Anesthetic Considerations for Elderly Patients Undergoing Vascular Surgery

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By 2030, one in five Americans will be aged 65 or older, nearly double the 12% in 2000 [1]. Lower extremity peripheral arterial disease (PAD) is now known to be associated with equal morbidity and mortality and comparable (or higher) health economic costs as coronary heart disease (CHD) and ischemic stroke [2, 3].

In contrast to coronary heart disease, relatively few genetic variants that influence susceptibility to PAD have been discovered due to greater clinical and genetic heterogeneity in PAD [4]. However, prolonged longevity will reveal physiologic strength, clinical variability, and genetic differences among individuals. Studies have revealed the prevalence of PAD to be 4.7% between the ages of 60 and 69 years, and 14.5% for the ages of 70 years and older [5]. In age- and gender-adjusted logistic regression analyses, black race/ethnicity, current smoking, diabetes, and poor kidney function were positively associated with prevalent PAD [5]. More than 95% of persons with PAD had one or more cardiovascular disease risk factors.

Modifiable risk factors in PAD include smoking, high blood pressure, hyperlipidemia, physical inactivity, obesity, diabetes, increased homocysteine levels [6], and hypothyroidism [7]. Judicious use of beta blockers, antiplatelet therapy, angiotensin-converting enzyme (ACE) inhibitors, statins is recommended for all patients with peripheral vascular disease. Decision to hold Acetylsalicylic Acid (Aspirin) (ASA), statins, ACE inhibitors should be made in conjunction with the surgical team on a case-to-case basis.

The prevalence of coronary artery disease (CAD) in PAD patients ranges from 14% to 90%, which clearly reflects differences in sensitivity of the detection technique for CAD [8]. In another study, 30% of all patients scheduled for aortic aneurysm resection, lower extremity revascularization, or extracranial reconstruction have severe CAD [9].

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Preoperative assessment should take into account all the factors listed above. Preoperative testing should be based on the degree of medical optimization of pre-existing diseases, nature of planned surgery, and the likelihood of substantial hemorrhage and disruption of the autonomic homeostasis of hemodynamic and metabolic function. The purpose of this chapter is to make the reader understand the standard preoperative assessment for patients undergoing vascular surgery and to familiarize them with anesthetic considerations for the most common vascular surgery procedures.

3.1 Local Anesthesia and Regional Anesthesia

3.1.1 Pathophysiology and Pharmacology Related to Aging

It is widely accepted that complex interaction in older patients between subtle changes in pharmacodynamics and altered age-related pharmacokinetics are responsible for drug effect.

Densities of myelinated and unmyelinated axons decrease markedly from birth to the end of the eighth decade, due to increasing size and separation of fibers during the first decade, axonal degeneration, and an increase in endoneurial collagen in the older age groups [10]. The conduction velocity in peripheral motor and sensory nerves slows progressively with advancing age. Spinal cord CVs decline sharply after age 60 [11]. These changes lead to increased sensitivity to local anesthetics in the elderly, reflecting changing pharmacodynamics in old age.

Age-related changes in pharmacokinetics of local anesthetics result in reduced clearance of local anesthetics. Free lidocaine concentration is prone to increase in elderly patients during continuous thoracic epidural anesthesia [12]. In a study by Veering et al. [13], pharmacokinetics of aging had minimal, if any, effect on the peak plasma concentration and the corresponding peak time after the epidural administration of bupivacaine. It demonstrated a marked effect of age on the clearance and a moderate effect on the terminal half-life of bupivacaine. However, this did not translate into a higher potential for systemic toxicity, since toxic threshold concentrations may alter with age. This study also showed faster caudad analgesia in older patients.

Elderly patients also have increased cephalad analgesia level than younger patients. This may be related to the decreased lateral escape of the local anesthetic solution due to the sclerotic intervertebral foramina.

A moderate correlation between the maximal cephalad height of analgesia and the age of the patients has been shown in various studies [13, 14].

Age did not influence the rate of regression of analgesia or the total time for recovery from analgesia. Neither could age be shown to affect the degree or time to recovery from motor blockade. However, a recent study by Paqueron et al. [15] showed age is a major determinant of duration of complete motor and sensory blockade with peripheral nerve block, perhaps reflecting increased sensitivity to local anesthetic agents (Fig. 3.1).

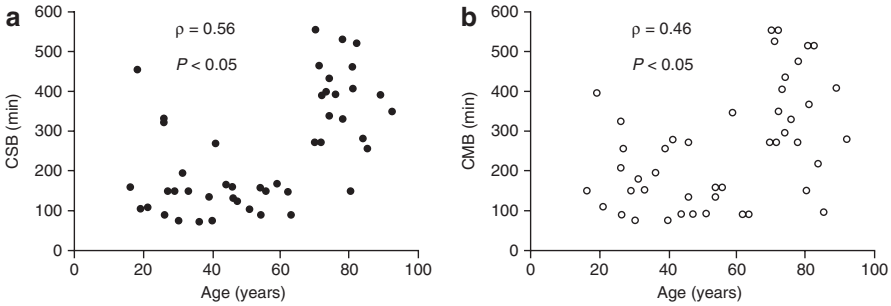

Fig. 3.1 The age of all patients (*x*-axis) in the elderly and young groups and complete sensory (*CSB*, **a**) or motor block (*CMB*, **b**) duration (*y*-axis) are shown. Postoperatively, sensory and motor blocks were assessed hourly for 9 h after time zero. Recovery from sensory and motor blocks was defined as any score greater than zero in any distribution of the radial, median, musculocutaneous, or ulnar nerve. The durations of complete sensory and motor blockade were calculated for each patient as follows: (onset time to a complete sensory or motor block) – (time of recovery from complete sensory or motor block). Durations of complete sensory ($\rho = 0.56$; $P < 0.05$) were significantly correlated with aging (Adapted from Paqueron et al. [15]; with permission)

3.1.2 Procedure-Specific Local and Regional Anesthesia

3.1.2.1 Arteriovenous Access for Hemodialysis and Permanent Vascular Access

Peripheral subcutaneous AV fistula or prosthetic graft is the current procedure of choice for patients requiring permanent hemodialysis access. The procedure is performed in supine position and is usually performed under local anesthesia with intravenous sedations. Elderly patients with chronic renal failure may present a great challenge to the anesthesiologists. Conditions like congestive heart failure, systemic hypertension, electrolyte imbalances, undetermined intravascular fluid volume status are fairly common in this age group. The presence of concomitant dementia, poor baseline cognitive function may make sedation and local anesthesia inadequate choice. In such patients, consideration should be made for brachial plexus block using an ultrasound-guided supraclavicular or infraclavicular approach. In a study by Mizrak et al. [16] when used for Arteriovenous Fistula (AVF) access surgery, infraclavicular brachial plexus block provides higher blood flow in the radial artery and AVF than is achieved with infiltration anesthesia. In another study by Malinzak and Gan [17,] it was also concluded that use of regional blocks may improve the success of vascular access procedures by producing significant vasodilatation, greater fistula blood flow, sympathectomy-like effects, and decreased maturation time. Significant vasodilation after regional block administration is seen in both the cephalic and basilic veins. These vasodilatory properties may assist with AVF site selection.

3.1.2.2 Minimally Invasive Vascular Surgery: Peripheral Arterial Stent Placement and Carotid Stent Placement

Anesthesia for peripheral arterial stent placement can be administered with intravenous moderate sedation and local anesthetic at the puncture site. A common

combination for sedation is 1–2 mg of midazolam (Versed) and 25–50 mcg of fentanyl, depending on the patient's size and response. Standard ASA monitoring with Monitored Anesthesia care is used for these procedures.

The anesthetic technique for Carotid artery stent involves minimal sedation with minimal or no midazolam as excessive sedation may contribute to hypotension in the post stent placement phase. Activated clotting time (ACT) is measured. After a baseline ACT, a small heparin bolus is administered IV to achieve an ACT of approximately twice as normal (250–300 s) to prevent thromboembolic complications. Protamine should be immediately available to treat hemorrhage, although it is not routinely used for the reversal of anticoagulation at the end of the case. Often an oral antiplatelet drug (ticlopidine, clopidogrel, or abciximab) is also given.

The anesthesiology team should also anticipate excessive bradycardia with carotid balloon angioplasty necessitating the pre-emptive use of Atropine 0.4–0.8 mg or Glycopyrollate 0.2–0.4 mg.

3.1.2.3 Lower Extremity Vascular Procedures Including Vein Stripping and Perforator Ligation, Lower Extremity Vascular Bypass, Amputation Procedures of the Lower Extremity

The lower extremity vascular procedures can be ideally performed under regional anesthesia. Regional anesthesia involves spinal, epidural anesthesia, lumbar plexus anesthesia, and regional nerve blocks involving the sciatic, femoral, popliteal fossa nerve blocks, and ankle blocks.

Vascular operations for the lower extremity constitute infrainguinal arterial bypass procedures. Use of an autogenous vein provides the best conduit for infrainguinal arterial bypass procedures. The principle is to have an inflow target that has no significant disease proximal to it that can interfere with the inflow into the bypass. The inflow vessel is usually the common femoral artery, profunda femoris artery, the superficial femoral artery, the popliteal artery, and, in some less common instances, one of the tibial vessels. The target recipient artery is either the popliteal artery or tibial, peroneal, or pedal vessel. These can be approached at the level of the knee or below with a medial incision or at mid tibial/malleolus level depending on the target. It requires administration of 10,000 units of heparin prior to distal anastomosis, followed by proximal anastomosis, arteriogram, partial reversal of heparin, and closure.

Spinal anesthesia provides excellent analgesia but since surgery can be unpredictable in complexity and duration, it may be beneficial to either perform a continuous spinal anesthesia [18, 19] with an intrathecal catheter or perform a combined spinal/epidural or just lumbar epidural catheter [20]. This allows the duration of anesthesia to be extended and may also provide postoperative analgesia. Surgical anesthesia involves L1–4 dermatomes, and a dermatomal level of T10-T12 is required. Hoff et al. [21] showed that spinal anesthesia using bupivacaine and tetracaine mixed in a single-injection technique can last 5 h at the T_{12} level without added untoward effects when compared with lower dose spinal anesthetics. Cautious fluid administration and vasoconstrictors use will limit fluid overload in elderly patients especially after sympathectomy resolves. Strict adherence to American Society of Regional Anesthesia (ASRA) anticoagulation guidelines as mentioned in

the previous section should be practiced before performing spinal anesthesia and prior to removal of the epidural catheter.

Yazigi et al. [22] showed in a series of 25 patients that infrainguinal bypass can be safely performed with combination femoral and sciatic nerve blockade without conversion to general anesthesia (GA). They [23] further did a prospective, randomized study comparing peripheral nerve blockade with general anesthesia for infrainguinal bypass and showed a statistically significant reduction in intraoperative myocardial ischemia in the group randomized to peripheral nerve blockade. Local anesthesia [24] and a combination [25] of a psoas compartment block, sciatic nerve block, and ipsilateral T12-L1 paravertebral block has also been shown to be successful in performing lower limb vascularization surgeries.

Thus local anesthesia and regional nerve blocks can be safely used for lower extremity vascularization procedures but larger randomized trials are needed to confirm the benefits over spinal and general anesthesia. The regional nerve blocks, neuraxial anesthesia, and local anesthesia are limited in their benefits in patients with moderate to severe chronic low back pain and in elderly patients with dementia, as such patients may be difficult to sedate and may require general anesthesia to perform the surgical procedures safely.

Considerable controversy exists over benefits of regional anesthesia over general anesthesia and many institutions have established different standards of care in managing anesthetic care for patients undergoing the above procedures. The goal of the following section is to clearly state the benefits of regional anesthesia in vascular surgery and specifically to geriatric population.

The advantages of spinal and epidural anesthesia using local anesthetic and/or opioids include avoidance of airway manipulation and pulmonary morbidity, and lower blood loss, which leads to reduction of the surgical stress response [26–28]. Urinary cortisol excretion, a marker of the stress response, was significantly diminished during the first 24 postoperative hours in the group receiving epidural anesthesia in a landmark study by Yeager et al. [29]. Reduction of surgical stress response leads to stable hemodynamics, reduced hypercoagulability, better wound healing, and less immunosuppression.

Further, vasodilation, secondary to sympathetic blockade, should be particularly helpful in sustaining graft patency.

In the Perioperative Ischemia Randomized Anesthesia Trial (PIRAT) [30], 100 patients were randomized to undergo lower extremity grafts under either epidural or general anesthesia found that revascularization rate was high in the GA group. Rosenfeld et al. [31], using patients from the PIRAT study, reported an increase in plasminogen activator inhibitor (PAI-1) in the general anesthesia patients but not in the regional anesthesia patients on the morning after surgery (Fig. 3.2).

A review of retrospective, prospective, and meta-analysis studies by Moraca et al. [32] showed significant reduction in perioperative cardiac morbidity (30%), pulmonary infections (40%), pulmonary embolism (50%), ileus (2 days), acute renal failure (30%), and blood loss (30%). Potential complications related to epidural anesthesia/analgesia ranged from minor issues like transient paresthesias (10%) to rare potentially devastating epidural hematomas (0.0006%).

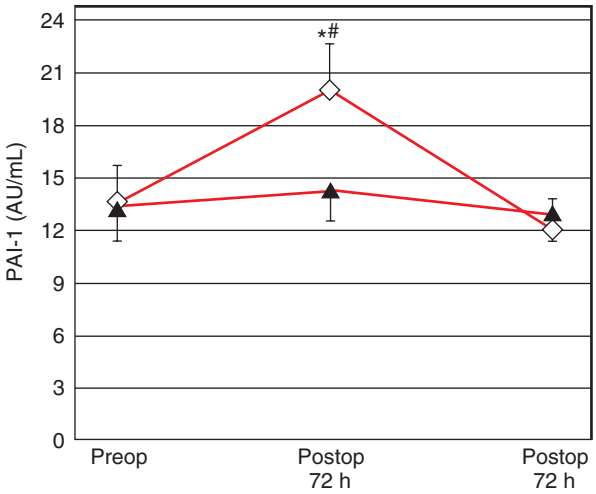

Fig. 3.2 Plasminogen activator inhibitor-1 levels in activity units per milliliter for general and regional anesthesia groups over time. Values are mean ± SEM. #*P*, 0.001 compared to preoperative and 72 h. **P* = 0.05 general anesthesia (GA) compared to regional anesthesia (RA) (From Rosenfeld et al. [31]; with permission; Source: Longnecker et al. [112], Copyright © The McGraw-Hill Companies, Inc. All rights reserved)

Thoracic epidural analgesia can enhance bowel motility not only by producing pain relief and lessening the systemic stress response, but also by creating a sympathectomy, resulting in unopposed parasympathetic innervations to the gut. Sympathetic stimulation, pain, opioids, nitrous oxide, inhalation anesthetics, and increased endogenous catecholamines all contribute to postoperative ileus, and all are blunted in patients treated with perioperative thoracic epidural analgesia [33].

Chery et al. [34] showed in a retrospective review of 407 consecutive patients who underwent above- or below-knee amputations at a single center. The study showed that regional anesthesia group which has older patients (76.6 vs.71.6) was associated with a lower incidence of overall postoperative pulmonary complications and postoperative arrhythmia. Duration of stay in the intensive care unit and hospital was significantly longer in the group receiving general anesthesia. No significant differences in postoperative myocardial infarction, venous thromboembolism, or mortality were seen between groups. Regional anesthesia included either spinal or combined spinal and epidural anesthesia. Nerve blocks were not used.

Singh et al. [35] did an analysis of a prospectively collected database by the National Surgical Quality Improvement Program (NSQIP) of the Veterans Affairs Medical Centers of all patients from 1995 to 2003 in the NSQIP database who underwent infrainguinal arterial bypass. Their results revealed that compared with general endotracheal tube anesthesia, spinal anesthesia (SA) was associated with superior 30-day graft patency, fewer cardiac events in patients without congestive heart failure but with normal functional status, less postoperative pneumonia, and decreased odds of returning to the operating room. In contrast, SA was significantly

better than epidural anesthesia only in the incidence of return to the OR. There was no significant difference in 30-day mortality among the three groups with univariate or multivariate analyses.

The use of neuraxial regional anesthesia (epidural) has shown to decrease incidence of elevated intraoperative blood pressure and variability in heart rate and blood pressure when compared to general anesthesia [36].

However, later Ghanami et al. [37] did observational analysis of 5642 patients to evaluate the effects of regional versus general anesthesia for infrainguinal bypass. The study showed no evidence to support the systematic avoidance of general anesthesia for lower extremity bypass procedures. In particular, graft thrombosis was found in 7.3% of patients, with an equal rate in both groups. Pulmonary morbidity occurred in 4% of patients and the rate of cardiovascular complications was 2.8% of general anesthesia patients and 2.2% of regional anesthesia patients. Venous thromboembolism rates were similar. These data suggest that anesthetic choice should be governed by local expertise and practice patterns.

Although neuraxial techniques confer some protection in the reduction in the rate of thromboprophylaxis as eluded in the PIRAT trial and large study by Singh et al., anticoagulant therapy has a major role in the in the maintenance of vascular graft patency in the perioperative period. Since anticoagulation has an important role in the decision making for neuraxial anesthesia, it is important to review the 2010 American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Third Edition) on regional anesthesia in patient receiving anticoagulant therapy and compare them with the latest guidelines published in Regional Anesthesia Pain Medicine 2015 [38]:

1. Combining neuraxial techniques with intraoperative anticoagulation with heparin during vascular surgery is acceptable with the following recommendations (Grade 1A):
   (a) Avoid the technique in patients with other coagulopathies.
   (b) Delay heparin administration for 1 h after needle placement.
   (c) Remove indwelling neuraxial catheters 2–4 h after the last heparin dose and assess the patient's coagulation status; re-heparin 1 h after catheter removal.
   (d) Monitor the patient postoperatively to provide early detection of motor blockade and consider use of minimal concentration of local anesthetics to enhance the early detection of a spinal hematoma.
   (e) Currently, insufficient data and experience are available to determine if the risk of neuraxial hematoma is increased when combining neuraxial techniques with the full anticoagulation of cardiac surgery. We suggest postoperative monitoring of neurologic function and selection of neuraxial solutions that minimize sensory and motor block to facilitate detection of new/progressive neurodeficits.

   Although the occurrence of a bloody or difficult neuraxial needle placement may increase risk, there are no data to support mandatory cancelation of a case. Direct communication with the surgeon and a specific risk-benefit decision about proceeding in each case is warranted.

2. There are no current contraindications to using neuraxial techniques in patients on subcutaneous heparin prophylaxis twice daily. Since there is no apparent difference between twice-daily subcutaneous unfractionated Heparin (UFH) with concurrent use of compression devices and thrice-daily subcutaneous UFH, it is advised that patients *not* receive three times a day of subcutaneous UFH while epidural analgesia is maintained. Rather, such patients can continue to be treated with twice-daily subcutaneous UFH and the use of compression devices.
3. Because heparin-induced thrombocytopenia may occur during heparin administration, we recommend that patients receiving heparin for more than 4 days have a platelet count assessed before neuraxial block and catheter removal.
4. For patients on low-molecular-weight heparin (LMWH), needle placement should occur at least 12 h after the last thromboprophylactic dose of LMWH and at least 24 h after the last therapeutic dose (enoxaparin 1 mg/kg every 12 h, enoxaparin 1.5 mg/kg daily, dalteparin 120 U/kg every 12 h, dalteparin 200 U/kg daily, or tinzaparin 175 U/kg daily).
5. In patients administered a dose of LMWH 2 h preoperatively (general surgery patients), we recommend against neuraxial techniques because needle placement would occur during peak anticoagulant activity.
6. The presence of blood during needle and catheter placement does not necessitate postponement of surgery. We suggest that initiation of LMWH therapy in this setting should be delayed for 24 h postoperatively and that this consideration be discussed with the surgeon.
7. Warfarin therapy should be discontinued 4–5 days before block placement, and coagulation status should be checked.
8. Clopidogrel should be discontinued for 7 days and ticlopidine for 14 days prior to neuraxial anesthesia.
9. In a patient on oral anticoagulation with warfarin, discontinue oral anticoagulation and verify PT normalization before neuraxial block. Monitor the PT and INR daily. Remove indwelling neuraxial catheters when the INR is <1.5 in order to assure that adequate levels of all vitamin-K-dependent factors are present.
10. In a patient on Fondaparinux, until additional clinical information is obtained, neuraxial techniques should be performed and managed under conditions utilized in clinical trials (single needle pass, atraumatic needle placement, and avoidance of indwelling neuraxial catheters). If this is not feasible, an alternate method of prophylaxis should be utilized.
11. While ASRA guidelines from 2010 provide no contraindication to performance of neuraxial blocks in patients taking ASA and NSAIDs, there are specific guidelines for high-risk (interventional pain) procedures as per guidelines published in 2015 in Regional Anesthesia and Pain Medicine. At our institution we perform neuraxial blocks routinely on patients taking ASA and NSAIDs without stopping either of them. Please examine recommendations from 2015 closely and refer to Table 3.1.

Although regional anesthesia (spinal and epidural anesthesia) has desirable effects, there is no sufficient data to recommend regional anesthesia over general anesthesia. With the advent of new anesthetic agents, general anesthesia can be

Table 3.1 Recommended time intervals for commonly prescribed anticoagulants

Drug	Half-life	Time of discontinuation	Time of resumption after pain procedure (h)
Coumadin	36–42 h	5 days and NR normalization	24
IV heparin	60–90 min	4 h	2
Subcutaneous heparin BID/TID	60–90 min	8–10 h	2
LMWH	4.5 h, but prolonged in renal failure	24 h	24
Fondaparinux	21 h	4 days	24
Darbigatron	8–17 h	4–5 days	24
Rivaroxaban	9–13 h	9–13 h	24
Apixaban	15 ± 8.5 h	3–5 days	24
Clopidogrel	6 h	7 days	24
Prasugrel	2–15 h	7–10 days	24
Acenocoumarol	11 h	3 days and INR normalization	24
ASA	6–20 h	6 days (primary prophylaxis) for high-risk procedure	24
NSAIDS	Variable	5 half-lives for high-risk procedures	24

Adapted from Ref. [39]

safely used with attention to detail throughout the perioperative period and aggressive management of hemodynamic changes.

It is clearly evident that severe pain after amputation is clearly associated with a higher prevalence of post-amputation pain [39, 40]. While there are numerous studies available showing decrease in incidence of phantom limb pain (PLP) [41, 42] with perioperative epidural analgesia, there are studies [43] which refute this observation. In a recent study by Karanikolas et al. [44,] optimized epidural analgesia or intravenous PCA, starting 48 h preoperatively and continuing for 48 h postoperatively, decreases PLP at 6 months.

3.2 General Anesthesia

All other major vascular procedures are performed under general anesthesia. In this section we will discuss preoperative assessment, pathophysiology, and pharmacology relevant to geriatric anesthesia and then in various subsections we will discuss vascular surgery-specific anesthesia management.

3.2.1 Preoperative Assessment

Patients undergoing peripheral and major vascular surgery constitute a particular challenge, as these patients have high prevalence of significant coronary artery disease. The usual symptomatic presentation for coronary artery disease in geriatric patients with vascular disease may be obscured by exercise limitations imposed by advanced age, intermittent claudication, or both. Perioperative hemodynamic changes like increases in blood pressure and heart rate, elevated preload, increased contractility, hypotension, tachycardia, anemia, and hypoxemia can predispose to myocardial ischemia, which is more pronounced in patients with underlying coronary disease.

The current standards for preoperative cardiac evaluation of these patients are the guidelines published by the American College of Cardiology (ACC) and these were revised in 2007 [45] and again in 2014 [46].

The 2007 Guidelines defined cardiac risk as combined incidence of cardiac death and nonfatal myocardial infarction and stratified it into low, intermediate (including carotid endarterectomy), and high risk (surgery for peripheral vascular diseases, aortic and other major vascular surgeries). In the absence of active cardiac conditions in a patient undergoing low-risk surgery, there was no indication for any further testing. The 2007 guidelines recommended that in patients undergoing intermediate risk or vascular surgery procedure, the presence of clinical risk factors determine further approach if their functional capacity was unknown or less than 4METS. Further invasive testing in patients undergoing vascular surgery should be considered only if it will change management.

The 2014 ACC guideline states that because recommendations for intermediate- and high-risk procedures are similar, classification into two categories, namely, low and elevated risk, simplifies the recommendations without loss of fidelity. A low-risk procedure is one in which the combined surgical and patient characteristics predict a risk of a major adverse cardiac event (MACE) of death or myocardial infarction (MI) of <1%. The lowest-risk operations are generally those without significant fluid shifts and stress. Plastic surgery and cataract surgery are associated with a very low risk of MACE. Procedures with a risk of MACE of >1% are considered elevated risk. Operations for peripheral vascular disease and aortic surgeries are generally performed among those with the highest perioperative risk. Some operations can have their risk lowered by taking a less invasive approach. For example, open aortic aneurysm repair has a high risk of MACE that is lowered when the procedure is performed endovascularly. In addition, performing an operation in an emergency situation is understood to increase risk.

A risk calculator has been developed that allows more precise calculation of surgical risk, which can be incorporated into perioperative decision making. The three most commonly used tools to calculate MACE risk are Revised Cardiac Risk Index (RCRI), American College of Surgeons National Surgical Quality Improvement Program (NSQIP), Myocardial Infarction and Cardiac Arrest (MICA), and American College of Surgeons NSQIP Surgical Risk Calculator.

The RCRI is a simple, validated, and accepted tool to assess perioperative risk of major cardiac complications (MI, pulmonary edema, ventricular fibrillation or primary cardiac arrest, and complete heart block). It has six predictors of risk for

major cardiac complications, only one of which is based on the procedure—namely, "Undergoing suprainguinal vascular, intraperitoneal, or intra thoracic surgery". A patient with zero or one predictor(s) of risk would have a low risk of MACE. Patients with >2 predictors of risk would have an elevated risk for adverse major cardiac events (MACE).

In a nutshell, 2014 ACC guidelines recommend that in a patient with known clinical risk factors for CAD scheduled for nonemergent elevated risk surgery (MACE > 1) and with poo r(<4METS) or unknown functional capacity, further pharmacological cardiac testing should be ordered if it will impact decision making or perioperative care. This follows a similar theme as 2007 guidelines.

It is also very important to note that implementation of the American College of Cardiology/American Heart Association guidelines has been associated with better perioperative outcomes.

Preoperative testing should not be determined by patient age alone [47]. Clinical yield of undirected or "routine" preoperative testing protocols is extremely low [48, 49]. Undirected or routine preoperative chest radiographs are unnecessary in elderly surgical patients.

Since older surgical patients are slightly more likely to be anemic, a complete blood count is mandatory for all vascular surgeries.

The prothrombin time (PT) and partial thromboplastin time (PTT) appear to have no value as screening tests in asymptomatic patients of any age with no evidence of liver disease and not taking anticoagulants. However, since most vascular surgery patients are on anticoagulants, a baseline measure of PT/PTT is required especially if planning spinal or epidural anesthesia.

Since many elderly patients may have concomitant renal dysfunction and diastolic dysfunction, a basic blood chemistry is prudent in the management of intraoperative fluid therapy.

ECG appears to be sufficiently cost-effective to warrant routine application in a geriatric population [50]. As per ACC guidelines of 2014, routine preoperative resting 12-lead ECG is not useful for asymptomatic patients undergoing low-risk surgical procedures. Since EG carries baseline information and is a prognostic standard, it is reasonable for patients with known coronary heart disease, significant arrhythmia, peripheral arterial disease, cerebrovascular disease, or other significant structural heart disease. There is poor concordance across different observational studies as to which abnormalities have prognostic significance including arrhythmias, pathological Q-waves, LV hypertrophy, ST depressions, QTc interval prolongation, and bundle-branch blocks. Likewise, the optimal time interval between obtaining a 12-lead ECG and elective surgery is unknown. General consensus suggests that an interval of 1–3 months is adequate for stable patients.

As per AHA guidelines it is reasonable to get an ECHO in the following patients:

Class IIa, dyspnea of unknown origin; Class IIa, known Congestive heart failure (CHF) with worsening dyspnea or other change in clinical status. Echo may be considered (Class IIb), reassessment in stable patients with previously documented LV dysfunction if not assessed within 1 year.

Exercise testing for ischemia may be considered (Class IIb) for patients with elevated risk and unknown or poor (<4METS) functional capacity if it will change

management. However, it is to be noted that vascular surgery patients may not be able to do exercise testing due to concomitant claudication. Patients able to achieve approximately 7 METs to 10 METs have a low risk of perioperative cardiovascular events, and those achieving <4 METs to 5 METs have an increased risk of perioperative cardiovascular events. Electrocardiographic changes with exercise are not as predictive.

Noninvasive pharmacological testing may be reasonable (Class IIa) for patients at elevated risk and have poor (<4 METs) functional capacity to undergo noninvasive pharmacological stress testing (either dobutamine stress echocardiogram (DSE) or pharmacological stress (MPI) if it will change management (Level of Evidence: B) The authors identified a slight superiority of stress echocardiography relative to nongated MPI with thallium in predicting postoperative cardiac events. In patients with abnormalities on their resting ECG for example: left bundle-branch block, LV hypertrophy with "strain" pattern, digitalis effect, concomitant stress imaging with echocardiography or MPI may be an appropriate alternative.

As per AHA guidelines consistent and clear associations exist between beta blocker administration and adverse outcomes, such as bradycardia and stroke. Beta blockers should be continued in patients undergoing surgery who have been on beta blockers chronically. In patients with intermediate- or high-risk myocardial ischemia noted in preoperative risk stratification test for instance three or more RCRI risk factors (e.g., diabetes mellitus, HF, CAD, renal insufficiency, and cerebrovascular accident), it may be reasonable to begin perioperative beta blockers 2–7 days before surgery. They recommend against starting beta blockers on the day of surgery in beta–blocker-naïve patients.

Perioperative initiation of statin use is reasonable in patients undergoing vascular surgery and statins should be continued in patients currently taking them.

The risk of coronary stent thrombosis in the perioperative period for both bare metal stent (BMS) and drug-eluting stent (DES) is highest in the first 4–6 weeks after stent implantation. Discontinuation of dual antiplatelet therapy (DAPT), particularly in this early period, is a strong risk factor for stent thrombosis. In patients undergoing urgent noncardiac surgery during the first 4–6 weeks after BMS or DES implantation, DAPT should be continued unless the relative risk of bleeding outweighs the benefit of the prevention of stent thrombosis. As such, use of DAPT or aspirin alone should be individualized on the basis of the considered potential benefits and risks. All elective surgeries should be delayed for minimum 30 days for BMS and 365 days for DES.

3.2.2 Geriatric Physiology

3.2.2.1 Cardiac Physiology

Changes in the cardiovascular system that accompany aging include decreased vascular and myocardial compliance due to fibrotic replacement of elastic tissues of the arteries and the ventricle. This leads to hypertension and diastolic dysfunction.

Due to hypertension there is progressive and sustained increase in left ventricular wall tension and myocardial workload resulting in symmetrical ventricular hypertrophy and increased ventricular mass. This can further complicate diastolic dysfunction.

The phases of diastole are isovolumic relaxation and the filling phase. The filling phase is divided into early rapid filling which is passive(70–80%), diastasis(5%), and atrial systole(15–25%).Early diastolic filling is driven by the left atrial (LA) to left ventricular (LV) pressure gradient.

The cause of diastolic dysfunction is that the stiffer ventricle and atrium do not permit complete chamber relaxation until relatively late in diastole. In elderly there is decreased early diastolic filling because of decreased LA-LV pressure gradient caused by impaired LV relaxation.

Consequently, passive ventricular filling, which occurs during the early phase of diastole, is significantly reduced in older adults. As a result, the elderly are particularly dependent on the synchronous atrial contraction of sinus rhythm for late ventricular filling and this also explains why cardiac rhythm other than sinus is often poorly tolerated in elderly individuals.

There is decrease in autonomic responsiveness namely there is increased vagal tone, and decreased sensitivity of adrenergic receptors leads to a decline in heart rate. Fibrosis of the conduction system and loss of sinoatrial node cells increase the incidence of dysrhythmias, particularly atrial fibrillation and flutter. In the absence of co-existing disease, resting systolic cardiac function seems to be preserved. The stiffer ventricle and atrium do not permit complete chamber relaxation until relatively late in diastole. Consequently, passive ventricular filling, which occurs during the early phase of diastole, is significantly reduced in older adults, producing a form of diastolic dysfunction. As a result, the elderly are particularly dependent on the synchronous atrial contraction of sinus rhythm for complete ventricular filling.

The elderly patient with diastolic dysfunction may poorly tolerate perioperative fluid administration, resulting in elevated left ventricular end-diastolic pressure and pulmonary congestion. Decrease venous capacitance due to stiffening reduces its ability to buffer changes in intravascular volume leading to exaggerated hypotension especially during induction of general anesthesia or spinal anesthesia.

Moderate hypotension can cause intolerable reduction in coronary, cerebral, and renal blood flow. Decreased β-receptor response in the elderly during exercise/stress cause the increased peripheral flow demand to be met primarily by preload reserve, thereby making the heart more susceptible to cardiac failure. As baseline and maximal achieved heart rate is limited, elderly patients will rely on alpha agonists to maintain blood pressure during moments of hypotension.

In older individuals, exercise-induced increases in cardiac output are achieved with a lower heart rate, higher EDV, and higher stroke volume.

3.2.2.2 Respiratory Physiology

Aging decreases the elasticity of lung tissue, allowing overdistention of alveoli and collapse of small airways. Residual volume, functional residual capacity increase with aging along with increased anatomic dead space, increased closing capacity, decreased diffusing capacity all leading to impaired gas exchange. Elderly have less

complaint chest wall. The elderly are more prone to respiratory impairment in the recovery after general anesthesia.

3.2.2.3 Nervous System Physiology

With aging there is gray and white matter atrophy, synaptic degeneration. The synthesis of neurotransmitters like acetylcholine and dopamine is reduced. Serotonergic, adrenergic, and gamma-aminobutyric acid-binding sites are also reduced. These changes may be responsible for increased sensitivity of elderly to general anesthetic and local anesthetics.

Such changes may also lead to age-related cognitive and behavioral deficits, and contribute to postoperative cognitive dysfunction in the elderly.

3.2.2.4 Renal and Hepatic Physiology

With normal aging there is progressive decrease in creatinine clearance but since muscle mass also decreases, serum creatinine remains relatively unchanged with aging. Therefore, serum creatinine is a poor predictor of renal function in elderly. Calculated creatinine clearance remains the most sensitive marker of renal function in the elderly.

Critical attention should be placed to perioperative fluid balance and electrolyte imbalance. As renal function declines with aging, the kidney's ability to excrete drugs also declines. The decreased capacity to handle water and electrolyte loads makes proper fluid management more critical during major vascular surgery.

Hepatic blood flow decreases by 10% per decade. Liver's ability to metabolize certain drugs also decreases with age. The rate of biotransformation and albumin production decreases. Plasma cholinesterase levels are reduced in elderly men.

3.2.3 Geriatric Pharmacology

Pharmacokinetic implies the relationship between drug dose and plasma concentration Pharmacodynamics implies the relationship between plasma concentration and clinical effect.

In older patients subtle changes in *pharmacodynamics* and altered age-related alpha phase redistribution *pharmacokinetics* are responsible for varied drug effect.

With aging [1], lean body mass decreases [2], body fat increases [3], and total body water decreases.

The reduced volume of distribution for water-soluble drugs can lead to greater plasma concentrations after rapid bolus or infusions. Conversely, an increased volume of distribution (due to increase in body fat) for lipid-soluble drugs could reduce their plasma concentration but lead to larger volume of distribution after prolonged infusions leading to increased drug effect. It is interesting to note that the decreased dose requirement of fentanyl in the elderly has a pharmacodynamic explanation, that is, elderly brain is more sensitive to opioids [51].Thus pharmacodynamics basis, increased brain sensitivity explains decreased minimum alveolar

concentration (MAC) of volatile anesthetics [52, 53], decreased dosing requirement of opioids [54] and benzodiazepines [55].

The prolonged duration of action of vecuronium [56] and rocuronium [57] in the elderly surgical patients is related to altered pharmacokinetics consistent with an age-related decrease in renal and hepatic functions. Recovery from pancuronium that depends on renal excretion may be delayed due to decreased drug clearance. Hofmann elimination, an organ-independent elimination pathway, occurs in plasma and tissue, and is responsible for approximately 77% of the overall elimination of cisatracurium besilate. Therefore, it provides most consistent clinical effects in the elderly. Proper neuromuscular monitoring with meticulous attention to train of four and reversal of neuromuscular blockade along with adherence to clinical criteria for extubation must be met prior to extubation of elderly patients. Complete recovery of neuromuscular function is more likely when anticholinesterases are administered early (>15–20 min before tracheal extubation) and at a shallower depth of block (train-of-four [TOF] count, 4) [58].

3.2.4 Anesthetic Management of Abdominal Aortic Aneurysm Repair

In a large US Veterans Affairs screening study, the prevalence of abdominal aortic aneurysm (AAA) was 1.4% [59].

Abdominal aortic aneurysms were the primary cause of *10,597 deaths* and a contributing cause in more than *17,215 deaths* in the United States in 2009 [60]. AAA repair involves the replacement or bypass of an aneurysmal section of abdominal aorta. There are two primary methods of AAA repair, open repair and endovascular repair (EVAR). Open AAA repair is well established as a definitive treatment, having been in use for over 50 years. Generally, EVAR is advocated for patients who are at increased risk with open repair.

3.2.4.1 Anesthetic Technique for Endovascular Repair

Many institutions initially performed endovascular surgery under general anesthesia. For both the surgeons and anesthesiologists, this was a natural choice due to the uncertain outcomes and possible complications related to the new procedure.

For the transfemoral approach, local anesthesia is well tolerated and provides greater hemodynamic stability than other anesthetic techniques. Henretta et al. [61] reported the first ever series that described the use of local anesthesia for the endovascular repair of infrarenal AAAs in patients with significant co-morbidities. They showed that the advantages of local anesthesia include decreased cardiopulmonary morbidity rates, shorter hospital stays, and lower hospital costs. Multiple other reports have shown decreased procedure times [62, 63], shorter hospital stays [62, 63], and fewer pulmonary complications [64] when local anesthesia is used in place of general anesthesia.

Spinal, epidural, and combined spinal–epidural techniques have been used for endovascular surgery especially with an iliac approach to EVAR. The sensory level at which anesthetic blockade is needed is T10 dermatome. The level of sensory

anesthesia required for endovascular surgery has fewer hemodynamic side effects than the high thoracic level needed for open surgical repair.

Aadahl et al. [65] showed that a single dose of spinal anesthesia combined with epidural anesthesia was effective for EVAR with no clinically significant period of hypotension in any case.

EVAR requires brief periods of intermittent apnea to obtain optimal imaging quality in digital subtraction angiography. Therefore, patient cooperation is essential, and a fine balance between optimal sedation and alertness when needed is essential. In patients with back pain or dementia, it may not be possible to maintain such a response, leading to anesthesiologist and surgical preference for general anesthesia.

The key elements in anesthetic management of EVAR include adequate hemodynamic monitoring with arterial line along with standard ASA monitors. Large-bore intravenous access should be obtained given the potential for significant blood loss and especially if a conversion to open surgery is indicated. Central venous access is not routinely required unless indicated by a patient's cardiac function or if a lengthy procedure is planned. General anesthesia typically consists of a balanced technique with a low-dose inhalational agent and opioids. Neuromuscular blocking agents are typically not necessary. A Foley catheter is required as a measure of volume status. Temperature should be closely monitored as patient is exposed and prepped for an open procedure if needed.

Blood loss during a simple infrarenal EVAR is usually minimal approximately 200–600 ml, and intraoperative transfusion is rare. Prolonged procedures and complex repairs have potential for ongoing blood loss from the access sites. Cell salvage should be available and should be used in long procedures due to propensity of extended blood loss. The occurrence of sudden hypotension should prompt immediate evaluation of access sites, followed by angiography to identify any possible causes of bleeding. It should be realized that blood loss can be difficult to quantify, as it is often lost around the sheaths and catheters, and can be retroperitoneal in the case of injury to femoral or iliac vessels. Since the incidence of renal failure is about 6.7% with EVAR [66], close intraoperative fluid management with early replacement of preoperative deficits and maintenance of intravascular volume is extremely essential during surgery especially because the surgery involves extensive use of iodinated contrast. Proper fluoroscopic protection should be provided to all personnel involved in the care of the patient during the procedure.

Postoperative patients can be discharged to a monitored bed but typically do not need intensive care. Analgesic requirements are minimal and rarely require intravenous opioids.

3.2.4.2 Anesthetic Management for Open AAA Repair

The key elements in anesthetic management of open AAA repair include adequate hemodynamic monitoring with arterial line along with standard ASA monitors (ECG, noninvasive blood pressure, pulse oximetry, capnography, and temperature). Arterial line is usually placed pre-induction in the radial artery of the arm which records he highest blood pressure via sphygmomanometer. At our institution we

also place a T8-10 thoracic epidural catheter for postoperative pain control. Nishimori et al. [67] found epidural superior to intravenous analgesia, with improved postoperative pain scores as well as a reduction in postoperative intubation times, acute respiratory failure rates, intensive care unit stay duration, and rates of cardiac, gastrointestinal, and renal complications. Central venous catheterization is performed after induction general anesthesia unless needed pre induction, as dictated by patient's cardiovascular status. Pulmonary artery catheterization (PAC) is preferred over Central venous catheterization (CVC) in patients with LV dysfunction (ejection fraction < 30%) and pulmonary hypertension. PACs with capability to monitor mixed venous oxygen saturation and continuous cardiac output may be helpful in hemodynamically unstable patients or patients with ruptured AAA.

Transesophageal echocardiography (TEE) can be used to evaluate regional wall motion abnormalities which may be indicative of ischemia. TEE also helps to accurately assess volume status in a hypotensive patient. Cardiac output measured by TEE correlates very well with thermodilution cardiac output derived from PAC in the absence of significant mitral regurgitation. TEE can be placed quickly if patients become hemodynamically unstable and can immediately provide information about ventricular function (acute myocardial infarction or pulmonary embolus), volume status (hypovolemia), or obstructive flow patterns (cardiac tamponade, LVOT). Rapid rescue [68] TEE has shown to improve outcomes after hemodynamic instability in noncardiac surgical patients and also provides additional diagnostic information in patients with intraoperative cardiac arrest which may directly guide specific, potentially life-saving therapy [69].

Anesthetic technique consists of a balanced technique with inhalational anesthetics and opioids. Agents available for blunting hemodynamic response, such as esmolol, sodium nitroprusside, nitroglycerin, and short-acting b-blockers such as esmolol, should be available for bolus and continuous infusion administration, as needed. Also a vasopressor like phenyepherine or norepinephrine should be available to counteract hypotension especially during unclamping. Since an epidural catheter is placed pre-induction, we typically avoid using long-acting opioids and activate the epidural catheter at the end of the procedure to avoid any hemodynamic consequences for epidural local anesthetic during the surgery. Depending on the location of the lesion, the cross-clamp can be applied to the supraceliac, suprarenal, or infrarenal aorta. Heparin is usually administered prior to aortic clamping. The effects of cross-clamping depend on the level of the clamp, patient's fluid status and baseline myocardial function. Patients with preexisting left ventricular dysfunction manifest more hemodynamic consequences than those with normal LV function.

The primary hemodynamic response [70] to aortic cross-clamping is an increase in mean arterial pressure due to an increase in afterload. Cardiac output often decreases in response to aortic cross-clamping. Preload changes as per the location of the clamp. If the aorta is clamped above the celiac artery, blood volume is shifted proximally to the clamp, the thereby increasing preload and blood flow to the lungs and the cranium.

During infraceliac clamping, the change in preload depends upon the tone of the splanchnic veins. If the splanchnic vascular tone is high, venous return to the heart increases. Alternatively, if splanchnic venous tone is low, a decrease in preload occurs as blood volume shifts into the compliant splanchnic vasculature. Due to increase in preload and after load, left ventricular decompensation can occur in patients with CAD and LV dysfunction. The blood flow to organs distal to the clamp depends on the flow from collateral vessels which depends on the perfusion pressure dictated by the proximal aortic pressure. Therefore, proximal hypotension should be avoided.

It has been shown [71] that infraceliac clamping produces minimal mean arterial pressure, ventricular filling pressures, and ejection fraction. In contrast, supraceliac clamping produces significant increases in proximal mean arterial pressure and filling pressures, and a decrease in cardiac ejection fraction and segmental left ventricular wall motion abnormalities.

The hemodynamic response to clamping and unclamping is less prominent in patients with aortic occlusive disease than in patients getting AAA repair [72].

Before aortic unclamping, the patient should be prepared for the side effects of reperfusion. The primary hemodynamic response to unclamping of the aorta is significant hypotension. The causes include significant decrease in afterload after clamp release, accumulation and release of vasodilating and myocardial depressant metabolites from the ischemic lower extremities, peripheral redistribution of blood volume into a vascular bed that is often vasodilated by hypoxia resulting in central hypovolemia.

Blood and fluid loss should be replaced before unclamping and patient's volume status should be optimized with blood, albumin, or crystalloid, based on patient's hemodynamics which will take in to consideration their CVP, BP, and TEE (if placed) to identify hypovolemia vs LV dysfunction as cause of hypotension. Epinephrine, phenylephrine, sodium bicarbonate, and calcium chloride should be available just before the release of the cross-clamp. The aortic cross- clamp can be gradually released and reapplied if significant hypotension occurs. Correction of metabolic acidosis with sodium may be required. Small boluses of neosynephrine, nor-epinephrine, or even epinephrine may be needed to correct significant hypotension but it should be remembered that correction of hypovolemia takes precedence as using vasopressors to increase the blood pressure without restoring blood volume may further decrease blood flow to coronary, renal, and hepatic circulations.

The incidence of renal failure is approximately 13% after suprarenal (SR) aortic cross-clamping and 5% after infrarenal (IR) clamping [73]. In a review of 1020 patients who underwent elective AAA repair, postoperative decline in renal function was 17.0% in SR vs 9.5% in IR ($P = .003$), however, new-onset dialysis was rare (0.6% SR, 0.8% IR, P = NS) [74]. The reason for the above changes is that aortic cross-clamping increases renal vascular resistance and decreases renal cortical blood flow. The degree of change does not correlate with changes in blood pressure or cardiac output. Gamulin et al. [75] showed an increase of 75% in renal vascular resistance and a decrease of 38% in renal blood flow after IR clamping, whereas systemic cardiovascular measurements did not change appreciably.

Anesthetic drugs including mannitol, ventilator parameters, pre-clamp blood volume were maintained in all. Suprarenal cross-clamping has been shown to reduce renal blood flow by 80%.

Urine output is routinely monitored intraoperatively; however, it does not predict the development of postoperative renal failure. A Cochrane database [76] review showed that there is not enough evidence to show the best fluid replacement to use during and following surgery on the abdominal aorta. Fluid replacement is needed to replace tissue fluids lost during surgery. Blood products, nonblood products, or combinations including crystalloid solutions and colloids are used. Combination therapy is most common. The incidence of postoperative renal insufficiency may be decreased by adequate volume loading, maintaining cardiac output, and aggressively treating hypovolemia based on hemodynamic parameters like CVP, cardiac output, or TEE imaging and reduce cross-clamp timing (<30 min [77] associated with minimal risk).

The incidence of spinal cord injury due to hypoperfusion or ischemia is rare in AAA repair, the incidence being higher in ruptured AAA repair. Three spinal arteries, one anterior and a pair of posterior spinal arteries from the vertebral arteries, supply the cord. The anterior spinal artery is the principal artery of the three [78], supplying the anterior two-thirds of the cord, including the critical motor area. Segmental arteries from subclavian, intercostal, upper lumbar, and branches from the internal iliac and middle sacral arteries regularly feed the anterior spinal artery. The largest of these segmental arteries is the great radicular artery of Adamkiewicz or arteria radicularis magna, which originates as a branch from a left intercostal artery between T9 andT12 in 75% of patients, T5 and T8 in 15%, and L1 and L2 in 10%. The injury to the anterior 2/3rd of the cord results in bilateral flaccid paraplegia and loss of pain and temperature sensation; proprioception and vibratory sensation is maintained.

The incidence of spinal cord damage was reported as 0.25% after abdominal aortic operations and the variation in origin of the artery of Adamkiewicz may explain the incidence of this complication [79].

Epidural opioids decrease incidence of atelectasis and epidural local anesthetics increase PaO2, decrease the incidence of pulmonary infections, pulmonary complications overall compared with systemic opioids [80]. Pain management at our institution is usually via a thoracic epidural catheter which is activated at the end of the procedure to avoid hemodynamic consequences from epidural bupivacaine. We use a dilute concentration of bupivacaine 0.0625% or 0.125% mixed with fentanyl 5 mcg/cc.

The other options for pain management include bilateral paravertebral catheters and bilateral transversus abdominis plane block.

3.3 Surgery on the Ascending Aorta and the Arch of Aorta

Surgery on the ascending aorta and the arch of the aorta uses median sternotomy and cardiopulmonary bypass. The conduct of anesthesia is similar to that for cardiac surgery involving cardiopulmonary bypass which is beyond the scope of this chapter.

3.4 Surgery Involving Thoracic Aortic Abdominal Aneurysm

Aneurysms of the TAAA aorta are primarily caused by atherosclerotic degenerative disease. The remainder can be caused by trauma or connective tissue diseases disorders such as Marfan syndrome, cystic medial degeneration, Takayasu arteritis, syphilitic aortitis, Turner's syndrome, polycystic kidney disease, and Loeys-Dietz syndrome.

The prevalence of TAAA is much less than that of infrarenal AAA. TAAAs have a much lower incidence of CAD, often cited as less than 30%.

The Crawford classification defines aneurysms as types I, II, III, and IV. Type I aneurysms involve all or most of the descending thoracic aorta and the upper abdominal aorta. Type II aneurysms involve all or most of the descending thoracic aorta and all or most of the abdominal aorta. Type III aneurysms involve the lower portion of the descending thoracic aorta and most of the abdominal aorta. Type IV aneurysms involve all or most of the abdominal aorta, including the visceral segment. Types II and III are the most difficult to repair because they involve both the thoracic and the abdominal segments of the aorta.

The anesthesia for open thoracoabdominal aneurysm (TAA) repair involves placement of cerebrospinal fluid drain, thoracic epidural catheter insertion for postoperative analgesia, general anesthesia with lung isolation (double lumen tube or bronchial blocker), placement of multiple large-bore (14 gage or smaller) IV catheters, arterial lines in the upper (radial artery) and lower (femoral artery) extremities, double lumen central venous catheter insertion, pulmonary artery catheterization, and transesophageal echocardiography for monitoring of hemodynamics. TEE is used to evaluate ventricular volumes, ventricular function, valvular abnormalities (regurgitation and stenosis), and optimize cardiac function during clamping and unclamping. Temperature monitoring at multiple sites using PA catheter, urinary catheter, and nasopharyngeal probe is advised.

A lumbar spinal drain is inserted before the procedure to monitor and control cerebrospinal pressure thereby maintaining spinal cord perfusion pressure during aortic occlusion and after the procedure. Moderate systemic hypothermia (34 °C or lower) is used to prolong spinal cord and organ ischemic tolerance.

One-lung anesthesia greatly facilitates surgical exposure. A left-sided double lumen tube (DLT) is usually preferred but endobronchial blocker is ideal in patients with difficult airway, as manipulation of the airway to exchange the endotracheal tube toward the end of the surgical procedure can be avoided. Placement of left-sided DLT or blocker on the left side should be avoided in patients with aneurysm compressing on the left bronchus with distortion of the anatomy. A right-sided DLT should be used in such situations.

If assisted circulation is required, a left heart bypass (LHB) [81] is needed. The goal of LHB, is to divert a portion of saturated blood from the patient's left atrium (LA) to a section of the arterial vasculature distal to the portion of the aorta that is being reconstructed (Fig. 3.1). The most common proximal cannulation site is the left inferior pulmonary vein, although the LA appendage, left ventricular (LV) apex, ascending aorta, or subclavian artery may be used. Distal cannulation is accomplished

with cannulation of the femoral artery. TEE is useful in confirming cannulae position. Once the proximal aorta is cross-clamped, the institution of LHB creates two parallel circulations, an upper and a lower. The "upper" consists of native flow from the LV and thence to the great vessels and heart. The "lower" consists of flow from the LA to the centrifugal pump, and then to the distal cannulation site (femoral artery or distal aorta) and/or any visceral vessels that have been selectively cannulated (Fig. 3.1). Lower body circulation also provides blood flow to the spinal cord anastomotic network through sacral vessels. Blood in the "lower" circulation will return to the right side of the heart primarily via the inferior vena cava (IVC). The upper circulation is dependent on the patient's underlying LV function. The lower circulation is dependent on LHB flows, which are typically 1.5–2.5 L/min. The adequacy of upper circulation is measured by the radial artery catheter and that of lower circulation by the femoral

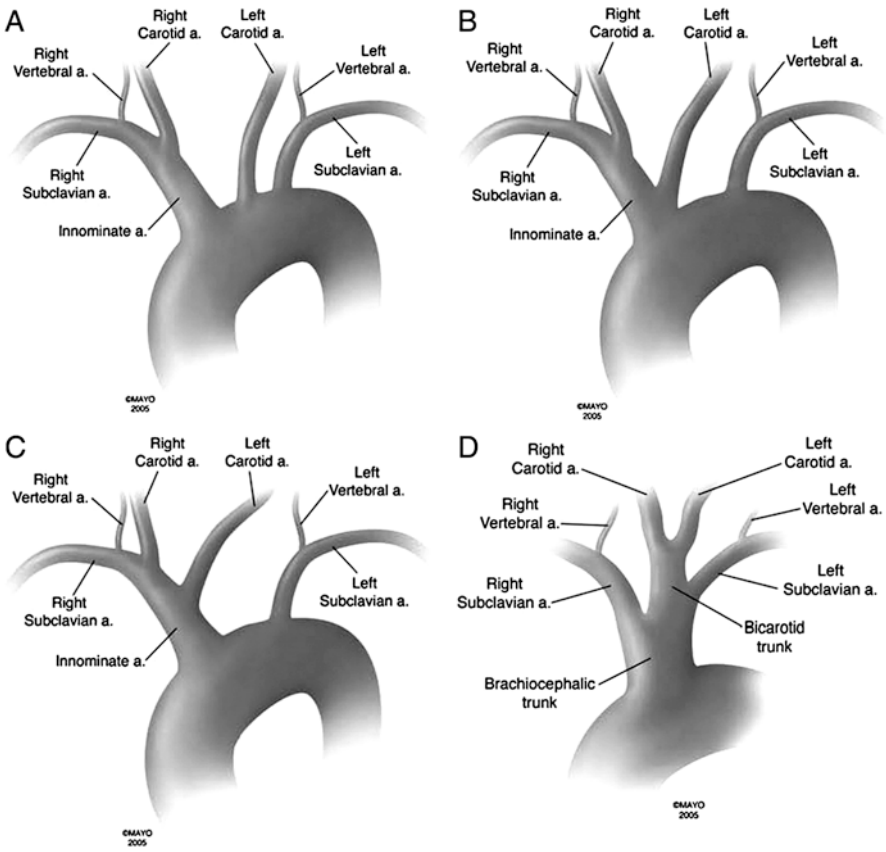

Fig. 3.3 Depiction of Left Heart Bypass. Note that blood from the left heart is shunted distal to the aortic cross-clamp via a centrifugal pump. The net effect is to create two parallel circulations: (1) an "upper" circulation to the brain and great vessels; and (2) a "lower" circulation below the aortic cross-clamp to the viscera and lower extremities (From Dwarakanath and Collard [81]; with permission)

artery catheter. Prior to LHB cannulation, the patient is first heparinized (100–150 units/kg) to achieve an activated clotting time of 200–250 s (Fig. 3.3).

Manipulation of intravascular volume, cardiac contractility, vascular tome proximal to cross- clamp and pump flows is done to optimize the upper and lower body circulation. For example, low flows in the upper circulation can be corrected by (1) volume loading the patient or (2) by decreasing flow to the lower circulation by reducing the rate of LHB flow assuming normal ventricular function. Excessive surgical bleeding and coagulopathy are not uncommon and volume resuscitation using cell saver, rapid infuser, and blood products are necessary in these patients.

Spinal cord protection during thoracic aortic surgery has been well described by Sinha and Cheung [82]. They recommend the following strategy:

1. Minimize spinal cord ischemia time:
   (a) Segmental reconstruction of the descending aorta
   (b) Distal aortic perfusion with a passive shunt (Gott shunt)
   (c) Partial left heart bypass
2. Increase tolerance to ischemia:
   (a) Deliberate mild systemic hypothermia
   (b) Deep hypothermic circulatory arrest
   (c) Selective spinal cord hypothermia by epidural cooling
   (d) Pharmacologic neuroprotection
3. Augmentation of spinal cord perfusion:
   (a) Deliberate hypertension
   (b) Lumbar cerebrospinal fluid (CSF) drainage
   (c) Reimplantation of intercostals and lumbar segmental arteries
   (d) Preservation of subclavian artery flow
4. Early detection of spinal cord ischemia:
   (a) Intraoperative motor evoked potential (MEP)
   (b) Intraoperative SEP monitoring, and serial postoperative neurologic examination

In conclusion, the goal of the anesthesiology team is to maximize tissue oxygen delivery by controlling intravascular volume, cardiac function, mean arterial pressure, in addition to maintaining spinal cord perfusion pressure by keeping adequate mean arterial pressure (MAP) and CSF pressure (below 10 cm H_2O). Transesophageal echocardiography provides real-time assessment of cardiovascular status and is vital to the intraoperative management of these patients.

3.5 Anesthesia for Carotid Endarterectomy

Atherosclerosis can involve the origins of both the internal and external carotid arteries as well as the bifurcation of the common carotid artery. The bifurcation of the common carotid artery is the most common site of atherosclerotic plaques that may lead to transient ischemic attacks (TIAs) or stroke.

The brain receives its blood supply from four major arteries. Eighty to ninety percent of the cerebral blood supply is delivered via the two internal carotid arteries with the majority of the remainder coming from the vertebrobasilar system. The carotid arteries and basilar artery unite to form the Circle of Willis at the base of the brain. This ring of arteries offers the brain considerable protection against the occlusion of one or another vessel; however, in patients with cerebrovascular disease one or more of the vessels within the circle maybe occluded by atheromatous plaque.

Carotid Endartrectomy (CEA) can reduce the risk of stroke in subgroup of patients. As per the guidelines published in the *Journal of Vascular Surgery* in 2011, CEA should be the first-line treatment for most symptomatic patients with stenosis of 50–99%. Symptomatic patients with 50–99% stenosis that are at high risk for CEA from anatomic (like prior neck surgery or radiation injury) or medical reasons should be offered carotid artery stenting.

CEA is first-line treatment for asymptomatic patients with stenosis of 60–99%. Asymptomatic patients at high risk for intervention or with <3 years' life expectancy should be considered for medical management as the first-line therapy.

According to the American Association of Neurological Surgeons and the American Stroke Association, treatment with CEA within 2 weeks of presentation for acute stroke is reasonable and appropriate.

The perioperative risk of stroke and death in asymptomatic patients must be <3% and for symptomatic patients <6%.

The risks associated with CEA involve neurological complications, hypertension, hypotension, hemorrhage, acute arterial occlusion, stroke, MI, venous thromboembolism, cranial nerve palsy, infection, arterial restenosis, and death. Risk is related mainly to the patient's preoperative clinical status. Symptomatic patients have a higher risk than asymptomatic patients, as do those with hemispheric versus retinal symptoms. Intracerebral hemorrhage may occur as a consequence of the hyperperfusion syndrome despite control of blood pressure. Cardiovascular instability has been reported in 20% of patients undergoing CEA, with hypertension reported in 20%, hypotension in 5%, and perioperative MI in 1% [83].

Most patients needing CEA are elderly with hypertension and associated CAD. Patient should be medically optimized with regard to their CAD, DM, and HTN. The patient should receive their blood pressure medications in the morning of surgery and their blood sugar should be well controlled. Standard cardiovascular monitoring should include continuous ECG with Lead II and V5 and arterial blood pressure. At our institution we prefer to place an arterial line pre-induction to keep tight control on blood pressure. Two large-bore IV catheters are placed and one of them is dedicated to running a vasodilator like nitroprusside or nitroglycerine and a vasoconstrictor like phenylephrine or norepinephrine. This surgery does not involve major fluid shifts and a central venous catheter or a pulmonary artery catheter is rarely needed. Patient's "baseline" mean arterial pressure (MAP) should be estimated from the preoperative visit, the patient's records, and the blood pressure in both arms should be measured. We aim to maintain MAP at, or up to 20% above, the documented baseline MAP during carotid cross-clamping using fluids, vasopressors, and hypotensive drugs as required The MAP are also regulated based on the

information available from EEG, other cerebral monitoring or neurological symptoms in awake patients. EEG monitoring is used during every CEA.

The carotid artery is temporarily completely occluded by a cross-clamp in order to perform the CEA. A temporary shunt may be inserted through the arteriotomy distally in the internal carotid artery and proximally in the common carotid artery to prevent cerebral hypoperfusion and impending ischemia during the cross-clamping of the carotid artery. Acute complications of shunt insertion include air or plaque embolization, intimal tears, and carotid dissection. There is an associated risk of local complications including hematoma, nerve injury, infection, and late carotid restenosis. Various modalities are used to monitor the need for shunting including EEG monitoring, SSEP, and carotid stump pressure monitoring.

In a study in 2002, it was shown that intraoperative EEG monitoring accurately (99.92%) identified patients who may safely have carotid endarterectomy without the need of a shunt. A statistically significant increase in intraoperative stroke rate was associated with the development of an abnormal EEG (1.1%), contralateral internal carotid artery occlusion (1.8%), and the combination of both abnormal EEG and contralateral internal carotid occlusion (3.3%) [84]. EEG was an excellent detector of cerebral ischemia and a valuable tool in guiding the need for shunting [85].

CEA performed with routine EEG monitoring and selective shunt placement is associated with a low risk of perioperative stroke. Identified predictors of significant EEG changes were anatomic factors including degree of contralateral carotid artery disease and moderate ipsilateral carotid artery stenosis (50–79%) [86].

Anesthetic agents and changes in temperature and blood pressure affect EEG. EEG monitoring is limited by the fact that most neurologic deficits after CEA is caused by thromboembolism rather than occlusion of blood flow during carotid clamping.

Somatosensory-evoked potentials reflect presence of intact sensory pathways from stimulated peripheral nerve to the cortex where the electrical activity is being recorded. There is evidence that distortion of these waveforms reflect ischemia, although these Somatosensory evoked potential (SSEP) changes may not reflect ischemia and may overestimate the need for shunting. SSEP may be superior in patients whose baseline EEG is not easily interpretable because of a previous stroke. Also SSEP may be affected by volatile anesthetics. In a recent study by Nwachuku et al. it was stated that patients with perioperative neurological deficits are 14 times more likely to have had changes in SSEPs during the procedure and intraoperative SSEP is a highly specific test in predicting neurological outcome following CEA [87].

The internal carotid stump pressure (pressure cephalad to the clamp) presumably reflects the pressure transmitted around the Circle of Willis. Studies have shown stump pressure to be specific but not sensitive at identifying patients who develop EEG changes consistent with cerebral ischemia upon carotid cross-clamping [88].

Transcranial Doppler (TCD) provides noninvasive assessment of the middle cerebral artery (MCA) by insonating the MCA through the thin petrous temporal bone using a specially designed Doppler probe. This helps to monitor both cerebral hemodynamics and the occurrence of emboli. However, the probe has to be

placed near the surgical site and may need constant adjustment. TCD may be used as a complement to EEG; it is to be realized that it is operator-dependent. TCD may be useful in predicting patients with cerebral hyperperfusion syndrome following CEA or CAS.

Near infrared spectroscopy (NIRS) allows continuous monitoring of regional cerebral oxygenation (rSO2) in the frontal lobe. It is not as reliable as other monitors of cerebral ischemia.

General anesthesia is the most commonly used technique at our institution. It allows reliable airway control, prevents hypoxemia and hypo- or hypercapnia, and provides optimal operating conditions for our surgical team. Induction of anesthesia should be slow with gradual titration of anesthetic drugs. Airway control should be expeditious with minimal hemodynamic alteration and avoidance of hypo or hypertension during induction and laryngoscopy. Hypercarbia causes vasodilation in normally reactive nonischemic areas of the brain and "steals" the blood away from the maximally vasodilated vessels in the territory of the occluded carotid. Hypocarbia may cause vasoconstriction of vessels in normally perfused areas of the brain and divert blood to the maximally vasodilated, unreactive areas of the brain but clinical trials have failed to show any benefit as leftward shift of oxyhemoglobin dissociation curve by hypocarbia decreases oxygen delivery to tissues. The current recommendation is to maintain normocarbia in CEA.

Maintenance of general anesthesia can be achieved with various agents as long as there is hemodynamic stability, cerebral ischemia is not enhanced and consideration is made for rapid emergence at the conclusion of surgery.

In general, volatile anesthetics are vasodilators and intravenous anesthetics are vasoconstrictors of the cerebral vasculature. Hence volatile anesthetics have the potential to cause vasodilation of normally perfused areas of brain and hence steal blood from ischemic areas. The risk of cerebral ischemia will be lessened by agents that cause decrease in cerebral metabolic oxygen consumption (CMRO2). Sevoflurane, desflurane, and isoflurane decrease CMRO2.

In a study, it was shown that times to extubation, movement on command, and consciousness were shorter after desflurane and sevoflurane than after isoflurane anesthesia; it was also noted that desflurane was associated with more hypertension and tachycardia [89].

Michenfelder et al. [90] defined the critical regional cerebral blood flow (rCBF) as the rCBF below which more than 50% of patients developed ipsilateral EEG changes of ischemia within 3 min of carotid occlusion. The critical rCBF varies depending on the volatile anesthetic used.

EEG monitoring for cerebral ischemia is feasible with 0.6–1.2% sevoflurane administered in 50% nitrous oxide, and it is similar to that determined with isoflurane and it may facilitate more rapid emergence [91].

Due to the advantages of the rapid emergence with sevoflurane, it appears to be a good alternative to isoflurane in CEA. We prefer to use remifentanil (0.05–2 mcg/kg/min) with sevoflurne or isoflurane in 0.5–1.0 MAC. There is evidence that cerebral autoregulation is impaired even with concentrations of volatile anesthetic agents <1.0 MAC, although this effect is more marked with isoflurane than with

sevoflurane. Which is another potential advantage of sevoflurane. Some centers avoid nitrous oxide as it increases the cerebral metabolic rate, increases cerebral blood flow and decreases the neuroprotective effect of other drugs. Anesthesia can be maintained using a combination of propofol and remifentanil. Propofol and sevoflurane at a concentration up to 1.0 minimum alveolar concentration produce comparable reductions in both the cerebral blood flow and the metabolic rate. Observations also indicate a hemodynamic advantage of propofol anesthesia during carotid clamping [92]. However, at a higher cost of propofol-remifentanil anesthetic it may offer little advantage over inhalational anesthesia for carotid endarterectomy [93]. It is to be noted that both propofol and volatile anesthetics afford neuroprotection during CEA, and anesthetic-induced neuroprotection [94] is an important research topic currently.

Surgical manipulation, traction of the carotid sinus or traction on the carotid artery can cause bradycardia and hypotension due to activation of the baroreceptor reflexes. Cessation of stimulus or infiltration of the carotid bifurcation with 1% lidocaine usually prevents further episodes. Episodes of hypotension during other phases of surgery can be handled by either decreasing depth of anesthesia, or giving vasopressors like phenylepherine or ephedrine. The goal is to maintain the MAP at or 20% above patient's baseline MAP. Hypertension can be treated by either deepening anesthesia or using antihypertensives like esmolol or labetalol.

Patients are extubated after neurologic integrity is confirmed. Emergence may be associated with marked hypertension and tachycardia, which may require aggressive pharmacologic intervention. If patient develops rapidly expanding hematoma at the site of surgery, consideration should be made to immediately intubate the trachea, as stridor is a late sign of airway compromise [95].

Neurologic deficits on emergence may necessitate angiography, reoperation, or both as determined by surgery team. Therefore, the key elements in anesthetic management of CEA involve maintaining normocarbia, controlling blood sugar [96], maintaining MAP equal to or greater that 20% baseline MAPs during clamping, and using neurologic monitoring as a guide to shunting. A controlled induction and emergence being critical to the success of the anesthetic and surgical course.

There is considerable debate on the efficacy of regional anesthesia over general anesthesia in CEA. Regional anesthesia is accomplished by blocking the C2 to C4 dermatomes by use of a superficial, intermediate, or deep cervical plexus block. Pandit et al. [97] showed that a superficial/intermediate block is safer than any method that employs a deep injection. The higher rate of conversion to general anesthesia with the deep/combined block may have been influenced by the higher incidence of direct complications.

A regional anesthesia audit for CEA showed that cervical plexus block is associated with a significantly lower frequency of anesthesia-related complications and should therefore be considered the regional anesthetic of choice. Cervical epidural anesthesia should not be performed except in extenuating circumstances [98].

The GALA (General Anesthetic Versus Local Anesthetic for Carotid Surgery) trial, failed to show a statistically significant improvement in stroke, MI, or death with combined superficial and deep cervical plexus block versus general anesthesia (GA) [99].

Studies comparing CEA under local anesthesia (LA) Vs GA showed a higher rate of shunt usage after GA compared with LA CEA. LA may protect the patient from early postoperative cognitive dysfunction and appears to be more cost-effective [100].

3.6 Postoperative Cognitive Dysfunction (POCD)

Decline in cognitive function after surgery in the elderly is becoming increasingly recognized with an increase in our aging population. The earliest recognition was associated with cardiac surgery but research has shown that cognitive dysfunction is seen after other major noncardiac surgeries. The diagnosis of POCD requires pre- and postoperative neuropsychological testing. The testing is diverse and includes learning and memory, verbal abilities, perception, attention, executive functions, and abstract thinking.

In a recent study, POCD was observed in 15.9% of adults 65 year or older 3 months after major noncardiac surgery [101]. This is similar to the findings of International study of postoperative cognitive dysfunction 1 (ISPOCD1) study of 1218 patients which found that the incidence of POCD after 3 months in age > 70 was 14% [102].

Monk et al. [103] reported that cognitive dysfunction is common in adult patients of all ages at hospital discharge after major noncardiac surgery, but only the elderly (aged 60 years or older) are at significant risk (12.7%) for long-term cognitive problems. They confirmed the findings of ISPOCD1, which found that advancing age and lower educational levels are risk factors for the development of cognitive decline after noncardiac surgery. They also found that asymptomatic patients with a history of stroke with no residual impairment, and POCD at hospital discharge had a higher incidence of late (3 months after surgery) POCD.

No significant difference was found in the incidence of cognitive dysfunction 3 months after either general or regional anesthesia in elderly patients [104].

Russo et al. also found that the type of anesthesia (general or epidural) does not affect the magnitude or pattern of postoperative cognitive dysfunction in older adults undergoing total knee arthroplasty [105].

ISPOCD1 also found no relation between different degrees and durations of hypoxemia or hypotension and early and late postoperative cognitive dysfunction.

The goal of future research should be to incorporate standard psychometric tests in pre anesthesia clinic especially for the elderly patient and follow up testing at 3 months to identify POCD. Currently no preventative strategies have been developed for POCD and proper identification and rehabilitation is the key to successful recovery.

3.6.1 Postoperative Delirium

Postoperative delirium can clearly be distinguished from POCD as the key characteristics are a change in mental status characterized by a reduced awareness of the

environment and a disturbance in attention. This may be accompanied by other, more florid, perceptual symptoms (hallucinations) or cognitive symptoms including disorientation or temporary memory dysfunction.

In elderly hip fracture patients, the incidence of delirium was reported to be between 28 and 41% [106, 107].

In a study by Litaker et al., delirium was detected in 11.4% patients aged 70 years or older, pre-existing cognitive impairment, greater preoperative functional limitations, and history of prior delirium [108].

Perioperative hypoxemia, hypotension, sepsis, hypoglycemia, electrolyte disturbances, administration of certain drugs like anticholinergics, barbiturates, benzodiazepines are believed to be risk factors for post-op delirium. There appears to be no difference in the incidence of postoperative delirium with either neuraxial or general anesthesia [109].

Treatment of post-op delirium relies on identifying the reversible medical causes, reassuring and re-orienting the patient to their current hospital environment [110].

Brief postoperative delirium lasting more than 6 weeks is a determining factor for poor long-term functional outcome after hip fracture repair, because it significantly impacts the ability to live independently [111].

Key Points

- Preoperative testing should not be determined by patient's age only.
- Goal of preoperative testing is to determine the functional reserve of each organ system and to identify need for optimization. The new ACC guidelines are helpful in performing a good preoperative evaluation.
- Regional anesthesia (spinal and epidural) and peripheral nerve blocks should be considered strongly in perioperative management as they will help improve postoperative pain control and decrease incidence of postoperative delirium in the elderly.
- Careful consideration should be made to anticoagulation guidelines published by ASRA before performing regional anesthesia/analgesia.
- Diastolic dysfunction and decreased autonomic responsiveness are important causes of perioperative cardiac dysfunction in the elderly.
- Transesophageal echocardiography is an important tool in identifying hypovolemia vs LV dysfunction as the cause of persistent hypotension during major vascular surgery.
- Key elements in anesthetic management of carotid endarterectomy involve maintaining normocarbia, controlling blood sugar, maintaining MAP equal to or greater that 20% baseline MAPs during clamping, and using neurologic monitoring as a guide to shunting.

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