Anesthetic Considerations for Complex Endovascular Aortic Repair

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Introduction

The rapid evolution of endovascular therapy for aortic pathology has dramatically changed the field of vascular surgery over the past two decades. In parallel, anesthesiology and perioperative medicine have adapted accordingly to the care of this complex patient population. Newer graft technology has enabled the field to expand the applicability of endovascular aortic aneurysm repair (EVAR) to more challenging anatomy. Patients with complex aneurysms who otherwise are deemed inoperable or with extreme risk of morbidity and mortality are now considered for a less invasive alternative that has paved the way to a new standard of care. Moreover, a number of prospective randomized trials have demonstrated short-term advantages over open aortic repair, including reduced blood loss, operative time, morbidity, and hospital length of stay [1, 2]. The survival advantage of EVAR is maintained at 3 years, but with current techniques, aortic-related reintervention rates are higher compared to open aortic repairs, whereas the latter is associated with more laparotomy-related complications [3, 4]. With the development of hybrid approaches, fenestrated, branched, and parallel graft techniques [5–7], there seems to be no limit to the endovascular treatment of aortic pathology. This chapter focuses on anesthetic and perioperative care considerations for these patients including anesthesia technique, specific hemodynamic and neurologic monitoring, spinal cord protection, and induced hypotension techniques to facilitate proximal aortic graft deployment.

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Anesthetic Considerations

Understanding of the aortic anatomy, aneurysm extension and morphology as well as open discussion of the procedural plan with the surgical team are essential. From risk calculation to cardiopulmonary testing, a thorough preoperative evaluation must take place, to determine the need and extent of perioperative hemodynamic and neurologic monitoring, need for spinal drain placement, as well as the most appropriate method for induced hypotension, if required. From the preoperative risk point of view, EVAR is considered an intermediate risk surgical category [8]. However, the risk of complex fenestrated, branched, and hybrid endovascular thoracoabdominal aortic approaches is likely underestimated by nonspecific risk calculators [9, 10] and more aggressive preoperative cardiopulmonary testing may be required.

Anesthesia Technique and Patient Setup

The choice of anesthesia technique is usually guided by baseline comorbidities, hemodynamic status, need for complex neurologic monitoring, aneurysm location, and patient preference. More often, general endotracheal anesthesia is the favored method for complex hybrid and fenestrated approaches. Specifically, if somatosensory (SSEP) or motor evoked potentials (MEP) are used [11], total intravenous anesthesia (TIVA) is the preferred anesthetic technique [12] with propofol (100–200 mcg/kg/min) and opioid (fentanyl, remifentanil, or sufentanil) infusions as traditional inhaled anesthetics at required doses inhibit the evoked potential signal. Muscle relaxants are avoided if MEPs are used. It is important to plan a fast-track anesthesia technique to allow for early postoperative neurologic examination, shortly after

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conclusion of the procedure. EVAR in patients with simple anatomy can be performed under monitored anesthesia care (MAC) with local anesthesia.

Cerebrospinal Fluid (CSF) Drainage

The use of CSF drainage to minimize risk of spinal cord injury was pioneered by Larry Hollier at the Mayo Clinic in the late 1980s. Although at that time the technique initially faced resistance and criticism, it is currently widely utilized for complex aneurysms involving risk of paraplegia. Despite the lack of clear prospective data, elective preoperative lumbar CSF drain placement for optimization of spinal cord perfusion (see below in Sect. Spinal Cord Protection) plays an important role in TEVAR [13]. Contraindications for spinal drain insertion include preoperative anticoagulation, intracranial process (tumor or bleeding), and infection at the insertion site (Table 22.1). After standard monitoring is in place, the lumbar drain is usually inserted before induction of anesthesia to allow for patient feedback as the needle is being inserted (Fig. 22.1). However, the timing of insertion after induction of anesthesia is largely dependent on institutional preference and it is described in some series [14, 15]. If using a lumbar CSF drain kit (Integra®, CODMAN®, etc.) the 0.7 mm ID drain is prepared in sterile fashion by flushing it with preservative free normal saline prior to inserting the flexible wire to reduce friction. The wire is then advanced to the catheter tip to provide support for drain insertion. Note the drain markings starting at 10 cm and drainage fenestrations up to 5 cm from the catheter tip. Once optimal patient positioning has been achieved (sitting or lateral decubitus with hip, knee, and neck flexion), using anatomical landmarks, the L4–5 interspace is located at the level of the iliac crest. This is the optimal location to avoid the conus medullaris (Fig. 22.2). Alternative insertion spaces are L3–4 or L5–S1. After standard asepsis with alcohol-based chlorhexidine solution and sterile draping, the 14-G beveled needle (lumbar CSF drain insertion kit) or 17-gauge epidural needle (if using a standard epidural catheter) is inserted and gently advanced between the L4–5 spinous processes into the thecal sac (see Fig. 22.1). The bevel is then rotated towards the head of the patient and the needle stylet is removed allowing for brisk CSF return. Once CSF return is noted, the drain with flexible wire (or epidural catheter) is advanced through the needle into the thecal sac. The catheter should be advanced at least 8 cm (up to 12 cm) into the subarachnoid space to ensure all fenestrations are in the thecal sac for optimal drainage [14, 15]. In some instances, such as presence of lumbar spine pathology (spinal stenosis) or prior spinal surgical procedures, the insertion of the lumbar drain is performed the day before surgery under fluoroscopy and advanced to T9–10 [16]. The distance from the patient's skin to the thecal sac should be noted and added to the length of catheter advanced in the subarachnoid space. The final catheter position secured at the skin should be recorded in the procedural note. If traumatic insertion or bloody return is encountered, discussions with the surgical team should include the possibility of delaying anticoagulation for at least 1 h, delaying the procedure for 1 day, or proceeding maintaining a higher index of suspicion for neuraxial hematoma [18]. After optimal catheter position is established, the wire is removed with gentle but firm traction while securing the drain position at the skin with gentle pressure such that the catheter is not retracted; the drain cap is placed, and secured with a small suture tie. The catheter is then secured at the skin with sterile dressing and the patient is placed in the supine position. Maintenance of adequate CSF drainage is confirmed by gentle aspiration with a 3-cc syringe and general anesthesia is induced. Additional hemodynamic lines are then placed and SSEP and MEP electrodes. During this time, the spinal drain is clamped (Fig. 22.3).

Once all monitors and lines are applied, the drainage system is primed with 10 mL of preservative free normal saline, the spinal drain is attached to the drainage system and the transducer is zeroed and opened to drain at 10 mmHg with a maximum CSF drainage volume of 10 mL/h or 20 mL/h during the ischemic period with avoidance of total drainage >130 mL for the case. There is some controversy regarding the optimal location to zero the transducer to guide spinal fluid drainage. Although there is rationale to use the phlebostatic axis (right atrium) due to the location of spinal cord at risk, there is a higher risk of over drainage and subsequent hemorrhagic complications [16, 17]. Moreover, zeroing at the tragus or external ear meatus (Fig. 22.4) is accurate in the supine position and would protect from over draining in any other position. At our institution, we utilize the tragus, and, if neurologic deficit develops, the patient is maintained supine.

Vascular Access, Hemodynamic and Neurologic Monitoring

Nearly all complex EVAR cases require arterial and central venous access for frequent arterial blood sampling and hemodynamic monitoring as well as large bore venous access for rapid volume administration. The site of arterial access should be discussed with the surgical team, as often, the left brachial artery is accessed for the procedure, limiting peripheral venous access and arterial catheter insertion to the right upper extremity. Depending on the patient's comorbidities and/or the need for rapid ventricular pacing, a pulmonary artery catheter may be required (see Sect. "Induced Hypotension For Precise Graft Deployment"). Transesophageal echocardiography (TEE) may also be required at the discretion of the surgeon or anesthesiologist, depending on location of aneurysm, monitoring wire in proximity of aortic valve and evaluation of dissection flaps.

Table 22.1 Guidelines for lumbar CSF drainage in TEVAR[a]

Subject	Recommendation
Assessment of contraindications/ preoperative considerations	
Current anticoagulation[18]	Ensure that: Platelets >100 K INR <1.3 Normal aPTT No LMWH for 24 h (high dose regimen); 12 h (low dose regimen) No clopidogrel for 7 days No ticlopidine for 10 days No abciximab for 24–48 h No eptifibatide or tirofiban for 4–8 h No dabigatran for 5 days No apixaban for 3 days No rivaroxaban for 3 days
Infection at the site	Consider alternative site or delay surgery
Intracranial process	Avoid drain placement if concern for intracranial hypertension
Potential placement issues	
Spinal pathology (spinal stenosis)	Consider elective fluoroscopic guided lumbar drain placement
Prior lumbar spine surgery	
Positioning limitations	
Drain insertion	Consider insertion prior to induction of anesthesia
Traumatic/blood puncture	Evaluate for delay of surgery for 24 h or anticoagulation delay for > 1 h
Intraoperative management	
Hemodynamics	Avoid hypotension Optimize SCPP
Zero transducer	External ear meatus (Tragus)—patient supine, avoid over drainage
CSF drainage	CSFP <10 mmHg and maintain SCPP >60 mmHg. Drain < 10–20 mL/h Avoid large volumes of CSF drainage Maximal drainage <130 mL
Postoperative management and monitoring complications	
Disposition	Intensive care unit
Neurological checks	Hourly
Early neurologic exam	Allow for fast-track anesthesia and early extubation
Position	Allow for head of the bed 20°–30° elevation if no neurologic deficit If neurologic deficit: supine
Hemodynamics	Avoid hypotension, augment BP if needed with vasoactive agents and fluids
CSF drainage	Monitor CSF drainage and calculate SCPP Open drain every hour for 15 min and record drainage If CSF drainage > 20 cc/15 min—clamp drain
Duration of CSF monitoring/drainage	<72 h to minimize infection risk 24 h if neurologic exam intact and Crawford type IV TAAA 48–72 h if neurologically intact and Crawford type I–III TAAA If neurologic deficit present, weigh risk of infection versus benefit of continued CSF drainage
Bloody CSF drainage	May indicate intracranial bleed, consider CT head and/or spine
Headache	Consider symptomatic intracranial hypotension Stop CSF drainage Consult neurology if neurologic deficit
New onset lower extremity neurologic deficit	Consider SCI vs. neuraxial hematoma Increase SCPP > 80 (MAP 90–100 and CSFP 0–5 mmHg) Consider imaging of neuraxis
DVT prophylaxis	SQ heparin ±SCDs
Drain removal	Stable neurologic exam after 6 h with drain clamped Ensure adequate coagulation profile: similar to insertion guidelines Delay removal 2–4 h after last heparin administration Ensure intact catheter tip (rule out catheter fracture) Hold heparin for 1 h after drain removal

Abbreviations: *LMWH* low molecular weight heparin, *SCPP* spinal cord perfusion pressure, *TAAA* thoracoabdominal aortic aneurysm, *MAP* mean arterial pressure, *CSFP* CSF pressure, *SQ* subcutaneous, *SCDs* sequential compression devices

[a]Modified from [19]

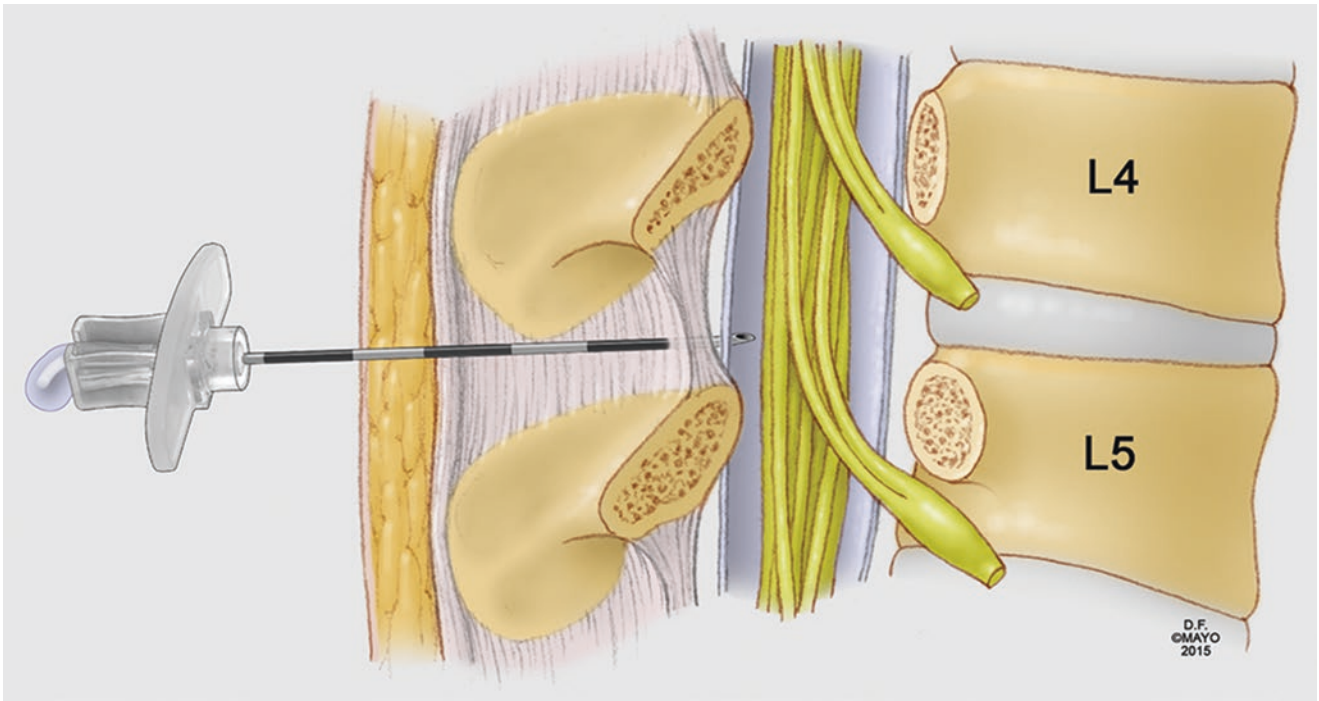

Fig. 22.1 Introduction of access needle in the L4–5 interspace with confirmation of clear cerebrospinal fluid prior to drain insertion. By permission of Mayo Foundation for Medical Education and Research. All rights reserved

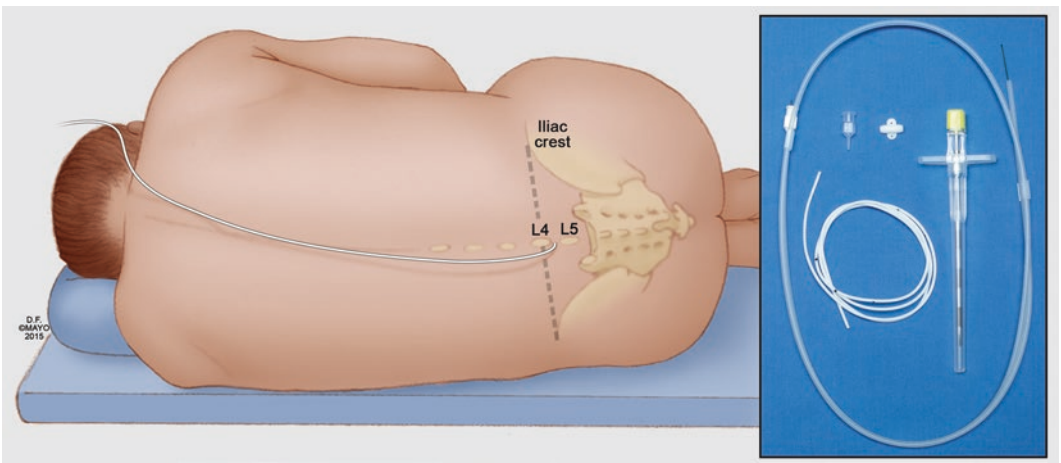

Fig. 22.2 Patient in the left lateral position for CSF drain placement and drain kit with ancillary tools. By permission of Mayo Foundation for Medical Education and Research. All rights reserved

Total intravenous anesthesia (TIVA) is the anesthesia technique of choice whenever SSEPs and MEPs are used to monitor the posterior column sensory proprioceptive and anterior motor pathways (Fig. 22.5). In those circumstances, or at the discretion of the anesthesiologist, a processed EEG monitor is applied in the patient's forehead to monitor anesthesia depth (see Fig. 22.3). The most common technology used is the Bispectral Index (BIS™) targeted anesthesia depth maintaining a BIS range of 40–60. Although TIVA is preferred, low dose inhalation agent (MAC < 0.5) can be used as well with minimal interference of SSEPs and MEPs once a good baseline has been obtained. After induction of anesthesia and placement of invasive hemodynamic monitors, the Intraoperative Neurophysiologic Monitoring (IOM)

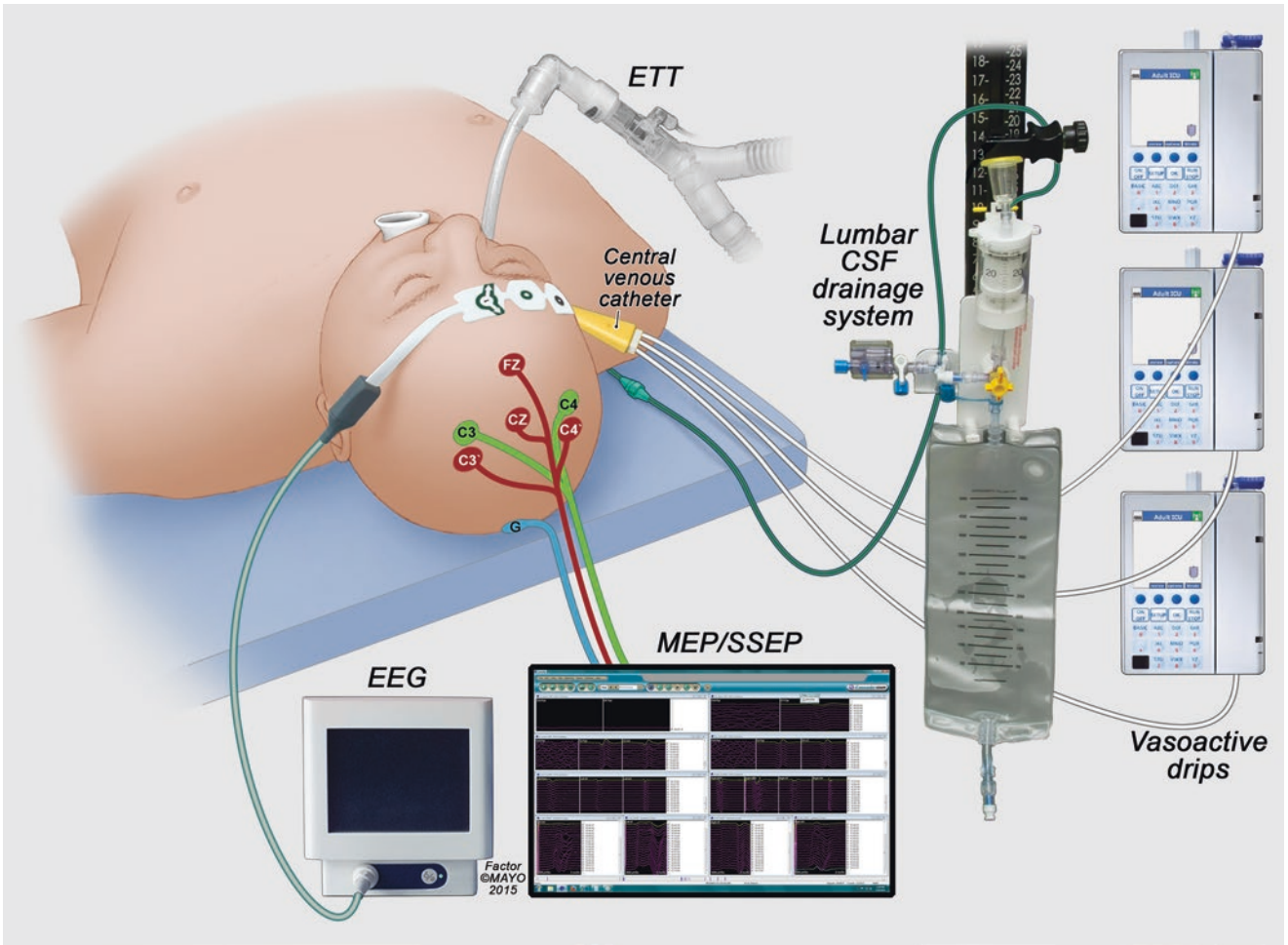

Fig. 22.3 Standardized set for CSF drainage and neuro-monitoring used during complex endovascular aortic procedures. By permission of Mayo Foundation for Medical Education and Research. All rights reserved

technicians place the respective electrodes (see Figs. 22.3 and 22.5). A constant current stimulator is recommended and either standard disk EEG electrodes or sterile subdermal needle electrodes may be used. Disk EEG electrodes should be applied to the scalp with collodion and sealed with tape or sheet to protect them from blood or other fluids. It is important to ensure that the OR personal is aware of the location of electrodes to avoid needle sticks.

During MEPs, electrical charges sent to the motor cortex through C3, C4 scalp electrodes stimulate the motor corticospinal pathway, which are recorded over multiple muscles in the upper and lower extremities at least every 10–15 min. An upper extremity muscle (extensor digitorum communis) is recorded to help differentiate neurogenic impairment such as spinal and lower limb ischemia from nonspecific changes. Bilateral lower extremity muscles are recorded using subdermal EEG electrodes placed in the hamstring, tibialis anterior, and abductor hallucis muscles (see Fig. 22.5). With SSEPs the electrical stimulus is generated at the ulnar or tibial nerve and travels from the distal extremity via the posterior column medial lemniscus pathway and is recorded over the neck and scalp. In patients where the lower extremity tibial SSEP are not present at the ankle, subdermal EEG electrodes can be positioned behind the knee (see Fig. 22.5).

Radiation Safety

The occupational radiation exposure is proportional to the complexity of the procedure and the use of endovascular suites and hybrid operating rooms. The key components of radiation safety include time, distance from radiation source and appropriate shielding. The inverse square law states that radiation scatter will decrease by the square of the distance to source of radiation; therefore, doubling the distance from a point source of radiation will decrease the exposure rate to one-fourth the original exposure rate. Anesthesia providers should practice similar safety to the operating surgical team and wear leaded aprons, thyroid shields and consider leaded eyewear.

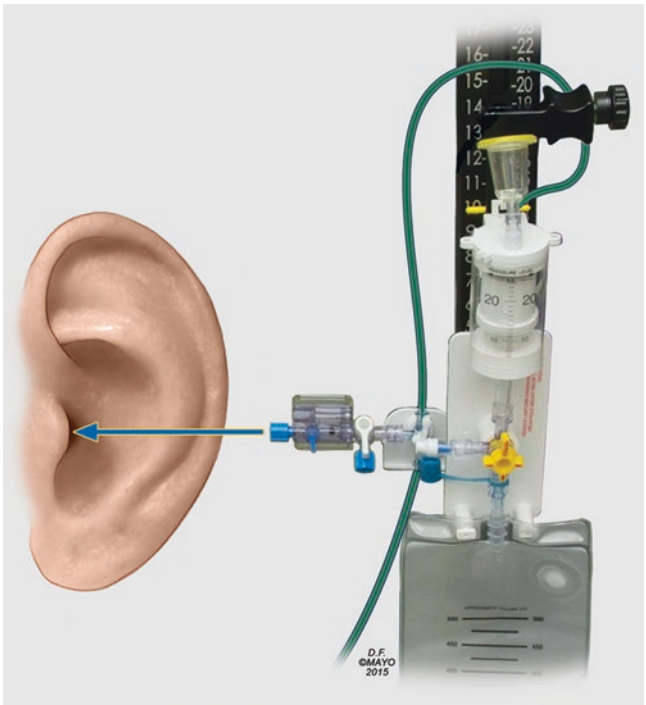

Fig. 22.4 Zeroing of CSF drainage for accurate measurement and monitoring. By permission of Mayo Foundation for Medical Education and Research. All rights reserved

Assessing and Managing the Risk for Contrast-Induced Nephropathy

Due to the requirement for iodinated contrast medium during EVAR, preprocedural renal protection strategies are considered for patients with preexisting kidney disease or at risk for acute kidney injury. Although there is no widely accepted definition of contrast-induced nephropathy (CIN), an increase in creatinine of 0.5 mg/dL or 25 % above baseline within 2–3 days from contrast exposure is likely consistent with CIN.

Patents risk factors for CIN include:

- Serum creatinine > 2 mg/dL.
- Congestive heart failure.
- Proteinuria.
- Dehydration.
- Renal transplant.
- Calcineurin inhibitors (cyclopropane, sirolimus, tacrolimus).

Procedural risk factors for CIN include:

- Large volume of contrast agent (>400 mL).

Preoperative hydration with intravenous fluid administration is the primary prevention strategy for contrast induced nephropathy (CIN) [20] and the efficacy of intravenous isotonic bicarbonate as the fluid of choice, or the administration of *N*-acetylcysteine in the prevention of CIN remains unclear despite earlier studies advocating their use [21]. Preprocedure renal optimization includes holding diuretics for 24 h before the procedure, as well as avoiding nephrotoxic medications and cautious periprocedural hydration with normal saline, lactate Ringer's, or isotonic sodium bicarbonate at 100 mL/h for 10 h, unless significant reduction in cardiac reserve or hypervolemia is present.

Spinal Cord Protection

Spinal cord ischemia (SCI) remains an important cause of morbidity after TEVAR [22]. Interestingly, the coverage of the thoracic aorta without revascularization of spinal arteries was expected to produce higher rates of spinal cord ischemia than what is actually observed, challenging traditional anatomical models of spinal cord perfusion [13]. It is important however to consider the extent of coverage of native aorta as an important risk factor for SCI. Despite lower rates of SCI compared with open thoracoabdominal aortic repairs, this phenomenon continues to represent an important cause of morbidity due to the inability to revascularize covered spinal arteries, the presence of hypotension during the procedure, risk of embolization from present atheromatous plaques, and the possibility of compromise of distal perfusion due to large-bore sheaths used for stent graft insertion. Independent risk factors for development of SCI include: perioperative hypotension defined as MAP < 70 mmHg, CSF drainage complications, prior abdominal aortic aneurysm repair (compromise of hypogastric arteries), preoperative kidney disease, left subclavian artery coverage without revascularization, and the use of >3 stent grafts (reflecting on the length and complexity of the procedure as well as the extent of aorta covered) [13]. Notwithstanding, others have demonstrated that the most important risk factor for symptomatic SCI is the simultaneous closure of two independent arterial spinal cord supplying vascular territories in addition to persistent intraoperative hypotension, emphasizing the concept of collateral network in the pathophysiology of this devastating condition [23]. Another important factor to consider is the chronicity of the disease; such as the use of TEVAR in acute aortic dissection compared to chronic aneurysmal atherosclerotic disease, in which a collateral network can be highly variable, including contributions from lumbar and pelvic arteries, with the possibility to compensate for spinal artery compromise [24].

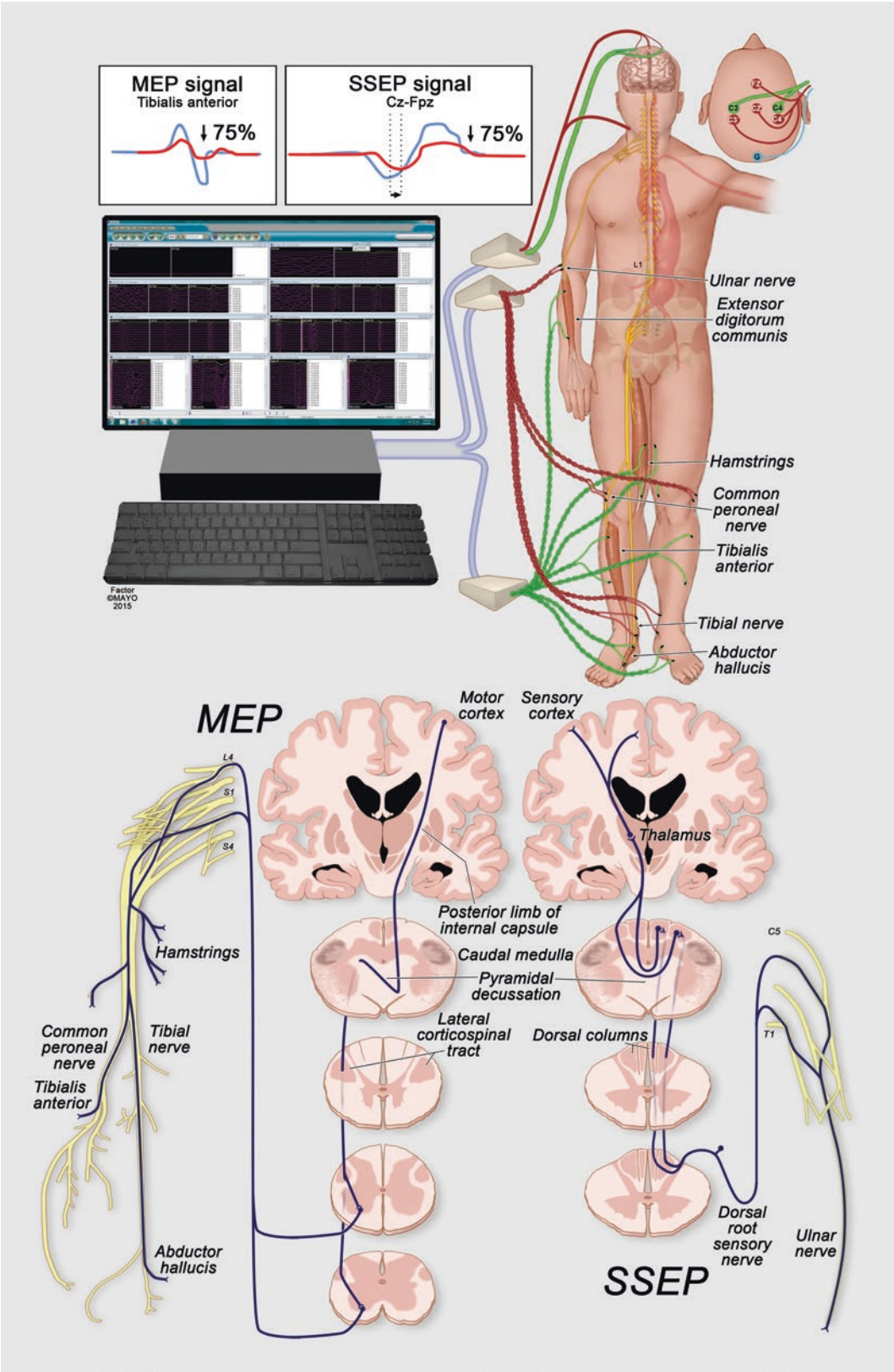

Fig. 22.5 Neuro-monitoring with somatosensory and motor evoked potentials is used routinely during extensive aortic coverage. By permission of Mayo Foundation for Medical Education and Research. All rights reserved

For optimal spinal cord protection, the intraoperative team focuses on strategies to maximize spinal cord perfusion with hemodynamic augmentation and CSF drainage, as well as strategies for early detection and management of SCI.

Strategies to Augment Spinal Cord Perfusion

The main therapeutic strategies to augment spinal cord perfusion aim to increase the collateral network pressure (CNP) and minimize cerebrospinal fluid or venous pressure according to the formula [25–27]:

$$\mathrm{SCPP} = \mathrm{MAP} - \left(\mathrm{CSFP\ or\ CVP}\left[\mathrm{whichever\ higher}\right]\right)$$

where SCPP = spinal cord perfusion pressure; MAP = mean arterial pressure (optimally distal aortic pressure); CSFP = cerebrospinal fluid pressure; and CVP = central venous pressure.

The CNP, which during TEVAR is the main driver of spinal cord perfusion, is only a fraction of the measured MAP (~70 %) [27]. Moreover, the CNP falls significantly more as a percentage of the MAP (~25 %) during the first 24 h after segmental spinal artery occlusion, and proportionally related to the number arteries sacrificed [27]. In general, it is imperative to avoid hypotension, maintain SCPP > 60 mmHg while avoiding large increases of CVP and drain CSF to maintain CSFP at ≤10 mmHg [19] (see Table 22.1). Institutions that perform complex TEVAR usually have protocols approved by the different members of the multidisciplinary team that care for these patients. These protocols vary between institutions from baseline monitoring pressure goals to optimal location to zero the transducer. At our institution, we continuously monitor the CSFP to allow for calculation of SCPP and open the drain to 10 mmHg, avoiding drainage >10 mL/h, or 20 mL/h during the ischemic period. As described above, the transducer is zeroed at the tragus or external ear meatus, which in the supine position correlates with the phlebostatic axis. If there is intraoperative evidence of SCI with changes on SSEPs and/or MEPs, the CSFP is lowered to 5 mmHg or 0 mmHg with care to avoid over draining and increase MAP to 90–100 mmHg (Fig. 22.6) Permissive systemic arterial hypertension is achieved with the use of vasoactive medications depending on cardiac function and hemodynamics. Usually, norepinephrine is initiated as a titratable infusion to increase SCPP >80 mmHg (or MAP 90–100 mmHg), despite paucity of data recommending its use over other vasoactive drugs. The practical advantages of norepinephrine include fast onset–offset of action and ability to titrate up and de-escalate acutely. Although not specific for SCI, at this time, norepinephrine remains the vasopressor of choice for acute neurologic injury [28]. Other adjuncts include vasopressin, a well-recognized and widely used vasopressor in vasodilatory shock. However, at doses >0.04 U/min, the risk of intestinal ischemia outweigh the benefits of permissive hypertension in SCI [29]. Phenylephrine has the potential for tachyphylaxis and therefore its use is limited to the operating room. In patients with significant left ventricular systolic dysfunction, an ionotrope, such as epinephrine or dobutamine with or without the addition of a vasopressor, may be appropriate. The choice of agent is largely dependent on the hemodynamic profile and cardiac function of the patient.

Strategies to Detect and Manage Spinal Cord Ischemia

The use of intraoperative neurologic monitoring (IOM) with SSEPs and MEPs (see Fig. 22.5) allows for detection of spinal cord ischemia and activation of a treatment plan while the patient is under anesthesia. As changes in blood flow correlate with changes in neuronal electrical activity, and there is a finite period of time (3–4 h) in which this can be reversed without permanent neurologic injury, IOM with SSEPs and MEPs has an important role in TEVAR with high risk for SCI [11]. Compared with the sensitivity of the neural tissue of the cerebral cortex to ischemia noted by electroencephalography (~20–30 s), the time for loss of response with ischemia is longer with SSEPs and MEPs (7–18 min and 11–17 min, respectively) [11]. Therefore, the evoked potentials are monitored every 10–15 min. A reduction of 50–75 % from baseline evoke potential amplitude is consider significant to trigger the SCI treatment algorithm (see Fig. 22.6). Once SSEP or MEP has confirmed a significant reduction of potentials, the SCPP is acutely maximized by increasing MAP >90–100 mmHg and decreasing CSFP to 5 mmHg. If these maneuvers improve the MEP and SSEP signals, the procedure is continued in standard fashion. If the optimization of SCPP fails to normalize the MEP/SSEP signals, the flow is restored to the pelvis and lower extremities by rearranging the sequence of target vessel stenting and consideration for arterial conduit is performed vs. staging the procedure.

Induced Hypotension for Precise Graft Deployment

Another important intraoperative technique unique to TEVAR is induced hypotension at the time of endovascular stent graft deployment to prevent the forward flow of blood from the heart causing the graft to move distal to the intended landing zone. Avoiding malposition of the stent graft secondary to pulsatile forces is critical. This is sometimes referred to as the "windsock effect." With newer graft designs and deployment techniques, the need for induced hypotension

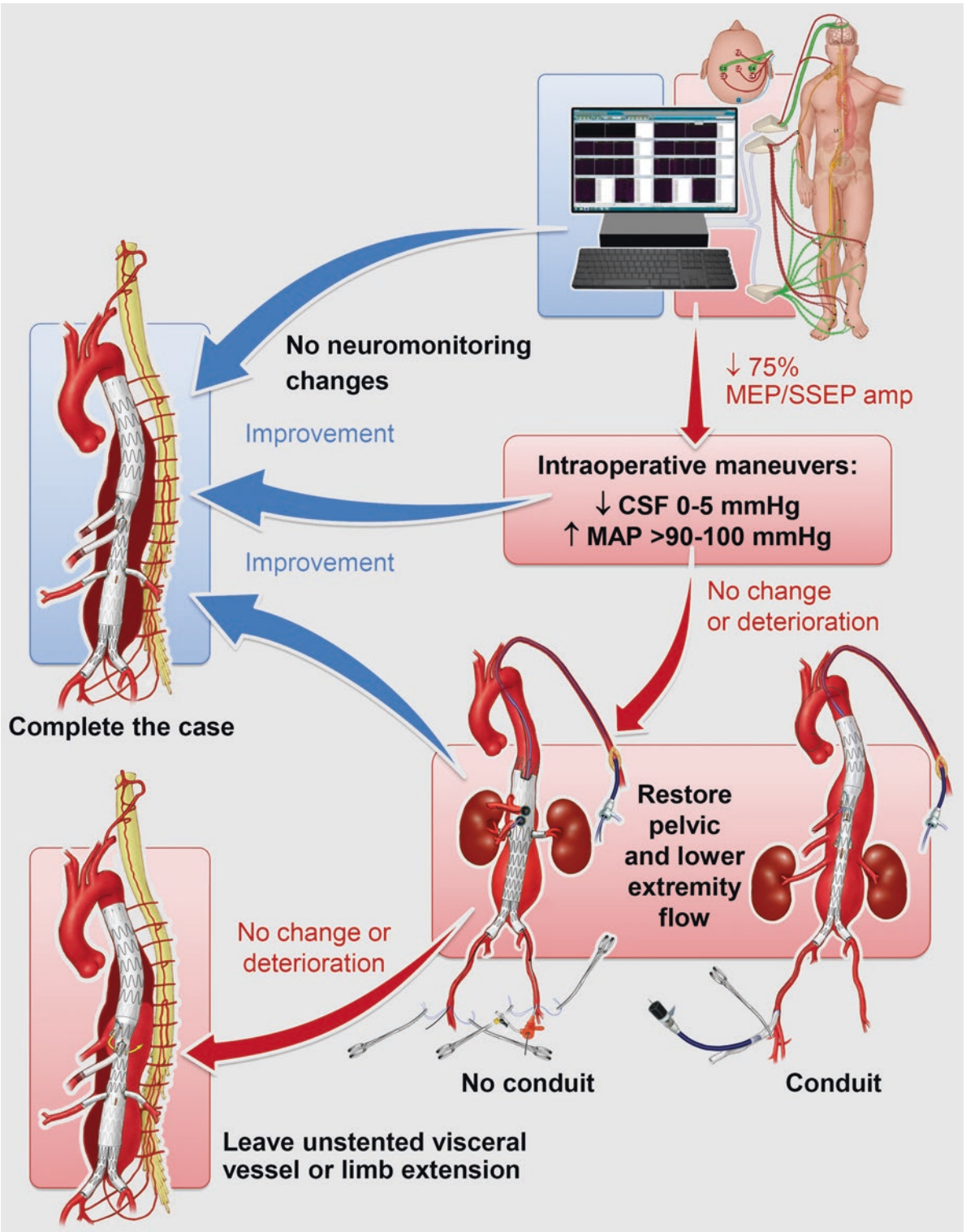

Fig. 22.6 Standardized Mayo Clinic protocol used in conjunction with neuro-monitoring for thoracoabdominal aortic repair. Note maneuvers include lowering CSF pressure and using hemodynamic augmentation to optimize spinal cord perfusion. By permission of Mayo Foundation for Medical Education and Research. All rights reserved

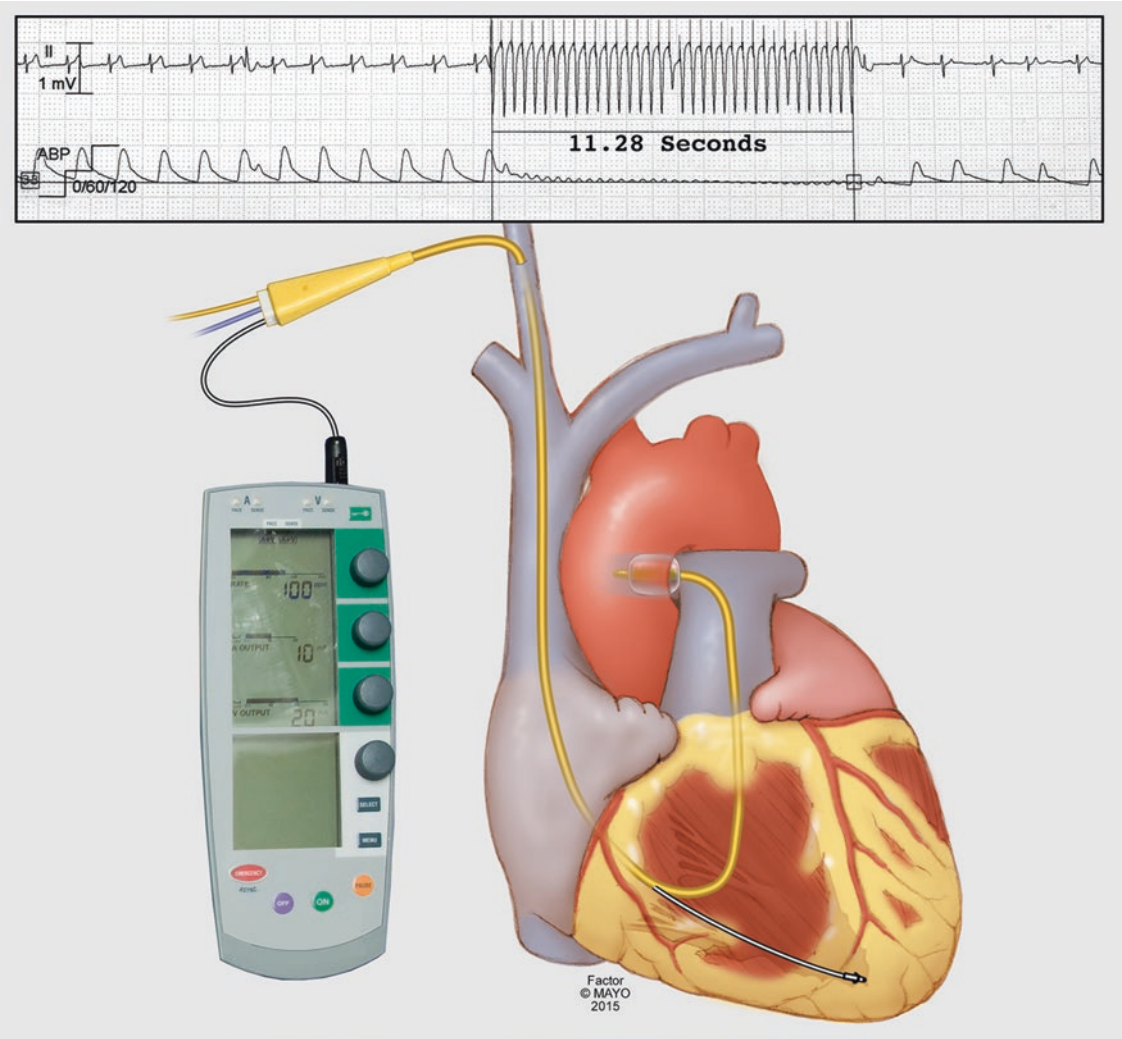

Fig. 22.7 For patients with ascending aortic and arch aneurysms with a landing zone in Zone 0, rapid ventricular pacing is used to allow controlled hypotension during stent deployment. By permission of Mayo Foundation for Medical Education and Research. All rights reserved

has been reduced to the proximal landing zones. Several methods are used to achieve induced hypotension including rapid ventricular pacing, right atrial-IVC balloon occlusion, and medication administration.

Rapid Ventricular Pacing

Rapid ventricular pacing provides a controlled reduction in cardiac output for a precise duration of time with hemodynamics returning to pre-pacing levels when the pacing is terminated. Pacing may be performed via a pulmonary artery catheter [30] (Fig. 22.7, rapid ventricular pacing) or via a transfemoral venous wire [31, 32]. Both cardiologists and anesthesiologist may safely provide rapid ventricular pacing for endograft deployment. Pulmonary artery catheter (PAC) rapid ventricular pacing utilizes a pacing wire placed through the RV pacing port of the catheter (see Fig. 22.7). The wire is deployed after the PAC is appropriately placed utilizing observation of hemodynamic waveforms or fluoroscopy. Capture is generally present when the wire is advanced 5 cm out of the RV pacing port. Regardless of the method of pacing, the pacing wires are tested prior to use for endograft deployment. Immediately prior to deployment pacing is commenced at rates between 160 to 200 beats/min and deployment is initiated when the blood pressure decreases to a mean arterial pressure of 40–50 mmHg and pulsatility is lost on the arterial waveform (see Fig. 22.7). After deployment is complete, pacing is terminated. The heart rate generally returns quickly to baseline, but patients may require temporary pacing at a back-up rate while their hemodynamics fully recovers. This process can be repeated as necessary for additional deployments or stent ballooning provided the patient continues to tolerate the pacing with

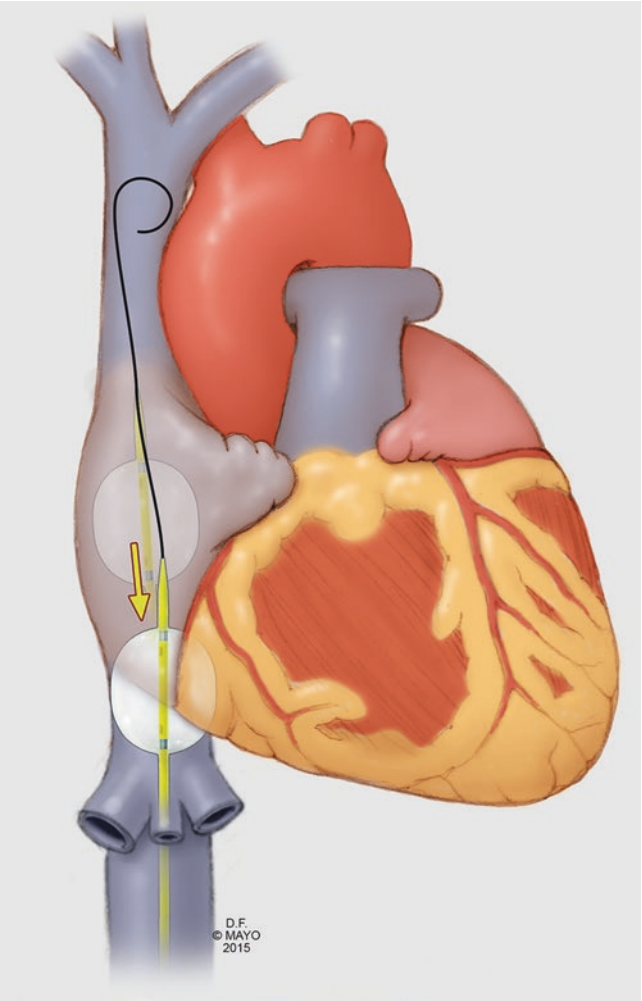

Fig. 22.8 An alternative for induced hypotension is balloon occlusion of the right atrium and caval junction decreasing preload and allowing systemic hypotension for stent deployment. By permission of Mayo Foundation for Medical Education and Research. All rights reserved

good recovery of hemodynamics after each episode. Following the procedure the pacing wire and PAC (if used) are removed.

Right Atrial Inflow Occlusion

Balloon occlusion of the right atrial-IVC junction has been described to decrease cardiac output for endograft deployment by decreasing preload [33, 34] (Fig. 22.8). The common femoral vein is accessed using standard Seldinger technique. Subsequently, a guidewire is advanced from the IVC to the SVC and a 12 Fr introducer sheath is placed (Coda® balloon catheter Cook Medical). The balloon is then advanced into the right atrium with fluoroscopic guidance and inflated. A mixture of contrast and saline may be used for inflation to confirm placement within the atrium. Immediately prior to endograft deployment traction is placed on the balloon pulling it into the IVC-right atrial junction and when resistance is felt the balloon is held in this position preventing blood return to the heart. This decrease in preload causes a decrease in cardiac output. When the deployment is complete the traction on the balloon is released allowing blood to resume flowing from the ICV to the heart and hemodynamics to normalize. It is important to ensure that the balloon is within the atrium at the time it is inflated and to not pull too vigorously on the balloon due to the risk of avulsing the IVC off of the right atrium.

Medications

Lastly, medications can also be used induce hypotension to facilitate endograft deployment. These include adenosine (inducing asystole), intravenous vasodilators (nitroprusside, nitroglycerin), and beta blockers (esmolol). All of these have been used safely to provide adequate conditions for graft deployment [35]. Adenosine-induced asystole results in no cardiac output for the duration of the drug effect; however, the duration is variable between patients. Titration of vasodilators and beta blockers allows for a gradual onset of hypotension and reduced cardiac output. After deployment, the patient is allowed to recover as the medication effects wear off or additional medications may be administered to improve the hemodynamics.

Each technique to alter hemodynamics and facilitate endograft deployment has unique benefits as well as risks. It is important that the team performing the hemodynamic maneuvers understand the physiology of the technique as well as the potential complications. The ability to prevent, recognize, and treat potential complications is paramount.

Postoperative Considerations

The disposition of a patient following endovascular stent graft placement depends on the nature of the procedure, the patient's preoperative state and comorbidities, need for strict neurologic monitoring with optimization of SCPP or development of complications at the time of surgery. At the completion of an endovascular stent graft procedure it is often ideal to evaluate the patient's neurologic status. The patient can either be awakened from anesthesia and extubated or the anesthesia can be lightened to a point where the patient is able to follow commands such as moving all extremities. The decision of whether the patient should be extubated at the end of the procedure should be based on the usual consideration such as hemodynamics, surgical course, and preoperative condition. If the patient is to remain intubated, the patient will go to the intensive care unit (ICU). A patient who is extubated would usually require ICU admission if blood pressure is being augmented by vasoconstrictors or inotropes, if a spinal

drain is in place, or if frequent neurologic monitoring is desired (see Table 22.1).

Complications following endovascular aortic stent grafting include endoleaks, ischemia secondary to inadvertent arterial blockage, other graft-related complications, bleeding, stroke, acute kidney injury, myocardial infarction, and spinal cord ischemia [36]. Spinal cord ischemia may be due to inadequate perfusion following the graft placement or due to spinal cord hematoma secondary to the spinal drain. Spinal cord injury after TEVAR usually occurs in the first 48 h. In a patient with a spinal drain who develops back pain and/or weakness, neuraxial hematoma, although rare, must be considered and appropriate imaging should be performed. More likely, however, new onset postoperative paraplegia is secondary to ischemia from suboptimal spinal cord and collateral network perfusion. At this time, it is appropriate to use the strategies to augment spinal cord perfusion as described above, include augmenting blood pressure with vasoconstricting agents to increase perfusion pressure, placement of a spinal drain if one is not being utilized, or increased CSF drainage in a patient with a functioning spinal drain. Due to the complex nature of these patients, both due to the surgical procedure and comorbidities, close monitoring and quick intervention is crucial to good outcomes.

Conclusion

As newer graft deployment technology is developed, anesthesia and perioperative medicine continues to evolve with the field. It is imperative to have an open discussion about the procedural plan, including the extent of aorta to be covered, the need for induced hypotension for graft deployment, and extremity access needed for graft deployment, as it will dictate the anesthesia technique, hemodynamic and neurologic monitoring required as well as the need for elective spinal drain placement with close vigilance to monitor and manage intraoperative spinal cord ischemia. The postoperative care depends largely on the complexity of the procedure, intraoperative complications, and need for permissive hypertension with vasoactive drips along with frequent neurovascular checks.

References

1. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. Lancet. 2005;365(9478):2179–2186.
2. Prinssen M, Verhoeven EL, Buth J, Cuypers PW, van Sambeek MR, Balm R, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med. 2004;351(16):1607–18.
3. Chang DC, Parina RP, Wilson SE. Survival after endovascular vs open aortic aneurysm repairs. JAMA Surg. 2015;2:1–7.
4. De Bruin JL, Baas AF, Buth J, Prinssen M, Verhoeven EL, Cuypers PW, et al. Long-term outcome of open or endovascular repair of abdominal aortic aneurysm. N Engl J Med. 2010;362(20):1881–9.
5. Oderich GS, Mendes BC, Gloviczki P, Kalra M, Duncan AA, Bower TC. Current role and future directions of hybrid repair of thoracoabdominal aortic aneurysms. Perspect Vasc Surg Endovasc Ther. 2012;24(1):14–22.
6. Oderich GS, Correa MP, Mendes BC. Technical aspects of repair of juxtarenal abdominal aortic aneurysms using the Zenith fenestrated endovascular stent graft. J Vasc Surg. 2014;59(5):1456–61.
7. de Souza LR, Oderich GS, Banga PV, Hofer JM, Wigham JR, Cha S, et al. Outcomes of total percutaneous endovascular aortic repair for thoracic, fenestrated, and branched endografts. J Vasc Surg. 2015;62(6):1442–9.
8. Fleisher LA, Fleischmann KE, Auerbach AD, Barnason SA, Beckman JA, Bozkurt B, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. J Am Coll Cardiol. 2014;64(22):e77–137.
9. Zhan HT, Purcell ST, Bush RL. Preoperative optimization of the vascular surgery patient. Vasc Health Risk Manag. 2015;11:379–85.
10. Bertges DJ, Goodney PP, Zhao Y, Schanzer A, Nolan BW, Likosky DS, et al. The Vascular Study Group of New England Cardiac Risk Index (VSG-CRI) predicts cardiac complications more accurately than the Revised Cardiac Risk Index in vascular surgery patients. J Vasc Surg. 2010;52(3):674-683, 683 e671-683 e673.
11. Sloan TB, Edmonds Jr HL, Koht A. Intraoperative electrophysiologic monitoring in aortic surgery. J Cardiothorac Vasc Anesth. 2013;27(6):1364–73.
12. Keyhani K, Miller 3rd CC, Estrera AL, Wegryn T, Sheinbaum R, Safi HJ. Analysis of motor and somatosensory evoked potentials during thoracic and thoracoabdominal aortic aneurysm repair. J Vasc Surg. 2009;49(1):36–41.
13. Etz CD, Weigang E, Hartert M, Lonn L, Mestres CA, Di Bartolomeo R, et al. Contemporary spinal cord protection during thoracic and thoracoabdominal aortic surgery and endovascular aortic repair: a position paper of the vascular domain of the European Association for Cardio-Thoracic Surgery. Eur J Cardiothorac Surg. 2015;47(6):943–57.
14. Estrera AL, Sheinbaum R, Miller CC, Azizzadeh A, Walkes JC, Lee TY, et al. Cerebrospinal fluid drainage during thoracic aortic repair: safety and current management. Ann Thorac Surg. 2009;88(1):9–15. discussion 15.
15. Cheung AT, Pochettino A, McGarvey ML, Appoo JJ, Fairman RM, Carpenter JP, et al. Strategies to manage paraplegia risk after endovascular stent repair of descending thoracic aortic aneurysms. Ann Thorac Surg. 2005;80(4):1280–8. Discussion 1288–1289.
16. Wynn MM, Mell MW, Tefera G, Hoch JR, Acher CW. Complications of spinal fluid drainage in thoracoabdominal aortic aneurysm repair: a report of 486 patients treated from 1987 to 2008. J Vasc Surg. 2009;49(1):29–34. Discussion 34–25.
17. Weaver KD, Wiseman DB, Farber M, Ewend MG, Marston W, Keagy BA. Complications of lumbar drainage after thoracoabdominal aortic aneurysm repair. J Vasc Surg. 2001;34(4):623–7.
18. Horlocker TT, Wedel DJ, Rowlingson JC, Enneking FK, Kopp SL, Benzon HT, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (third edition). Reg Anesth Pain Med. 2010;35(1):64–101.
19. Fedorow CA, Moon MC, Mutch WA, Grocott HP. Lumbar cerebrospinal fluid drainage for thoracoabdominal aortic surgery: rationale and practical considerations for management. Anesth Analg. 2010;111(1):46–58.
20. Weisbord SD, Palevsky PM. Prevention of contrast-induced nephropathy with volume expansion. Clin J Am Soc Nephrol. 2008;3(1):273–80.

21. Sterling KA, Tehrani T, Rudnick MR. Clinical significance and preventive strategies for contrast-induced nephropathy. Curr Opin Nephrol Hypertens. 2008;17(6):616–23.
22. Greenberg RK, Lu Q, Roselli EE, Svensson LG, Moon MC, Hernandez AV, et al. Contemporary analysis of descending thoracic and thoracoabdominal aneurysm repair: a comparison of endovascular and open techniques. Circulation. 2008;118(8):808–17.
23. Czerny M, Eggebrecht H, Sodeck G, Verzini F, Cao P, Maritati G, et al. Mechanisms of symptomatic spinal cord ischemia after TEVAR: insights from the European Registry of Endovascular Aortic Repair Complications (EuREC). J Endovasc Ther. 2012;19(1):37–43.
24. Jacobs MJ, Elenbaas TW, Schurink GW, Mess WH, Mochtar B. Assessment of spinal cord integrity during thoracoabdominal aortic aneurysm repair. Ann Thorac Surg. 2002;74(5):S1864–1866. Discussion S1892–1868.
25. Mutch WA. Control of outflow pressure provides spinal cord protection during resection of descending thoracic aortic aneurysms. J Neurosurg Anesthesiol. 1995;7(2):133–8.
26. Augoustides JG, Stone ME, Drenger B. Novel approaches to spinal cord protection during thoracoabdominal aortic interventions. Curr Opin Anaesthesiol. 2014;27(1):98–105.
27. Etz CD, Zoli S, Bischoff MS, Bodian C, Di Luozzo G, Griepp RB. Measuring the collateral network pressure to minimize paraplegia risk in thoracoabdominal aneurysm resection. J Thorac Cardiovasc Surg. 2010;140(6 Suppl):S125–130. Discussion S142–S146.
28. Muzevich KM, Voils SA. Role of vasopressor administration in patients with acute neurologic injury. Neurocrit Care. 2009;11(1):112–9.
29. Obritsch MD, Bestul DJ, Jung R, Fish DN, MacLaren R. The role of vasopressin in vasodilatory septic shock. Pharmacotherapy. 2004;24(8):1050–63.
30. Ricotta 2nd JJ, Harbuzariu C, Pulido JN, Kalra M, Oderich G, Gloviczki P, et al. A novel approach using pulmonary artery catheter-directed rapid right ventricular pacing to facilitate precise deployment of endografts in the thoracic aorta. J Vasc Surg. 2012;55(4):1196–201.
31. Nienaber CA, Kische S, Rehders TC, Schneider H, Chatterjee T, Bünger CM, et al. Rapid pacing for better placing: comparison of techniques for precise deployment of endografts in the thoracic aorta. J Endovasc Ther. 2007;14(4):506–12.
32. Chen J, Huang W, Luo S, Yang D, Xu Z, Luo J. Application of rapid artificial cardiac pacing in thoracic endovascular aortic repair in aged patients. Clin Interv Aging. 2014;9:73–8.
33. Lee WA, Martin TD, Gravenstein N. Partial right atrial inflow occlusion for controlled systemic hypotension during thoracic endovascular aortic repair. J Vasc Surg. 2008;48(2):494–8.
34. Cires G, Noll Jr RE, Albuquerque Jr FC, Tonnessen BH, Sternbergh 3rd WC. Endovascular debranching of the aortic arch during thoracic endograft repair. J Vasc Surg. 2011;53(6):1485–91.
35. Nicolaou G, Forbes TL. Strategies for accurate endograft placement in the proximal thoracic aorta. Semin Cardiothorac Vasc Anesth. 2010;14(3):196–200.
36. Bell D, Bassin L, Neale M, Brady P. A review of the endovascular management of thoracic aortic pathology. Heart Lung Circ. 2015;24(12):1211–5.