Postoperative Care Following Major Vascular Surgery

Elrasheed S. Osman and Thomas F. Lindsay

Introduction

 Patients with vascular disease are among those at highest risk for postoperative complications. Postoperative management following major vascular surgery has seen a fundamental shift towards a more efficient use of postoperative care facilities. Minimally invasive surgical techniques such as endovascular repair of complicated aortic aneurysms and the use of local and/or regional blocks, together with improvements in perioperative anesthesia, have all led to fewer patients requiring routine admission to intensive care units (ICUs). Despite best practices and planning in the preoperative assessment and in the intraoperative hemodynamic optimization, vascular surgical patients remain among the highest at risk for postoperative myocardial infarction (MI), stroke, renal failure, and bleeding complications. Better preoperative evaluation, triage, and preparation provide appropriate environment for early identification and treatment of complications. This chapter concentrates on the common postoperative management strategies in vascular surgical patients and highlights procedure-specific perioperative risks.

Division of Vascular Surgery, Department of Surgery, Toronto General Hospital, Toronto, ON, Canada e-mail: [elrasheed.osman@mail.utoronto-ca;](mailto:elrasheed.osman@mail.utoronto-ca) elrasheedosman@gmail.com

Preoperative Triage

 The preoperative medical comorbidities and perioperative hemodynamic stability are the major factors that determine the most appropriate location for postoperative admission and monitoring.

With proper risk stratification, selective rather than obligatory use of intensive care and step-down units can provide safe and cost-effective care [1]. Age and comorbid conditions such as coronary artery disease, congestive heart failure, chronic obstructive pulmonary disease (COPD), and chronic renal failure are predictors of increased mortality in vascular surgical patients $[2]$. Moreover, poor preoperative nutritional status directly correlates with the increased incidence and severity of the systemic inflammatory response syndrome (SIRS) seen in some patients after major vascular surgery [3].

 Major open vascular surgery carries a 30-day mortality rate of 5 %—mostly secondary to cardiac events $[4]$. Perioperative markers for direct ICU admission include sustained hemodynamic instability (systolic blood pressure <90 mmHg), clinically significant cardiac ischemia, ventilatory failure, hypothermia (<35 °C), and transfusion of multiple blood products [5]. Patients with preoperative severe coronary artery disease (ejection fraction <40 %, congestive heart failure, New York Heart Association [NYHA] class III or IV angina), COPD (forced expiratory volume 1 [FEV $_1$] <1 L), and chronic renal failure (dialysis-dependent) should be admitted directly to an ICU postoperatively following elective abdominal aortic aneurysm (AAA) repair [5]. Patients undergoing vascular procedures not associated with major fluid shift or blood loss, such as carotid endarterectomy (CEA), still require close monitoring in the immediate postoperative period as they are at highest risk of stroke and MI. The event rate for these patients is highest in the first 8 h postoperatively. Thus, patients having CEA and uncomplicated EVAR are normally monitored in high dependency units for 8–12 h, after which they are often discharged home once they are deemed medically and neurologically stable $[6]$.

E.S. Osman, MBBS, FRCSI

T.F. Lindsay, MDCM, MSc, FRCS, FACS (\boxtimes) Division of Vascular Surgery, Department of Surgery, University of Toronto, 200 Elizabeth St EN 6-228, Toronto, ON, Canada, M5G 2C4 e-mail: thomas.lindsay@uhn.ca

General Postoperative Considerations

Postoperative Hemodynamics and Bleeding

An essential first step is to provide an appropriate environment that facilitates maintenance of intraoperative and postoperative hemodynamic stability. It is therefore critical to know the patient's preoperative heart rate, blood pressure, detailed cardiac status including myocardial function as these parameters will have great impact on postoperative management. Commonly seen adverse outcomes such as prolonged hypotension, hypertension, tachycardia, and hypothermia can be modified by careful perioperative planning and execution. Perioperative hemodynamic monitoring is satisfactorily achieved through simple variables such as heart rate, mean arterial pressure, and urine output. The implementation of " Goal-Directed Therapy ," which requires the use of advanced monitoring technologies such as pulmonary artery catheterization, is not essential and has produced conflicting results in cardiac and vascular surgery patients [7].

 Blood pressure targets must be balanced to avoid both hypotension and hypertension. Before considering any therapeutic intervention, common causes of postoperative hypertension —such as pain, agitation, hypoxia, hypercarbia, hypervolemia, and bladder or gastric distention—must be ruled out and treated $[8]$. Many authors recommend targeting a systolic blood pressure between 20 mmHg above and 20 mmHg below the patient's baseline. It is important to avoid high blood pressures for many reasons including the potential to place undue shear stresses on new grafts and increase hemorrhage risk at fresh anastomotic sites. Mean arterial pressure greater than 20 % above preoperative levels should be treated with antihypertensives [9]. Different categories of antihypertensive agents are available for use and it is customary that most treating vascular surgeons are familiar with the safe initial management of hypertension before involving the specialists. Nitrates, beta blockers, calcium channel blockers, angiotensin converting enzyme inhibitors, and vasodilators are among the most commonly used antihypertensive medications. Following major vascular surgery, it is obviously as critical to avoid hypotension and hypovolemia. Low-flow states increase the risk of vascular graft thrombosis and renal dysfunction. Hypotension increases risks of myocardial ischemia, renal failure, and may be a major cause of stroke $[10]$. It is crucial to rule out hemorrhage as the major cause of hypotension in patients who require greater than expected intravenous fluids in the early postoperative period [11].

The authors recommend aggressive fluid resuscitation with crystalloids in the early postoperative period with the judicious use of vasopressors. Several recent studies analyzed the concept of restrictive blood transfusion practices [12]. The American Association of Blood Banks (AABB)

strongly recommends—with high-quality evidence—adhering to restrictive transfusion strategy (7–8 g/dL) in hospitalized, stable patients. The guidelines did not find strong evidence for or against restrictive transfusion strategy in patients at risk of perioperative myocardial infarction and hence gave a weak recommendation to transfusing patients with preexisting cardiovascular disease only if their hemoglobin is less than 8 g/dL or they have symptoms $[13]$.

Myocardial Ischemia

 Myocardial infarction (MI) is the most common cause of death following major vascular surgery. This is not surprising as the prevalence of coronary artery disease in these patients approaches 90 $\%$ [11, 14]. Many postoperative myocardial infarctions in vascular patients occur without chest pain and may not be preceded by changes in heart rate or blood pressure [14]. An elevated postoperative cardiac troponin I is a consistent predictor of increased cardiac events and mortality following major vascular surgery. A single Troponin I on each of the first 3 postoperative days will identify patients who have silent myocardial ischemia. Even in the absence of other signs of clinically significant cardiac ischemia, an elevated troponin has been shown to correlate with increased 30-day mortality $[15]$. There is time between the positive troponin I increase and death, allowing for changes in management and intervention.

 Intraoperative hypothermia has been demonstrated to be an independent predictor of postoperative myocardial ischemia $[16]$. However, following acute myocardial infarction or cardiac arrest, early establishment of "therapeutic" or induced hypothermia is thought to be a safe technique that promotes cardioprotection, probably by improving microvascular reperfusion of previously ischemic heart tissue [17].

 Perioperative cardiovascular optimization is a crucial step in reducing postoperative myocardial events. American and European guidelines recommend the use of beta blockers (target heart rate 60–65 bpm while avoiding hypotension) in patients undergoing vascular surgery [18]. However the most recent meta-analysis of beta blocker therapy of secure trials documented that perioperative beta blocker initiation prior to high and intermediate risk noncardiac surgery was associated with a 27 $\%$ increased risk in all-cause mortality [19–22]. Hypotension and stroke are increased by beta blockers but non-fatal MIs are reduced in secure trials. Preoperative initiation of beta blockers is no longer considered to be the gold standard in myocardial prevention. A recent trial of clonidine to blunt central sympathetic signal outflow which is activated during and after surgery with the goal of reducing myocardial infarction and postoperative death, in noncardiac surgical patients, was not associated with a reduction in myocardial infarction or death at 30 days $[23]$. It is important to note that withdrawal of beta blocker therapy perioperatively is associated with increased mortality $[24]$. Thus to date there are no antihypertensive medications that been proven to reduce perioperative myocardial infarction in vascular patients.

 Guidelines from the American College of Cardiology/ American Heart Association/Society for Cardiovascular Angiography and Interventions recommend dual antiplatelet therapy with aspirin and clopidogrel following percutaneous coronary intervention (PCI). The recommended duration is at least 4 weeks following bare metal stents (BMS) and 12 months for drug eluting stents (DES) , with aspirin continued indefinitely $[25]$. The authors recommend that aspirin can be continued perioperatively in all vascular patients because the risk of perioperative bleeding in this setting is much smaller, compared with the cardiovascular thromboembolic risks associated with its withdrawal. A recent trial examined the use of ASA in the perioperative period $[26]$. Although approximately one-third of patients in the trial had a history of vascular disease (CAD, PAD, or stroke), only 5 % were undergoing vascular procedures. There was no benefit of ASA in the perioperative period in patients with vascular disease and bleeding was increased in those on ASA. Because most patients underwent lower risk nonvascular procedures, this trial is insufficient to change practice. Neuraxial blocks can be safely performed on patients taking aspirin [27], whereas it is safer to discontinue clopidogrel 7 days and ticlopidine 14 days prior to any such procedure $[28]$. Unlike cardiac surgery, discontinuation of clopidogrel prior to major vascular procedures (when neuraxial blockade/anesthesia will not be used) is not associated with increased bleeding risk $[29-31]$. In addition to clopidogrel, vascular surgeons must be familiar with the bleeding risks of patients on other antiplatelet agents such as prasugrel, ticagrelor, and glycoprotein IIb/IIIa inhibitors (GPI) (e.g., abciximab, eptifibatide, and tirofiban).

 Based on several primary and secondary prevention studies, the recommendations from the National Cholesterol Education Program (NCEP) and the ACC/AHA guidelines, statins are used in vascular surgery patients for their apparent protective effect in the reduction of perioperative cardiac complications. Several studies have demonstrated the beneficial effects of statins in reducing the risk of perioperative cardiac events in patients undergoing major noncardiac arterial surgery $[32-38]$. In addition to their lipid-lowering role, the pleiotropic properties of statins that result in stabilized atherosclerotic plaques, improved endothelial function, and decreased platelet aggregation are well known [39–42]. Even though it is now commonplace to start statins in vascular surgery patients who are not on it perioperatively (statinnaïve), the ideal timing and optimal dosage is not clear. Some studies suggested commencing statins two weeks preoperatively [39]. Following chronic statin therapy, withdrawal or accidental omission postoperatively has the potential for increasing the cardiovascular risk [43, 44].

Renal Failure

 Acute renal failure is more common following vascular, cardiac, and major abdominal surgery with a mortality rate of 60–80 $\%$ if renal replacement therapy is required [45]. Abnormal renal function prior to major vascular surgery has been shown to be a strong predictor of postoperative acute renal failure [46]. Recent studies have demonstrated that even minimal increases in serum creatinine are associated with higher short- and long-term mortality rates, irrespective of partial or full recovery of renal function at the time of discharge [47]. Postoperative acute renal failure is multifactorial. However, perioperative hypotension (pre-renal azotemia) and ischemic acute tubular necrosis (ATN) are central to most episodes. Examples of periprocedural factors that could potentially lead to ATN include aortic cross-clamping time, renal ischemia secondary to supra renal clamping, volume of contrast given, and the use of other nephrotoxins.

 A meta-analysis combining the results of 61 studies (3359 patients) failed to show any benefit for renal-dose dopamine (potent vasodilator acting on dopamine 1 and 2 receptors in the renal cortex) in reducing mortality or need for renal replacement therapy despite an increase in urine output [48]. Small studies using fenoldopam, a potent renal vasodilator (dopamine 1 receptor agonist) and natriuretic, have demonstrated improved renal outcome when given intraoperatively and postoperatively [49, [50](#page-8-0)]. However, Kidney Disease Improving Global Outcomes (KDIGO) gave level 2C suggestion not to use fenoldopam to prevent or treat acute kidney injury (AKI) [51]. The FENO HSR is an interesting randomized controlled study that has been recently completed but not yet published. It is trying to show whether fenoldopam is beneficial in reducing the incidence of renal replacement therapy (RRT) or mortality in patients with or at risk for AKI following cardiac surgery. There is high quality of evidence that during AAA repair, forced diuresis, whether using mannitol or furosemide, has probably no benefit in reducing the risk of postoperative AKI. However, in a recent meta-analysis of 47 studies, the evidence for forced diuresis being beneficial in reducing the need for renal replacement therapy (RRT) is quoted as weak [52]. On the other hand, it has been suggested that the perioperative timely introduction of continuous infusion of furosemide in cardiac surgery patients does not increase the incidence of renal impairment $[52]$.

N -acetylcysteine (NAC) is known for its safe and harmless anti-oxidant and renal vasodilator properties. Several comprehensive reviews failed to find sufficient evidence to support its use in prevention of contrast-induced nephropathy. However, NAC should never replace adequate hydration [53]. Thus, the optimal perioperative strategy remains to ensure adequate intravascular volume and to avoid the administration of possible nephrotoxins such as non- steroidal anti-inflammatory drugs (NSAIDs) or aminoglycosides.

Respiratory Complications

 Postoperative respiratory failure is associated with increased mortality, both in the short and long term $[54, 55]$ $[54, 55]$ $[54, 55]$. Johnson et al. reported a reduction in the median long-term survival by 87 % in patients who suffered from a pulmonary complication in the first 30 days following surgery $[54]$. Deepbreathing exercises, incentive spirometry, and continuous positive airway pressure are associated with a reduction in pulmonary complications [56, [57](#page-8-0)]. Postoperative analgesia in the form of continuous infusions of low dose morphine in combination with propofol commonly ensures relative comfort and sedation without respiratory depression for those who require intubation. This in turn helps in early extubation and effective return of function. Currently, most patients are extubated in the operating room following major vascular surgery. Once in the ICU, patients who are still intubated should be rewarmed aggressively and stabilized. Patients who require prolonged mechanical ventilation are generally those who have suffered a postoperative MI, renal failure, bowel ischemia, sepsis, or early acute respiratory distress syndrome (ARDS). The patients at highest risk for early postoperative ARDS are those who are hemodynamically unstable (e.g. ruptured AAA repair). This is because of the "two-hit" nature of AAA rupture and repair in addition to the large volumes of crystalloid and blood products administered [58]. Patients who suffer from early ARDS will benefit from a lung-protective ventilation strategy that combines low tidal volumes and relatively high respiratory rates with reduction in ventilator-associated lung injury (barotrauma, volutrauma, atelectrauma, biotrauma). This strategy leads to improved recruitment and oxygenation and subsequently higher rate of weaning from mechanical ventilation.

 Patients who need prolonged ventilation may require tracheostomy, which is correlated with poor outcome and increased in-hospital mortality in this patient cohort [59].

Gastrointestinal Complications

 Two potentially lethal postoperative gastrointestinal (GI) complications worthy of special mention are bowel ischemia and abdominal compartment syndrome. One cannot ignore the risk of upper GI hemorrhage when discussing GI complications and this needs to be treated with proton pump inhibitors gastroscopy with or without intervention as required.

These complications are most commonly associated with patients who undergo aortic surgery for a ruptured AAA. Colonic ischemia affects less than 2 % of patients undergoing elective AAA repair but carries a mortality rate of 40–65 $%$ [60]. Patients operated on for a ruptured AAA have rates of colonic ischemia that range from 15 to 65 % [60, [61](#page-8-0)]. As previously stated, excessive fluid requirements may be an early indication of bowel ischemia . Certainly, persistent acidosis, low bicarbonate level, and hypotension are worrisome markers. It is important to maintain a high index of suspicion at an early stage, before the patient develops elevated lactate, acidosis, GI bleeding, portal-venous gas on plain abdominal X-ray, or pneumatosis intestinalis on computed tomography (CT) scan. Flexible sigmoidoscopy and colonoscopy are the most useful tools in making the diagnosis of bowel ischemia. Mild bowel ischemia limited to colonic mucosa can be treated conservatively. More extensive ischemia with or without hemodynamic instability requires urgent resection. If the diagnosis is delayed, the patient can progress to full transmural ischemia, which carries a mortality rate of 80–100 $\%$ [62].

 A second serious complication is abdominal compartment syndrome (discussed in detail in Chap. [48\)](http://dx.doi.org/10.1007/978-3-319-19668-8_48). Normal intra-abdominal pressure is less than 7 mmHg and it is normally measured using bladder pressure. A bladder pressure greater than 12 mmHg is indicative of intra-abdominal hypertension and pressures above 25 mmHg place patients at high risk for abdominal compartment syndrome $[63]$. Patients who have received massive volume resuscitation with fluids and blood products (e.g., ruptured AAA) are at particularly high risk. Classical cases of full blown abdominal compartment syndrome present with increased airway pressures when on a ventilator, decreased cardiac output, hypotension, and oliguria. This can lead to ischemia of intraabdominal organs. Early recognition using bladder pressure monitoring and treatment with decompressive laparotomy can significantly improve patient survival. Recent reports of aspiration of significant volumes of blood and/or fluid at the conclusion of RAAA treated by Endovascular Aneurysm Repair (EVAR) before clotting occurs may prevent the development of the syndrome.

Pain Management

Effective analgesia has a significant impact on the overall postoperative outcome. Patients with adequate postoperative analgesia based on pain scores have lower rates of myocardial ischemia [64]. Epidural analgesia is frequently used in most major abdominal vascular operations. The benefit of thoracic epidurals is the achievement of effective analgesia with lower doses of local anesthetic drugs with an end result of fewer subsequent hypotensive events. They tend to maintain hemodynamic stability; whereas lumbar epidurals can worsen cardiac segmental wall-motion abnormalities [65]. Reduced incidence of delayed gastric emptying, reduced postoperative ileus and increased mobility are among many advantages of epidural analgesia over the intravenous method. Increased mobility has the further theoretical benefit of reduced thrombotic and pulmonary complications.

Multisystem Organ Failure

 Currently, multisystem organ failure occurs most frequently after repair of ruptured aortic aneurysms or other major open vascular procedures. No interventions have been demonstrated to prevent this condition in this cohort. Although respiratory failure is common in the early postoperative period however it is frequently not the main cause of early death, whereas renal dysfunction constitutes an early sign of poor prognosis. Hepatic dysfunction occurring near the end of the first week postoperatively was found to be a negative prognostic indicator $[66]$.

Surgery-Specific Considerations

Open AAA Repair

 One-third of patients who have undergone elective repair of an AAA will suffer from one or more postoperative complications $[67]$. Some of these complications depend on the location and duration of the aortic clamp. The incidence of renal failure and other complications is much higher with a suprarenal or supra celiac clamp. Renal failure following elective infrarenal AAA repair is reported to be around 5.4 %, with less than 1 % of patients requiring hemodialysis [68]. An infrarenally placed aortic clamp can decrease renal blood flow by 40 $%$ [69]. Prior to aortic cross-clamping, it is vital to maintain sufficient intravascular volume to ensure adequate renal perfusion. Another common problem following AAA involves the gastrointestinal system. In the absence of high gastric outputs (>500 cc/24 h) or clear evidence of paralytic ileus, nasogastric (NG) tubes should be routinely removed on the first day following elective AAA repair and patients will often tolerate sips of fluid. Gastric emptying has been shown to return to normal by 18 h following elective AAA, with small bowel function normalizing by the third day $[70]$. Early oral nutrition is one of the components of the enhanced recovery after surgery (ERAS) program, which is well established in colorectal surgery. A systematic review is looking at the evidence to support future implementation in the postoperative care of patients undergoing major vascular surgery $[71]$.

 In patients who require prolonged mechanical ventilation, low-volume tube feeding should be started early and progressed based on the patient's residuals or established ICU feeding protocols.

Ruptured AAA Repair

 Despite recent advances in the perioperative management of patients with vascular disease, mortality rate following open ruptured AAA remains around 40–50 %. More than 8000 patients die each year in the USA from AAA ruptures [72]. These are the most challenging postoperative vascular patients to manage and they have the highest rates of all of the aforementioned complications. They are frequently cold and coagulopathic on leaving the operating room, hence rapid rewarming and stabilization is essential. Aggressive reversal of medical coagulopathy with blood products is vital to prevent further bleeding. Third-space fluid losses can be significant, and patients may require a large amount of volume resuscitation. Acidosis is common and must be addressed with adequate volume resuscitation in an early goal-directed fashion with prompt adjustments in ventilatory support, and appropriate usage of bicarbonate infusions. Large fluid and blood-product administration puts patients at risk for abdominal compartment syndrome and early ARDS. The development of abdominal compartment syndrome is significant after ruptured AAA because of the large retroperitoneal hematoma that remains after repair and bowel edema. A planned delayed abdominal closure can prevent this complication, although this may require use of mesh or a vacuum-assisted closure technique. It is important to maintain a high index of suspicion for complications that require intervention such as stress ulceration and intestinal (particularly left colon) ischemia.

Endovascular Aortic Repair

 The endovascular approach to treat AAA has been in clinical practice for over 20 years. Initially this was only offered to patients considered too "high risk" for traditional open repair. Currently the treatment of aortic aneurysms continues to shift towards endovascular repair. In the USA, more than 70 % of elective AAA repairs are now being performed by this method [73]. This will increase further as new techniques of endovascular aortic repair (e.g. fenestrated and branched EVAR) become widely disseminated to deal with infrarenal aneurysms with short neck lengths. Instead of making a large abdominal incision, EVAR is generally done through the groins, either by making small groin incisions or percutaneously, allowing the devices to be introduced via the femoral arteries. Fluoroscopy is used to guide the devices to the proper location, where they are deployed; relining the diseased aorta and excluding the aneurysm $[74]$. The early advantages of EVAR include less pain, reduced blood loss, decreased rates of postoperative medical complications, and rapid recovery. A recent meta-analysis of 41 studies (total population of nearly 60,000) reported an advantage of EVAR over open repair [\[75](#page-9-0)]. EVAR was found to be associated with significantly lower inhospital mortality, respiratory complications, and acute renal failure, in addition to lower requirements of intraoperative blood transfusion. It also showed a trend towards a reduced cardiac complications and mesenteric ischemia [75]. It is important to monitor these patients for postoperative acute limb ischemia secondary to dissection of the femoral artery, thrombosis, or embolization. Furthermore, the deployment of the devices involves the introduction of several guide wires, sheaths, and catheters via the femoral arteries through to the level of the aneurysm and beyond. Extreme care is required, as this carries an increased risk of iatrogenic damage of vessels even remote from the site of the aneurysm. Although a completion angiogram is performed at the end of the procedure, such injuries can be missed.

 Ischemic colitis can occur as the origin of inferior mesenteric artery is covered by the device, but fortunately the natural presence of rich collaterals makes this infrequent. Nevertheless it is a deadly complication and early identification and therapy is critical to patient survival.

 Finally, because these patients receive radiocontrast during the procedure, it is imperative to maintain adequate hydration before the contrast is given and maintain urine output postoperatively to minimize the risk of contrast nephropathy.

 Repairing ruptured AAA (rAAA) by EVAR is seen more commonly and at least 33 % are performed through this route in the USA. Several studies have reported lower perioperative mortality (50 % open vs. 23 % EVAR) and morbidity (90 % vs. 53 %) in selected patients $[76, 77]$. A recent study, from the USA, comparing open and endovascular approaches in treating rAAA reported that EVAR is a safe and superior approach to open surgery for the management of patients with rAAA. On multivariate analysis, patients who underwent open repair were at significantly increased risk of morbidity and mortality [78]. However, when referring to such "favorable" studies, it is critical to take into account factors like patient's hemodynamic stability, comorbidities, and other parameters such as the lowest blood pressure at presentation. Although the recent multi-center UK-based IMPROVE trial failed to show any statistically significant difference in 30-day mortality between EVAR and open repair of rAAA on an intention to treat basis, it was demonstrated that EVAR is a valid option in treating this cohort of patients [79]. The IMPROVE trial study demonstrated that EVAR was associated with reduce mortality (38 % open vs. 25 % EVAR) but this was on a procedure

basis not the intention to treat analysis. EVAR for rAAA is promising, but future expansion is limited by logistical barriers, such as the availability of a dedicated endovascular team with round the clock access to resources together with expertise in performing EVAR in this setting.

Thoracoabdominal Aortic Repair

 Open thoracoabdominal aneurysm repair carries substantial risk of perioperative morbidity and mortality. Compared to infrarenal AAA repair, postoperative renal, pulmonary, visceral, and cardiac complication rates and mortality are significantly higher. Pulmonary complications are the most common and impact significantly on patient outcome. The left lung is commonly not ventilated to facilitate repair and it frequently requires time to recover. Division of the diaphragm is associated with prolonged postoperative ventilation and this is found to be independent of known pulmonary risk factors [80]. Etz et al. performed early tracheostomy, on postoperative days $5-7$, on all patients with significant pulmonary problems in order to aid care and early mobilization [81]. Coselli and his group recently published a retrospective analysis of 823 patients who underwent open TAAA [82]. Their relatively superior "early outcome" results clearly explain "volume-related outcome," a concept well known, but more striking in thoracoabdominal aneurysms. A recent review of long-term follow-up of these patients demonstrated that renal failure, neurologic events, and ventricular dysfunction decreased late as well as early survival [83]. A devastating complication following this operation is spinal cord ischemia. Spinal drains are used for a period of 72 h with a target pressure of $<$ 10 mmHg to reduce cerebrospinal fluid (CSF) pressure and improve spinal cord perfusion. Their use is generally coupled with maintenance of adequate preload and mean blood pressure above 80 mmHg for optimum tissue perfusion. Any hypotensive precipitant, occurring even days to weeks postoperatively, could lead to delayed neurological deficits. Despite being universally used, a recent meta-analysis of three randomized controlled trials on the topic of "CSF drainage" in TAAA reported that there is only limited support for its use $[84]$. Spinal cord function can be monitored using motor evoked potentials (MEP) which can provide an intraoperative indication that the spinal cord is adequately perfused prior to leaving the operating room.

Carotid Endarterectomy

 CEA is one of the most frequently performed vascular operations in the USA $[85]$. Monitoring in a high dependency unit postoperatively with adequate nurse-to-patient ratio is desirable as most of the adverse events are mani-

fested within 8 h of surgery [86]. Patients on preoperative aspirin and clopidogrel have been shown to experience a fi vefold increased risk of postoperative bleeding after CEA [87]. However, recent reports suggest that a single 75-mg dose of clopidogrel the night before surgery in addition to regular 75 mg aspirin postoperative may reduce the risk of thromboembolic stroke [88].

 Patients need to be monitored for early hemorrhage, which can present with a rapidly expanding neck hematoma and lead to airway compromise. It is important to facilitate the expeditious patient transfer back to the operating room, which is the optimal location to secure a potentially difficult airway, as well as to deal with source of bleeding. Even in the absence of hemorrhage, airway swelling and edema can still occur. The common causes of stroke after CEA are embolization or thrombosis, or as secondary to a low-flow insult from hypotension. Development of stroke symptoms in the immediate postoperative period demands urgent attention. Urgent duplex scanning to look for endarterectomy-site thrombosis is a mandatory initial step. If duplex scanning is not immediately available, the rapid return to the operating room for assessment of thrombosis and thrombectomy is mandatory. Perioperative statin use prior to CEA has been shown to significantly reduce 30-day stroke, transient ischemic attack (TIA), and mortality [89]. The second most common postoperative complication after CEA is myocardial infarction. Thirteen percent of patients undergoing CEA have an increased postoperative troponin I, which correlates with a worse prognosis, as discussed earlier in this chapter [90]. Bradycardia is common following CEA because of the increased blood pressure sensed at the endarterectomized carotid bulb. In the absence of hypotension, bradycardia alone is not treated unless severe. Hypotension with bradycardia requires therapy to prevent precipitation of stroke and MI. Reduction in the sensitivity of the baroreceptor reflex can also cause postoperative hypertension. One concern is an excessive increase in cerebral perfusion, causing hyperperfusion syndrome. Hyperperfusion syndrome can affect 1–3 % of patients following CEA $[91]$. These patients have a dramatic increase in postoperative cerebral blood flow; with velocities in the middle cerebral artery almost double the preoperative values $[91]$. The constellation of symptoms and signs includes ipsilateral headache, hypertension, seizures, and focal neurological deficits. Without immediate aggressive antihypertensive treatment, patients may suffer from cerebral edema, intracerebral or subdural hemorrhage, or may even die. In symptomatic patients, immediate blood pressure targets of $140/90$ mmHg should be the goal $[92]$. Patients with severe headache following CEA, without hemodynamic changes, should have urgent imaging studies to rule out intracranial hemorrhage. Cranial nerve deficits can complicate up to 12.5 % of CEA $[93]$. The most common nerves involved are the hypoglossal, recurrent laryngeal,

superior laryngeal, marginal mandibular, and greater auricular. These are mostly secondary to traction injuries, the majority of which resolve by 6 months [91].

Revascularization for Peripheral Artery Disease

 Reconstructions for peripheral artery disease (PAD) are divided into supra inguinal (aortofemoral) and infra inguinal (femoral popliteal or tibial) reconstructions. Those cases requiring abdominal aortic surgery differ little from those requiring open aneurysm repairs. All patients who require leg reconstructions for critical limb ischemia have advanced atherosclerosis and must have aggressive perioperative risk reduction. After revascularization, graft patency must be carefully monitored. Early graft failure generally implies a technical problem and patients should be returned to the operating room for exploration, thrombectomy, or revision. Postoperative patients should be continued on aspirin and statin therapy with selective use of IV anticoagulation, which may be beneficial in specific high-risk patients $[94]$.

 Compartment syndrome in the revascularized limb is most common in the emergent setting, such as postembolectomy or thrombectomy. Early identification requires a high index of suspicion by the treating surgeon and continuous assessment by the nursing staff. Early symptoms include pain with passive or active flexion or extension, decreased movement, and paralysis. In this setting, urgent four-compartment leg fasciotomies are indicated. Fasciotomy wounds can often be managed with delayed primary closure. If they cannot be closed, management with suction-type wound closure devices is effective or simple wet-to-dry dressings and delayed skin grafting. Rhabdomyolysis secondary to muscle injury and necrosis can complicate the reperfusion of the acutely ischemic limb [95]. Myoglobin released into the circulation can cause direct renal tubular damage if it precipitates in tubules. When urine pH is <6.0 or during low urine output states, myoglobin precipitation occurs leading to ATN. Testing the urine for myoglobin is unreliable because the qualitative test can be negative. Thus, serum creatinine kinase (CK) levels are a better marker of patients at risk. Preventive therapy with adequate hydration and urine alkalinization is best if begun intraoperatively.

Conclusion

Major vascular surgery is a leading cause of significant perioperative morbidity and mortality due to age and comorbid conditions these patients present with. The common perioperative complications following major vascular surgery are cardiac, pulmonary, and renal; however, all systems must be carefully observed. Meticulous preoperative evaluation and implementation of risk reduction strategies are the cornerstone of a successful postoperative management. Understanding procedure-specific factors is crucial. Patients undergoing more invasive procedures like open thoracoabdominal aneurysm and ruptured AAA repair are at the highest risk, creating the greatest management challenges. Every effort should be made to reduce myocardial oxygen demand postoperatively through adequate volume resuscitation, rewarming, and effective analgesia. It is equally important to avoid both hypotension and hypertension. Maintenance of a high index of suspicion and early treatment for common and procedure-specific complications will significantly improve the outcomes of these patients.

References

- 1. Bakoyiannis CN, Tsekouras NS, Georgopoulos S, Klonaris C, Bastounis EE, Filis K, et al. ICU transfer after elective abdominal aortic aneurysm repair can be successfully reduced with a modified protocol. A fourteen year experience from a University Hospital. Int Angiol. 2011;30:43–51.
- 2. Hadjianastassiou VG, Tekkis PP, Goldhill DR, Hands LJ. Quantification of mortality risk after abdominal aortic aneurysm repair. Br J Surg. 2005;92:1092–8.
- 3. Hassen TA, Pearson S, Cowled PA, Fitridge RA. Preoperative nutritional status predicts the severity of the systemic inflammatory response syndrome (SIRS) following major vascular surgery. Eur J Vasc Endovasc Surg. 2007;33:696–702.
- 4. Sprung J, Abdelmalak B, Gottlieb A, Mayhew C, Hammel J, Levy PJ, et al. Analysis of risk factors for myocardial infarction and cardiac mortality after major vascular surgery. Anesthesiology. 2000; 93:129–40.
- 5. Lawlor DK, Lovell MB, DeRose G, Forbes TL, Harris KA. Is intensive care necessary after elective abdominal aortic aneurysm repair? Can J Surg. 2004;47:359–63.
- 6. Angevine PD, Choudhri TF, Huang J, Quest DO, Solomon RA, Mohr JP, et al. Significant reductions in length of stay after carotid endarterectomy can be safely accomplished without modifying either anesthetic technique or postoperative ICU monitoring. Stroke. 1999;30:2341–6.
- 7. Giglio M, Dalfino L, Puntillo F, Rubino G, Marucci M, Brienza N. Hemodynamic goal-directed therapy in cardiac and vascular surgery. A systematic review and meta-analysis. Interact Cardiovasc Thorac Surg. 2012;15:878–87.
- 8. Papia G, Klein D, Lindsay TF. Intensive care of the patient following open abdominal aortic surgery. Curr Opin Crit Care. 2006;12:340–5.
- 9. Gopalan PD, Burrows RC. Critical care of the vascular surgery patient. Crit Care Clin. 2003;19:109–25.
- 10. Krul JM, van Gijn J, Ackerstaff RG, Eikelboom BC, Theodorides T, Vermeulen FE. Site and pathogenesis of infarcts associated with carotid endarterectomy. Stroke. 1989;20:324–8.
- 11. McArdle PJ, Sanders KD. Postoperative care of vascular surgery patients. Anesthesiol Clin North America. 2004;22:333–47.
- 12. Carson JL, Carless PA, Hebert PC. Transfusion thresholds and other strategies for guiding allogeneic red blood cell transfusion. Cochrane Database Syst Rev. 2012;4, CD002042.
- 13. Carson JL, Grossman BJ, Kleinman S, Tinmouth AT, Marques MB, Fung MK, et al. Red blood cell transfusion: a clinical practice guideline from the AABB. Ann Intern Med. 2012;157:49–58.
- 14. Mangano DT. Perioperative cardiac morbidity. Anesthesiology. 1990;72:153–84.
- 15. van Waes JA, Nathoe HM, de Graaff JC, Kemperman H, de Borst GJ, Peelen LM, et al. Myocardial injury after non cardiac surgery and its association with short-term mortality. Circulation. 2013; 127:2264–71.
- 16. Frank SM, Beattie C, Christopherson R, Norris EJ, Rock P, Parker S, et al. Epidural versus general anesthesia, ambient operating room temperature, and patient age as predictors of inadvertent hypothermia. Anesthesiology. 1992;77:252–7.
- 17. Mottillo S, Sharma K, Eisenberg MJ. Therapeutic hypothermia in acute myocardial infarction: a systematic review. Can J Cardiol. 2011;27:555–61.
- 18. Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof EL, Fleischmann KE, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for non-cardiac surgery: executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2007;116:e418–500.
- 19. Bouri S, Shum-Shin MJ, Cole GD, Mayet J, Frances DP. Metaanalysis of secure randomised controlled trials of β-blockade to prevent perioperative death in non-cardiac surgery. Heart. 2014;100:456–64.
- 20. Angeli F, Verdecchia P, Karthikeyan G, Mazzotta G, Gentile G, Reboldi G. β-Blockers reduce mortality in patients undergoing highrisk non-cardiac surgery. Am J Cardiovasc Drugs. 2010;10:247–59.
- 21. Dunkelgrun M, Boersma E, Schouten O, Koopman-van Gemert AW, van Poorten F, Bax JJ, et al. Bisoprolol and fluvastatin for the reduction of perioperative cardiac mortality and myocardial infarction in intermediate risk patients undergoing non cardiovascular surgery. Ann Surg. 2009;249:921–6.
- 22. Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof EL, Fleischmann KE, et al. 2009 ACCF/AHA focused update on perioperative beta blockade: a report of the American college of cardiology foundation/American heart association task force on practice guidelines. Circulation. 2009;120:e169–276.
- 23. Devereaux PJ, Sessler DI, Leslie K, Kurz A, Mrkobrada M, Alonso-Coello P, et al. Clonidine in patients undergoing noncardiac surgery. N Engl J Med. 2014;370:1504–13.
- 24. Hoeks SE, Scholte Op Reimer WJ, van Urk H, Jörning PJ, Boersma E, Simoons ML, et al. Increase of 1-year mortality after perioperative beta-blocker withdrawal in endovascular and vascular surgery patients. Eur J Vasc Endovasc Surg. 2007;33:13–19.
- 25. Levine GN, Bates ER, Blankenship JC, Bailey SR, Bittl JA, Cercek B, et al. 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions. J Am Coll Cardiol. 2011;58(24):e44–122.
- 26. Devereaux PJ, Mrkobrada M, Sessler DI, Leslie K, Alonso-Coello P, Kurz A, et al. Aspirin in patients undergoing noncardiac surgery. N Engl J Med. 2014;370:1494–503.
- 27. Kotsovolis G, Komninos G, Kyrgidis A, Papadimitriou D. Preoperative withdrawal of antiplatelet treatment in lower limb vascular patients prior to surgical management under epidural or spinal anesthesia: an evidence based approach and systematic review. Int Angiol. 2010;29:475–81.
- 28. Benzon HT, Fragen R, Benzon HA, Savage J, Robinson J, Puri L. Clopidogrel and neuraxial block: the role of the PFA 2 and P2Y12 assays. Reg Anesth Pain Med. 2010;35:115.
- 29. Fleming MD, Stone WM, Scott P, Chapital AB, Fowl RJ, Money SR. Safety of carotid endarterectomy in patients concurrently on clopidogrel. Ann Vasc Surg. 2009;23:612–5.
- 30. Stone DH, Goodney PP, Schanzer A, Nolan BW, Adams JE, Powell RJ, et al. Vascular Study Group of New England. Clopidogrel is not

associated with major bleeding complications during peripheral arterial surgery. J Vasc Surg. 2011;54:779–84.

- 31. Saadeh C, Sfeir J. Discontinuation of preoperative clopidogrel is unnecessary in peripheral arterial surgery. J Vasc Surg. 2013;58: 1586–92.
- 32. O'Neil-Callahan K, Katsimaglis G, Tepper MR, Ryan J, Mosby C, Ioannidis JP, et al. Statins decrease perioperative cardiac complications in patients undergoing non-cardiac vascular surgery: the Statins for Risk Reduction in Surgery (StaRRS) study. J Am Coll Cardiol. 2005;45:336–42.
- 33. Chopra V, Wesorick DH, Sussman JB, Greene T, Rogers M, Froehlich JB, et al. Effect of perioperative statins on death, myocardial infarction, atrial fibrillation, and length of stay: a systematic review and meta-analysis. Arch Surg. 2012;147:181–9.
- 34. Paraskevas K, Veith F, Liapis C. Perioperative/periprocedural effects of statin treatment for patients undergoing vascular surgery or endovascular procedures: an update. Curr Vasc Pharmacol. 2013;11:112–20.
- 35. Schouten O, Kertai MD, Bax JJ, Durazzo AE, Biagini E, Boersma E, et al. Safety of perioperative statin use in high-risk patients undergoing major vascular surgery. Am J Cardiol. 2005;95: 658–60.
- 36. Kertai MD, Boersma E, Westerhout CM, Klein J, Van Urk H, Bax JJ, et al. A combination of stations and beta-blockers is independently associated with a reduction in the incidence of perioperative mortality and nonfatal myocardial infarction in patients undergoing abdominal aortic aneurysm surgery. Eur J Vasc Endovasc Surg. 2004;28:343–52.
- 37. Poldermans D, Bax JJ, Kertai MD, Krenning B, Westerhout CM, Schinkel AF, et al. Statins are associated with a reduced incidence of perioperative mortality in patients undergoing major non cardiac vascular surgery. Circulation. 2003;107:1848–51.
- 38. Ward RP, Leeper NJ, Kirkpatrick JN, Lang RM, Sorrentino MJ, Williams KA. The effect of preoperative statin therapy on cardiovascular outcomes in patients undergoing infrainguinal vascular surgery. Int J Cardiol. 2005;104:264–8.
- 39. Durazzo AE, Machado FS, Ikeoka DT, De Bernoche C, Monachini MC, Puech-Leão P, et al. Reduction in cardiovascular events after vascular surgery with atorvastatin: a randomized trial. J Vasc Surg. 2004;39:967–75.
- 40. Ito MK, Talbert RL, Tsimikas S. Statin-associated pleiotropy: possible beneficial effects beyond cholesterol reduction. Pharmacotherapy. 2006;26:85S–97.
- 41. Cannon CP, Braunwald E, McCabe CH, Rader DJ, Rouleau JL, Belder R, et al. Intensive versus moderate lipid lowering with statins after acute coronary syndromes. N Engl J Med. 2004;350: 1495–504.
- 42. Schwartz GG, Olsson AG, Ezekowitz MD, Ganz P, Oliver MF, Waters D, et al. Effects of atorvastatin on early recurrent ischemic events in acute coronary syndromes: the MIRACL study: a randomized controlled trial. JAMA. 2001;285:1711–8.
- 43. Le Manach Y, Godet G, Coriat P, Martinon C, Bertrand M, Fléron MH, et al. The impact of postoperative discontinuation or continuation of chronic statin therapy on cardiac outcome after major vascular surgery. Anesth Analg. 2007;104:1326–33.
- 44. Schouten O, Hoeks SE, Welten GM, Davignon J, Kastelein JJ, Vidakovic R, et al. Effect of statin withdrawal on frequency of cardiac events after vascular surgery. Am J Cardiol. 2007;100: 316–20.
- 45. Calvert S, Shaw A. Perioperative acute kidney injury. Perioper Med (Lond). 2012;1:6.
- 46. Mangano CM, Diamondstone LS, Ramsay JG, Aggarwal A, Herskowitz A, Mangano DT. Renal dysfunction after myocardial revascularization: risk factors, adverse outcomes and hospital resource utilization. Ann Intern Med. 1998;128:194–203.
- 47. Ishani A, Nelson D, Clothier B, Schult T, Nugent S, Greer N, et al. The magnitude of acute serum creatinine increase after cardiac surgery and the risk of chronic kidney disease, progression of kidney disease, and death. Arch Intern Med. 2011;171:226–33.
- 48. Friedrich JO, Adhikari N, Herridge MS, Beyene J. Meta-analysis: low-dose dopamine increases urine output but does not prevent renal dysfunction or death. Ann Intern Med. 2005;142:510–24.
- 49. Halpenny M, Rushe C, Breen P, Cunningham AJ, Boucher-Hayes D, Shorten GD. The effects of fenoldopam on renal function in patients undergoing elective aortic surgery. Eur J Anaesthesiol. $2002.19.32 - 9$
- 50. Miller Q, Peyton BD, Cohn EJ, Holmes GF, Harlin SA, Bird ET, et al. The effects of intraoperative fenoldopam on renal blood flow and tubular function following suprarenal aortic cross-clamping. Ann Vasc Surg. 2003;17:656–62.
- 51. Bove T, Paternoster G, Conte M. Letter to the editor. The FENO-HSR study: details of statistical analyses. HSR Proc Intensive Care Cardiovasc Anesth. 2013;5:55–6.
- 52. Gandhi A, Husain M, Salhiyyah K, Raja SG. Does perioperative furosemide usage reduce the need for renal replacement therapy in cardiac surgery patients? Interact Cardiovasc Thorac Surg. 2012;15:750–5.
- 53. Ad-hoc Working Group of ERBP, Fliser D, Laville M, Covic A, Fouque D, Vanholder R, et al. A European Renal Best Practice (ERBP) position statement on the Kidney Disease Improving Global Outcomes (KDIGO) clinical practice guidelines on acute kidney injury: part 1: definitions, conservative management and contrastinduced nephropathy. Nephrol Dial Transplant. 2012;27:4263–72.
- 54. Johnson RG, Arozullah AM, Neumayer L, Johnson RG, Arozullah AM, Neumayer L. Multivariable predictors of postoperative respiratory failure after general and vascular surgery: results from the patient safety in surgery study. J Am Coll Surg. 2007;204:1188–98.
- 55. Khuri SF, Daley J, Henderson W, Barbour G, Lowry P, Irvin G, et al. The national veterans administration surgical risk study: risk adjustment for the comparative assessment of the quality of surgical care. J Am Coll Surg. 1995;180:519–31.
- 56. Khuri SF, Henderson WG, DePalma RG, Mosca C, Healey NA, Kumbhani DJ, et al. Determinants of long-term survival after major surgery and the adverse effect of postoperative complications. Ann Surg. 2005;242:326–41.
- 57. Lawrence VA, Cornell JE, Smetana GW. Strategies to reduce postoperative pulmonary complications after non cardiothoracic surgery: systematic review for the American College of Physicians. Ann Intern Med. 2006;144:596–608.
- 58. Lindsay TF, Luo XP, Lehotay DC, Rubin BB, Anderson M, Walker PM, et al. Ruptured abdominal aortic aneurysm, a "two-hit" ischemia/reperfusion injury: evidence from an analysis of oxidative products. J Vasc Surg. 1999;30:219–28.
- 59. Cambria RP, Clouse WD, Davison JK, Dunn PF, Corey M, Dorer D. Thoracoabdominal aneurysm repair: results with 337 operations performed over a 15-year interval. Ann Surg. 2002;236:471–9.
- 60. Champagne BJ, Darling 3rd RC, Daneshmand M, Kreienberg PB, Lee EC, Mehta M, et al. Outcome of aggressive surveillance colonoscopy in ruptured abdominal aortic aneurysm. J Vasc Surg. 2004;39:792–6.
- 61. Björck M, Lindberg F, Broman G, Bergqvist D. pH monitoring of the sigmoid colon after aortoiliac surgery: a five-year prospective study. Eur J Vasc Endovasc Surg. 2000;20:273–80.
- 62. Kehlet H, Moesgaard F. Prophylaxis against postoperative complications in gastroenterology. Scand J Gastroenterol Suppl. 1996; 216:218–24.
- 63. Karkos CD, Menexes GC, Patelis N, Kalogirou TE, Giagtzidis IT, Harkin DW. A systematic review and meta-analysis of abdominal compartment syndrome after endovascular repair of ruptured abdominal aortic aneurysms. J Vasc Surg. 2014;59:829–42.
- 64. Mangano DT, Siliciano D, Hollenberg M, Leung JM, Browner WS, Goehner P, et al. Postoperative myocardial ischemia: therapeutic trials using intensive analgesia following surgery – the Study of Perioperative Ischemia (SPI) Research Group. Anesthesiology. 1992;76:342–53.
- 65. Kock M, Blomberg S, Emanuelsson H, Lomsky M, Strömblad SO, Ricksten SE. Thoracic epidural anesthesia improves global and regional left ventricular function during stress-induced myocardial ischemia in patients with coronary artery disease. Anesth Analg. 1990;71:625–30.
- 66. Maziak DE, Lindsay TF, Marshall JC, Walker PM. The impact of multiple organ dysfunction on mortality following ruptured abdominal aortic aneurysm repair. Ann Vasc Surg. 1998;12:93–100.
- 67. Vemuri C, Wainess RM, Dimick JB, Cowan Jr JA, Henke PK, Stanley JC, et al. Effect of increasing patient age on complication rates following intact abdominal aortic aneurysm repair in the United States. J Surg Res. 2004;118:26–31.
- 68. Gelman S. The pathophysiology of aortic cross-clamping and unclamping. Anesthesiology. 1995;82:1026–60.
- 69. Alpert RA, Roizen MF, Hamilton WK, Stoney RJ, Ehrenfeld WK, Poler SM, et al. Intraoperative urinary output does not predict postoperative renal function in patients undergoing abdominal aortic revascularization. Surgery. 1984;95:707–11.
- 70. Avrahami R, Cohen JD, Haddad M, Singer P, Zelikovski A. Gastric emptying after elective abdominal aortic aneurysm surgery: the case for early postoperative enteral feeding. Eur J Vasc Endovasc Surg. 1999;17(3):241–4.
- 71. Gotlib Conn L, Rotstein OD, Greco E, Tricco AC, Perrier L, Soobiah C, et al. Enhanced recovery after vascular surgery: protocol for a systematic review. Syst Rev. 2012;1:52.
- 72. Mureebe L, Egorova N, McKinsey JF, Kent KC. Gender trends in the repair of ruptured abdominal aortic aneurysms and outcomes. J Vasc Surg. 2010;51:9S–13.
- 73. Giles KA, Pomposelli F, Hamdan A, Wyers M, Jhaveri A, Schermerhorn ML. Decrease in total aneurysm-related deaths in the era of endovascular aneurysm repair. J Vasc Surg. 2009;49:543–51.
- 74. Woody JD, Makaroun MS. Endovascular graft limb occlusion. Semin Vasc Surg. 2004;17:262–7.
- 75. Antoniou GA, Georgiadis GS, Antoniou SA, Pavlidis P, Maras D, Sfyroeras GS, et al. Endovascular repair for ruptured abdominal aortic aneurysm confers an early survival benefit over open repair. J Vasc Surg. 2013;58:1091–105.
- 76. Alsac JM, Desgranges P, Kobeiter H, Becquemin JP. Emergency endovascular repair for ruptured abdominal aortic aneurysms: feasibility and comparison of early results with conventional open repair. Eur J Vasc Endovasc Surg. 2005;30:632–9.
- 77. Larzon T, Lindgren R, Norgren L. Endovascular treatment of ruptured abdominal aortic aneurysms: a shift of the paradigm? J Endovasc Ther. 2005;12:548–55.
- 78. Speicher PJ, Barbas AS, Mureebe L. Open versus endovascular repair of ruptured abdominal aortic aneurysms. Ann Vasc Surg. 2014;28:1249–57.
- 79. IMPROVE Trial Investigators, Powell JT, Sweeting MJ, Thompson MM, Ashleigh R, Bell R, et al. Endovascular or open repair strategy

for ruptured abdominal aortic aneurysm: 30 day outcomes from IMPROVE randomised trial. BMJ. 2014;348:f7661.

- 80. Huynh TT, Miller 3rd CC, Estrera AL, Porat EE, Safi HJ. Thoracoabdominal and descending thoracic aortic aneurysm surgery in patients aged 79 years or older. J Vasc Surg. 2002;36:469–75.
- 81. Etz CD, Di Luozzo G, Bello R, Luehr M, Khan MZ, Bodian CA, et al. Pulmonary complications after descending thoracic and thoracoabdominal aortic aneurysm repair: predictors, prevention, and treatment. Ann Thorac Surg. 2007;83:S870–6.
- 82. Lemaire SA, Price MD, Green SY, Zarda S, Coselli JS. Results of open thoracoabdominal aortic aneurysm repair. Ann Cardiothorac Surg. 2012;1:286–92.
- 83. Schepens MA, Kelder JC, Morshuis WJ, Heijmen RH, van Dongen EP, ter Beek HT. Long-term follow-up after thoracoabdominal aortic aneurysm repair. Ann Thorac Surg. 2007;83:S851–5.
- 84. Khan SN, Stansby G. Cerebrospinal fluid drainage for thoracic and thoracoabdominal aortic aneurysm surgery. Cochrane Database Syst Rev. 2012;10, CD003635.
- 85. Rothwell PM, Eliasziw M, Gutnikov SA, Fox AJ, Taylor DW, Mayberg MR, et al. Analysis of pooled data from the randomized controlled trials of endarterectomy for symptomatic carotid stenosis. Lancet. 2003;361:107–16.
- 86. Sheehan MK, Baker WH, Littooy FN, Mansour MA, Kang SS. Timing of post carotid complications: a guide to safe discharge planning. J Vasc Surg. 2001;34:13–6.
- 87. Hale B, Pan W, Misselbeck TS, Lee VV, Livesay JJ. Combined clopidogrel and aspirin therapy in patients undergoing carotid endarterectomy is associated with an increased risk of postoperative bleeding. Vascular. 2013;21:197–204.
- 88. Naylor AR, Sayers RD, McCarthy MJ, Bown MJ, Nasim A, Dennis MJ, et al. Closing the loop: a 21-year audit of strategies for preventing stroke and death following carotid endarterectomy. Eur J Vasc Endovasc Surg. 2013;46:161–70.
- 89. McGirt MJ, Perler BA, Brooke BS, Woodworth GF, Coon A, Jain S, et al. 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors reduce the risk of perioperative stroke and mortality after carotid endarterectomy. J Vasc Surg. 2005;42:829–36.
- 90. Motamed C, Motamed-Kazerounian G, Merle JC, Dumérat M, Yakhou L, et al. Cardiac troponin I assessment and late cardiac complications after carotid stenting or endarterectomy. J Vasc Surg. 2005;41:769–74.
- 91. Howell SJ. Carotid endarterectomy. Br J Anaesth. 2007;99:119–31.
- 92. Scozzafava J, Hussain MS, Yeo T, Jeerakathil T, Brindley PG. Case report: aggressive blood pressure management for carotid endarterectomy hyperperfusion syndrome. Can J Anaesth. 2006;53:764–8.
- 93. Ballotta E, Dagiau G, Saladini M, Bottio T, Abbruzzese E, Meneghetti G, et al. Results of electroencephalographic monitoring during 369 consecutive carotid artery revascularizations. Eur Neurol. 1997;37:43–7.
- 94. Dagher NN, Modrall JG. Pharmacotherapy before and after revascularization: anticoagulation, antiplatelet agents, and statins. Semin Vasc Surg. 2007;20:10–4.
- 95. Zimmerman JL, Shen MC. Rhabdomyolysis. Chest. 2013;144: 1058–65.