Abdominal Aortic Aneurysm

Abdominal aortic aneurysm is a dilatation of the abdominal aorta generally below the level of the renal arteries. Aneurysms pose an ever-present threat to life because of their unpredictable tendency to rupture or embolize. Aggressive medical, surgical and anesthetic management may be necessary.

ANESTHETIC CONSIDERATIONS:

- Elective vs. emergency / unruptured vs ruptured
- High risk surgery with >5% probability of perioperative cardiac complication and significant risk of postoperative respiratory failure
- High Risk patient population and significant comorbid medical disease
- Hemodynamic effects of cross clamping and unclamping
- Significant risk of blood loss, fluid shifts, transfusion and coagulopathy
- Postoperative pain management (neuraxial, PCA, nurse administered analgesia)
- Peri/postoperative ischemic complications: paralysis and spinal cord ischemia, postoperative renal dysfunction, ischemic bowel, hepatic ischemia, myocardial ischemia

ANESTHETIC GOALS:

1. Preoperative cardiac and respiratory optimization
2. Maintain stable hemodynamics to avoid rupture prior to clamp and maintain end organ perfusion
    a. invasive monitoring (art line, central line, echo/PA cath)
    b. vasopressors/inotropes and appropriate access to administer
    c. Anticipate hemodynamic changes with cross clamping and release
3. Adequate IV access for rapid fluid resuscitation
4. Perioperative blood conservation techniques (cellsaver) and blood product availablility for massive transfusion
5. Consider neuraxial technique combined with GA for sympathomodulation and postoperative pain control
6. Maintain normothermia
7. Consider postoperative disposition to monitored/ICU setting
8. If ruptured, consider salvage surgery and clamping prior to aggressive resuscitation

HISTORY

- Details of aneurysm (anatomy, level, size, symptoms)
- Exercise tolerance / functional capacity (> 4METs?)
- Active cardiac conditions (unstable coronary syndromes – unstable or severe angina, recent MI, decompensated heart failure, significant arrhythmias, and severe valvular disease)
- Assess for co-existing comorbidities (CAD, DM, HTN, PVD, smoking, COPD, stroke, renal insufficiency) and preoperative organ dysfunction
- AMPLE history**,** Anesthetic history, Surgical history
- Contraindications to neuraxial techniques

PHYSICAL

- Vitals
- Airway exam
- Focused cardiorespiratory exam: Assess for HTN, pulmonary edema secondary to CHF, murmurs, limb perfusion

INVESTIGATIONS

- Labs: CBC, coags, crossmatch, lytes, BUN, Cr
- Cardiac:
    - EKG
    - Non-invasive testing depending on functional capacity, presence of active cardiac conditions and cardiac clinical risk factors
        - High risk surgery (>5% cardiac risk, as per 2007 AHA guidelines)
        - Patients often have co-existing "clinical risk factors" (heart disease, CHF, CVD, DM, renal insufficiency)
- Respiratory:
    - PFT/ABG as dictated by history and coexisting respiratory diseases
- Imaging:
    - CT and aortography for definitive diagnosis of aortic dissection or ruptured AAA
    - Review CT for location of aneurysm/cross clamp location for elective cases
    - Endovascular stent-grafting of the aorta requires preoperative angiography and CT/MRI to precisely evaluate and delineate the aortic anatomy

OPTIMIZATION

- For elective repair, optimize volume status, correct anemia (Miller: maintain Hb>9.0 g/dL), optimize coagulation
- Consult cardiology, nephrology, pulmonary as necessary to optimize comorbid conditions
- Medications:
    - Continue beta-blockers, hold ACEI/ARB
    - Hold anticoagulants/antiplatelet agents and bridge if necessary
- Consider **Endovascular Aneurysm Repair** (EVAR) for high-risk patients
    - 50-60 % patients with infra-renal AAA have suitable anatomy
    - The endovascular approach can be undertaken without the large incisions, extensive dissections, prolonged aortic cross-clamp times, and significant blood loss and fluid shifts associated with open repair
    - Better short-term outcomes vs open AAA
        - Less 30 day operative mortality (~1.7% vs 4.7% in the DREAM and EVAR-1 trials) but the perioperative survival advantage with endovascular repair vs open repair is not sustained after the first postoperative year
        - Shorter hospital stay
        - Less peri-operative systemic complications

- However, the EVAR-2 trial concluded that in patients deemed unfit for open surgical repair, that endovascular repair did not improve survival over no intervention, had little effect on health-related quality of life, and was associated with a need for continued monitoring and reintervention, at substantially increased cost
- Significant aneurysm neck angulation, short neck length, large neck diameter, and severe aortic calcification currently exclude many patients from endovascular repair
- More secondary interventions required with EVAR due to migration, kinking, thrombosis and endoleak
- Long-term data still lacking, current ongoing trials
- Rate of conversion to open is 1-3%

ANESTHETIC OPTIONS

- GA only
- Regional only eg. can be done under thoracic epidural anesthesia
- Combined general+epidural anesthesia and general+spinal anesthetics
  - Some clinicians are reluctant to use epidural anesthesia for supraceliac aortic reconstruction because of concerns about concurrent heparinization and the associated incidence of paraplegia; may consider use of peridural narcotics without local anesthetics which can preserve sensory and motor function and can allow early assessment of neurologic integrity
  - Also consider the high incidence of hypotension with supraceliac aortic cross-clamping and epidural (could use epidural opioids without local anesthetics during clamping, and epidural local anesthetic can be given later, after aortic unclamping, when hemodynamics and intravascular volume have stabilized)
  - No evidence for improved organ function/morbidity/mortality or decreased length of stay with regional techniques/combined techniques vs GA/PCA
- For endovascular aneurysm repair, either local (+sedation), regional, or general anesthesia techniques can be used depending on the length and complexity of the procedure
  - A variety of regional techniques have been used, including paravertebral, spinal, continuous spinal, epidural, and combined spinal/epidural
  - Use of local or regional anesthesia may reduce ICU admission, hospital length of stay, and early complications; A mortality benefit may exist when local or regional anesthesia is used
  - The surgeon will typically gain access to the aorta via the femoral or iliac arteries

ANESTHETIC SETUP

- **Location:** The standard operating room environment for both open surgical repair of AAA and for EVAR
- **Monitors and lines:**
  - Standard CAS monitors plus 5-lead EKG and temperature monitoring
  - Large bore IV access proximal to cross-clamp
  - Pre-induction arterial line
  - Central line, CVP
  - Consider a pulmonary artery catheter:
    - The routine, nonselective use of PA catheter monitoring is not recommended for patients undergoing infrarenal abdominal aortic reconstruction
      - Only consider for patients with significant LV dysfunction (EF <30%), a history of congestive heart failure, significant renal impairment (preoperative creatinine >2.0 mg/dL), or cor pulmonale
    - Generally, CVP estimates LV filling in patients with normal LV function and pulmonary compliance
    - Some centers routinely insert a PA catheter for all patients requiring supraceliac aortic cross-clamping
    - With selective use, accurate interpretation of data, and rational treatment strategies, pulmonary artery catheter monitoring may be beneficial in high-risk patients undergoing complex aortic reconstruction; but the clinical value of pulmonary artery catheter monitoring in high-risk patients has not been established in studies
  - 2D TEE has been used intraoperatively to assess global ventricular function, guide fluid therapy, and monitor for MI
- **Drugs:**
  - Standard emergency meds
  - Vasopressors/ inotropes
  - Vasodilators (e.g. nitroglycerine, nitroprusside) and antihypertensive drugs
  - Mannitol for renal/pulmonary protection
  - Heparin and protamine
- **Other:**
  - Blood immediately available (at least 4 units in fridge or in room if emergency case)
  - Blood conservation: Cell Saver
  - Fluid warmer, forced air warmer
- **For Endovascular Aneurysm Repair:**
  - O.R. must be equipped with endovascular supplies, portable radiologic imaging tools, and an angiographic table
  - Lines and monitors:
    - Right radial arterial line for all endovascular aortic repairs (should be placed on the right side because a catheter may be placed percutaneously in the left brachial artery for aortic angiography)
    - Central venous and pulmonary artery catheter monitoring is not routine
    - Two large-bore peripheral iv's
  - The possibility of acute aortic rupture necessitates the availability of fluids, blood, and a rapid infusion device
  - Monitoring of urine output can help guide fluid management
  - Active patient warming is frequently necessary to prevent hypothermia, particularly with longer procedures

MANAGEMENT OF ANESTHESIA

Induction:

- Maintain stable hemodynamics on induction and intubation – avoid hypertension + hypotension
  - Prehydration may limit variations in blood pressure on induction of anesthesia
  - Keep the patient's vital signs within 20% of his or her normal range, as long as the heart rate does not exceed 80 to 90 beats per minute and signs of organ ischemia are absent
  - The heart rate should be maintained at or below baseline because myocardial ischemia is often related to the heart rate
- Risk of cardiovascular collapse on induction for ruptured aneurysms- have patient prepped & surgeon present
- A variety of intravenous anesthetics (thiopental, etomidate, propofol) are suitable
- Addition of a short-acting, potent opioid such as fentanyl (3-5 μg/kg) usually provides stable hemodynamics during and after induction
- Volatile anesthetics may be administered in low concentration before intubation during assisted ventilation as an adjunct to blunt the hyperdynamic response to

laryngoscopy and endotracheal intubation
- Esmolol (10 to 25 mg), sodium nitroprusside (5 to 25 µg), nitroglycerin (50 to 100 µg), and phenylephrine (50 to 100 µg) should be available for bolus administration during induction if needed to maintain appropriate hemodynamics

Maintenance:
- Any technique reasonable as long as vital organ perfusion and function is maintained
    - Technique should allow rapid and precise control of hemodynamic parameters
    - May be accomplished with a combination of a potent opioid (fentanyl or sufentanil) and an inhaled anesthetic (sevoflurane, desflurane, or isoflurane) i.e., balanced anesthesia
    - Patients with severe left ventricular dysfunction may benefit from a pure opioid technique
    - Nitrous oxide can be used to supplement either an opioid or an inhaled anesthetic (In general, nitrous oxide decreases cardiac output and arterial pressure while increasing systemic vascular resistance)
    - Volatile anesthetics provide a means of controlling afterload and preload but can increase need for intravascular volume
    - Volatile anesthetics may improve preconditioning mechanisms and reduce the size of MI, should it occur (these effects have been documented in animal models and in CABG surgery in humans, but not in vascular surgery patients)
- Consider effect of epidural on hemodynamics
    - If high or prolonged cross clamp, may want to wait until re-perfusion prior to loading epidural
    - For low thoracic or high lumbar epidural catheters, the initial bolus should be limited to 6 to 8 mL of local anesthetic. Additional local anesthetic is administered by continuous infusion at 4 to 6 mL/hr with adjustments based on hemodynamics and inhaled anesthetic requirements during surgery
- Prior to cross clamp
    - Avoid over-resuscitation (remember blood volume redistribution)
    - It is crucial to maintain temperature homeostasis (unless hypothermia is desired for its potential spinal cord protection)
    - Patient will require Heparin at ~ 100U/kg to achieve an ACT>300
    - For the half hour immediately prior to cross-clamping, keep patient slightly hypovolemic by examining the ventricular volume by means of echocardiography or by keeping pulmonary capillary wedge pressure at 5 to 15 mm Hg
- Cross clamp
    - Physiologic response:
        - Increased systemic blood pressure (HTN) and systemic vascular resistance up to 50%
            - This is attributed to a sudden increase in impedance to aortic flow (afterload), activation of renin, and release of catecholamines, prostaglandins, and other active vasoconstrictors
        - No change in hr, decreased CO
        - Decreased total body oxygen consumption, increased mixed venous oxygen saturation
        - Increased preload from collapse and constriction of venous vasculature distal to cross-clamp
        - Increased filling pressures in patients with CAD or myocardial dysfunction: increased CVP, pulmonary capillary occlusion pressure, LVEDP
        - Changes in MAP, end-diastolic and end-systolic LV area and EF, and wall motion abnormalities are minimal during infrarenal aortic cross-clamping but dramatic during supraceliac aortic cross-clamping
            - 10 to 20% of "infrarenal" aortic disease will actually involve the suprarenal portion of the aorta, necessitating suprarenal clamping
            - Ruptured aneurysms often must be controlled initially by supraceliac clamping because of anatomic considerations
        - To tolerate the increase in afterload and preload, an increase in myocardial contractility and an autoregulatory increase in coronary blood flow are required
            - If coronary blood flow and myocardial contractility cannot increase, LV dysfunction is likely
            - Strategies for myocardial preservation during and after aortic cross-clamping include decreasing afterload and normalizing preload, coronary blood flow, and contractility
            - Monitor for myocardial ischemia (increased PAP, large V-waves indicative of MR)
    - Increase anesthetic depth prior to cross clamping and have vasodilators or antihypertensives ready (e.g., may require 0.3 to 0.7 µg/kg of nitroprusside or 200 to 600 µg of nicardipine or 50 µg/kg of milrinone over 10 minutes to offset the hypertensive response after clamping)
    - Consider using 15-cm of PEEP immediately before cross-clamp, with removal just before unclamping; this approach facilitates volume loading and reduces hypotension after unclamping
    - Carefully monitor blood loss replace replace with crystalloid or colloid, warmed cell-saver blood, or banked blood to keep the hematocrit slightly above 30% because it will decrease to 30% in the postocclusion period
        - However, maintaining increased central venous or pulmonary capillary wedge pressure *during* the cross-clamp period is not indicated and may result in significant overtransfusion of fluids and blood products

Table 42-4 from Barash	PERCENT CHANGE IN VARIABLE, BY LEVEL OF AORTIC OCCLUSION		
Cardiovascular Variable	**Supraceliac**	**Suprarenal Infraceliac**	**Infrarenal**
Mean arterial blood pressure	54	5	2
Pulmonary capillary wedge pressure	38	10	0
End-diastolic area	28	2	9
End-systolic area	69	10	11
Ejection Fraction	-38	-10	-3
Abnormal motion of wall, % of patients	92	33	0
New myocardial infarctions, % of patients	8	0	0

- Removal of cross clamp
    - Physiologic response:
        - Decrease in systemic vascular resistance and systemic blood pressure
        - Cardiac output may increase, decrease, or remain unchanged
        - Left ventricular end-diastolic pressure decreases and myocardial blood flow increases
        - The principal causes of declamping hypotension include (1) central hypovolemia caused by pooling of blood in reperfused tissues, (2) hypoxia-mediated vasodilation causing an increase in vascular capacitance in the tissues below the level of aortic clamping, and (3) accumulation of vasoactive and myocardial depressant metabolites in these tissues
        - Vasodilation and hypotension may be further aggravated by the transient increase in carbon dioxide release and oxygen consumption in these tissues following unclamping
    - Anticipate clamp removal (duration & location of aortic clamp determine degree of hypotension observed)

- Therapy prior to unclamping:
  - Aggressive volume resuscitation - Crystalloid, colloid, or blood is administered just before reperfusion guided by filling pressures or echocardiographic estimates of volume
  - Reduce anesthetic and stop vasodilators
  - Have vasopressors (phenylephrine, ephedrine, epinephrine) and calcium chloride (300-500 mg) available
  - Allow higher blood pressure (raising BP 20-30% above baseline often necessary to avoid significant hypotension)
- Upon unclamping, washout of vasoactive and cardiodepressant mediators from reperfusion of ischemic tissues are washed back into circulation
  - Make prophylactic ventilatory adjustments (hyperventilate) to accommodate acid load
  - Profound acidosis my require sodium bicarbonate to adjust pH until acidosis resolves
    - However, administration of bicarbonate does not prevent immediate postunclamping hypotension and can sometimes exacerbate it, probably due to the initial increase in intracellular myocardial acidity
  - Frequent ABGs necessary to monitor acidosis
- Gradual release of cross clamp will help minimize effects and if unable to control hypotension, surgeon may have to re-apply cross clamp (be ready for rebound hypertension)
- Correction of metabolic acidosis does not significantly influence the degree of hypotension following aortic declamping

- After unclamping reverse heparin after discussion with surgeon
- Endovascular repair:
  - Prior to device insertion, systemic anticoagulation will be started with a typical heparin dose of 5,000 units with a goal of ACT ≥200 seconds
  - At the time of device deployment, awake patients will be asked to hold their breath, and mean arterial pressure is often lowered to decrease the risk of distal migration of the stent
  - After device deployment, anticoagulation is reversed and activated coagulation time rechecked

Emergence:
- Hypertension and tachycardia are aggressively controlled during emergence to prevent bleeding or damage to the anastamoses by the use of short-acting agents such as esmolol, nitroglycerin, and sodium nitroprusside
- Prophylactic beta-blockade is continued into the postoperative period, as tolerated
  - In unstable patients, esmolol may be infused; in more stable patients, metoprolol 5 to 10 mg intravenously every 6 hours, as long as heart rate is >55 beats per minute and blood pressure is >110 mm Hg
- Consider loading the epidural and ensure the epidural is working prior to extubation
- Hemodynamic, metabolic, and temperature homeostasis must be achieved before skin closure and extubation; otherwise, patients are transported to the ICU intubated and their ventilation controlled
- Ensure patients have met all extubation criteria prior to extubation, including resolution of metabolic acidosis (persistent acidosis could indicate bowel ischemia)
- Extubation of the trachea is not generally attempted in patients with supraceliac aortic cross-clamp times longer than 30 minutes, patients with poor baseline pulmonary function, or patients requiring large volumes of blood or crystalloid during surgery

Anesthetic Management for EVAR
- As with open aortic repair, maintenance of vital organ perfusion and function by the provision of stable perioperative hemodynamics is probably more important to overall outcome than the choice of anesthetic technique is
- Important to monitor urine output:
  - High volumes of heparinized flush solution and radiographic contrast material are used
  - Diuretics (i.e., mannitol or furosemide) may be administered
  - Isotonic bicarbonate infusion is often used in patients with renal dysfunction to reduce the incidence of contrast-induced nephropathy
- Fluid management is directed primarily at maintaining normovolemia
  - Be prepared for rapid blood loss or aortic rupture with massive blood loss
- For GA, a balanced technique using relatively short-acting agents maximizes management flexibility
  - Opioid requirements are usually minimal (2 to 4 μg/kg fentanyl), and postoperative pain is easily managed
  - Esmolol, sodium nitroprusside, nitroglycerin, and phenylephrine should be available and used to maintain appropriate hemodynamics
- For regional/local:
  - The surgeon will require an immobile patient as he or she strives to position modular graft components
  - Rarely, retroperitoneal vascular access is necessary, which typically makes local anesthesia inadequate

Anesthetic Management for Ruptured Abdominal Aortic Aneurysm
- Rapid diagnosis with immediate laparotomy and control of the proximal aorta are of the highest priority
- In emergency resection the crucial factor for patient survival is first, rapid control of blood loss and reversal of hypotension, and then preservation of myocardial function last
- Euvolemic resuscitation may be deferred until the aortic rupture is surgically controlled in the operating room because euvolemic resuscitation and the resultant increase in blood pressure without surgical control of bleeding may lead to loss of retroperitoneal tamponade, further bleeding, hypotension, and death
- Considerations of supraceliac clamp apply
- Massive fluid and blood resuscitation can cause hypothermia
- If hypotension related to poor myocardial contractility or coagulopathy develops, administration of calcium may be therapeutic
- Vasopressin may be particularly effective in restoring blood pressure when hemorrhagic shock is resistant to catecholamines

DISPOSITION & MONITORING
- Require close post-operative monitoring in either an intermediate or intensive care unit
  - Postoperative care depends on the physiologic derangements incurred during the perioperative period (i.e., depression of consciousness, hypothermia, fluid overload, incisional pain, ileus, and respiratory depression), as well as on the development of certain less common, but more severe postoperative complications (i.e., MI, pneumonia, sepsis, renal failure, and decreased tissue perfusion)
- There is no evidence that epidural anesthetic and analgesic techniques for aortic surgery offer any major advantage or disadvantage over general anesthesia and intravenous patient-controlled analgesia

COMPLICATIONS
- Majority of complications occur within first 2 days of surgery (MI, ARF, bowel ischemia, spinal cord ischemia, hepatic ischemia)
- Most post-op myocardial ischemia is silent
  - Peak incidence: early (day 0-3), and late (day 4-7)
  - Subendocardial ischemia most common (ST depression)
- Post-operative renal failure

- ARF (majority ATN) occurs in ~3% of patients undergoing elective infrarenal AAA with mortality > 40%
- Postoperative mortality is 4-5 fold higher in those who develop acute renal failure when compared with those who do not
- The degree of preoperative renal insufficiency remains the strongest predictor of postoperative renal dysfunction
- In addition to aortic cross-clamping–induced reductions in renal blood flow, ischemic reperfusion injury, intravascular volume depletion, embolization of atherosclerotic debris to the kidneys, and surgical trauma to the renal arteries all contribute to renal dysfunction
- The level of aortic clamping (ARF in 5% postinfrarenal clamping, 13% postsuprarenal clamping) and the avoidance of prolonged hypotension are important of all the factors, as they markedly impact renal blood flow
- Decreased renal blood flow during cross-clamp:
  - *Infra-renal*: 38-45% (due to increased renal vascular resistance 70%)
  - *Supra-renal:* 80-90%
- Intraoperative urine output does not predict postoperative renal function
- There is no renal protective strategy proven to yield superior outcome
  - Mannitol:
    - Improves renal cortical blood flow during infrarenal aortic cross-clamping and reduces ischemia-induced renal vascular endothelial cell edema and vascular congestion
    - No human studies showing a renal protective effect
    - Administer 0.125-0.25g/kg prior to aortic cross clamp
  - Furosemide:
    - Induces diuresis, theoretical benefit but no outcome studies
  - Dopamine:
    - Lack specific renal hemodynamic effects and does not appear to improve postoperative renal dysfunction
    - Low doses (3 mcg/kg/min)
  - Fenoldopam:
    - Selective DA-1 agonists that preferentially dilates the renal and splanchnic vascular beds
    - Has shown some early promise as a renoprotective drug but its role in the prevention of renal dysfunction after aortic surgery is not known
    - Generally reserved for patients with pre-existing renal dysfunction
    - Dose: 0.03 mcg/kg/min
  - Statins:
    - Associated with preserved renal function after suprarenal aortic cross-clamping
  - One of the most important factors for preventing postoperative renal failure remains good hydration (as the most important factor for maintaining renal blood flow) during clamping and postclamp release
  - If prolonged renal ischemia is anticipated, selective profound hypothermia with direct intra-arterial infusion of 4°C Ringer lactate into the kidneys may decrease the incidence of postoperative renal impairment
  - Ischemic preconditioning:
    - Intermittent cross-clamping of the internal iliacs prior to the insult may provide a stimulus for both renal and myocardial preconditioning
    - Using this technique, the incidence of renal insufficiency decreased by 23% in one study

- Spinal cord ischemia
  - Spinal cord ischemia occurs in 1 to 11% of operations involving a distal aortic repair
  - Cross-clamping the aorta distal to the left subclavian artery associated with ~90% decrease in spinal cord blood flow
    - Decrease in distal aortic-anterior spinal artery pressure and an increase in CSF pressure
  - The definitive preventive measures to spinal cord ischemia are short cross-clamping time, fast surgery, maintenance of normal cardiac function, and higher perfusion pressures
- Pulmonary damage associated with aortic cross-clamping and unclamping
  - Reflected by an increase in pulmonary vascular resistance (particularly with unclamping of the aorta), an increase in pulmonary capillary membrane permeability, and development of pulmonary edema
  - The mechanisms involved may include pulmonary hypervolemia and the effects of various vasoactive mediators.
  - Incidence of pulmonary complications is 5-14% with thoracoabdominal aneurysm repair
  - Mannitol administration before and after unclamping may be beneficial in preventing increased pulmonary vascular resistance (and increased CVP/PAP) because of its function as a hydroxyl free radical scavenger
- Bowel ischemia
  - Associated with a high postoperative mortality rate, approaching 25%
  - Factors contributing to the pathogenesis of visceral ischemia include pre-existing medical conditions, renal dysfunction, stage of aortic disease and level of aortic cross-clamping, the duration of cross-clamping, and perioperative hypotension
  - Hypoxic insult to the intestines during aortic occlusion leads to gut permeability and bacterial translocation as evidenced by activation of polymorphonuclear leukocytes
  - With endovascular repair, intestinal ischemic events seem less profound
  - High doses of methylprednisolone at induction of anesthesia may be beneficial in reducing the inflammatory response, including C-reactive protein and T-cell activation levels, but have been shown to negatively impact postoperative renal function
- Complications with endovascular aneurysm repair:
  - Endoleak: the inability to obtain or maintain complete exclusion of the aneurysm sac from arterial blood flow; the concern is that any pressurization of the aneurysm sac (i.e., endotension) can lead to aneurysm enlargement and rupture
  - Severe complications, including paraplegia, aortic rupture, stroke, renal failure, and respiratory failure, have been reported for endovascular repair involving the descending thoracic aorta

PATHOPHYSIOLOGY

- An *aneurysm* is a dilatation of all three layers of an artery
  - The most common definition is a 50% increase in diameter compared to normal
- Increased prevalence in men, up to 8% in elderly
- Risk Factors: advanced age, male, family history of AAA, HTN, atherosclerosis, smoking, low serum high-density lipoprotein cholesterol, high level of plasma fibrinogen, and low blood platelet count
- Chronic inflammation plays a fundamental role in the degradation of elastin in the aortic wall
- Rare causes: mycotic infection, syphilitic infection, Marfan's, Ehler's-Danlos
- Half of patients have asymptomatic disease but aneurysms may occasionally produce symptoms because of compression of surrounding structures
- Annual risk of rupture directly related to diameter and rate of expansion
- Larger aneurysms grow at faster rate, but will generally grow between 0.2–0.4 cm/year

Annual Risk of Rupture	
Size	Risk of Rupture (%)
< 4 cm	0
4.0 – 4.9	0.5 – 5
5.0 – 5.9	3 – 15
6.0 – 6.9	10 – 20
7.0 – 7.9	20 – 40
> 8.0	30 - 50

- Indications for surgery:
    - Aneurysms >5.5cm, as operative risk < risk of rupture
    - Aneurysms < 5.5 cm which grow at a rate of >0.5cm/year
    - Symptomatic aneurysms:
        - Tender
        - Associated with abdominal or back pain (suggestive of recent expansion)
        - Co-existing iliac or femoral artery aneurysms that require treatment
        - Co-existing occlusive disease or thrombotic / embolic complications
- Elective AAA operative mortality rate 1.2-2.4%
- Late survival rates after repair of nonruptured abdominal aortic aneurysms are 92% at 1 year and 67% at 5 years
- Ruptured AAA's:
    - With aortic dissection, blood moves through a tear in the aortic intima and separates the intima from the adventitia
        - A false lumen results, which can reconnect with the true lumen anywhere along the course of the dissection
    - For ruptured AAA's, perioperative mortality of nearly 50% has not changed significantly over the last 4 decades
    - Symptoms of ruptured AAAs include:
        - Pain in the back, abdomen, or both
        - Hypotension
        - Pulsatile mass
        - Faintness or frank collapse
        - Vomiting
    - Rupture site:
        - Most commonly occur into the left retroperitoneum, which permits tamponade of the hemorrhage
        - However, retroperitoneal hemorrhage and subsequent hematoma can displace the left renal vein, inferior vena cava, and intestine, possibly leading to damage to these structures during the surgical approach
        - ~25% of aneurysms rupture into the peritoneal cavity, a site associated with a great degree of exsanguination
        - Other sites of rupture include adjacent structures after formation of fistulae with the inferior vena cava, iliac veins, renal veins, or bowel
    - Venous hemorrhage is often much more difficult to control than arterial hemorrhage
    - Hypovolemic shock frequently accompanies rupture; however, the absence of hypotension does not rule out the possibility of rupture, and shock may occur suddenly

REFERENCES

- Co-existing Chapter 8
- Barash Chapter 42
- Miller Chapter 62