Endovascular Aortic Repair in the Descending Thoraco-Abdominal Aorta
A Guide to Perioperative Management

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I Introduction
In 1991, the first cases of endovascular repair of the abdominal aorta, using a synthetic tube graft were described (1). Despite the smaller surgical incision, the absence of aortic cross clamping and a dramatic decline in fluid shifts, a large proportion of patients with thoraco-abdominal aneurysms were not candidates for endovascular exclusion of their aortic aneurysms, mainly due to challenges in creating an adequate seal with these stents to avoid continuous aneurysm expansion (endoleaks), while avoiding the risk of graft migration and major vessel occlusion.

In the last 20 years several generations of endovascular stent grafts have helped extend the inclusion criteria to elective and emergent repair of thoraco-abdominal aneurysms (2,3,4), type B aortic dissections (5) as well as traumatic aortic injuries. (6)

II Evolution and Types of Endovascular Aortic Grafts
Endovascular stent grafts are fabric or synthetic tube grafts reinforced by a wire frame that can be collapsed within a catheter for delivery and deployed within the aortic lumen via expansion with a ballooning catheter.

The endovascular stent graft is designed to be deployed within the aorta to span the length of the aneurysm and exclude blood flow into the aneurysm cavity.

Endovascular stent repair requires the existence of a 1-cm long non tapered region of aorta on either end of the aneurysm, often called the aneurysm neck, to provide a landing zone for each end of the graft. Furthermore, aneurysms that span aortic branch vessels require either extra-anatomic bypass (also called aortic debranching) or coverage of the branch vessels to accomplish endovascular stent repair.

Endovascular aortic repair (EVAR) in the thoracic location is typically referred to as TEVAR (Thoracic endovascular aortic repair) while EVAAR refers to endovascular abdominal aortic repair.

Tube grafts The first “endografts” were aorto-aortic tube grafts with balloon-expandable stents stitched to either end. These were effective in the short term but implantation sites in the distal aorta proved to be subject to a high failure rate due to underestimation of atheromatous disease in the aorto-iliac segment and continued expansion of the distal aorta.

Aorto-uni-iliac stent grafts This triggered the use of aorto-uni-iliac stent grafts which required simultaneous occlusion of the contralateral iliac segment combined with a femoro-femoral cross over graft to maintain blood flow to the contralateral leg.

Modular bifurcated stent grafts In the mid-1990s, modular bifurcated endografts were developed to preserve the normal anatomic configuration of the aorto-iliac segments and to enable devices to be applied to a wide range of vascular anatomy by allowing the pairing of different iliac sizes with each modular “body” section(7). They may consist of two or three components (modules), depending on whether one or both iliac limbs are modular. Proximal fixation may consist of an infrarenal sealing stent (with or without hooks) or a suprarenal bare stent to provide “transrenal fixation.”

Fenestrated stent grafts Endovascular grafts with fenestrations, which are openings within the graft fabric to accommodate visceral arteries, have been developed to improve the proximal seal by incorporating segments of the visceral arteries into the proximal sealing zone.

Fenestration refers to the creation of a hole within the stent graft (8). The challenge is to line up this hole with the orifice of the branch artery, thereby maintaining end-organ perfusion while excluding the aneurysm (9).

Branched stent grafts Branched stent-grafts are used in the endovascular repair of aortic aneurysms that involve the origins of vital arteries (e.g.: abdominal viscera) (10). While fenestrated stent-grafts have no branches at all, just holes (i.e., fenestrations) in the wall of the primary graft, branches are added at the time of insertion when covered stents are used to bridge the gap between each fenestration and each target artery.
III Surgical Procedures to Expand the Eligibility for Endovascular Stent Grafting

1- Retroperitoneal incision/approach

While conventional endografts are generally introduced via the femoral vessels, introduction via the iliac vessels or the infrarenal aorta may be necessary if the femoral vessels are small in diameter, are heavily calcified or highly tortuous. In these cases, a Dacron graft conduit is generally sewn onto the common iliac artery or aorta via a retroperitoneal approach to allow stent graft introduction; the conduit is usually oversewn at the end of the case.

2- Hybrid Procedures

In cases of aortic aneurysms involving major branches that originate from the aorta, adequate sealing of the aneurysm requires coverage of the major aortic branches that are involved in the aneurysm. To avoid ischemic complications that occur as a result of coverage of these vessels, “hybrid” approaches, i.e.: a combination of open surgical and endovascular stenting procedures have been developed to expand the “anatomic” suitability for endovascular stenting (11, 12, 13, 14). Even though these procedures involve open surgical procedures, they are typically thought to be less invasive than traditional open surgical repair. Hybrid procedure can either be staged or can be done simultaneously in the same operative setting.

A - Left carotid Subclavian Bypass:

Up to 40 % of patients undergoing TEVAR have lesions requiring coverage of the ostium of the left subclavian artery (LSA) to obtain proximal seal (proximal landing zone) (12). In these patients as well as in selected patients who have an anatomy that compromises perfusion to the brain, spinal cord, heart, or left arm, routine preoperative LSA revascularization is strongly recommended prior to TEVAR.

B- Staged Elephant Trunk Procedures:

For patients with aneurysms involving the transverse arch with no adequate proximal landing zone but adequate distal landing zone, a first-stage total arch replacement (stage I elephant trunk procedure) may be performed to treat the proximal aortic pathology and create a proximal landing zone using cardiopulmonary bypass and deep hypothermic circulatory arrest. This is followed at a later setting with the second stage endovascular repair utilizing the elephant trunk graft as the proximal landing zone (14).

C- Aortic Visceral Debranching

To allow for endovascular stenting of aortic aneurysms that involve major visceral branches such as the celiac, superior mesenteric, inferior mesenteric vessels and renal vessels without the need for conventional open repair that involves aortic cross-clamping, a visceral debranching procedure can be used, either at the same setting immediately prior to endovascular stenting or as a first part of a “staged” procedure (11). Following a midline laparotomy, and systemic heparinization to a goal ACT >300 seconds, the visceral debranching procedure is performed using a custom designed multibranched graft with distal anastomoses to the left renal artery, SMA, celiac axis, and right renal artery. Inflow for visceral debranching is typically performed via a single proximal anastomosis from the left iliac system, infrarenal aorta, or existing infrarenal aortic graft.

IV Indications and Contraindications to EVAR

Elective surgical intervention (open or endovascular) is usually indicated for abdominal aortic aneurysms more than 5 cm in diameter (>5.5 cm for TAA), those growing by more than 1 cm yearly (3 mm yearly for TAA) as well as for symptomatic aortic aneurysms (15, 16, 17).

Surgical candidates with significant comorbid medical diseases (also referred to as physiologic risk) are typically considered for EVAR due to the high surgical morbidity and mortality associated with open surgical repair in that patient population.

EVAR is also indicated in type B aortic dissections and traumatic aortic injuries (5, 6, 18, 19)

Before a decision can be made on EVAR eligibility (table 1), aortic imaging and angiographic “roadmapping” are performed (sometimes referred to as anatomic risk, which denotes the suitability of patients’ anatomy for EVAR). Aortic imaging can identify patients with aortic anatomy that either precludes the use of endovascular stents, requires the use of custom fenestrated or branched stent grafts, or necessitates the use of staged and or hybrid procedures.
Table 1: Assessment of EVAR eligibility by Imaging:

1- Proximal aneurysm neck:
   - Length: > 15 mm
   - Diameter: < 30 mm
   - Angulation: < 60 degree angulation in long axis
   - Thrombus: < than 2 mm layer of mural thrombus

2- Distal landing zone: Adequate diameter and length

3- Iliac arteries: Absence of aneurysms and occlusive disease

4- Access arteries: Adequate diameter, absence of occlusive disease

V Preoperative evaluation and preparation of patients undergoing EVAR

Patients presenting for EVAR typically have a high incidence of medical comorbidities (cardiac, pulmonary, renal, neurologic).

Despite the fact that EVAR is associated with less fluid shifts, absence of aortic cross clamping and a smaller surgical incision, it should be considered a high risk surgery (based on a perioperative risk of major adverse cardiac events > 5%) and patients should undergo functional testing when indicated based on the ACC/AHA guidelines for preoperative evaluation of patients undergoing non cardiac surgery, especially if the results of functional testing will alter management (20).

Perioperative renal protection:

Acute kidney injury (AKI) following EVAR is multi-factorial in nature and can occur as a result of hypoperfusion, mechanical encroachment of the stent graft on the renal vessels and emboli to the renal arteries. One of the common causes of acute kidney injury in patients undergoing EVAR is radiographic contrast induced nephropathy and occurs in 2-10 % of patients exposed to intravascular radiographic contrast agents (21, 22). Reduced GFR is an independent predictor of mortality in patients undergoing EVAR (24).

Strategies to reduce the incidence of contrast induced AKI are listed in table 3. Perioperative hydration and maintenance of a normal cardiac output and stable hemodynamics appear to be the most important strategies in preventing contrast induced AKI (24). (Table 5)

Table 3 Strategies to reduce the incidence of acute kidney injury following EVAR

1- Ensure perioperative euvolemia
2- Maintain cardiac output and blood pressure
3- Limit contrast dye exposure
4- Use iso-osmolar non-ionic contrast dye
5- Pharmacologic strategies especially in patients with baseline chronic kidney disease:
   a. N-Acetylcysteine
   b. Sodium bicarbonate
   c. Statin drugs

VI Choice of Anesthetic Management

EVAR has been safely done using local infiltration anesthesia, central neuraxial blockade (spinal, epidural) as well as general anesthesia.

A- Local anesthesia with monitored anesthesia care

Several centers perform EVAR cases safely under local anesthesia (25, 26). It is important to note that the mode of anesthesia should not reduce the level of perioperative monitoring (see section on choice of intraoperative monitoring). Table 4 lists the conditions that should be present before local anesthesia for EVAR is attempted

Table 4 Factors affecting the use of local anesthesia for EVAR

- Patient is able to lay in the supine position for 1 – 2 hours (no orthopnea)
- Patient cooperation and understanding that a “deep level of monitored anesthesia care” will not be feasible, since patients will be periodically asked to hold their breath during angiography
- Favorable iliofemoral anatomy making a retroperitoneal incision for iliac artery access unlikely
- Favorable aneurysm anatomy (no need for fenestrated or branched stent grafts), with an expected surgical duration less than 2 hours.
B-Central Neuraxial Blockade:
Both spinal and lumbar epidural anesthesia has been safely used in EVAR cases (27). Some centers have reported beneficial effects with the use of regional anesthesia, e.g. less vasopressor use, shorter ICU and hospital stay. Other studies have reported equal efficacy and safety when compared to general anesthesia (27, 28). It is likely that there is a selection bias in the non randomized trial documenting better outcomes with regional anesthesia since patients with less comorbidities undergoing "straight forward" EVAR are more likely to receive central neuraxial anesthesia. Lumbar epidural anesthesia allows the administration of titrated doses to avoid a sympathectomy and subsequent hypotension. It also allows the use of short and intermediate acting local anesthetics (lidocaine, mepivacaine) so that post operative neurologic examination for absence of lower extremity neurologic deficits can be performed as early as possible (Table 5).

Table 5 Factors influencing the choice of central neuraxial anesthesia for EVAR
(In addition to the factors influencing the choice for local anesthesia):
- No contraindications to central neuraxial blockade, e.g.: patient approval, patients not on anticoagulation or platelet inhibitors (thienopyridines)
- No need for transesophageal echocardiography
- No need for MEP or SSEP monitoring
- No need for measures to achieve a motionless field (use of adenosine or transvenous pacing) during stent deployment.

C-General Anesthesia:
Factors influencing the choice of general anesthesia are listed in table 6.
Regardless of the mode of anesthesia, the intraoperative anesthetic goals during EVAR are to maintain hemodynamic stability, and preserve perfusion to vital organs including the brain, heart, spinal cord, kidney and splanchnic vessels. Avoidance of hypertension and tachycardia reduces the imbalance in myocardial oxygen supply demand relationship and avoids the resultant ischemic acute coronary events. In addition, avoidance of hypertension and tachycardia are essential in reducing dp/dt in patients with both aortic aneurysms and dissections.
During induction of general endotracheal anesthesia, blunting of the hemodynamic response to intubation can be accomplished with short acting beta blockers such as esmolol. Newer agents such as the short acting dihydropyridine clevidipine can reduce the blood pressure without a reflex increase in heart rate. It is important to avoid long acting antihypertensive agents since postinduction hypotension commonly occurs in this patient population. Maintenance of intravascular volume and early identification and management of bleeding is essential, especially that most bleeding in EVAR is concealed under the drapes. Table 7 lists common causes of hypotension during EVAR by pathophysiologic mechanism.

Table 6 Factors influencing the choice of General Anesthesia:
- Complicated EVAR with planned fenestrated or branched endografts due to the expected long duration
- Need for iliac artery access (through a retroperitoneal incision) since a high level of central neuraxial blockade is necessary which increases the respiratory side effects
- Planned use of Transesophageal Echocardiography
- Planned hemodynamic manipulations to create a motionless field during stent deployment
- Planned SSEP and/or MEP monitoring
- History of difficult airway especially if EVAR procedures are performed outside the operating room suite (i.e. interventional radiology or cardiology suite) where immediate expert help may not be available and access to the airway may be delayed by the fluoroscopy machine

Table 7 Causes of intraoperative hypotension during EVAR (TEVAR and EVAAR):
Hypovolemic:
- Absolute Hypovolemia due to Surgical Bleeding: (may be occult)
  - Iliac artery rupture during device introduction (more in females with small iliofemoral diameter)
  - Accidental “withdrawal” of the device during fluoroscopic manipulation resulting in bleeding from the femoral artery under the drapes (may also occur during brachial artery access)
  - Rupture of the aortic aneurysm
  - Retroperitoneal bleeding especially following retroperitoneal dissection for iliac artery conduit

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- Relative Hypovolemia due to lactic acidosis:
  - Reperfusion of lower extremities following stent deployment and device withdrawal after endovascular aortic repair
  - Visceral Ischemia during branched stent graft introduction (celiac, superior mesenteric) or coverage during TEVAR
  - Reperfusion syndrome following endovascular repair of acute aortic type B dissection

Cardiogenic
  - Perioperative myocardial ischemia
  - Guidewire manipulation near the aortic arch (aortic baroreceptors) with resultant arrhythmias
  - Over advancement of guidewire into the left ventricle resulting in hemopericardium and cardiac tamponade (29)
  - Retrograde Type A aortic dissection during TEVAR (30)

Iatrogenic:
  - Intra-arterial injection of nitroglycerin (per surgeon) into major aortic branches (e.g.: renal vessels) to prevent vasospasm
  - Maneuvers to temporarily interrupt blood flow and create a motionless field during device deployment:
    - Intravenous injection of adenosine
    - Rapid ventricular pacing to create a motionless field during device deployment
    - Right atrial inflow occlusion

Neurogenic:
  - Acute spinal artery syndrome causing paraplegia and neurogenic shock

Distributive:
  - Abdominal compartment syndrome following TEVAR for type B aortic dissection:
    - Re-establishing blood flow to ischemic gut with massive edema exacerbated by massive fluid resuscitation to treat hypotension - can result in a vicious cycle until the abdomen is opened surgically to relieve the pressure

VII Choice of Intraoperative Monitoring

CSF drainage
Cerebrospinal fluid drainage theoretically increases spinal cord blood flow by decreasing CSF pressure resulting in an increased spinal cord perfusion pressure. Spinal cord perfusion pressure is defined as distal mean aortic pressure minus CSF pressure.

The blood supply of the spinal cord is made up of 2 posterior spinal arteries (originating from the vertebral or posterior inferior cerebellar artery) and one anterior spinal artery. From the caudal end, the anterior spinal artery receives arterial collateral supply from the internal iliac artery and its branches, the middle sacral artery, and the inferior mesenteric artery, while the thoracic portion of the anterior spinal artery is supplied by radicular branches of the intercostal arteries.

The largest of the radicular branches, the artery of Adamkiewicz (arteria radicularis magna), arises directly from the aorta at T9-T12 in the majority of cases, but can arise anywhere between T5 and L5. Exclusion of this artery during aneurysm stenting can result in paraplegia (Table 8). Other postulated mechanisms are the occurrence of hypoperfusion as a result of hypotension as well as thrombosis or embolization of the arteries supplying the anterior spinal artery. The injury seen after ischemia of the spinal cord (anterior spinal artery syndrome) is manifested by loss of motor function and pinprick sensation and preservation of vibratory and position sense.

Table 8 Factors believed to cause or contribute to the development of spinal cord ischemia after TEVAR:

A- The extent of the descending thoracic aorta covered by graft due to exclusion of critical intercostal arteries at the T6 to T12 vertebral levels that supply the anterior spinal artery;
B- Previous abdominal aortic aneurysm repair explained by compromised collateral vascular supply to the spinal cord from the pelvic and hypogastric circulation as a consequence of prior sacrifice of the inferior mesenteric artery or sacrifice of the median sacral artery,
C- Injury to the external iliac artery from intravascular delivery of the stent or severe pre-existing occlusive
disease of the femoral or iliac arteries, because spinal cord collaterals originating from the iliac arteries
may be compromised.

D- Hypotension associated with an occult retroperitoneal bleed due to reduction in CSF perfusion pressure

E- Severe atherosclerosis of the thoracic aorta due to the increased risk of emboli to the anterior spinal artery

While several randomized controlled trials and meta analyses documented the efficacy of CSF drainage (to a
pressure of 10 mmHg) in reducing the incidence of spinal cord ischemia after open thoraco-abdominal repair (31,
32, 33), evidence to support the efficacy of lumbar CSF drainage to decrease the incidence of spinal cord ischemia
after EVAR procedures is limited (34, 35). There are, however, multiple reports of reversal of paraplegia after
institution of CSF drainage in patients undergoing EVTAR (36, 34). In light of proven efficacy in the open thoracic
repair literature, it is unlikely that randomized controlled trials will be conducted to prove efficacy in EVAR.

**Table 9 Indications of CSF drainage in TEVAR:**
1. Long segment thoracic aortic exclusion especially close to T6-T12 (Types I-III Crawford
   Classification)
2. Prior open or endovascular abdominal or thoracic repair due to limited remaining collateral circulation

**Table 10 Management of paraplegia following TEVAR:**
1. Elevation of Mean Arterial Blood Pressure (> 80 mmHg using alpha agonist e.g.: norepinephrine or
   phenylephrine)
   - Improves spinal cord perfusion pressure
   - Counteracts neurogenic shock caused by autonomic dysfunction as a consequence of spinal cord ischemia
2. Therapeutic CSF drainage (5-10 mmHg)
3. Repeated neurologic examination for evidence of reversal of paraplegia
4. Avoid abrupt cessation of CSF drainage by allowing gradual increase in CSF pressure followed
   by capping of CSF catheter prior to its removal.

Complications associated with lumbar CSF catheters include meningitis, epidural abscess, persistent CSF leak and
intracranial hypotension (temporal downward herniation with kinking of the posterior cerebral artery resulting in an
acute brain infarction or death, breakage or retention of catheter fragments (38), intradural spinal, epidural
hematoma, subdural (39) or intracerebellar hematoma (40).

**Somatosensory and Motor Evoked Potential Monitoring (MEP and SSEP)**

Motor and somatosensory evoked potential monitoring is utilized in patients undergoing EVAR with a high risk for
postoperative paraplegia whether due to a planned long segment thoracic exclusion (type I and II Crawford
classification) or due to limited collateral circulation in patients with prior open or endovascular aortic repair (41).
The main goal of intraoperative neurophysiologic monitoring is to ensure adequate spinal cord perfusion throughout
the EVAR procedure and to immediately identify spinal cord ischemic changes that require prompt intervention,
typically by augmenting MAP and instituting CSF drainage. (42).

Central neuraxial anesthesia is contraindicated if SSEP or MEP is planned. During general anesthesia, inhaled
anesthetic concentrations should be maintained at 1/2 minimum anesthetic concentration (MAC) when performing
SSEP since high inhaled anesthetic concentrations attenuate cortical signals and neuromuscular blocking drugs
should be avoided when performing MEP neuromonitoring. Hypothermia attenuates signals with MEP and SSEP
and should be avoided.

Loss of SSEP and MEP signals is not specific to spinal cord ischemia and (amplitude) can also occur as a result of
peripheral nerve ischemia and lower extremity vascular malperfusion whether due to arterial cannulation or
atheroemboli to lower extremities.

Since most endovascular stents are non retrievable (cannot be retrieved once deployed), some centers do not use
intraoperative SSEP or MEP and rely on early postoperative identification and management of neurologic deficits.
However, the inflation of a balloon in the intended area of aortic segment exclusion can mimic stent deployment and
can identify the occurrence of SEP and or MEP changes before permanent endovascular stenting, triggering early
augmentation of spinal cord perfusion pressure (by increasing MAP and reducing CSF pressure)

**Transesophageal Echocardiography**
Although the primary diagnosis of aortic aneurysm or aortic dissection will have already been established in patients undergoing EVAR in the abdominal or thoracic location, TEE has multiple roles in the perioperative management of these patients.

**Indications and applications of TEE in EVAR** (43, 44, 45)
- Avoidance of high contrast dye load exposure:
- Diagnosis and confirmation of aortic pathology:
- Identification of guidewire, sheath and endograft location within the aorta:
- Detection of endoleaks:
- Cardiac assessment:

**VIII Hemodynamic Manipulation during Aortic Endograft Deployment**

Newer generations of self-expanding aortic endografts only require a temporary reduction in mean arterial pressure and heart rate during stent deployment. It is important to note that the goal is not to reduce blood pressure per se but to temporarily reduce blood flow (cardiac output) in the aorta during deployment until maximum expansion, apposition and sealing of the endograft against the aortic wall occurs. The titrated use of short acting agents to reduce heart rate (e.g.: esmolol gtt with a target heart rate of 50 - 60 /min) and systemic blood pressure (e.g.: sodium nitroprusside, nitroglycerin, clevidipine with a target mean arterial pressure of 60 -70 mmHg) can safely achieve this goal while avoiding residual postdeployment hypotension with its deleterious effects on organ perfusion (including spinal cord perfusion).

For endograft deployment in the proximal descending thoracic aorta, as well as for newer endograft technique involving the aortic arch and the ascending aorta, endograft deployment requires an entirely “motionless” field. The rationale for this approach is threefold:

1- Once deployed, almost all thoracic endografts cannot be repositioned
2- Proximal and distal landing zones of thoracic and aortic arch endografts are typically dangerously close to major vessels originating from the aorta (aortic branch vessels) with catastrophic effects if stent migration or encroachment occurs.
3- Even with the stent graft perfectly positioned, deployment of the graft can be complicated secondary to the high volume of blood flow in the thoracic aorta and the potential for the “windsock effect,” which is the tendency for the graft to be pulled distally before deployment is complete.

Techniques used to achieve a motionless field include the use of adenosine, transvenous pacing as well as right atrial inflow occlusion (46)

**IX Complications of EVAR**

**Early complications:**
Include iliofemoral lacerations, acute kidney injury, pelvic and lower extremity ischemia, myocardial ischemia, paraplegia, stroke and postimplantation syndrome.

Postimplantation syndrome occurs during the early postoperative period and is characterized by leukocytosis, fever, and elevation of inflammatory mediators such as C-reactive protein, interleukin-6, and tumor necrosis factor (47). For TEVAR, development of either unilateral or bilateral reactive pleural effusions is not uncommon, with a reported incidence of 37 to 73% (48).

While cases of thrombocytopenia as a result of “consumption” of platelets in the fabric graft have been reported, clinically significant thrombocytopenia, coagulopathy and fibrinolysis after EVAR are rare (47).

**Late complications:**
Include device migration, endoleaks with resultant aneurysm rupture, endograft infection, as well as long term effects of radiation exposure (carcinogenesis, skin burns) (49).

Endoleak is defined as the persistence of blood flow outside the lumen of the endograft but within an aneurysm sac or adjacent vascular segment being treated by the graft.

**Classification of endoleaks**
Type I endoleak: Involves the proximal or distal seal zones. Further ballooning or placement of another graft may be necessary to achieve seal. Vigorous proximal ballooning may be hazardous; retrograde proximal aortic dissection has been reported.
Type II endoleak: Unusual in the thoracic aorta, but due to retrograde flow from intercostal arteries into the sac. Typically resolves with conservative management.

Type III endoleak: Occurs with inadequate overlap and seal between modular components. Usually responds with further ballooning or additional graft or stent placement.

Type IV endoleak: Occurs due to porosity of the graft, which is a rare occurrence with current-generation devices.

Type V endoleak: Otherwise known as “endotension,” occurs in the setting of continued sac expansion despite absence of an identifiable endoleak on subsequent imaging studies and may be due to the limitations in imaging technology.

While types II and IV are considered benign, especially if not associated with an increase in aneurysm sac diameter, types I and III require interventions such as placement of extension cuffs to prevent aneurysm rupture.

X Outcomes

Most trials comparing EVAAR with open surgery have shown a reduction in short term (perioperative and or 30 day) mortality\(^{(50, 51, 52, 53, 54)}\). Most studies show that the beneficial mortality trend is lost over time, as early as at one year \(^{(55)}\) in some trials and as late as at 4 years in others \(^{(53)}\). In a recent study, however, the early perioperative mortality advantage was not offset by increased morbidity and mortality in the first 2 years after repair \(^{(54)}\). Longer term data are needed to fully assess the relative merits of the 2 procedures.

The Dutch Randomized Endovascular Aneurysm Management (DREAM) trial \(^{(50)}\) randomly assigned 351 patients with asymptomatic AAAs greater than 5 cm in diameter with anatomy suitable for EVAR to open or endovascular repair. A strong trend toward a 30-day benefit in mortality favored EVAR in this study (1.2% for EVAR vs 4.6% for open surgery; \(P = 0.10\)). Two-year follow-up data demonstrated that by 1 year, this trend toward improved survival was lost, with no mortality benefit using EVAR \(^{(55)}\).

The EVAR Trial 1 (EVAR 1), compared EVAR to open surgical repair in 1082 patients with suitable EVAR anatomy and aneurysms \(\geq 5.5\) cm \(^{(51)}\). Blood product use and length of hospital stay favored EVAR, as did perioperative mortality (1.7% for EVAR vs 4.7% for open surgery; \(P = 0.009\)) \(^{(52)}\). However, the primary end point of all-cause mortality did not show a lasting benefit for EVAR at the 4-year study conclusion.

Long-term complication and reintervention rates also were higher in the EVAR group, but a reduction in aneurysm-related death was noted (3.5% for EVAR, 6.3% for open surgery; \(P = 0.02\)) \(^{(51)}\).

Using Medicare administrative data, Schermerhorn et al \(^{(53)}\) compared patients undergoing open repair with those undergoing endovascular repair in the United States between 2001 and 2004. 22,830 matched patients were available for study in each cohort. Perioperative mortality was 1.2% after EVAR and 4.8% after open repair (95% CI, 3.51–4.56; \(P < 0.001\)). Long-term survival rates reflected an early mortality benefit for EVAR that lasted more than 3 years, at which time mortality rates converged. By the fourth year, the rate of AAA rupture was significantly higher in the EVAR group (1.8% for EVAR vs 0.5% for open repair; \(P < 0.001\)), as were reintervention rates related to AAA (9.0% for EVAR vs 1.7% for open repair; \(P < 0.001\)).

Finally, Lederle et al \(^{(54)}\), in a multicenter clinical trial, randomized 881 veterans from 42 Veterans Affairs Medical Centers with eligible AAA to EVAR versus open repair. Perioperative mortality (30 days or inpatient) was lower for endovascular repair (0.5% vs 3.0%; \(P = .004\)), but there was no significant difference in mortality at 2 years (7.0% vs 9.8%, \(P = .13\)). Patients in the endovascular repair group had reduced median procedure time (2.9 vs 3.7 hours), blood loss (200 vs 1000 mL), transfusion requirement (0 vs 1.0 units), duration of mechanical ventilation (3.6 vs 5.0 hours), hospital stay (3 vs 7 days), and intensive care unit stay (1 vs 4 days), but required substantial exposure to fluoroscopy and contrast. There were no differences between the 2 groups in major morbidity, procedure failure, secondary therapeutic procedures, aneurysm-related hospitalizations, health-related quality of life, or erectile function.

Most randomized trials reporting on intermediate and long term mortality only included EVAR in the abdominal position (EVAAR) versus open surgery and did not randomize patients with thoraco-abdominal aneurysms, mainly because endografts in the thoracic position have only received FDA approval in March 2005. Despite promising results of TEVAR versus open repair in the reduction of perioperative morbidity, mid term results are less promising and large randomized trials addressing long term outcomes are still needed \(^{(56, 57, 58, 59)}\).
In a retrospective database review, Stone et al (60) compared the perioperative and 48 months mortality of patients undergoing TEVAR with those undergoing open surgery during the same time period. Operative mortality was significantly lower in the TEVAR group (7.6%) versus open surgery group (15.1%) ($P$ = .09). 48 months survival was similar for both cohorts (~60%). Reinterventions were required at a nearly identical rate for open repair and TEVAR (10%) and both groups experienced similar rates of spinal cord ischemic complications (~7%). Fenestrated and branched endografts are considered investigational devices and have not been approved for general use. In a recently published retrospective review, Bakoyiannis et al (61) reported on outcomes of 155 patients who underwent fenestrated and branched endograft insertion for thoraco-abdominal aneurysms. The 30-day mortality was 7.1%, while the one year mortality was 16.1%. with 18.4% patients developing type I endoleak, 5.8% developing renal failure and 3.2% of patients developing permanent lower extremity neurologic deficits, highlighting the high morbidity and mortality associated with these procedures.

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