

   An important point is that especially when heart failure has not been excluded, the beta-blockade should be started slowly titrating the dose, which implies that the treatment should ideally be started between 30 days and at least 1 week before surgery. Therefore these should be interpreted within the constraints of logistics and infrastructure that allow to see the patient sufficiently long in advance preoperatively.

   In general terms, patients should be maintained on their usual cardiovascular medications throughout the perioperative period. Antiplatelet therapy requires special consideration and must be individualized to each patient.
3. Guidelines on perioperative cardiovascular evaluation and care suggest that coronary intervention is rarely necessary to simply lower the risk of surgery unless such intervention is indicated irrespective of the preoperative context. Current evidence does not support the role of prophylactic coronary revascularization as a means to reduce perioperative or long-term morbidity after major vascular surgery.

   Caregivers will increasingly be reminded that additional testing of the patient is only relevant if they may lead to substantial alterations of perioperative management.
4. Non-invasive testing can also be considered prior to any surgical procedure for patient counselling, or change of perioperative management in relation to type of surgery and anaesthesia technique.

1. Unstable coronary syndromes 1.1 unstable or severe angina 1.2 recent myocardial infarction (within 30 days)
2. Decompensated heart failure
3. Significant arrhythmias 3.1 high-grade atrioventricular block 3.2 symptomatic ventricular arrhythmias 3.3 supraventricular arrhythmias with uncontrolled ventricular rate (> 100 bpm at rest) 3.4 symptomatic bradycardia 3.5 newly diagnosed ventricular tachycardia
4. Severe valvular disease 4.1 severe aortic stenosis (mean pressure gradient > 40 mm Hg, area < 1 cm2 or symptomatic) 4.2 symptomatic mitral stenosis

Table 1. Active cardiac conditions that necessitate further evaluation and treatment before non-cardiac surgery

A. High risk (cardiac risk > 5%) A.1 Aortic surgery A.2 Major vascular surgery A.3 Peripheral vascular surgery
B. Intermediate risk (cardiac risk 1 – 5%) B.1 Abdominal B.2 Carotid B.3 Peripheral arterial angioplasty B.4 Endovascular aneurysm repair B.5 Head and neck surgery B.6 Major neurologic/orthopedic B.7 Pulmonary B.8 Major urologic
C. Low risk (cardiac risk < 1%) C.1 Breast C.2 Dental C.3 Endocrine C.4 Eye C.5 Gynecology C.6 Reconstructive C.7 Minor orthopedic C.8 Minor urologic

Table 2. The risk of cardiac death and non-fatal myocardial infarction for non-cardiac operations.

1. History of ischemic myocardial disease or angina pectoris
2. Current stable or history of heart failure
3. History of cerebrovascular disease / transient ischemic attack
4. Diabetes requiring insulin therapy
5. Renal failure (serum creatinine > 2 mg / dL or a creatinine clearance <60 ml/min)

Table 3. Clinical risk factors

3. Intraoperative management of Abdominal Aortic Aneurysm

An important part of perioperative patient care resides in the possible impact of intra-operative patient care on the outcome. However, strong evidence remains to be gathered of the influence of intra-operative anaesthetic management on short and long-term postoperative outcome. For instance, while epidural analgesia is generally considered a valuable tool in the perioperative pain treatment it remains to be proven that the treatment positively affects perioperative morbidity and mortality. But, the increasing preoperative use of antiplatelet and anti-coagulant drugs will interfere with the possibilities to freely apply neuro-axial techniques. It is mandatory to realize that the choice of anaesthetic techniques will influence the potential for starting and / or continuing anti-coagulant therapy. Thus, the anaesthesiologist is required to consider his/her work in the context of the total perioperative course. Further, the important question of fluid management has not resulted in specific recommendations due to the combination of conflicting and lacking evidence.

The importance (lack of) of perioperative respiratory interventions is not evident. Several studies have shown that postoperative pulmonary complications are associated with worse outcome. However, there has been conflicting evidence to which degree preventive measures have been effective, a fact which is mirrored in the Guidelines. There are studies reporting effectiveness of early application of non invasive continuous positive airway pressure in presence of acute lung injury.

Infrarenal abdominal aortic aneurysms (AAAs) are more common (70%). The abdominal aorta is aneurismal when its diameter is greater than 3.0 cm. The disorder is more common in men than in women. Sakalihasan reports that the prevalence of AAAs is rising and is between 1.3% and 8.9% in men and between 1.0% and 2.2% in women over the age of 65.

Sakalihasan et alt. also refer that most deaths due to ruptured AAAs are potentially preventable since elective repair can be performed with an perioperative mortality rates in the 2% to 4% range even in contemporary experience due to cardiac complications. Population-based series employing state wide or national databases indicate higher mortality, in the 4% to 8% range. By contrast, with the progress in elective repair mortality, no improvement in operative mortality of ruptured aneurysms has been reported during the past decades remaining as high as 30–70%. Mortality depends on the haemodynamic status of the patient at the time of surgery. Overall mortality from ruptured abdominal aortic aneurysms is about 80% associated to hypovolemic shock with an operative survival of 50%. Mortalities occurring at the scene of rupture, during transfer, shortly after admission to the emergency department, and during surgery are combined, then only 18% of patients with ruptured aortic aneurysms survive.

Most patients with AAAs are asymptomatic and are discovered incidentally when other examinations are performed. Patients presenting with back, abdominal or groin pain in the presence of a pulsatile mass require urgent evaluation to exclude a rupture or dissection. The main risk factors for developing AAAs are advancing age, family history, smoking and hypertension.

3.1 Surgical indication

Current guidelines of American Association for Vascular Surgery for the treatment of abdominal aortic aneurysms are to offer operative intervention when the aneurysm exceeds 5.5 cm. The risk for rupture of a 5.5-cm aneurysm (per year) is equal to or greater than the risk for perioperative mortality. The risk of rupture of a 6 cm or greater exceeds 20%. The bigger the aneurysm is, the more tendency to the rupture is.

The incidence of rupture in small aneurysms less than 5 cm is 2%. There is no survival benefit from early surgical intervention. Patients with small aneurysms should undergo regular ultrasound scanning to monitor the aneurysm size.

Open repair remains the gold standard treatment. Endovascular aneurysm repair (EVAR) is emerging as a minimally invasive treatment for some AAAs that are anatomically suitable.

3.2 Preoperative evaluation

We must search risk factors and co-morbidities. Patients presenting for abdominal vascular surgery have a high incidence of co-morbidities:

- Coronary artery disease often with impaired ventricular function is observed in the 80% of patients scheduled for vascular surgery
- Cerebrovascular disease
- Hypertension
- Pulmonary disease (often related to smoking)
- Renal impairment
- Diabetes mellitus
- Obesity
- Dislipemy

Careful preoperative assessment is required by the surgeon and anaesthetist to identify high risk patients and to optimise medical management according the guideline in previous section:

- Lifestyle advice should be given.
- Cessation of smoking and structured exercise programmes may improve cardio-respiratory fitness.
- Patients should receive antiplatelet medication to protect against thromboembolic complications.
- Statins should be prescribed due to plaque stabilisation.

Patients with inducible ischemia on pharmacological stress testing have improved outcomes if prescribed b blockers. The success of modern medical treatments in coronary artery

disease offers benefits over coronary revascularisation prior to non cardiac surgery. Preoperative coronary artery bypass surgery should only be performed if indicated on prognostic grounds (severe left main stem disease or severe triple vessel disease with impaired left ventricular function). This should be performed 1-2 months prior to surgery.

- Patients should receive all their regular medication on the day of surgery.
- Correct premedication to decrease anxiety
- Antibiotic prophylaxis
- Intestinal cleanse to ensure vascular prosthesis viability.

3.3 Anaesthetic management of open surgery repair (OSR)

3.3.1 Aims

Haemodynamic stability, according to the patient with CAD preventing tachyarritmies, high and low blood pressure, sympathetic stimulation, myocardial depression or coronary steal.

Normothermia. Perioperative hypothermia is associated with myocardial ischemia and dysrhythmias. It contributes to a coagulopathy and increases wound infections. Shivering can increase oxygen consumption up to six fold placing excessive demands on the cardiovascular system. Forced air warming devices, fluid warmers and increasing ambient theatre temperatures are used to minimise heat loss. The legs should not be actively warmed during cross clamping.

Pain free patient on completion of surgery.

Balanced general anaesthetic technique is usually used (high dose opioid, oxygen, air, low dose volatile agent) with a thoracic-lumbar epidural oriented to haemodinamic management with deep neuromuscular relaxing. Epidurals ameliorate the stress response to surgery, reducing cardiovascular demands and provide high quality postoperative analgesia, facilitating early extubation and reducing the incidence of pulmonary complications. But, there is no evidence that epidurals reduce mortality. It is safe to insert epidurals on patients taking aspirin and less clear with clopidogrel. Patients should be monitored closely for the symptoms and signs of spinal haematoma (back pain, bladder dysfunction, leg weakness)

3.3.2 Monitoring

Regular observations of the patients and the information provided by monitoring devices must be undertaken and documented. Electronic record keeping is recommended. Monitoring itself cannot prevent adverse reactions during anaesthesia, but basic monitoring reduces the risk of incidents by providing an early warning if the patient's condition worsens. However, human error is inevitable and a number of studies of critical incidents and mortality associated with anaesthesia have shown that adverse events are often attributable to this type of error.

Basic physiologic monitoring in anaesthesia includes continuous display of the electrocardiogram (ECG), intermittent non-invasive measurement of blood pressure (NIBP),

pulse oximetry and capnography. Sensitivity increases when a five-lead ECG is used and leads II and V5 are continuously monitored and routine use of a five-lead ECG is strongly recommended in patients with known or suspected coronary artery disease. In patients with one or more cardiac risk factors a pre-operative baseline ECG is recommended to monitor changes during the perioperative period.

Monitoring of anaesthetic equipment comprises the use of an oxygen analyser, capnography, and a disconnection alarm. The use of a vapour analyser is essential if a volatile anaesthetic agent is used.

Then, basic physiologic monitoring with a 5 lead ECG will aid detection of ST segment changes.

In addition to standard monitoring, direct measurement of arterial and central venous pressure, temperature and urine output is mandatory.

Cardiac output during AAA surgery with pulmonary artery flotation catheters, transoesophageal echocardiography (TOE), oesophageal doppler monitoring or pulse contour analysis cardiac output monitoring (LiDCO™ and PiCCO ™) can be considered.

3.3.3 Heparinisation

A dose of 100units/kg of heparine prior to crossclamping has reduced thrombotic and embolic events in the prothesis. Heparin needs to be available five minutes before aortic crossclamping. We must check ACT (activated clotting time) baseline, 3 minutes after heparin given and every 30 minutes thereafter while cross-clamped. Additional heparin may be required in the presence of prolonged clamp times. Heparin can be reversed by protamine if bleeding. Protamine should be used with caution as it may lead to myocardial depression, anaphylaxis and pulmonary hypertension.

3.3.4 Fluid replacement - haemorrhage and blood product management

We have fluid negative balance due to, intestinal cleanse, fasting, osmotic diuresis by previous contrast, intestinal oedema and big lost due to evaporation. We must manage this balance according to aortic crossclamping and diuresis (1,25ml/kg/h).

Blood loss during AAA surgery is highly variable from 500ml to various litters. It is greater in suprarenal aneurysms. Significant bleeding can occur when opening the native aorta due to backbleeding from the lumbar arteries. Blood loss can also result from malpositioned clamps, from leaking anastomosis, excessive heparine or coagulopathy. Homologous blood transfusion can be minimised by intraoperative cell salvage (ICS). Since vascular patients have a high incidence of coronary disease the haematocrit should be kept greater than 27% (Hb > 9g/dl).

Massive haemorrhage more than 2000ml results in a dilutional coagulopathy requiring fresh frozen plasma, cryoprecipitate and platelet transfusions and calcium. Appropriate administration of clotting factors is best guided by near patient testing using a thromboelastograph if possible. Laboratory based coagulation tests which often lag behind the clinical picture. Appropriate goals are an International Normalised Ratio (INR) of less than 1.5, a platelet count of greater than 50 x 10^9/l and fibrinogen levels greater that 1g/dl.

3.3.5 Crossclamping

Aortic cross clamping is necessary in open AAA surgery. The increased vascular resistance results in arterial hypertension. Higher the crossclamping is, higher haemodinamic repercussion and higher hypoperfussion on vital organs. Blood pressure typically rises by 7-10%and until 50% if supraceliac aortic crosscalmping. Blood pressure decreases under the crossclamping about 80%. Aortic crossclamping also increase intracranial pressure.

A diseased coronary system may be unable to respond to increases in cardiac workload resulting in cardiac failure. This may be exacerbated by over fluid administration prior to cross-clamping. Studies show reductions of cardiac output of between 9-33% after infra-renal cross clamping.

We must minimize these haemodinamics effects with: 1.Vasodilators (e.g. urapidil, glyceryl trinitrate, sodium nitroprusside) that may exacerbate organ ischaemia by reducing perfusion pressure in the collateral circulation. 2. Deeper anaesthesia. 3. Small hypovolemia. Aortic crossclamping has compensatory changes as liberation of vasoactive substances due to intestinal ischemia, prostaglandins liberation due to aortic dilatation, hormonal liberation due to ventricular dilatation and vasoactive intestinal peptides and prostaglandins released by the mesenteric traction and evisceration.

Patients with severe aorto-occlusive disease often have a well developed collateral circulation and show minimal response to cross clamping.

3.3.6 Unclamping

Unclamping may result in a dramatic reduction in blood pressure. The causes for this are: a decrease in systemic resistance due to the removal of the cross clamp and the release of vasoactive cytokines and metabolites from ischaemic tissues; central hypovolemia due to sequestration of blood in the reperfused organs and the release of myocardial depressant factors.

The severity of hypotension is proportional to cross clamp time. It also depends on the level of clamp. We must prepare the patient: optimizing and ensuring adequate fluid resuscitation (fluids and blood); with a gradual release of the cross clamp; vasopressors may with the potential disadvantage of preferential vasoconstriction of the vasculature above the clamp; increasing $F_i O_2$; with lighter anaesthesia; reducing metabolic acidosis with HCO_3^- or $CaCl_2$. Reclamping may be required in resistant hypotension.

We must hyperventilate the patients due to metabolic acidosis and also because there are an increase of CO_2. During clamp the arterial CO_2 decreases secondary to deep anaesthesia, no venous return from the inferior part of the body and hypothermia.

3.3.7 Regional circulation

3.3.7.1 Renal system

The incidence of renal failure after AAA surgery is from 0.2% to 5.4% for infrarrenal crossclamping (up to 40% renal blood flow decrease) and 13% for suprarenal crossclamping (80% renal blood flow decrease). The level of aortic crossclamping is the more important risk factor. Other renal risk factors are: Pre existing renal disease; Prolonged clamp time;

Hypotension; Massive haemorrhage; Severity of renal artheriosclerosis; The type of Aortic reconstruction; Increasing age; Contrast nephropathy or Drugs (NSAID's ACEI, aminoglycosides)

Loop diuretics (e.g. furosemide), dopamine, mannitol, fenoldapam and N-acetylcysteine are proposed renal protective agents. There is no Level 1 evidence to support their use. The mainstay of renal preservation is by adequate fluid resuscitation, to diminish clam time, and, the avoidance of nephrotoxins (NSAID's, ACEI, aminoglycosides). Intraoperative diuresis can led fluid therapy but is a bad predictor of the renal perfusion and the postoperative renal function.

3.3.7.2 Digestive system

Bowel complications as intestinal ischemia increase the postoperative mortality in 25%. In the majority of surgeries the inferior mesenteric artery is removed causing ischemic colitis of descendent colon and sigma that can reache 6% if there are not collateral flow. The diagnosis is late due to a high incidence of paralytic ileus. The infrarenal clamp also decreases mesenteric flow due to a regulatory vasoconstriction.

Predisposing factors are: previous medical condition; renal insufficiency; high degree of artheriosclerosis, the level of clamp; time of clamp; hypoblood pressure.

During the clamp ischemia disturbs intestinal permeability and favours bacterial translocation.

3.3.7.3 Nervous system and medullary ischemia

The patient has increased risk of neurological complication if the aorta is clamped above the Major Anterior Segmental Medullary Artery (Artery of Adamkiewicz) which has variable origin: T5-8 15%, T9-12 60%, L1-2 25%.

Anterior spinal syndrome caused by anterior motoneurons ischemia consists of loss of motor (different degree of paraplegia) and pinprick sensation but preservation of vibration and proprioception. In men is frequent unnoticed erectile dysfunction

We can improve ischaemia with a small time of clam; fast surgery; increasing blood perfusion pressure; decreasing CSF; decreasing medullar metabolism by barbiturics or hypothermia.

3.3.8 Postoperative care

Patients require close monitoring after abdominal vascular surgery. Traditionally this has been provided in an Intensive Care Unit. However, careful patient selection coupled with improvements in anaesthetic and surgical techniques allow early extubation and transfer to a High Dependency Units (HDU). Early enteral nutrition is encouraged to maintain gut mucosal integrity and reduce bacterial translocation. Nasogastric tubes are not routinely required. Tight glycaemic control, temperature and analgesia are important. Appropriate antacid and thromboembolic prophylaxis must be prescribed.

3.4 Emergency AAA surgery

Ruptured AAA classically presents with back or abdominal pain, shock and an expanding pulsatile abdominal mass. If the patient is stable the diagnosis can be confirmed by CT

scanning. Shocked patients require immediate transfer to the operating theatre and laparotomy. Surgery may be futile in patients with severe pre-existing co-morbidity.

Emergent EVAR should be considered for treatment of a ruptured AAA, if anatomically feasible. Endovascular aneurysm repair (EVAR) is increasing in popularity as it avoids the need for a laparotomy in a group of patients who usually have significant co-morbidity. At present, there are no large, multi-centre, prospective, randomized data assessing the efficacy of EVAR in the treatment of ruptured AAA but many small series have demonstrated a trend towards decreased mortality compared with open repair.

3.4.1 Anaesthetic management

Insertion of two wide bore cannulas. Rapid fluid infusor. Cell salvage blood equipment. Aggressive preoperative fluid resuscitation is contraindicated as it will only serve to increase bleeding and dilute clotting factors.

- Baseline bloods (blood count, electrolytes, coagulation screen).
- Insertion of urinary catheter.
- Forced air warming device.
- Drugs and Fluids: 6 - 10 units of cross matched blood, FFP and platelets; Routine anaesthetic drugs, crystalloids and colloids; Inotropes and vasopressors.

The patient is draped and skin prepared prior to a rapid sequence induction. Loss of abdominal tone combined with the negative inotropic and vasodilatory effects of the anaesthetic agents may result in severe hypotension post induction. Skin incision is made as soon as the airway is secure. Aortic cross clamping is a life saving manoeuvre. For EVAR: Do not induce anaesthesia until aorta is occluded by the balloon, if possible.

After the aorta is cross clamped aggressive fluid resuscitation can be instituted with blood and colloid solutions. A dilutional coagulopathy should be anticipated and FFP and platelets ordered.

Heparinization is not required.

Once haemodynamic stability is obtained arterial and central venous catheters can be inserted, a nasogastric tube passed, and temperature monitoring commenced.

3.4.2 Postoperative care

All patients should be transferred to ICU postoperatively where supportive care includes optimization and maintenance of circulating volume. Re-warming will continue until normal body temperature is achieved and respiratory support is usually required for up to at least 24 h and frequently several days. Renal function, coagulation, haemoglobin, and acid-base balance are monitored closely. There is a high incidence of myocardial ischemia and renal failure. Renal replacement therapy is required in a significant proportion of patients and those with a coagulopathy may require continuing blood product transfusion. Other important issues include an anticipated prolonged ileus and analgesia. Prolonged stays are common due to multi-organ failure.

Patients are particularly prone to developing intra-abdominal hypertension (intra-abdominal pressure> 12 mmHg) and abdominal compartment syndrome (ACS, defined as

IAP > 20 mmHg). Factors which contribute to the development of ACS include anaemia, prolonged hypotension, cardiopulmonary resuscitation, hypothermia, severe acidosis (base deficit > 14 mEq) and aggressive fluid resuscitation (>4 l/h). The International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome recommends that decompression should be considered at the time of laparotomy in patients who demonstrate multiple risk factors for IAH/ACS (grade 1C) as patients with ruptured abdominal aneurysm. These patients may benefit from laparastoma or mesh closure of the abdominal wall with delayed secondary surgical closure after 2–3 days. No prospective randomized studies are available to validate the concept of the open abdomen protocol. However, retrospective data in the form of case and cohort studies do exist as Rasmussen et al. report. Performing a mesh closure initially in these patients reduces the incidence of multiorgan failure when compared with patients who require a second operation for ACS in the postoperative period. Monitoring of IAP should be considered in all patients and consideration given to parenteral nutrition if ileus is prolonged.

Predictors of survival to discharge include patient age, total blood loss and postoperative hypotension.

3.5 Endovascular aortic aneurysm repair (EVAR)

EVAR was developed as a less invasive alternative to open repair. Modular bifurcated stent grafts are placed via open femoral arteriotomy. This is a combined surgical and radiological procedure which may be performed in theatre or the angiography suite.

Physiological disturbances are reduced as there is no requirement for laparotomy or crossclamping of the aorta. The last guideline for the care of patients with abdominal aortic aneurysm of The Society for Vascular Surgery points the EVAR technique, as a minimally invasive technology that would be associated with lower inhospital and 30-day mortality rates as compared to OSR. Specifically, in-hospital mortality rates were 1.7% - 1.2% for EVAR and 6% - 4.6% for OSR. Procedural blood loss is less for EVAR (414 mL) when compared to OSR (1,329mL), with reductions in ICU (0.7 vs 1.6 d) and hospital (4.2 days vs 9.9 days) stays.

The society of Vascular Surgery also refers that major medical complications are lower after EVAR than OSR. A meta-analysis of observational studies conducted prior to 2002 demonstrated an incidence of systemic complications of 9% after EVAR, as compared with 22% after OSR, largely attributable to fewer cardiac and pulmonary events. It has been observed a lower incidence of cardiac complications in a statewide review of patients treated by EVAR in 2002 (3.3% vs 7.8%). In addition, the latter study noted a reduction in the incidence of pneumonia (9.3% vs 17.4%), acute renal failure (5.5% vs 10.9%), and need for dialysis (0.4% vs 0.5%) among those treated by EVAR.

3.5.1 Indications

A number of reports have documented that EVAR can be performed with low rates of perioperative mortality and morbidity in patients at high risk for open surgery repair (OSR). Additional research is needed to define objective criteria that identify patients who are unfit for OSR and whose anticipated life expectancy limits benefit from EVAR. At present EVAR should be reserved for fit elderly patients (age > 80) and those patients in whom previous abdominal surgery may make open access to the abdominal aorta difficult. The inclusion criteria also depends on the anatomy of the aneurism.

3.5.2 Anaesthetic management of EVAR

EVAR can be safely performed under general, epidural, or local anaesthesia. Lesser degrees of anaesthesia may be of benefit. Mortality differences have not been observed. Local anaesthesia was associated with shorter operative times, reduced ICU admission, shorter hospital stay, and fewer systemic complications. However, the anaesthetist should consider: the problems of anaesthesia in the angiography suite; the requirement for short periods of apnoea; prolonged bilateral femoral occlusion resulting in ischemic pain; the risk (1%) of conversion to an open procedure and the average surgical time of 3 hours with the necessity of an immobile patient.

3.5.3 Monitoring

Basic physiologic monitoring with a 5 lead ECG will aid to detect ST segment changes. In addition to standard monitoring, direct measurement of arterial, temperature and urinary catheterisation is required as the high contrast load may result in nephropathy.

Large bore venous access is necessary as rupture of the aorta or of an iliac artery are reported complications.

3.5.4 Heparinisation

Anticoagulation is recommended. Heparin can be reversed by protamine if bleeding. Protamine should be used with caution as it may lead to myocardial depression, anaphylaxis and pulmonary hypertension.

3.5.5 Fluid replacement - haemorrhage

We must consider fasting and osmotic diuresis by contrast specially in the postoperative cares that can led to a prerenal failure.

Blood loss during is less for EVAR (414 mL) when compared to OSR (1,329mL). We must be aware to the unnoticed blood lost under the drapes and sometime they are important.

Massive haemorrhage can arrive after a vascular rupture.

3.5.6 Radio-contrast injection

Radio-contrast injection can cause allergic reactions in patients with history of asthma, atopy, allergy or previous exposition to up than 20gr of contrast. We can do prophylaxis with dexchlorpheniramine, ranitidine and prednisone.

Contrast is nephrotoxic and can cause acute tubular necrosis. We must prevent it with adequate hydration and n-acetylcysteine 600mg 12h before and the day of surgery.

Radio-contrast can induce osmotic diuresis and polyuria with secondary hypovolemia.

3.5.7 Critical moments during surgery

During surgery we must pay attention to some events: Skin incision, prothesis introduction, device implantation where migration must be prevented by severe bradichardia and

hypotension and ballon insuflation to fix the device that results similar to crossclamping but much shorter in time.

3.5.8 Complications

Endoleak, or persistent blood flow in the aneurysm sac outside of the endograft, is the most frequent complication after EVAR and has been reported in nearly one in four patients at some time during followup. It is one of the most common abnormalities identified on late imaging and used to justify lifelong followup of these patients. Type I endoleak occurs in the absence or loss of complete sealing at the proximal (Type IA) or distal (Type IB) end of the stent graft. Type I endoleak is associated with significant pressure elevation in the sac and has been linked to a continued risk of rupture. It should be made to resolve Type I endoleaks noted at the time of EVAR before the patient leaves the intervention suite. On occasion, small persistent Type I endoleaks may be observed and if endovascular intervention has been unsuccessful, the only alternative is surgical conversion. Type II endoleaks are the most common form of endoleak and arise from retrograde filling of the sac by lumbar branches or the inferior mesenteric artery. For those detected at the time of EVAR, further treatment is not indicated, since spontaneous resolution is possible. Type III endoleaks arise from poorly seated modular connections or from disconnection and separation of components. All Type III endoleaks should be treated, typically with limb components, as they represent a lack of exclusion of the aneurysm with repressurization of the aneurysm sac. Type IV endoleaks represents self-limiting blood seepage through the graft material due to porosity and treatment is not required. Typically, this form of endoleak is only noted at the time of repair on post-implantation intra-operative angiography. An endoleak noted on follow-up imaging should not be considered a Type IV endoleak.

Vascular rupture

Technical difficulties for putting the endoguide with the endograf to the right position through the aorta artery.

Device migration

Contrast-induced nephropathy occurs infrequently after EVAR.

The incidence of local vascular or device related complications, as well as the 30-day re-intervention rate is greater after EVAR than OSR. Similar findings have been reported in several observational studies with local or vascular complications occurring in 9% to 16% of patients after EVAR. In the EVAR-1 and EVAR-2 trials, reintervention within 30 days of EVAR occurred in 9.8% and 18% of patients, respectively. Groin and wound complications are the most frequent event. Stents, endografts, or surgical repair may be required if severe vascular access injuries occur.

Distal embolization is now rare with lower profile introducer systems. Limb occlusion occurs more frequently in patients with aortoiliac occlusive disease, a small (<14 mm) distal aorta and tortuous vessels and when unsupported endografts are used.

Post-implantation syndrome, characterized by fever, malaise, back or abdominal pain after EVAR, may last up to 10 days, but appears to be a relatively rare phenomenon. It has been attributed to the release of cytokines after aneurysm sac thrombosis.

3.5.9 Postoperative care

Patients can be transferred to the ward after a brief period of observation in a HDU. Hospital stay is reduced to 24-48h. The Pain is minimum and patient can food almost immediately. We must watch the diuresis and the blood lost. There is a 65% absolute reduction in early (30 day) mortality compared to open repair. This early survival advantage must be balanced against the risk of endograft related complications, in particular endoleak which necessitates annual CT surveillance.

In summary, the recognized benefits of EVAR, including reduced morbidity, ICU and hospital length of stay, as well as observed lower perioperative mortality rates, especially among elderly patients, has led to widespread adoption of this technology. Nonetheless, it is recommended that elective EVAR is best performed at centers that have a documented in-hospital mortality of less than 3% and a primary conversion rate to OSR of less than 2% for elective repair.

Further research is needed to improve EVAR devices and related techniques to reduce complications and long-term follow-up; to identify whether EVAR outcomes vary with respect to endograft type or aneurysm features; and to define the relationship of hospital and physician volume to outcomes after EVAR.

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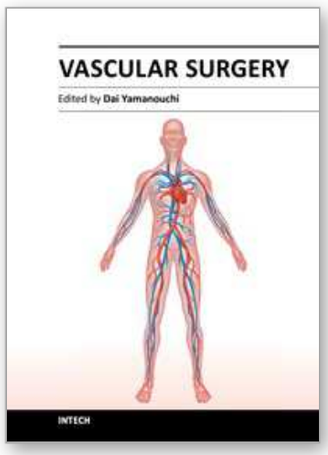
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This book aims to provide a brief overview of conventional open vascular surgery, endovascular surgery and pre- and post-operative management of vascular patients. The collections of contributions from outstanding vascular surgeons and scientists from around the world present detailed and precious information about the important topics of the current vascular surgery practice and research. I hope this book will be used worldwide by young vascular surgeons and medical students enhancing their knowledge and stimulating the advancement of this field.

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