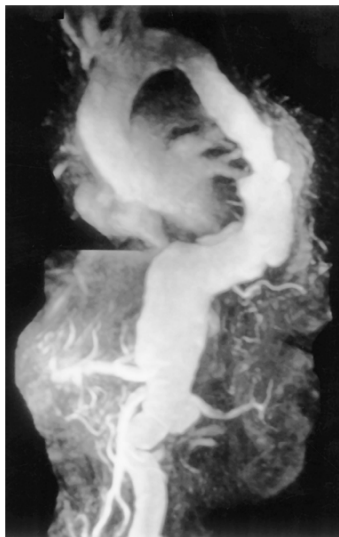




Torn Apart; Caring for the Patient with a Non-Traumatic Thoracic Aneurysm or Dissection



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Not always what you think (6)

You and you partner are called to the home of a 76 y/o male for chest pain.

Assessment:

- 10/10 “crushing” CP that radiates to his back and shoulder
- A&Ox3, tells you the pain was what aroused him from his night’s sleep
- Vitals:
 - Mild difficulty breathing, in the 30’s. BS clear

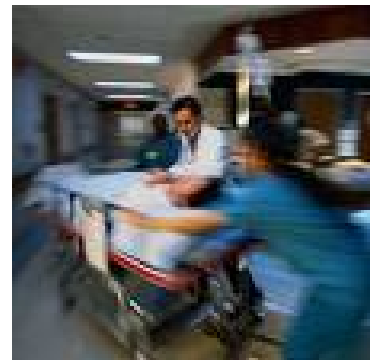


Not always what you think (cont.) ⁽⁶⁾

- CV
 - Skin: pale, cool, and moist
 - No palpable radial pulses, palpable carotid
 - Unable to obtain an accurate BP
 - 12-lead: Slight anterior ST elevation
 - HR: Tachy around 110, regular
- GI
 - Vomiting
- GU/MS
 - Benign
- PMH:
 - HTN, Gastritis, Prostate CA, Sleep Apnea, and Tobacco Use
 - Had a “complete” physical and stress test with his PCP recently, reported to be in “excellent” shape.



Differential Diagnosis





Lots of Possibilities

- Gastritis/GI event
- PE
- Pneumonia/Septic Shock
- MI
- Internal hemorrhage
- And of course, an aneurysm/dissection

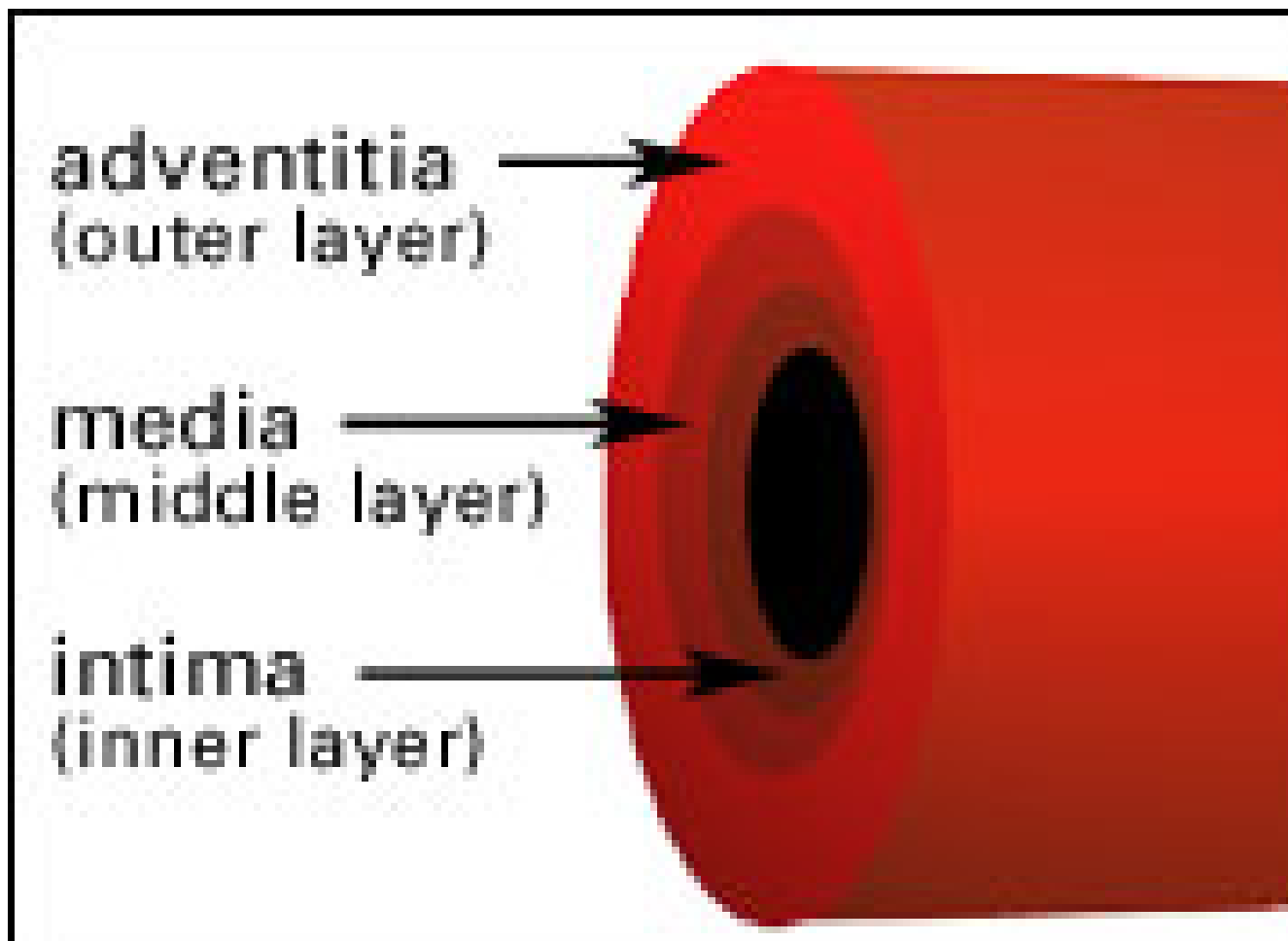


Objectives

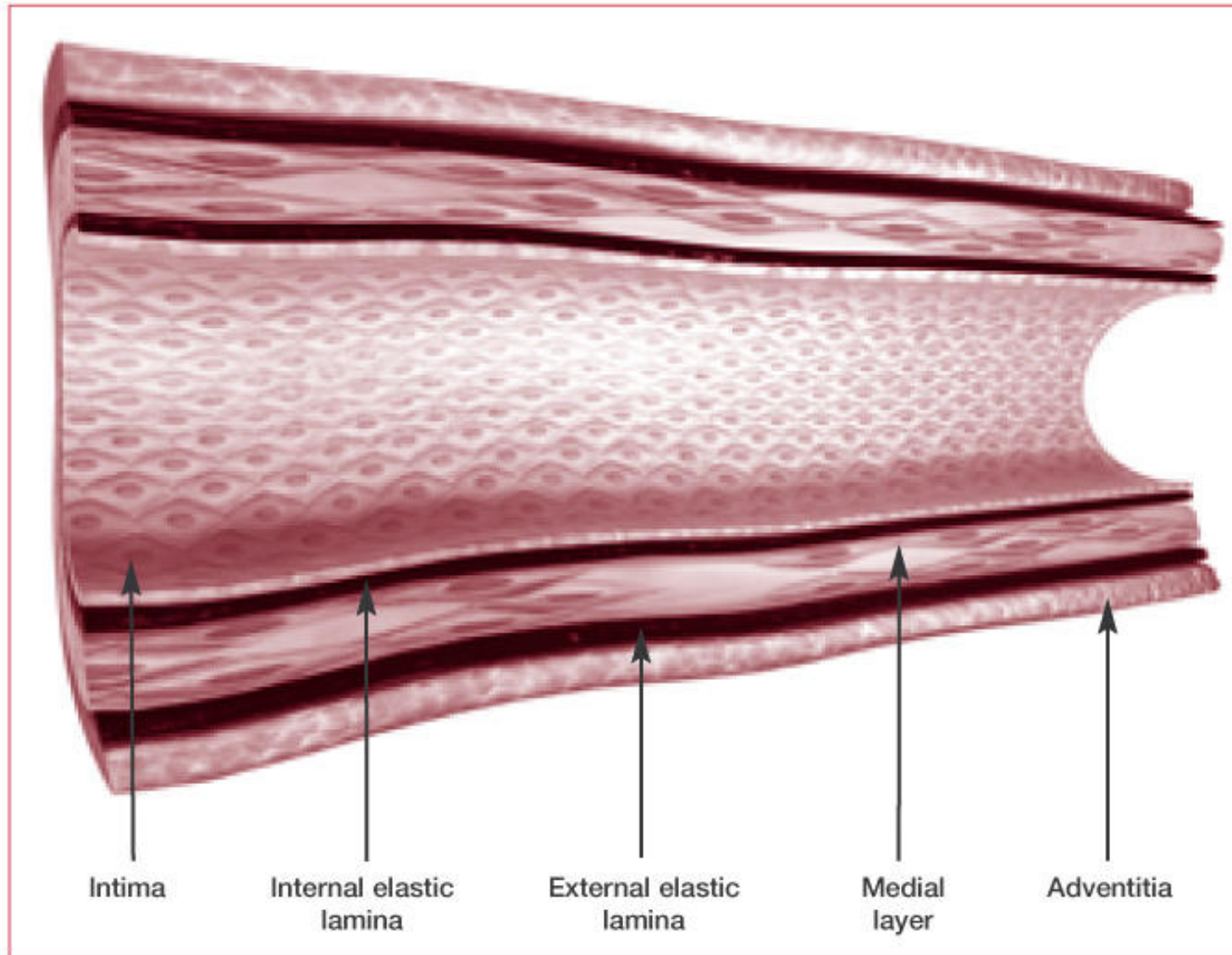
The participant will be able to:

- Identify at least 2 unique characteristics of a non-traumatic TAA
- Assess radiological and clinical findings of a patient experiencing a TAA
- Familiar with 1st line therapy for the pt having a TAA

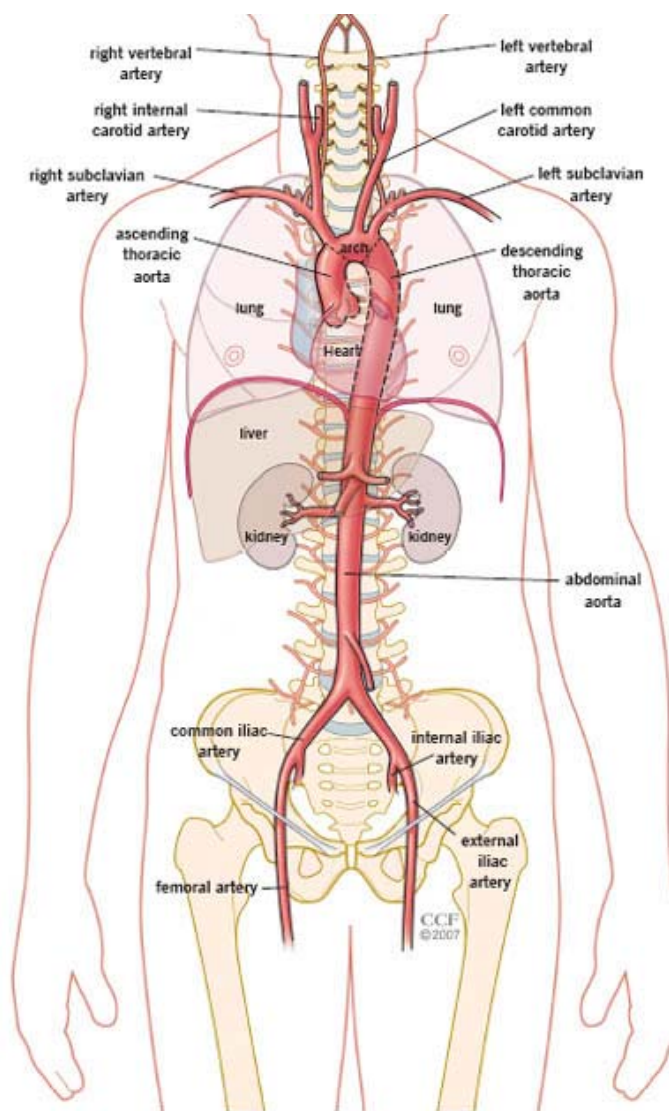
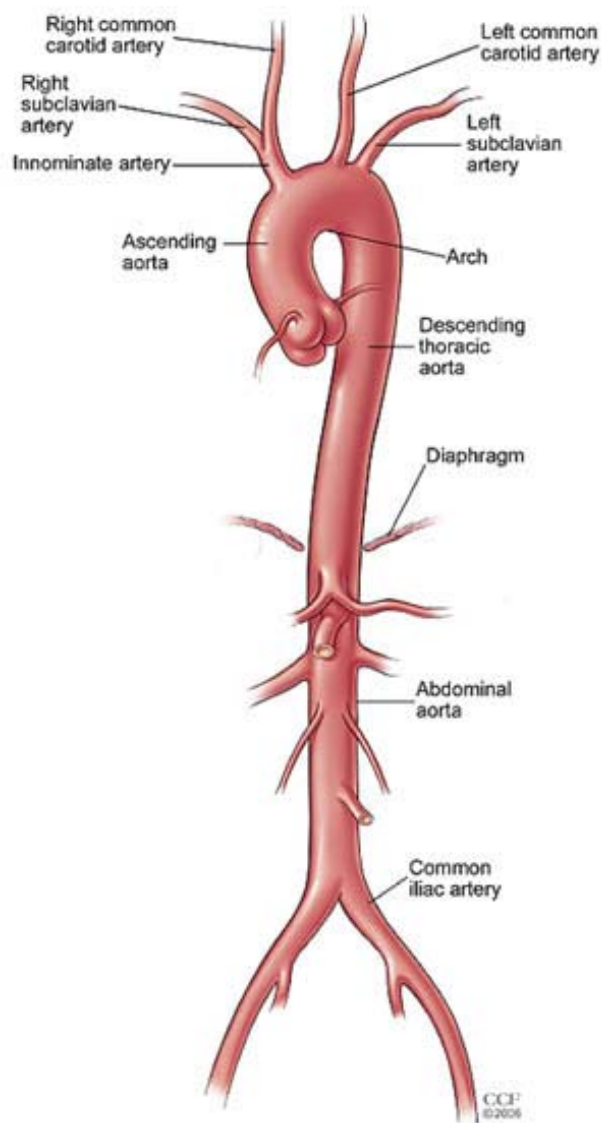
Let's Review



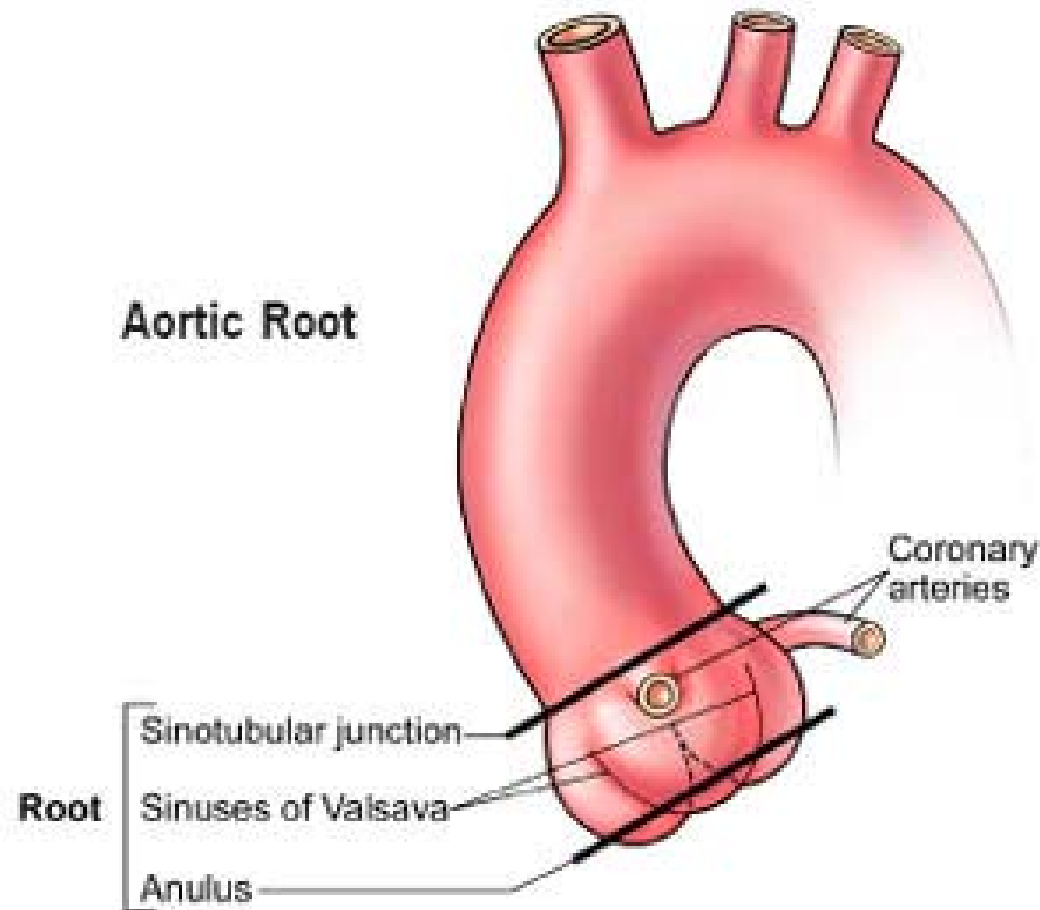
Let's Review: The Aorta



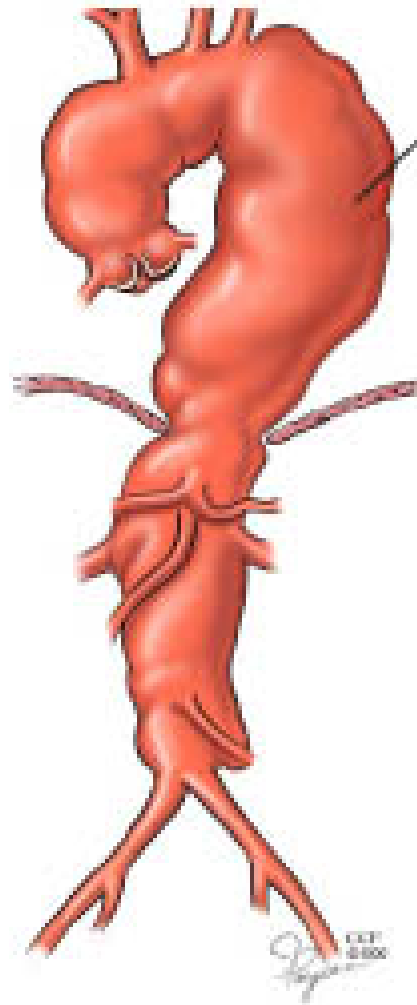
The Thoracic Aorta



Thoracic Aorta: Coronary Flow



Aneurysms



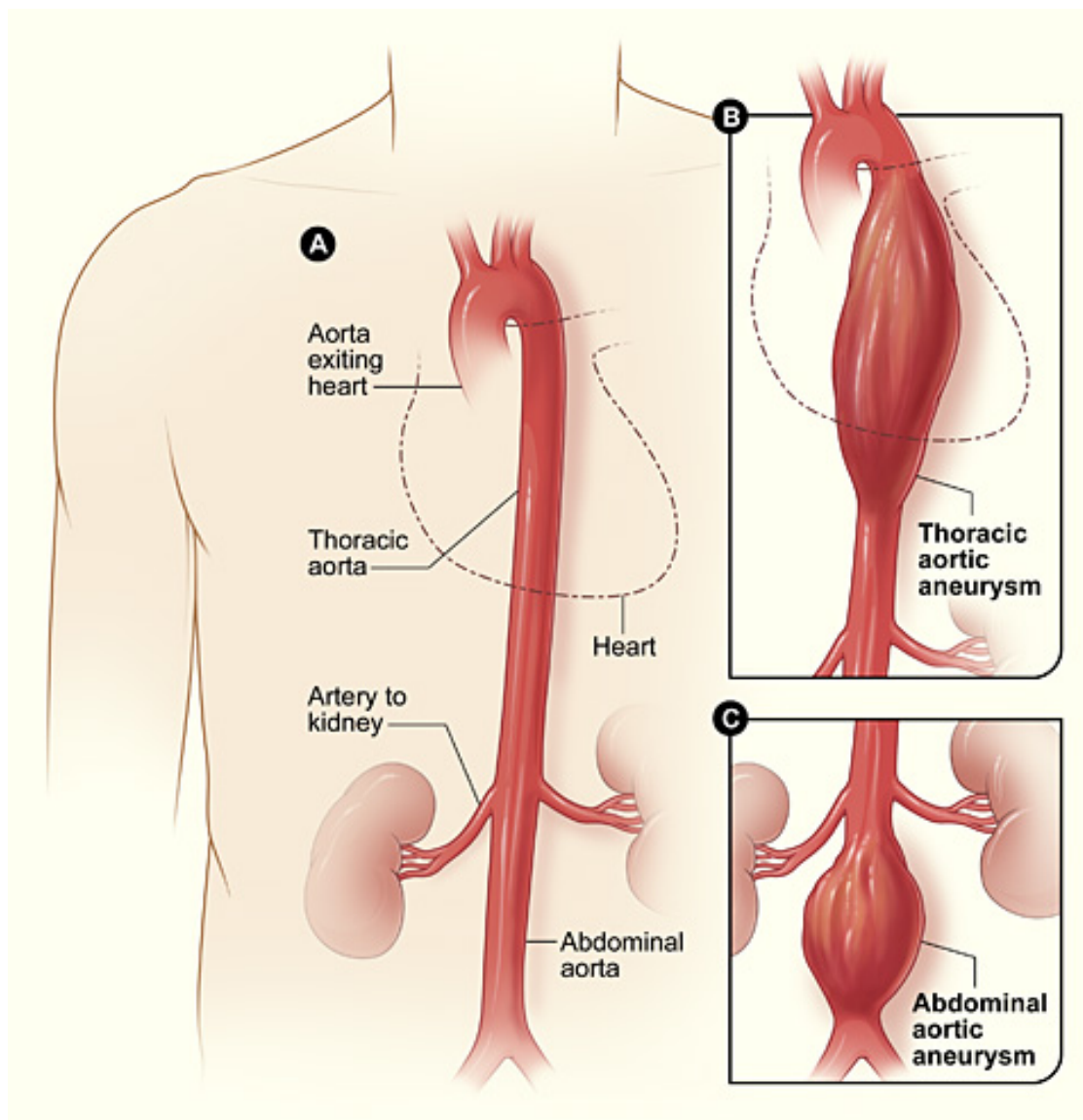
Aortic Aneurysm

An aortic aneurysm is an abnormal enlargement or bulging of the wall of the aorta.

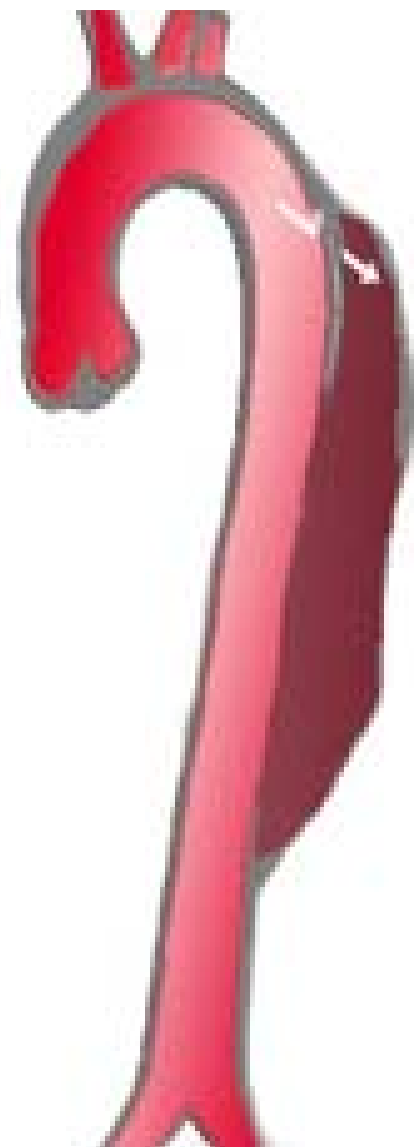
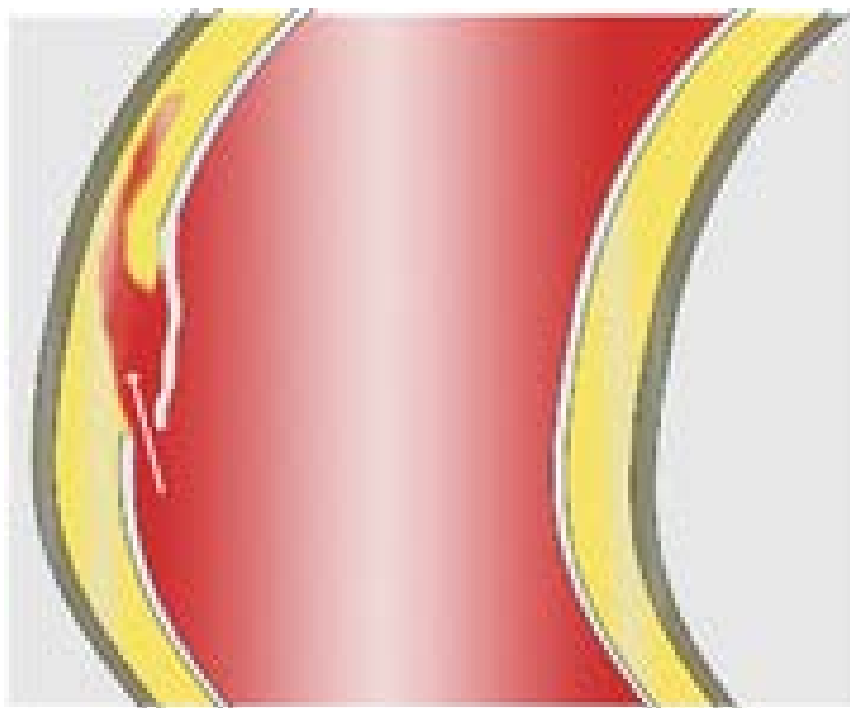
An aneurysm can occur anywhere in the vascular tree.

Once an aneurysm is diagnosed, treatment may be needed, depending on the size of the aneurysm. Ruptured aneurysms require emergency surgery to stop the bleeding.

Where are Aneurysms?



Dissection



Type III or B

Classification of Dissections

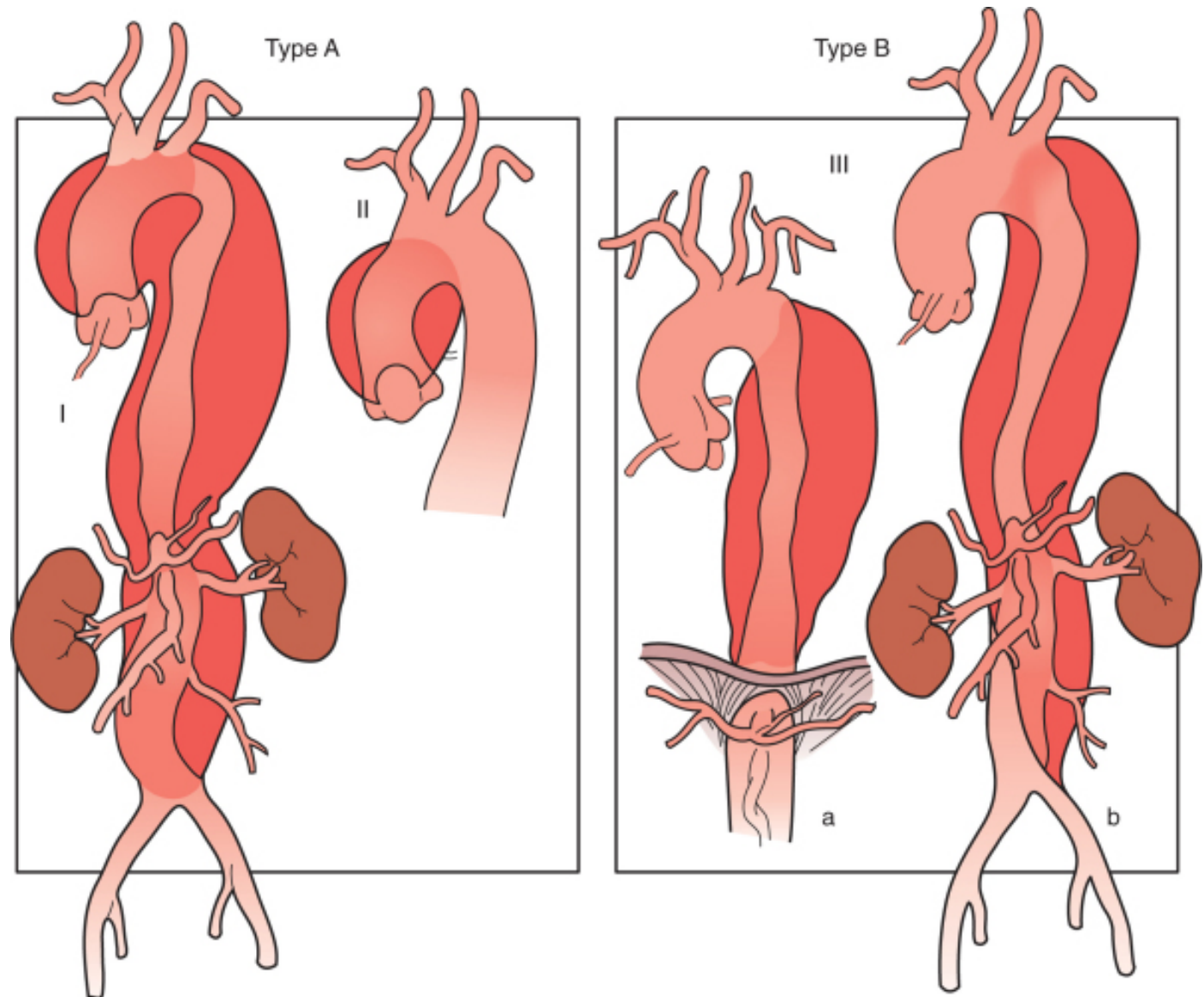
DeBakey:

I, II, III

Stanford: (more
commonly used)

A (I and II)

B (III)





Acute Aortic Syndromes

Conditions that lead to acute or chronic changes to the lining of the aorta

- The “Big” three
 - Aortic Dissection
 - Intramural Hematoma
 - Penetrating Atherosclerotic Ulcer

Aortic Rupture

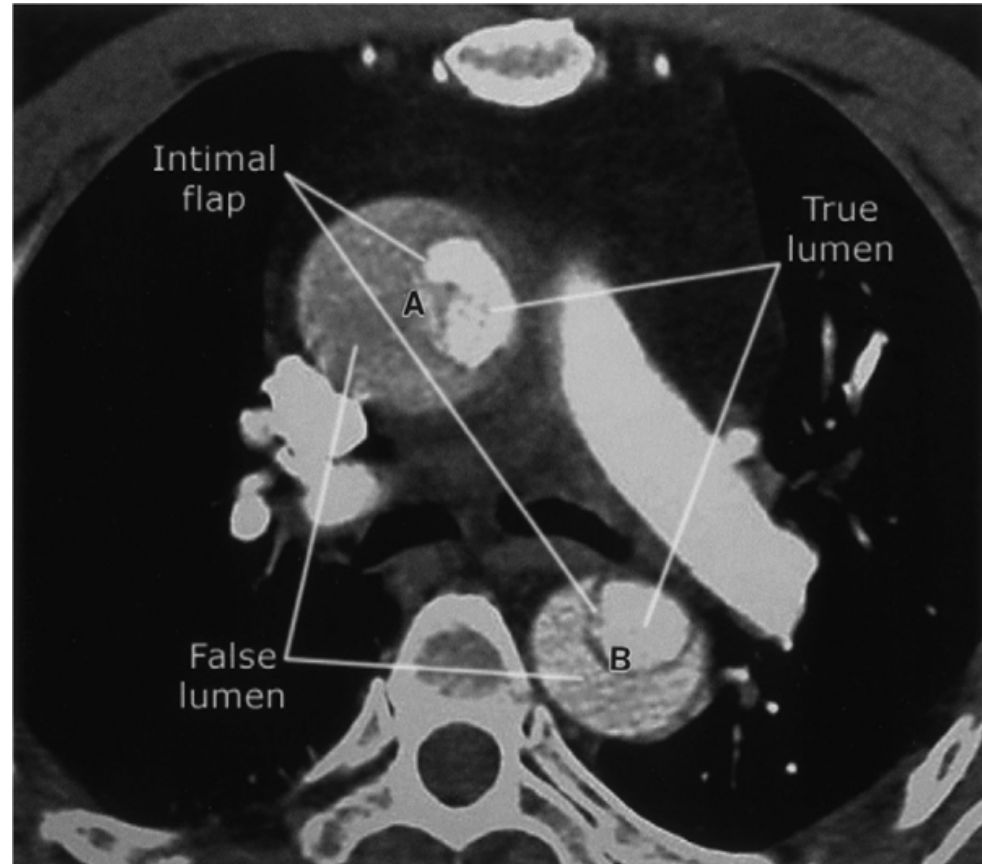
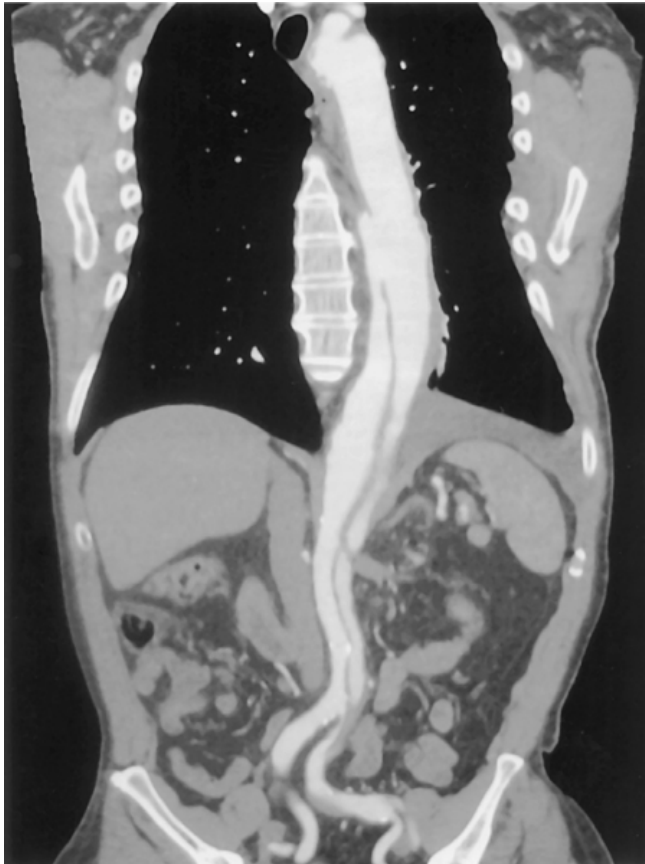
100% Mortality if not repaired



Aortic Dissection⁽¹⁾

- Intimal damage from disease leading to elastic degeneration and smooth muscle cell loss. As the intima damage continues there is separation on the layers allowing blood to enter (A false lumen occurs).
- Common cause
 - The combination of hypertension and atherosclerosis
 - Leads to necrosis and fibrosis of the elastic components leading to stiffness and weakness

Aortic Dissection

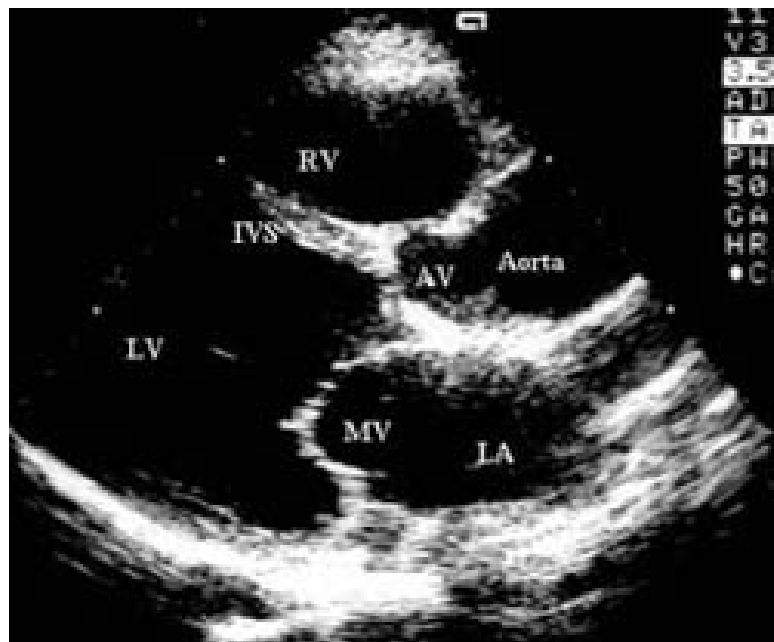




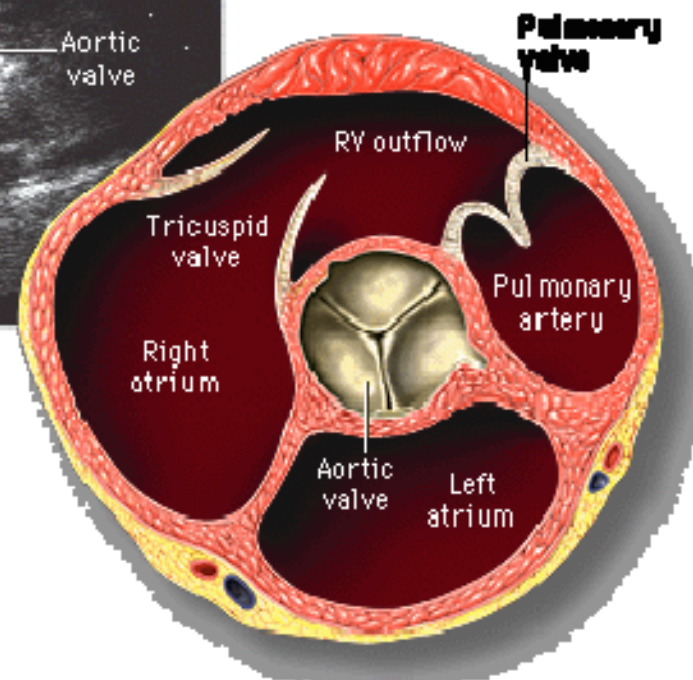
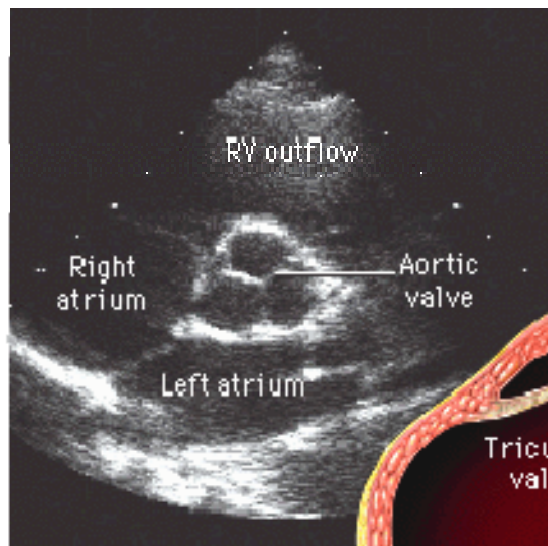
Type B Dissection MRA

[Radiology Rounds Type B Aortic Dissection \(MRA\)](#)

Normal ECHO



Pacific Health and Wellness 2007 - 2008



1999, Yale University

Dissection (ECHO)

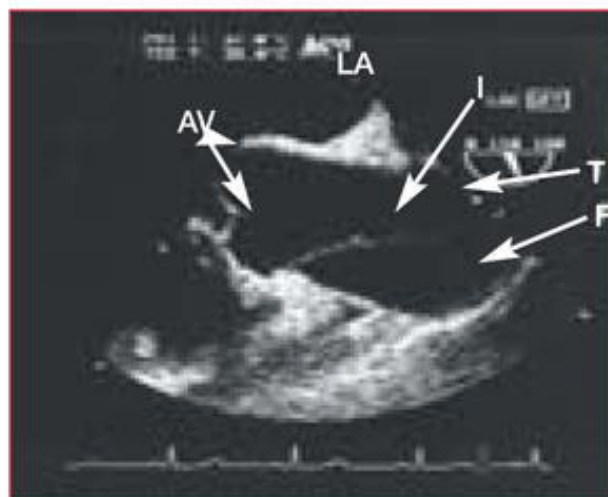
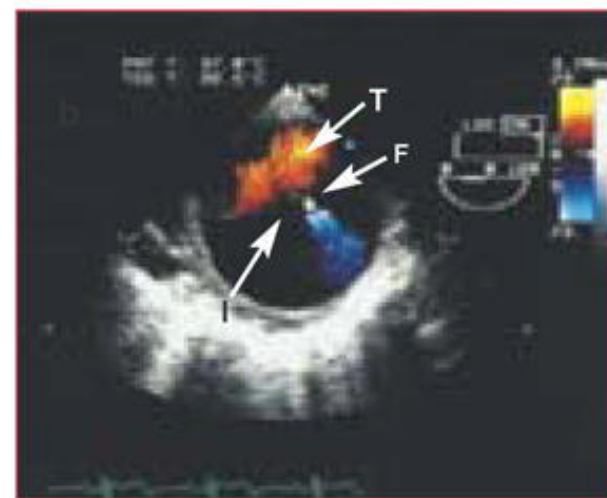
AV: aortic valve;

F: false lumen;

I: intimal flap;

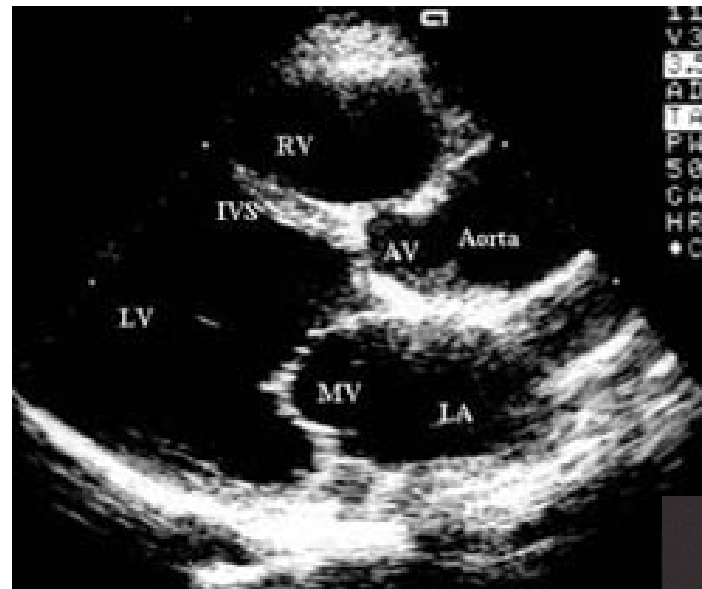
LA: left atrium;

T: true lumen.

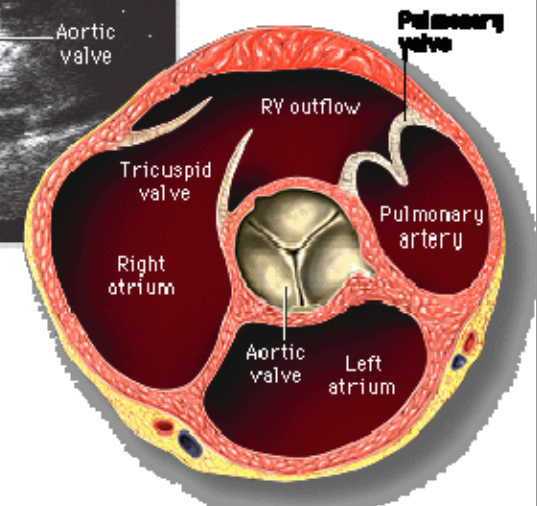
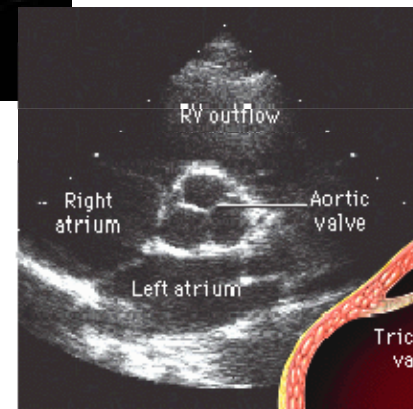


ECHOS

Normal ECHO



Dissecting Ascending TAA





Causes of Dissections⁽¹⁾

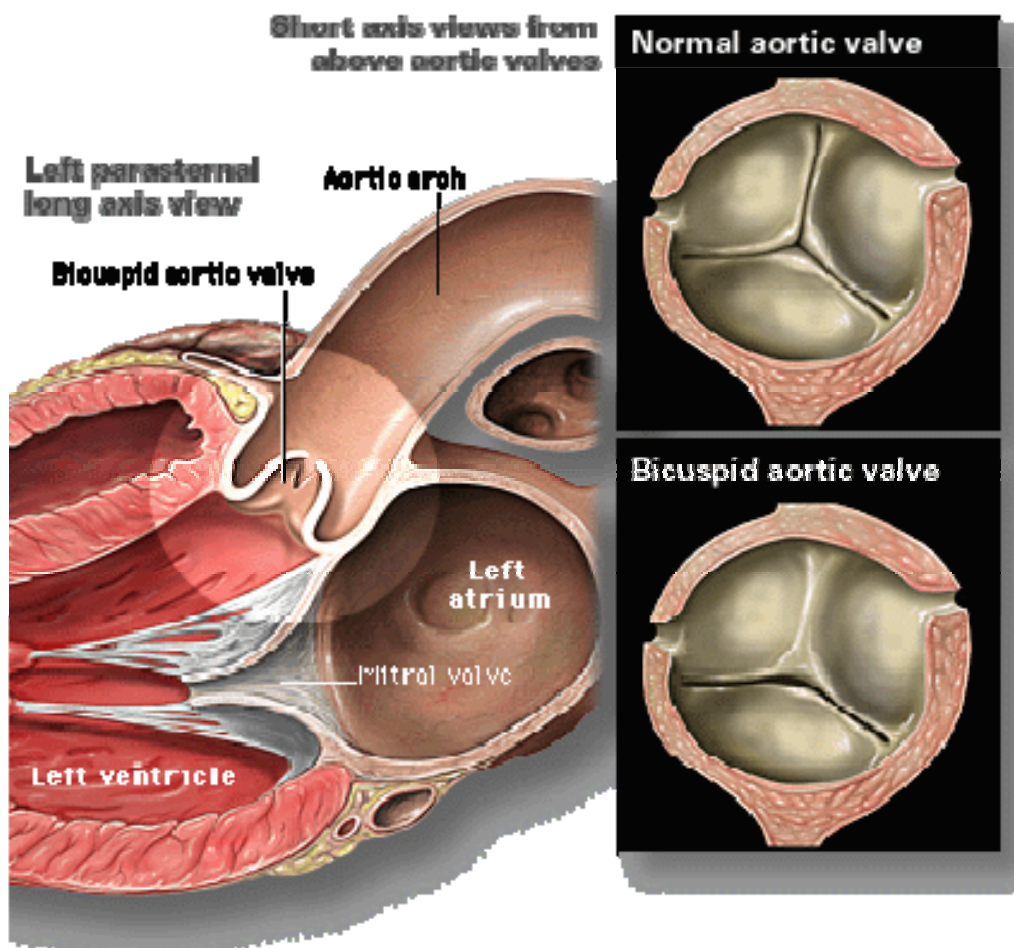
- #1: Uncontrolled Hypertension
 - Causes high shear stress
 - 75% of pt with dissections have h/o HTN
 - Bicuspid aortic valves most common congenital abnormality leading to a dissection (2% population)
- The others
 - Blunt trauma
 - Tobacco Use
 - Hyperlipidemia
 - Cocaine use
 - Pregnancy (rare, 3rd trimester)
 - Mechanical causes: IAPB, Cardiac Surgery, Caths
 - Genetic Causes (discussed later)



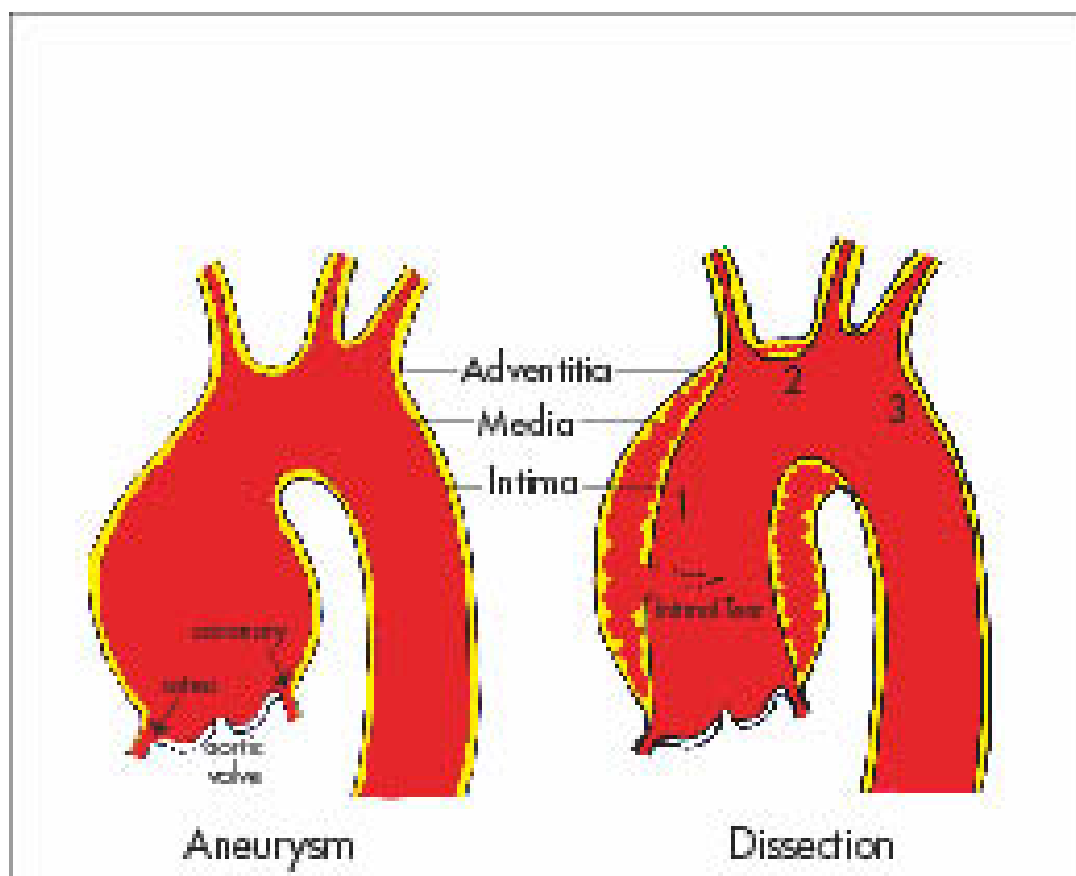
How does a Dissection Occur? ⁽¹⁾

- Cystic Medial Necrosis
 - Progressive focal degeneration of the elastic and muscle tissue within the tunica media
 - In turn leading to Aortic wall weakening and dilation 2nd to increased blood flow pressures.
 - People born with bicuspid aortic valves are pre-disposed to cystic medial necrosis
 - Up to 50% will develop dilatation of the ascending aorta
 - Often dissect under age 40 w/o a hx of HTN

Bi-Cuspid Aortic Valve



How does a Dissection Occur?





Incidence^{(1),(4)}

- 25% of all aneurysms are Thoracic, the remaining are abdominal
- ~15,000 people per year are diagnosed with a TAA
- Only 20-30% of ruptured TAA make it to the hospital alive
- Incidence increases with age (6th and 7th decade)
- More common in men vs. women



Genetic Causes of TAA⁽²⁾

- Marfan Syndrome
- Ehlers-Danlos Syndrome
- Familial Aortic Disease
- Annuloaortic Ectasis



Marfan Syndrome⁽²⁾

- The most common of the genetic causes
- A deficiency in the Fibrillin (FBN) gene
- Leads to connective tissues disorder and breakdown of the elastic component of the Aortic wall (loss of elastic fibers)
 - 1 in 5,000 people have the condition, some literature reports 1 in 10,000

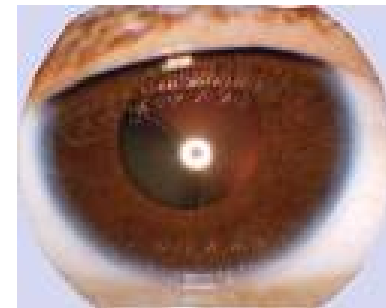
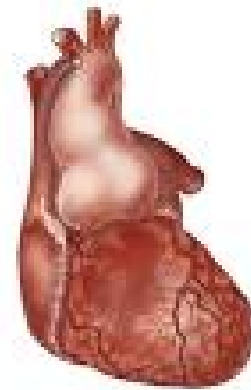


Abraham Lincoln



Characteristics of Marfan's

1. Aortic aneurysm or dissection often occurring at the base of the aorta
2. Dislocation of lenses: Vision issues
3. Long thin extremities





Marfan's Outcomes⁽²⁾

- In the 1970's most patient's died in their 30's
- With improvements in medical and surgical procedures; life expectancy is now in the 70's



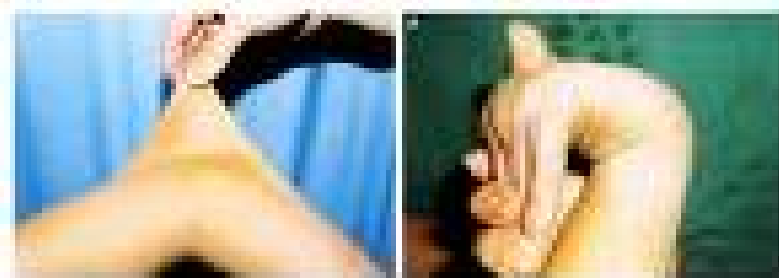
Ehlers-Danlos Syndrome⁽¹⁾

- Also a connective tissue disorder
 - Related to a defect in the type III procollagen
- 11 different types
- Type IV is greatest risk for aortic rupture because the normal aorta is rich in type III procollagen
- Characteristics:
 - Skin Hyperelasticity
 - Hypermobile joints

Ehlers-Danlos Syndrome

Incident:

- ~1/5000
- Slightly higher than Marfan's
- More common in African Americans





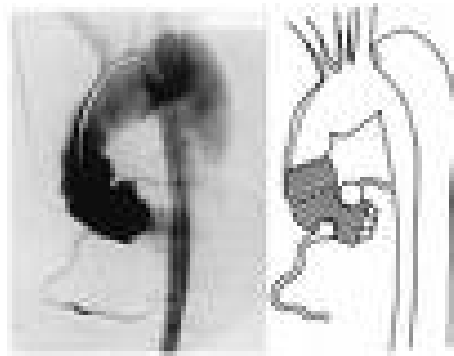
Familial Aortic Disease⁽¹⁾

- Connective disorder diseases with characteristics to either Marfans or Ehlers-Danlos.
- Common variable:
 - The breakdown of elastic fibers
- Example is Loeys-Dietz syndrome
 - Similar to Ehlers-Danlos syndrome



Annuloaortic Ectasia⁽¹⁾

- Dilation of the proximal ascending aorta and aortic annulus
 - Caused by degenerative changes in the elastic fibers



Cocaine₃

- ~0.5% of all TAA's are r/t to Cocaine Abuse
- Usually a Type “B” dissection (descending)
- Crack Cocaine higher incidence
- Average age ~41
- More common in:
 - African Americans
 - Hypertension history
 - Tobacco use (smoking)





Dissection with Cocaine₃

- Cocaine is thought to weaken the elastic media (middle) layer of the Aorta
- Extreme sheer forces
 - Sudden and severe hypertension
 - Profound sympathetic stimulation may cause nicks or tears in the intima layer (almost immediate with Crack Cocaine)
 - tears may occur most often at the ligamentum arteriosum (fixed structure of Aorta)
- Long Term Exposure:
 - premature atherosclerosis may cause dissection

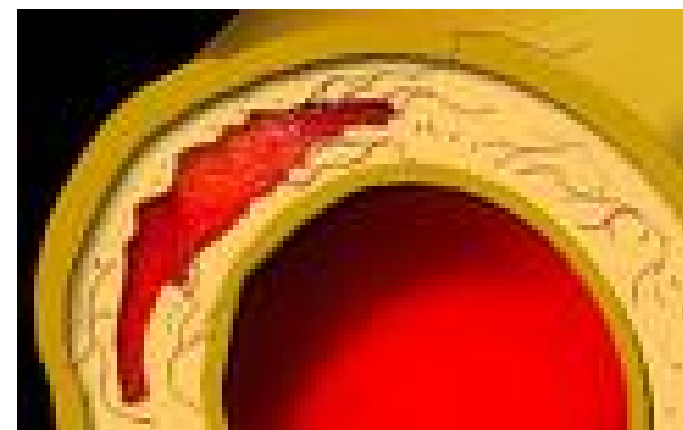


Cocaine₃ (cont.)

- Patient presentation
 - >50,000 ED visits yearly
 - Most present with chest discomfort (CP)
 - MI's and arrhythmias are much more common
 - Only tiny fraction will experience a dissection
 - ***sudden onset of chest or back pain (especially when severe and sharp or stabbing) is suggestive of aortic dissection***
 - ***Other findings:***
 - ***BP differentials, absent pulses, aortic mumur, syncope, abnormal CXR***

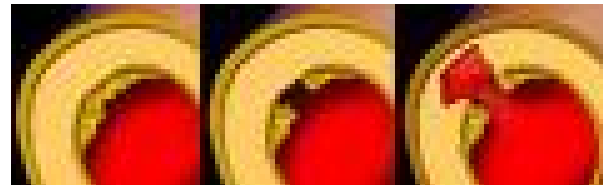
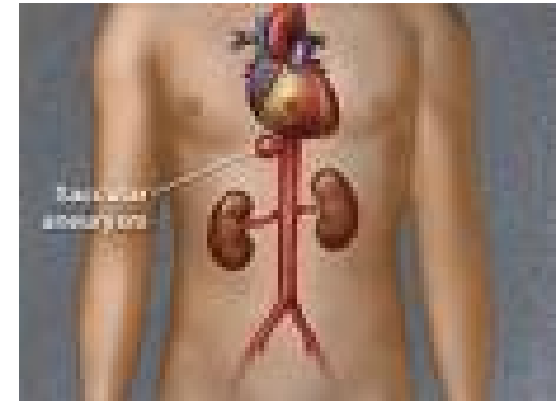
Intramural Hematoma⁽¹⁾

- Caused by the rupture of the Vaso Vasorum of the medial wall but no tear in the intima layer
- Leads to Aortic Infarct
- ~33% of time will lead to an aortic dissection
- Most occur in the descending aorta

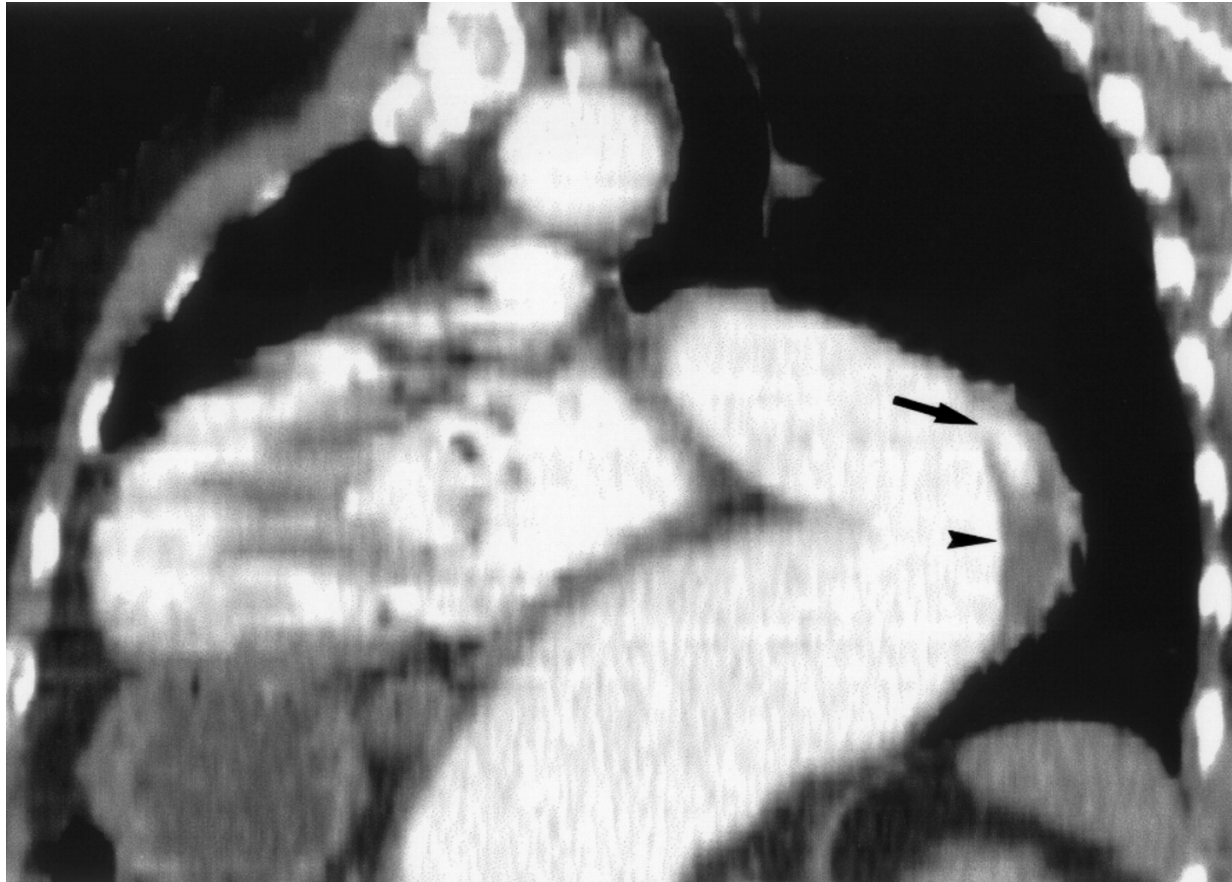


Penetrating Atherosclerotic Ulcer⁽¹⁾

- Plaque formation that over time destroys the intima layer and tunnels into media layer
- A hematoma may form in the aortic wall
- May lead to a pseudo-aneurysm
- 90% occur in the descending aorta

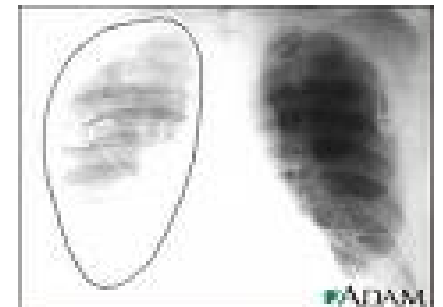
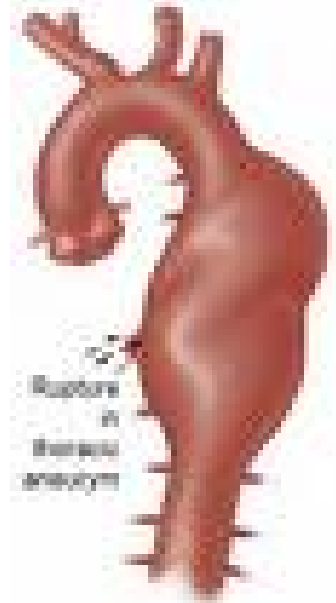


Penetrating Atherosclerotic Ulcer



Aortic Rupture (non-traumatic)⁽¹⁾

- ~15,000 people die each year
- 100% death w/o repair
- Survival rate
 - In hospital: 35%
 - Pre-hospital: 10%
- Common symptom
 - excruciating pain that begins high in the back and spreads down the back and into the abdomen





MI vs. Dissection: Presentation

- MI

- Pain

- Pain may be slow or fast onset
 - Subtle pain, maybe sharp. Crescendo like
 - Heaviness/Pressure in chest w/ possible radiation to back/shoulder
 - Syncope: Uncommon
 - Abdominal Pain: Uncommon

- Dissection

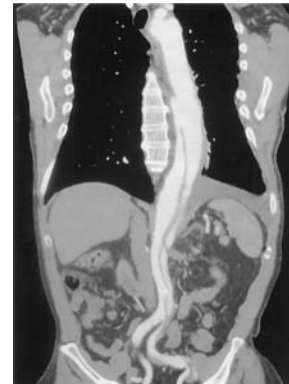
- Pain

- Sudden onset (90%)
 - Severe sharp chest pain: Tearing or ripping
 - Chest pain with type A
 - Back and abdominal pain with type B
- 13% of Type A
 - More common
 - 22% type A
 - 43% type B



MI vs. TAA/Dissection⁽¹⁾

- 30% of patients found to have a TAA/dissection were diagnosed to have other medical conditions
- Peak time for dissection: 08:00 am to 09:00 am
- Higher incidence during Winter months





MI vs. Dissection: Vitals

- MI's
 - BP: normal to hypertensive
 - Could be hypotensive
 - Pulses:
 - Present and = bilaterally
 - Cap Refill
 - Normal or delayed
- Dissection
 - Usually hypertensive
 - Profoundly low if ruptured
 - Possible deficits noted
 - Poor prognosis
 - 30% of cases
 - Normal or delayed



MI vs. Dissection: Labs

– Labs

- H/H usually WNL
- Coags: WNL

– BUN and creatinine

- WNL

– Creatine kinase (CK) and troponin

- Likely elevated and rising

- May be lower, especially with rupture
- Coagulopathy possible

- May be elevated, if renal hypoperfusion

- May be normal, but could be elevated
- If dissection extends to



MI vs. Dissection: Symptoms⁽¹⁾

- Myocardial Infarction
 - 12-lead: Possible elevation
 - CXR: Normal
 - CT: Normal if performed
- Dissection/Rupture
 - Normal (usually)
 - May see ischemia
 - MI possible, Type A
 - Mediastinal widening, blurring of the aortic knob, Pleural effusions
 - specificity is 93% for detecting aneurysms



Emergent Care ⁽¹⁾

- Key points:
 - Blood pressure control
 - Hemodynamic stability
 - Strong assessment skills
- High mortality with acute Type A TAA's
 - Usually require surgical repair within 24 hours
 - Mortality increase 1-2% for each hour after onset
- Type B may often be medically managed
 - Not always surgically repaired unless pain cannot be controlled, cont HTN, or cont. dissection



Pre-Hospital Recommendations for an Acute TAA Dissection⁽¹⁾

- High suspicion for a dissection
 - Put all the puzzle pieces together
- Rapid notification and transport to a center with on-call Vascular Surgery
- High flow O₂
- BP goal (if sure it is a dissection):
 - Systolic BP between 100 and 120 mm/Hg
 - Be careful with BP, can pt function with a BP of 100
 - Contact Med Command
 - Nitrates and/or Beta Blockers
- Large bore IVs, Fluids KVO for desired BP



Pre-Hospital Recommendations for an Acute Thoracic Aortic Rupture ⁽¹⁾

- High likelihood of mortality
- If certain is a rupture then
 - Rapid notification and transport to a center with on-call Vascular Surgery
 - O2 / Intubate
 - Large bore IV's
 - Fluids W/O
 - PUHA or Medevac



Emergency Dept Care: Dissection₍₁₎

SBP: 100-120 and HR ~60

- Aggressive Beta blockade (if not contraindicated)
 - Labetalol IV
 - » Alpha and Beta Blocker
 - Esmolol IV
 - » Short ½ life
 - » Careful with COPD patients
 - Calcium Channel Blockers
 - » Diltiazem IV, Verapamil IV, Nicardipine IV
- Vasodilators
 - SNP
 - » If Beta Blockers not indicated or not successful
 - » Don't use SNP w/o Beta Blockers for it may allow LV ejection to rapidly rise worsening the dissection



Emergency Dept Care: Dissection Cont.⁽¹⁾

- O2/Intubation
- Large bore IV's/Central Lines/Cordis
- Fluids PRN
- Labs
 - Blood PRN
 - Especially if concerned with a ruptured TAA
 - Electrolytes PRN
- CXR
- CT/CTA
- MRI/MRA



Cocaine: Emergency Care ³

- Treatment
 - Key is early recognition
 - BP control to limit aortic shear forces and trans-aortic pressures
 - BP <120/80 and HR <60
 - Aggressive Beta blockade
 - Recommend Labetolol to block the unopposed alpha
 - Vasodilators
 - SNP
 - Also consider IV Nitro and IV Verapamil

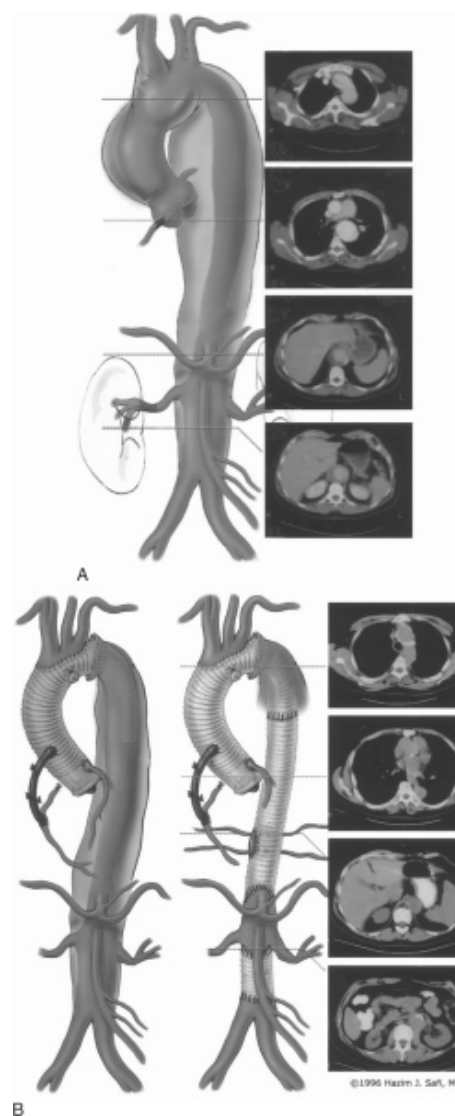


Surgical Options ⁽¹⁾

- Type A (I/II) and IMH in ascending aorta are more likely to be repaired
- Type A repairs
 - Open Repair with a graft
 - Often the Aortic Valve is repaired or replaced
 - CABG may be needed
 - Circulatory Arrest if arch repaired/replaced
 - Mortality ~10-35% (50% w/o repair)

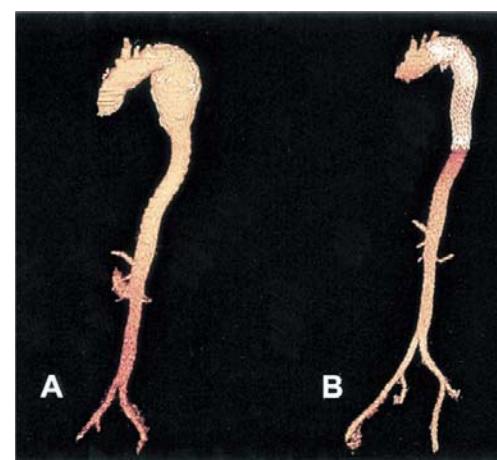
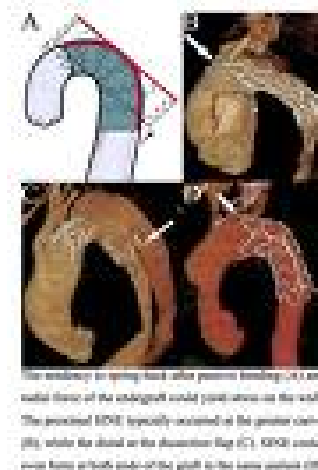
Surgical Options ⁽¹⁾

- Type A (I/II) Repair



Surgical Options ⁽¹⁾

- Type B (III) Repairs
 - Most can be treated with aggressive medical therapy
 - High surgical mortality rate (up to 32%)
 - Spinal Cord Ischemia Risk
 - Stent grafts are becoming more common in Type B repairs (Endovascular repair)



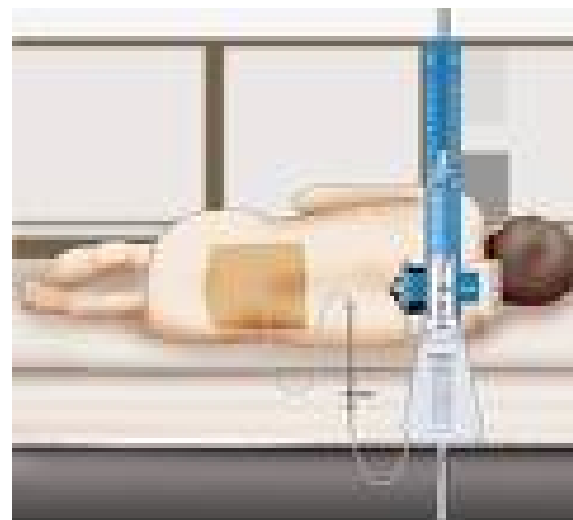
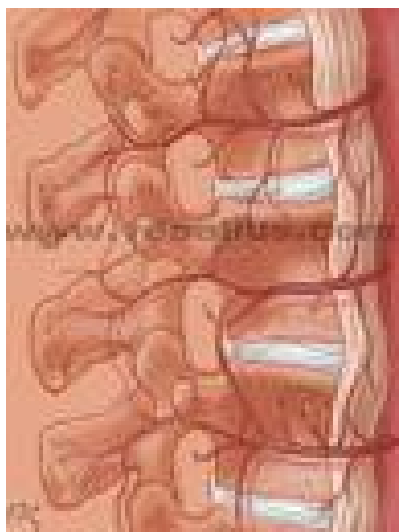


Post Surgical Paralysis⁽⁵⁾

- Between 5% and 21% of TAA repair patients experience spinal cord ischemia after endovascular repair
- May be transient or permanent
- Usually occurs with descending TAA repairs (Type B)
- Blood flow to the thoracic spinal column/nerves are supplied from the descending Thoracic aorta

Lumbar Drainage⁽⁵⁾

- To help decrease the ischemia Lumbar drains are placed to drain CSF and to keep spinal pressures “ICP” 10mm/Hg or less



Lumbar Drainage



Case Study Revisited (6)

You and you partner are called to the home of a 76 y/o male for chest pain.

Assessment:

- 10/10 “crushing” CP that radiates to his back and shoulder
- A&Ox3, tells you the pain was what aroused him from his night’s sleep
- Vitals:
 - Mild difficulty breathing, in the 30’s. BS clear
 - No palpable Radial pulse

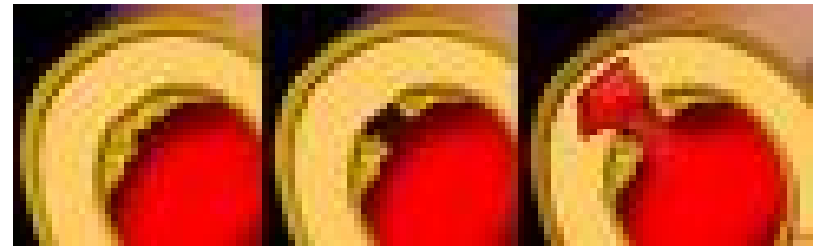


Case Study Outcome (6)

- EMS Care:
 - 15 liters O2 via NRB
 - 2 large bore IVs
 - 1000ml NS
 - BP still in 60's
 - Dopamine Infusion
 - 5 then 7.5 then 10 mcg/kg/min
 - BP ~70 on arrival in the ED

Case Study Outcome (6)

- In the ED
 - FAST Ultrasound
 - Inconclusive
 - CXR
 - Widened mediastinum, possible TAA
 - CT revealed:
 - Type A Aortic Dissection
 - Pericardial Effusion
 - Aortic Ulcer
 - PE
 - Emergently given blood, cont. Dopa, and rushed to OR
 - Type A Open Repair
 - Made a full recovery





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4. **Etiology, Pathogenesis and Management of Thoracic Aortic Aneurysm.** Huseyin Ince and Christopher Nienaber. *Nature Clinical Practice Cardiovascular Medicine*. 4(8):418-427, August 2007.
5. **Cerebrospinal Fluid Drainage to Reverse Paraplegia After Endovascular Thoracic Aortic Aneurysm Repair.** Kurt Tiesenhausen, MD; Wilfried Amann, MD; Günter Koch, MD; Klaus A. Hausegger, MD; Peter Oberwalder, MD; and Bruno Rigler, MD. *Journal of Endovascular Therapy*: Vol. 7, No. 2, pp. 132–135.
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Questions





Thank You

