

VASCULAR SURGERY: AAA, CAROTID DISEASE AND PERIPHERAL ARTERIAL DISEASE (PAD)

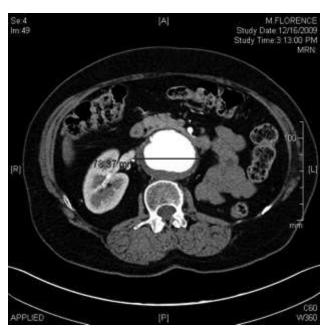
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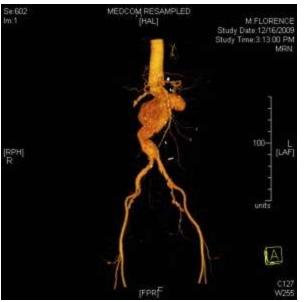
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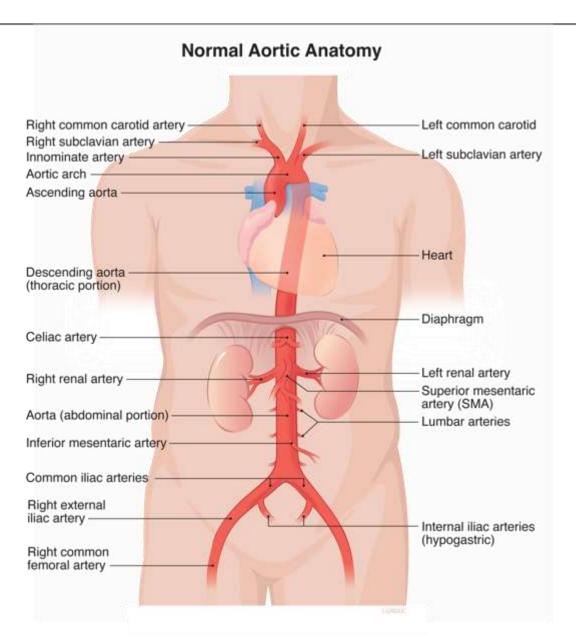




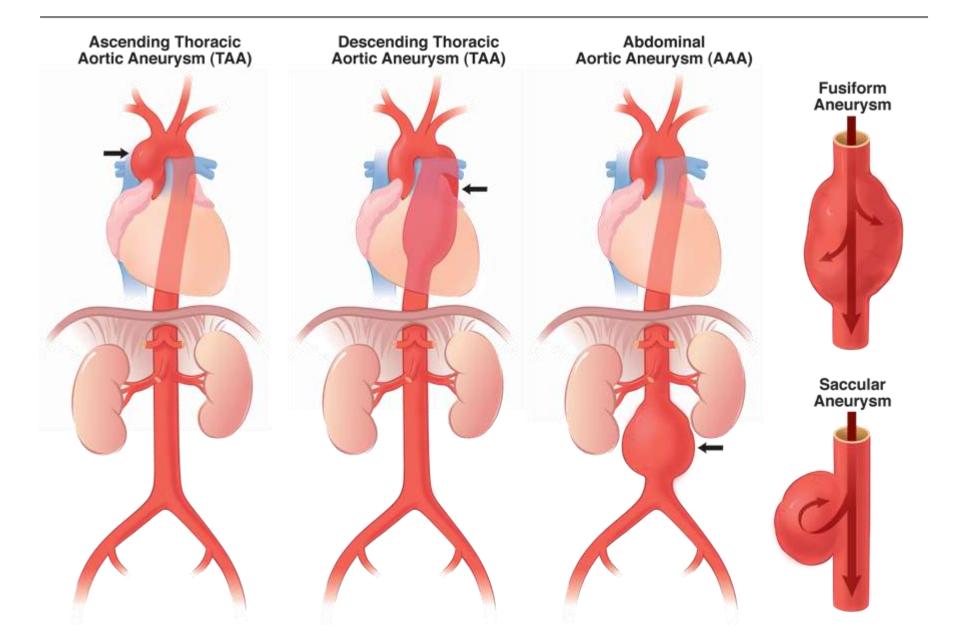






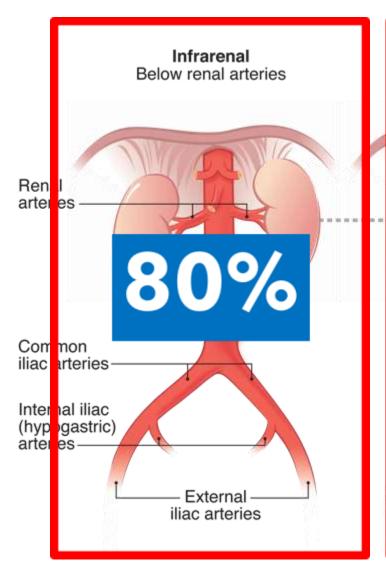


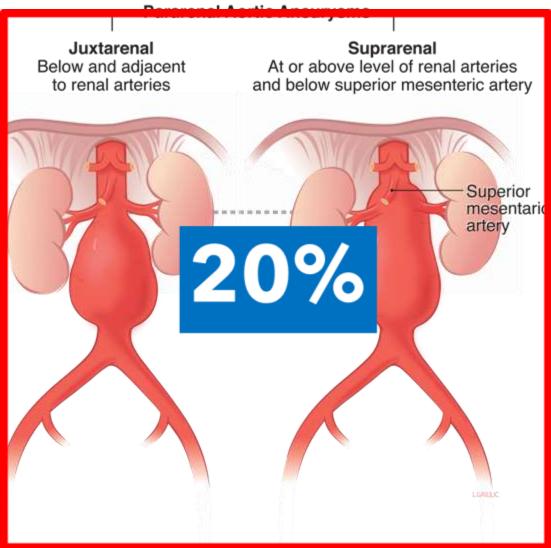


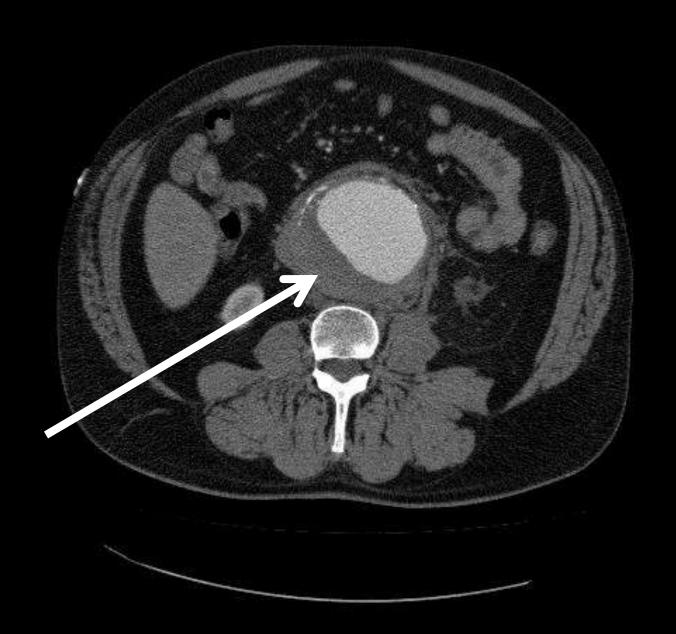


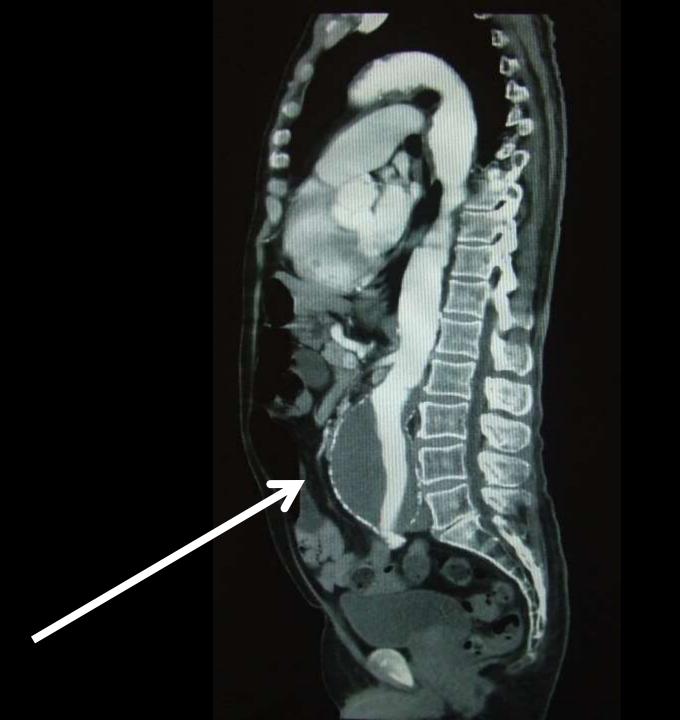


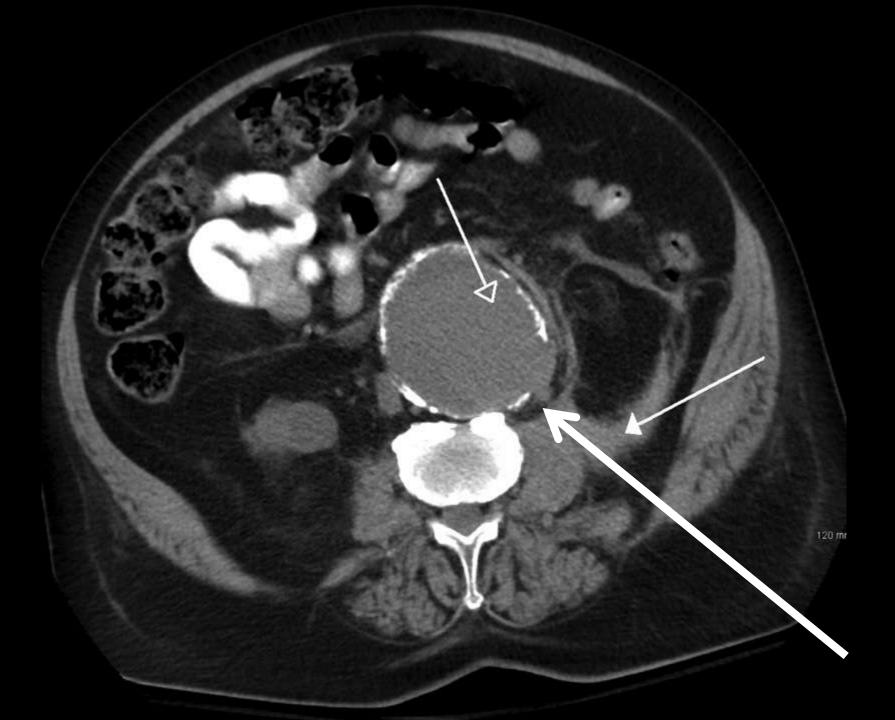
Types of Abdominal Aortic Aneurysms













- 50% of all patients die of ruptured AAA before reaching the hospital
- 30-40% die who reached the hospital
- Overall 80-90% mortality in ruptured AAA

AORTIC ANEURYSMS FACTS



- Infrarenal aorta: 1.8-2.2 cm in diameter
- Aneurysm defined as 2X normal diameter
- Growth rate 4.5 cm AAA (0.4 cm/yr, 10%/yr)
- Peripheral aneurysms in 3.5% (pop > femoral)

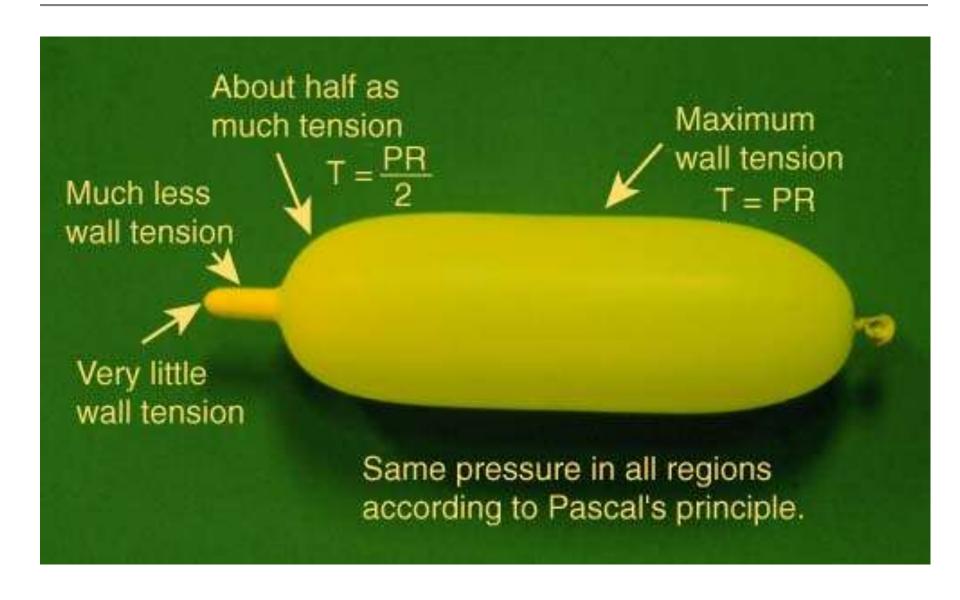
AAA RISK FACTORS



- Male gender (6X > females)
- Smoking (5X nonsmokers)
- Caucasian (2.5X > African American)
- Family History (1.9X)
- Age (1.7X per 7 years)
- CAD (1.5X)
- Hypercholesterolemia (1.4X)
- COPD (1.2X)

LAPLACE LAW





AORTIC ANEURYSM FACTS



- Rationale for repair is prevention of rupture!
- Laplace's Law
- Risk of Rupture

Size (cm)	Annual Rupture Risk
<4.0	0.3%
4.0-4.9	1.5%
5.0-5.9	6.5%
6.5	20%
7.5	35%

FACTORS ASSOCIATED WITH RUPTURE



- Female gender (4.5X > Male)
- Smoking
- COPD
- Family History
- Hypertension
- Connective Tissue Disease (Marfans, Ehlers-Danlos)



Society of Vascular Surgery, Medicare Screening

- Any Male who has smoked (65-75 yo)
- Any Male or Female with a family history of AAA
- Eliminates referral requirement as part of the Welcome to Medicare Physician Exam
- Eliminates the one-year time limit for all beneficiaries who are at risk for AAA
- This remains a one-time screening

ASSESSING THE AAA PATIENT



- Ultrasound
 Used to diagnose and monitor AAA until
 aneurysm approaches size at which repair
 considered
- Computed Tomography
 Used in preop assessment of AAA
- * 3-4 mm difference between US and CT scan

SURGERY VS OBSERVATION



- 1. Risk for AAA rupture without surgery
- 2. Operative risk of repair
- 3. Patient's life expectancy
- 4. Personal preference of patient

1. RISK OF RUPTURE



- Size matters:
 - Aneurysm > 5cm 6-16% and > 7cm 33%
- Saccular aneurysm have higher rupture rate
- HTN, COPD, active smoking is independent predictors of rupture
- (+) Family Hx tend to rupture
- Expansion rate (> 5 mm over 6 months)

2. OPERATIVE RISK OF REPAIR



- Mortality after:
 - Elective open AAA ~ 5%
 - EVAR 1.7%

3. PATIENTS LIFE EXPECTANCY



 Very difficult to assess due to patient's comorbidities

Age	Life Expectancy after AAA repair (years)
60	13
70	10
80	6

4. PERSONAL PREFERENCES



- Fear of AAA vs. Fear of surgery
- Anecdotal experiences of friends and family

MEDICAL MANAGEMENT OF AAA



- Smoking Cessation Single most important modifiable risk factor
- Exercise Therapy Evidence suggests may benefit small aneurysms
- Beta Blockers May decrease the rate of expansion?
- ACE inhibitors Evidence is mixed, however, implicated in less aneurysm rupture

MEDICAL MANAGEMENT OF AAA



- Doxycycline Antibiotic activity against chlamydia species and suppresses expression of MMP
- Statins associated with reduced aneurysm expansion rates. Decreases MMP-9 in aneurysm wall

CLINICAL PRESENTATION



- Asymptomatic
- Pulsatile mass
- Embolic phenomenon
- Compressive symptoms
- Acute onset of abdominal and back pain
- Hemorrhage and cardiovascular collapse

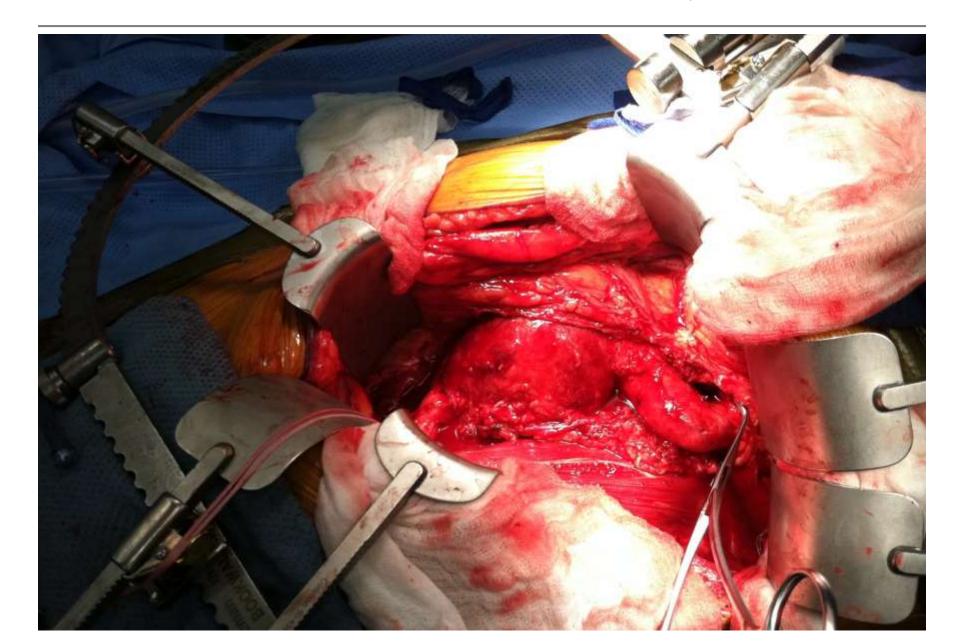
WHEN TO OPERATE?



- >5.5 cm (5.0 cm)
- UK Small AAA Trial (Lancet 1998) & Aneurysm Detection and Management Trial (ADAM) (NEJM 2002)
- No difference in survival in 4.0-5.5 cm AAA when randomized to a strict surveillance vs immediate repair (follow-up 5 yrs)
- >60% of surveillance pts underwent repair

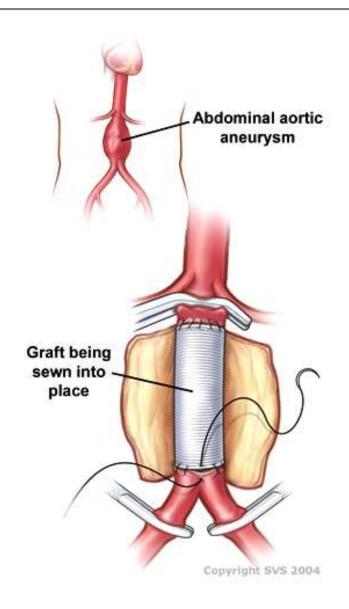
OPEN REPAIR AAA





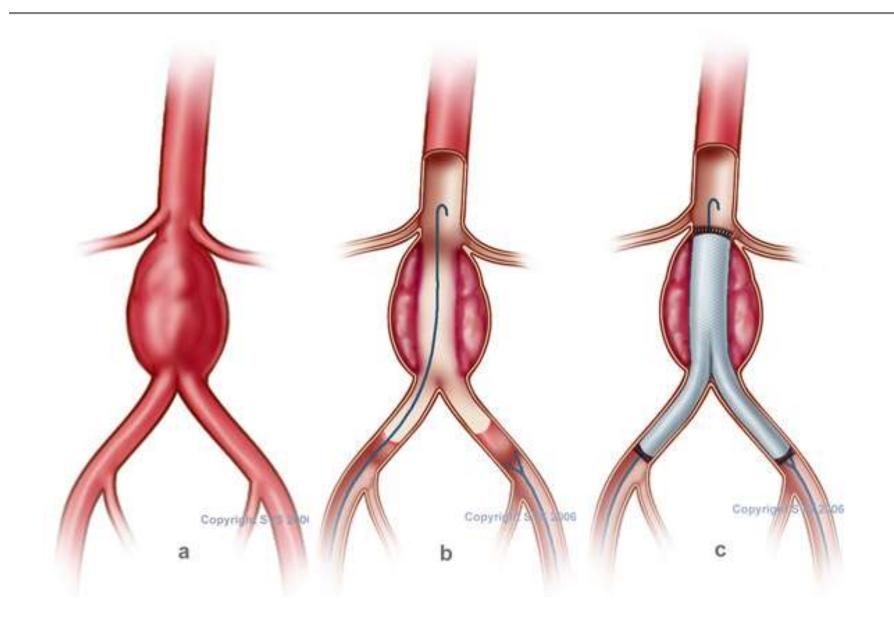
OPEN REPAIR AAA





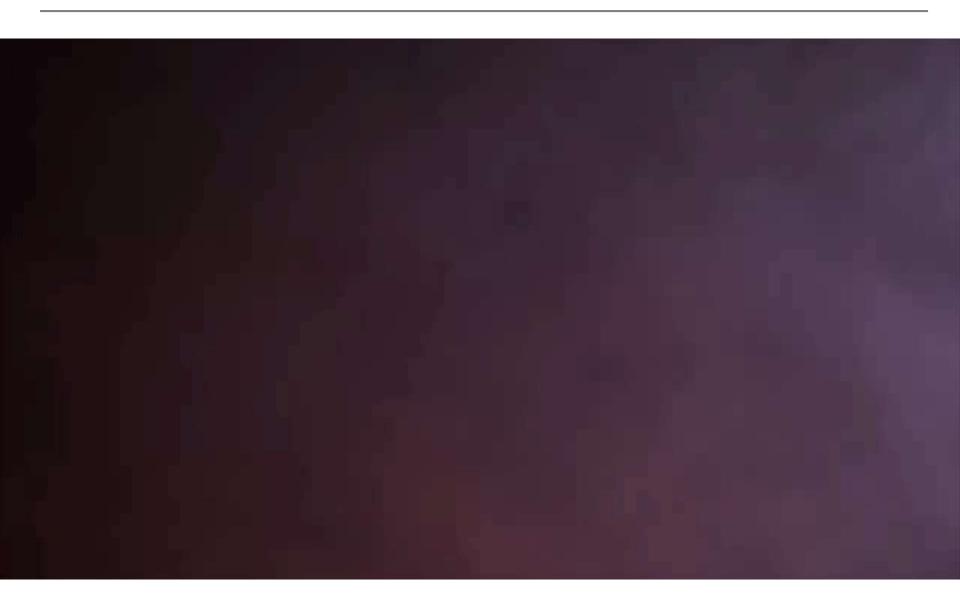
EVAR REPAIR AAA





EVAR REPAIR AAA





EVAR REPAIR AAA







AAA: WHEN TO OPERATE?



- Repair must outweigh the risk of rupture
- 30-day in-hospital mortality (3.8% to 8.2%) for elective <u>open</u> repair
- Same size criteria for elective open vs endovascular repair (EVAR)
- EVAR is associated with decreased morbidity and mortality in the early post-operative period

OPEN VS EVAR

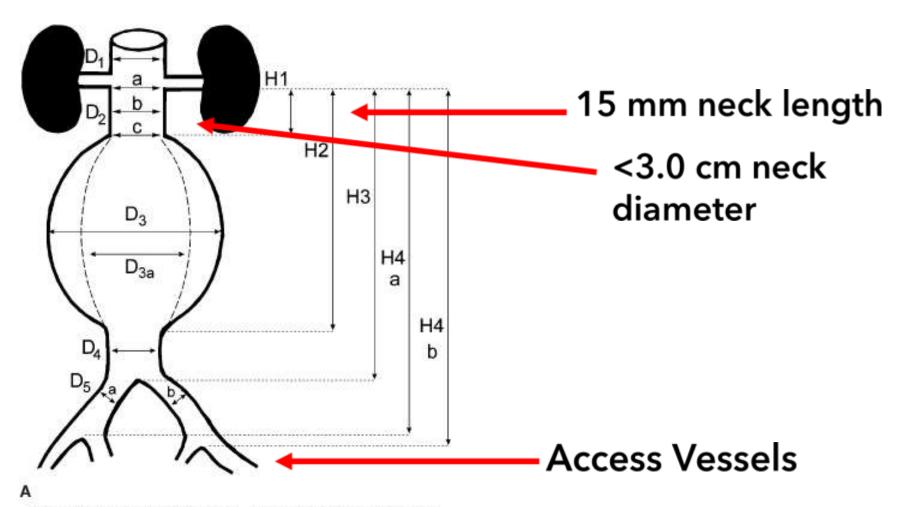


	EVAR I		DREAM	
	Open	EVAR	Open	EVAR
N	539	543	178	173
30 Day Mortality	4.7%	1.7%*	4.6%	1.2%*
Late All Cause Mortality	29% (4yr)	26% (4yr)	11% (2yr)	11% (2yr)
Late AAA Related Mortality	7% (4yr)	4% (4yr)	5%(2yr)*	2% (2yr)

^{*} $p \le 0.05$

EVAR ELIGIBILITY REQUIREMENTS





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CONTRAINDICATION TO EVAR



- Short proximal neck
- Thrombus present in proximal landing zone
- Conical proximal neck
- >120° angulations of the proximal neck
- Critical inferior mesenteric artery
- Significant iliac occlusion
- Tortuosity of iliac vessels

AAA DEVICE SELECTION









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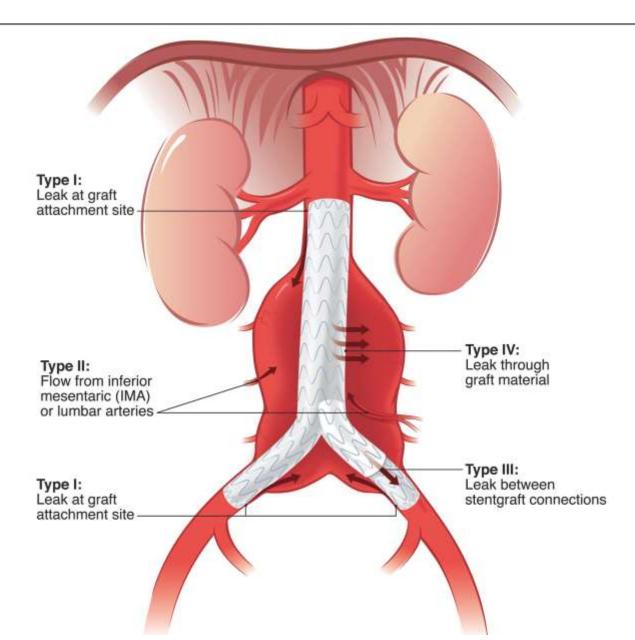
EVAR COMPLICATIONS



- 30 day mortality ranges 0-6% (1.2%)
- Reduced cardiac/pulmonary complications
- Contrast nephropathy
- Stent deployment issues
- Local trauma to access vessels
- Endoleaks

AORTIC ANEURYSM ENDOLEAKS





EVAR POSTOPERATIVE FOLLOWUP



- Successful exclusion and depressurization with shrinkage of the aneurysm sac
- CTA at 6 months and yearly
- Renal issues
- Patient compliance issues



EVAR

Lower perioperative mortality/morbidity

Shorter hospital stay

No ICU/Ventilator Care

Fewer transfusions

Strict followup

Shorter operative time

No ventral hernias

Open Repair

Less frequent need for reintervention

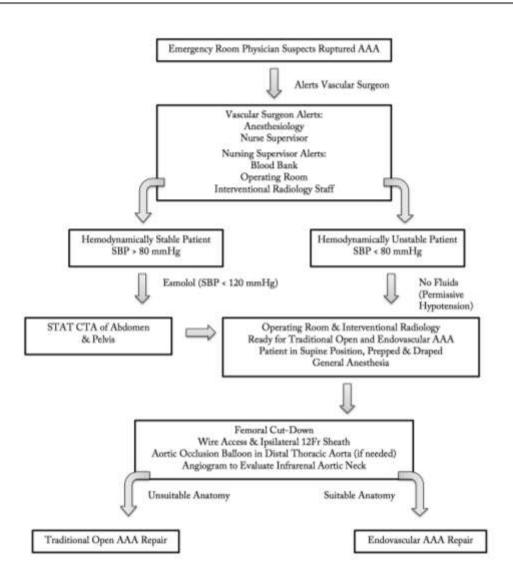
No routine follow-up imaging

No endoleaks

? Lower Cost

RUPTURE PROTOCOL

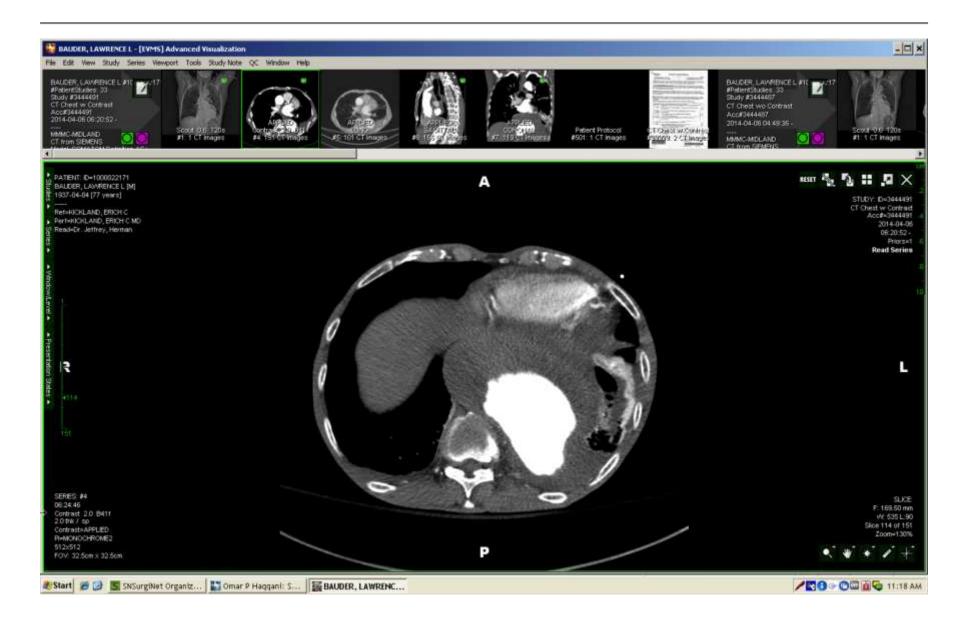




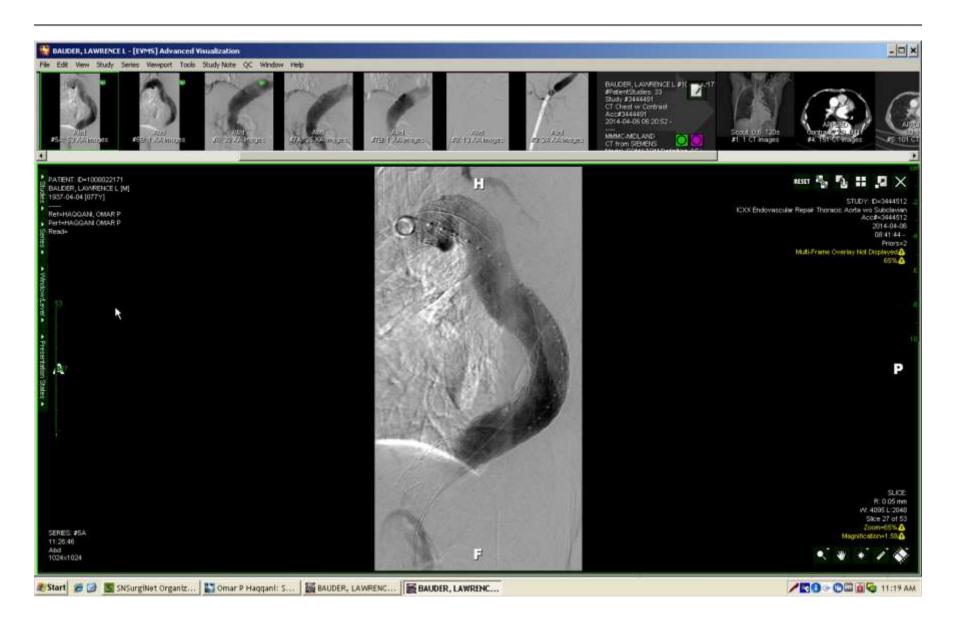














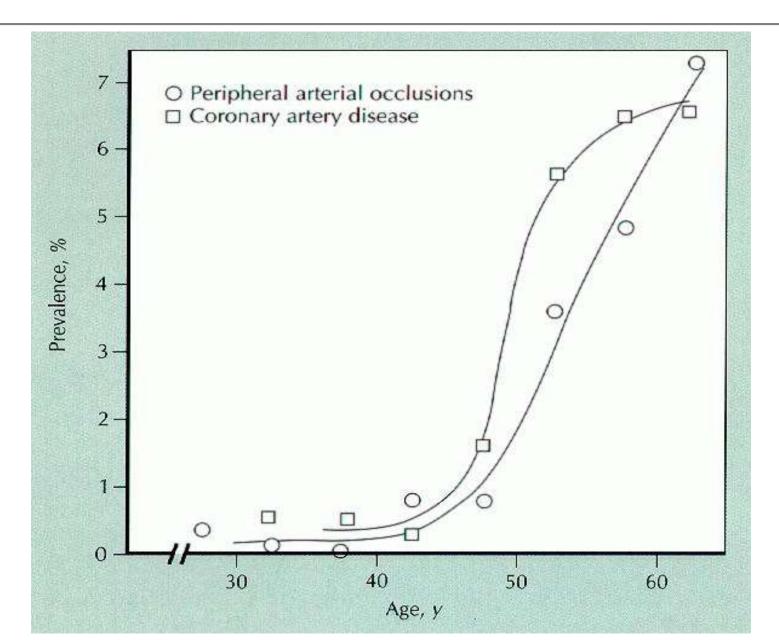




PERIPHERAL ARTERIAL DISEASE

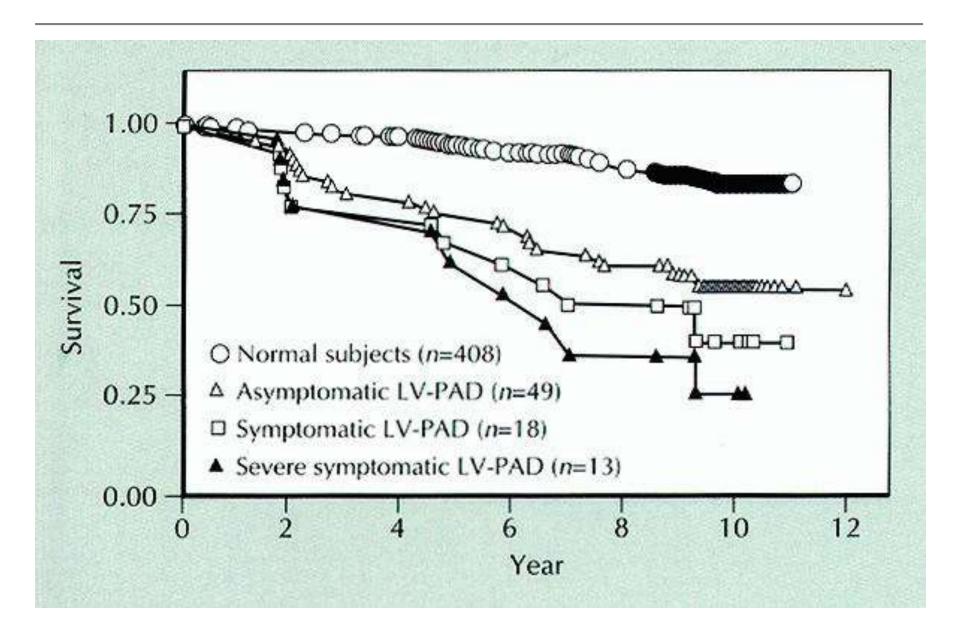
AGE DISTRIBUTION OF PAD





IMPACT OF PAD ON MORTALITY





ATHEROSCLEROTIC RISK FACTORS



Traditional Risk Factors

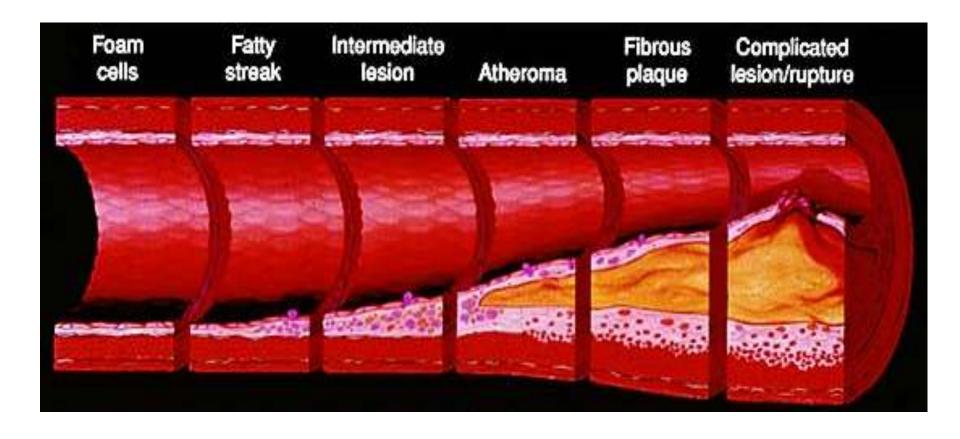
- Age
- Family history of CAD
- Cigarette smoking
- Hypertension
- Low HDL-C
- High total and LDL-C
- Diabetes mellitus
- Obesity

Novel Risk Factors

- D-Dimer
- Lipoprotein (a)
- Serum amyloid A
- CMV, HSV, C.
 Pneumoniae
- Homocysteine
- Estrogen deficiency
- Plasma fibrinogen
- Factor VII
- C-reactive protein

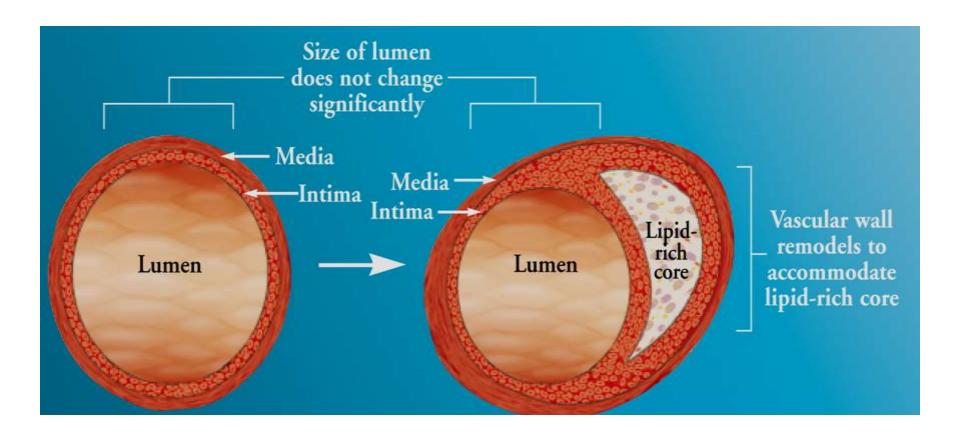
ARTERIAL PATHOPHYSIOLOGY





ARTERIAL PATHOPHYSIOLOGY





ARTERIAL PATHOPHYSIOLOGY



Autoregulation:

- dilation of peripheral arterioles distal to stenosis
- collateral pathway formation
- enhanced muscle metabolism/O2 extraction

Claudication and rest pain:

 accumulation of anaerobic metabolites lactic acid, adenosine monophosphate

MANAGEMENT OF PAD PATIENTS



Lifestyle modification

- Smoking cessation
- Regular exercise training
- Diet

Pharmacological treatment

- Antiplatelet therapy
- Control risk factors (e.g. hypertension, blood glucose)
- Vasodilators for symptomatic relief?

CLAUDICATION



- Pain in calf, thigh, or buttock
- Occurs after consistent level of exercise
- Resolves with rest
- Aching, cramping sensation
- Benign condition
- 3% to 15% amputation rate over 3 years (depends on tobacco use)

REST PAIN



- Dull, aching pain of foot or toes
- Relieved by dependency, worsened by supine position
- May have paresthesia and pallor
- Demands prompt attention
- Does not lead to limb loss in 100%

LIMB THREATENING ISCHEMIA



- Pulselessness
- Pallor
- Pain
- Paralysis
- Paresthesia (or anesthesia)
- Demands immediate attention

PHYSICAL EXAMINATION

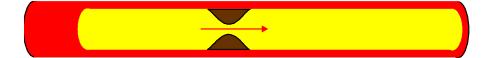


- Decreased pulses
- Bruits (carotid, femoral, abdominal)
- Hair loss, muscle atrophy
- Atrophic, shiny skin
- Ulceration/gangrene (toes or foot)
- Dependent rubor

PHYSICAL EXAMINATION



Low Flow: rest



High Flow: exercise



Increased flow leads to greater energy loss across stenosis and hence greater pressure drop

DIABETIC FOOT INFECTION



- Neuropathy
- Impaired immune function
- Greater prevalence of arterial disease
- Impaired inflammation

DIABETIC NEUROPATHY



- Segmental demyelinization: distal > proximal
- Paresthesia, loss of touch, vibration, temperature
- "Stocking" distribution
- Affects motor and autonomic nerves

DIABETIC NEUROPATHY



- Weakening of intrinsic foot muscles
- Pes Cavus deformity, "Rocker-bottom foot"
- Hammer toe, extensor subluxation
- Maldistribution of weight over plantar surface

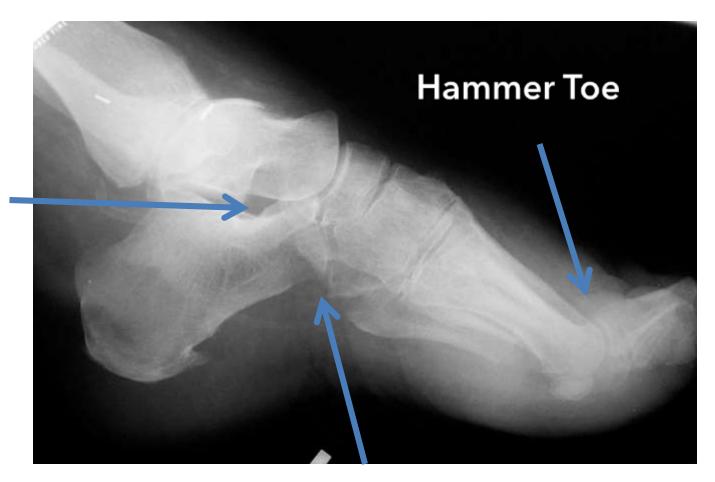
DIABETIC NEUROPATHY







Subluxed tarsal bone



Pes Cavus deformity

MECHANISM OF INJURY



- Poorly fitted shoes
- Foreign body in shoe
- Unrecognized trauma, burn
- Injudicious pedicure
- Problems compounded in elderly population with poor vision

IMPACT OF ISCHEMIA



- Digital or foot gangrene
- Necrosis with trivial injury or infection
- Neglected ulcers and infection can rapidly progress

PATIENT ASSESSMENT



- Careful examination
- Signs of toxicity
- Fever, leukocytosis
- Hyperglycemia most sensitive indicator
- Frequent delay in diagnosis
- Minimal pain and inflammation
- Casual observer can easily miss deep abscess

PATIENT ASSESSMENT



- Careful assessment by vascular surgeon
- Aggressive manipulation, probing
 - unroof eschars, bullae
- Radiologic assessment
 - soft tissue swelling, osteomyelitis, tissue gas
- Aggressive drainage and debridement
- Broad spectrum antibiotics





























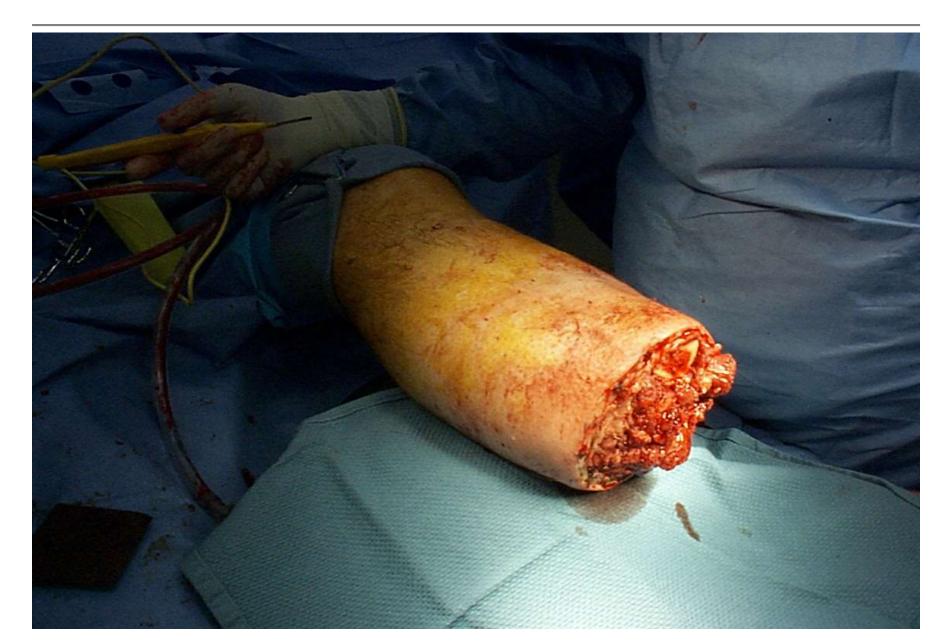












PHYSICAL EXAMINATION



- Segmental pressures
- Pulse volume recording
- Duplex ultrasound
- Magnetic Resonance Angiography (MRA)
- Contrast Angiography
- Spiral CT

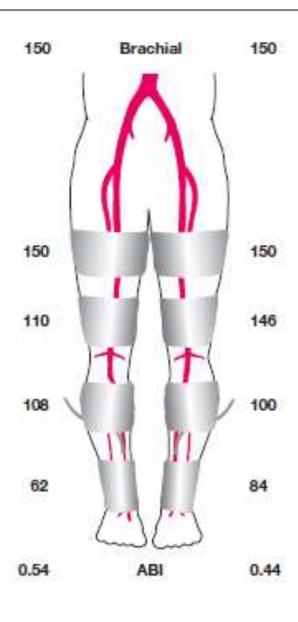


- Insonate best pulse at ankle/foot level
- DP, PT, Peroneal
- Cuff inflation until systolic flow disappears
- Slowly release until flow detected
- Repeat at all levels











Limitations of SLP measurements include:

- Nondetection of isolated moderate stenoses (usually iliac) that produce little or no pressure gradient at rest
- Falsely elevated pressures in patients with diabetes calcified, incompressible arteries
- The inability to differentiate between arterial stenosis and occlusion



Ankle Brachial Index (ABI)

- Doppler systolic occlusion pressure at ankle
- Quantifies arterial obstruction
- Falsely elevated with calcified arteries (diabetics, dialysis patients)





ANKLE BRACHIAL INDEX



ABI	Symptoms	
> 0.92	Normal	
0.5 - 0.9	Claudication	
< 0.4	Rest Pain	
< 0.4	Tissue loss	

TOE PRESSURES AND TOE BRACHIAL INDEX

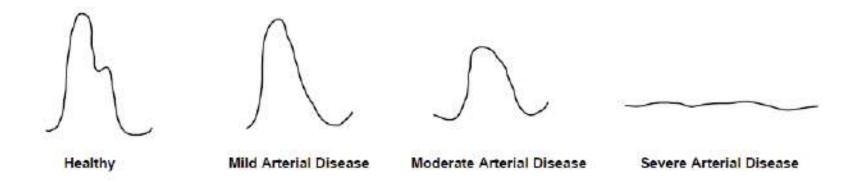


- Diabetes, renal failure results in incompressible tibial arteries (≥250 mmHg) or ABI >1.40
- Toe pressures provides an accurate measurement of distal limb systolic pressures in vessels that do not typically become noncompressible
- A special small occlusion cuff is used proximally on the first or second toe

PULSE VOLUME RECORDING (PVR)

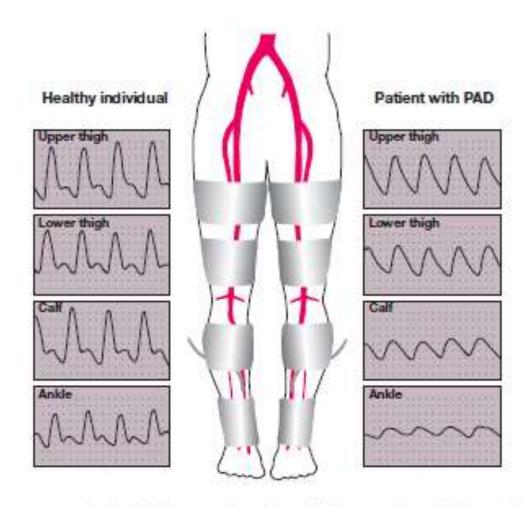


- Measures changes in pressure reflecting arterial pulsatility
- Aid in localizing significant occlusive lesions
- Not affected by calcified vessels as the ABI



PULSE VOLUME RECORDING (PVR)





In the presence of arterial disease, the slope of the waveforms flattens, the pulse width widens, and the dicrotic notch is lost

CHARACTERISTICS OF ARTERIAL IMAGING



Modality	Availability	Relative risk and complications	Strengths	Weaknesses	Contraindications
X-ray contrast angiography	Widespread	High risk Access site complications Contrast nephropathy Radiation exposure	'Established modality'	2D images Limited planes Imaging pedal vessels and collaterals in the setting of occlusion requires prolonged imaging and substantial radiation	Renal insufficiency Contrast allergy
MDGTA	Moderate	Moderate risk Contrast nephropathy Radiation exposure	Papid imaging Sub-millimeter voxel resolution 3D volumetric information from axial slices Plaque morphology	Calcium causes 'blooming artifact' Stented segments difficult to visualize	Renal insufficiency Contrast allergy

CHARACTERISTICS OF ARTERIAL IMAGING



Modality	Availability	Relative risk and complications	Strengths	Weaknesses	Contraindications
MRA	Moderate	None	True 3D imaging modality; infinite planes and orientations can be constructed Plaque morphology from proximal segments with additional sequences Calcium does not cause artifact	Stents cause artifact but alloys such as nitinol produce minimal artifact	Intracranial devices, spinal stimulators, pace-makers, cochlear implants and intracranial clips and shunts are absolute contradications
Duplex	Widespread	None	Hemodynamic information	Operator-dependent and time-consuming to image both lower extremities Calcified segments are difficult to assess	None

MANAGEMENT OF PAD INTERVENTIONS



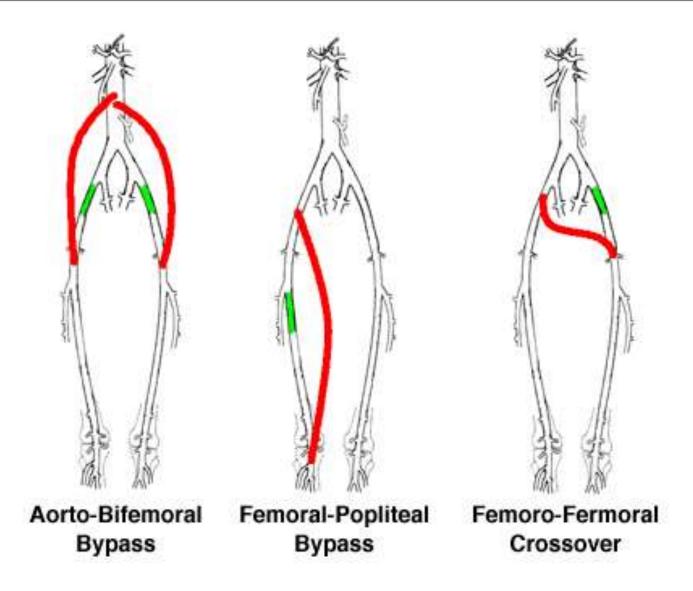
Endovascular

- Revascularization (angioplasty)
- Stent placement

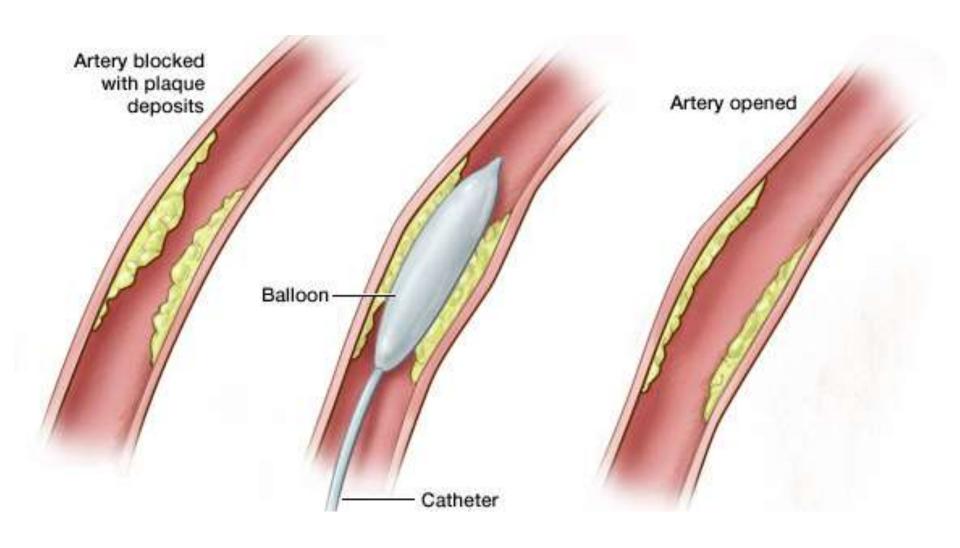
Surgical

- Endarterectomy
- Peripheral bypass graft
- Amputation

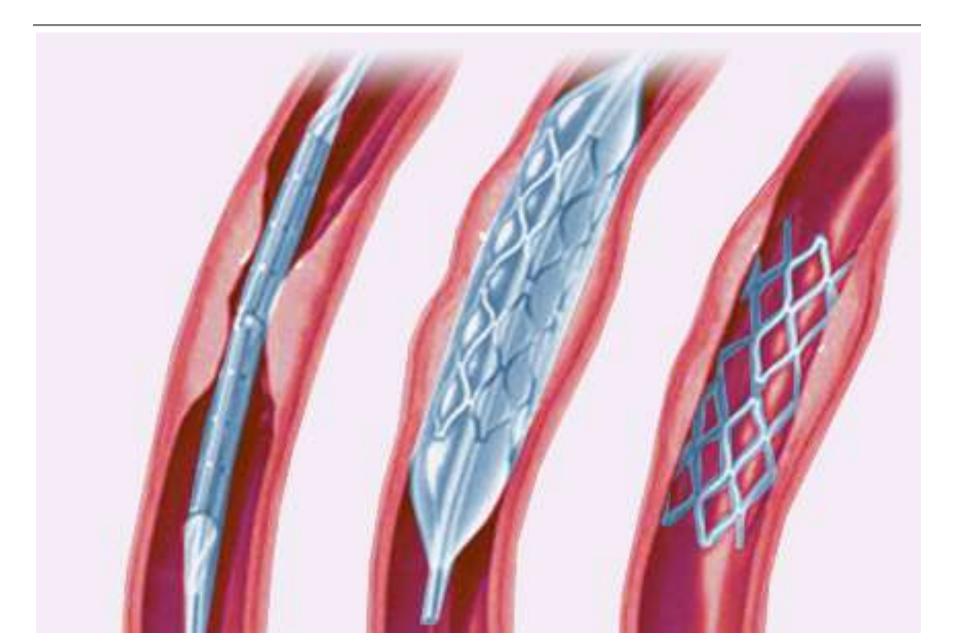












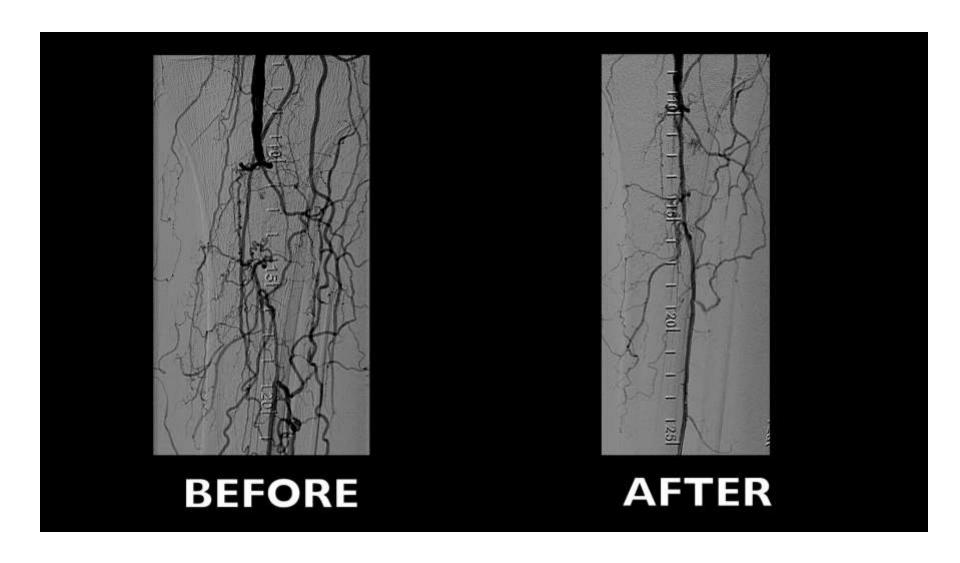




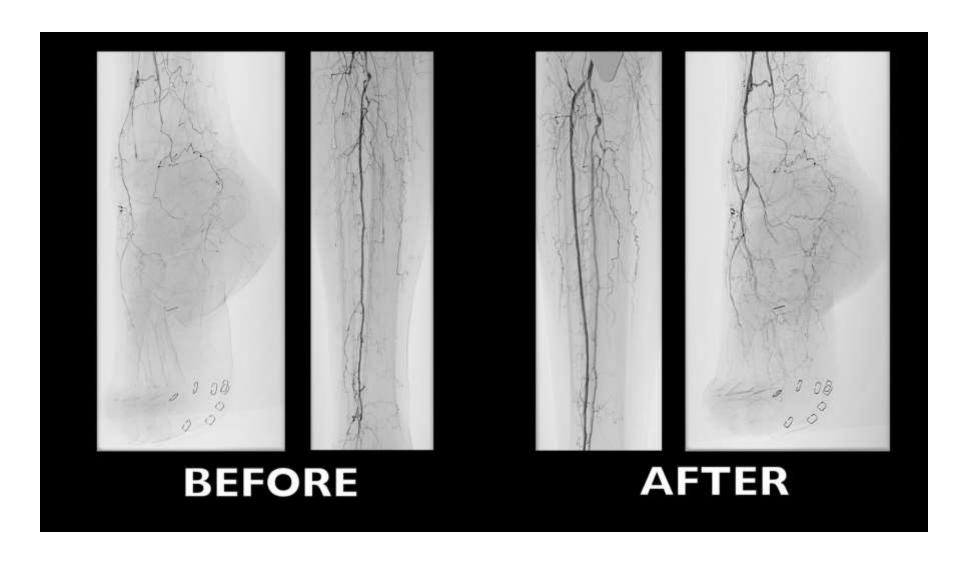




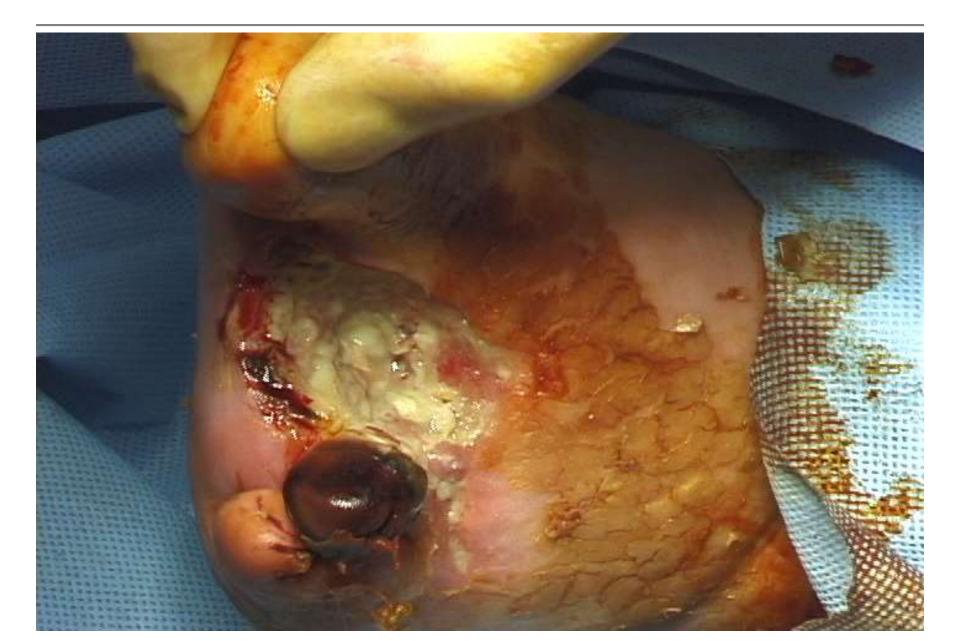




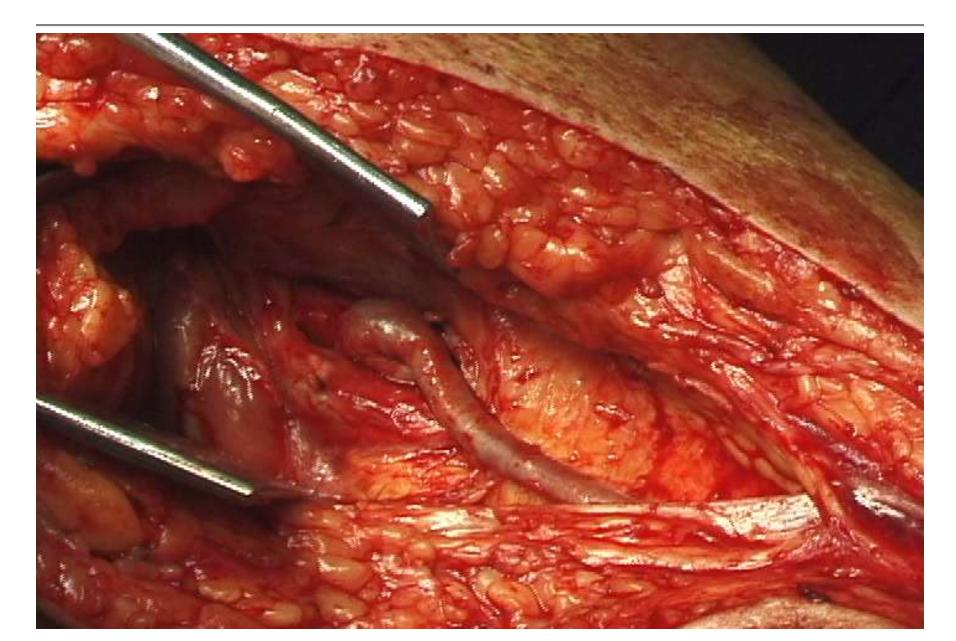




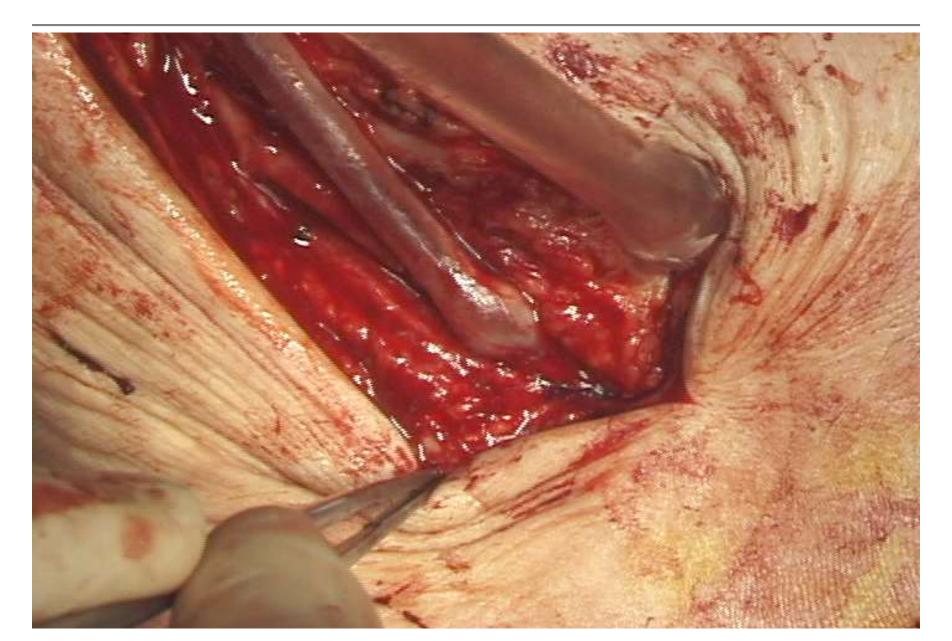


























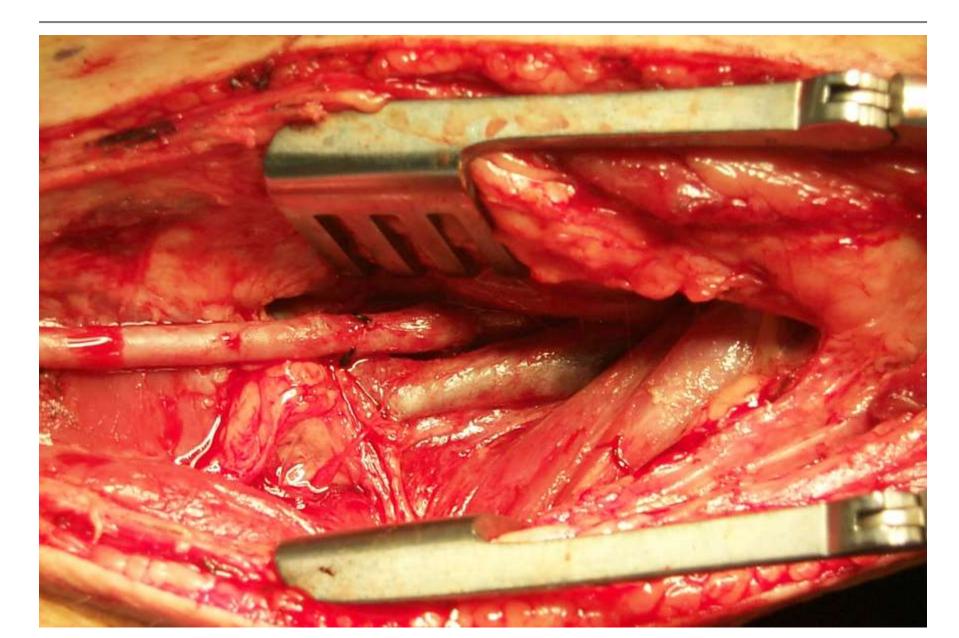












37 YO DM WITH ESRD





37 YO DM WITH ESRD







RESULTS OF ARTERIAL RECONSTRUCTION



To prevent a delay in treatment early vascular consultation and revascularization important as >85% of amputations may be prevented by early detection and appropriate treatment

CRITICAL LIMB ISCHEMIA



Emergency Room Physician Suspects Critical Limb Ischemia



Alerts Vascular Surgeon

Vascular Surgeon Alerts: Anesthesiology Nurse Supervisor Nursing Supervisor Alerts:

Operating Room
Interventional Radiology Staff (possible)



- · STAT CTA Abdomen/Pelvis with bilateral lower extremity runoff
- · Hydrate patient with NS 150 cc/hr (unless renal failure)
- Administer 100 Units/kg as loading dose Heparin IV followed by 30 Units/kg/hour Heparin gtt
- Aspirin 81 mg po
- Type and Cross for 2 units PRBC
- NPO



Operating Room (Ensure C-Arm and Angio table)

- · 1 gram Vancomycin, Esmolol, Neosynephrine, Heparin gtt
- · A Line, 2 Large Bore IV's in holding
- Maintain patient SBP 120 140 mmHg during surgery by titrating drips rather than bolus



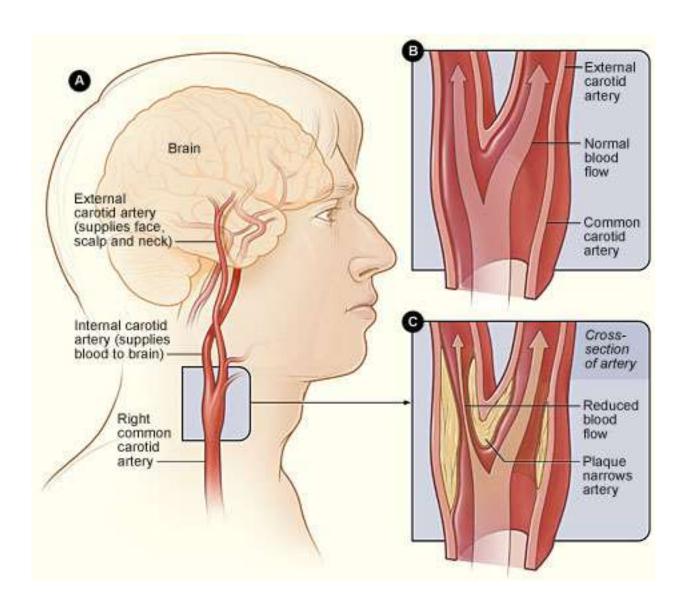
Expeditious Revascularization

Open vs Endovascular Repair

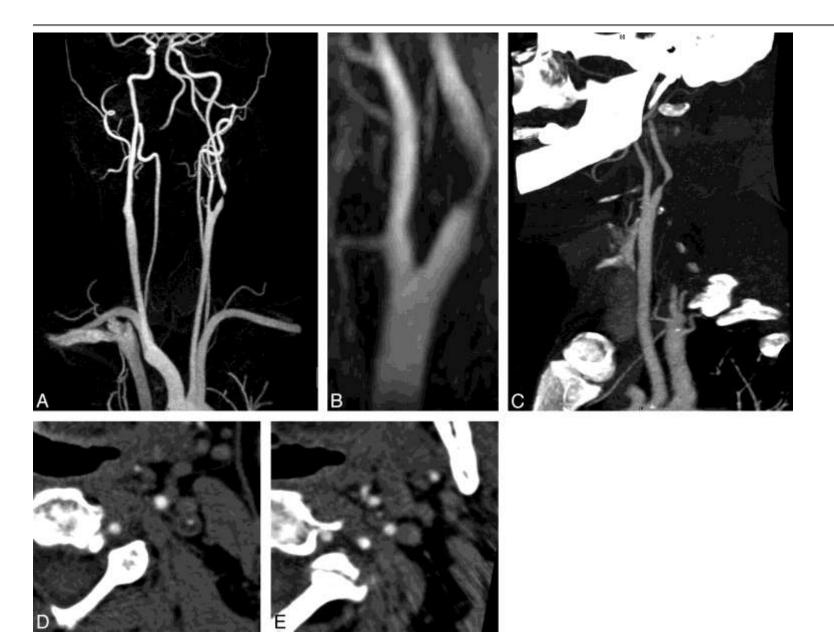
Prophylactic fasciotomy if greater than 6 hours of ischemic time



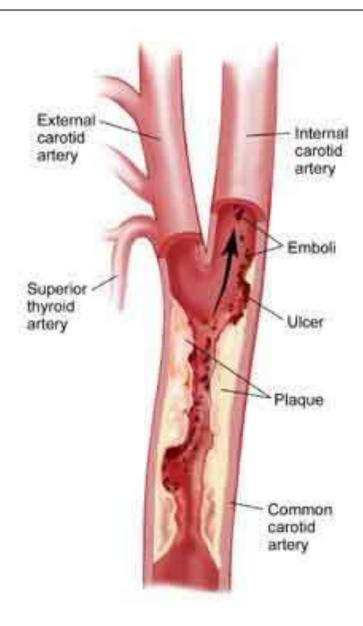




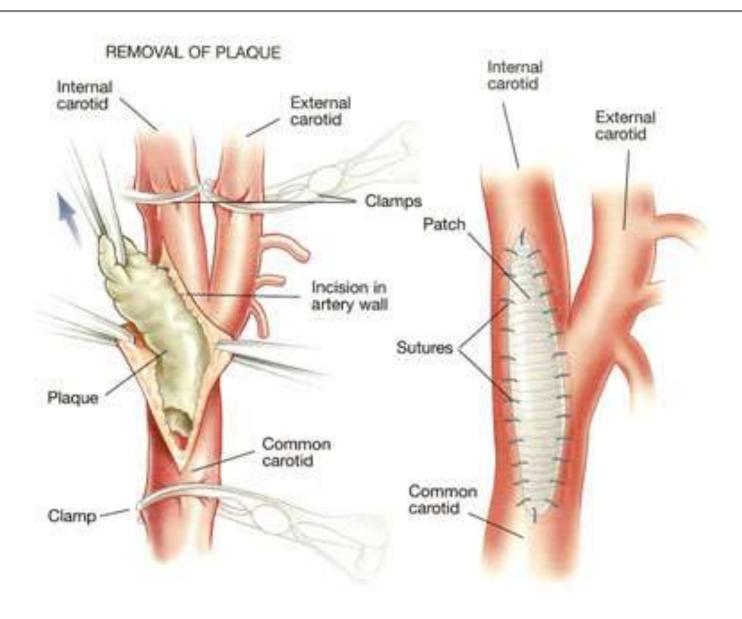
















RANDOMIZED CEATRIALS



Symptomatic disease

- NASCET 1991, 1998
- VAST 1991
- ECST 1991

Asymptomatic

- VAAT 1993
- ACAS 1995
- ACST 1995

NORTH AMERICAN SYMPTOMATIC CAROTID ENDARTERECTOMY TRIAL (NASCET)



- 50 centers in US and Canada
- Stratified patients into 30-69% and 70-99% stenosis

Eligibility:

TIAs or minor strokes within 3 mo. of entry into study

NASCET: 70-99% 2-YEAR RESULTS



	Medical Rx Alone	Surgical Rx (+BMM)	P Value
Ipsilateral	61/331	26/328	<0.001
stroke	(26%*)	(9%*)	
Any stroke	64/331	34/328	<0.001
	(27.6%*)	(12.6%*)	
Any stroke or death	73/331	41/328	<0.001
	(32.3%*)	(15.8%*)	

^{*} Kaplan-Meier Estimate

NASCET: 50-69% 2-YEAR RESULTS



	Medical Rx Alone	Surgical Rx (+BMM)	P Value
Ipsilateral	80/428	57/430	0.045
stroke	(22.2%*)	(15.7%*)	
Any stroke	113/428	85/430	0.026
	(32.3%*)	(23.9%*)	
Any stroke or death	156/428	120/430	0.005
	(43.3%*)	(33.2%*)	

^{*} Kaplan-Meier Estimate

NASCET: <50% 2-YEAR RESULTS



	Medical Rx Alone	Surgical Rx (+BMM)	P Value
Ipsilateral	110/690	89/678	0.16
stroke	(18.7%*)	(14.9%*)	
Any stroke	151/690	148/678	0.88
	(26.2%*)	(25.7%*)	
Any stroke or death	209/690	208(678)	0.97
	(37.0%*)	(36.2%*)	

^{*} Kaplan-Meier Estimate

EUROPEAN CAROTID SURGERY TRIAL (ECST)



- 10 yr period, 2518 patients with CVAs, TIAs or retinal infarctions were randomized to medical vs. surgical + BMM treatment with 3 yrs. follow up
- 80 medical centers in 14 countries
- Medical treatment left up to discretion of physician
- 6mo interval of qualifying event (vs 3mo in NASCET)

ECST: >70% 2-YEAR RESULTS



	Medical Rx Alone	Surgical Rx (+BMM)	P Value
Ipsilateral stroke	16.8%	2.8%	<0.0001
Disabling or fatal stroke	11%	6%	<0.05
Any stroke or death	21.9%	12.3%	<0.01

ASYMPTOMATIC CAROTID ATHEROSCLEROSIS TRIAL (ACAS)



 1662 men and women with asymptomatic ≥ 60% stenosis were randomized to best medical therapy vs. surgery plus BMM

NASCET: >60% 3-YEAR RESULTS



	Medical Rx Alone	Surgical Rx (+BMM)	P Value
Ipsilateral	92/834	42/825	0.006
stroke	(11%*)	(5.1%*)	
(+periopera			
tive death/strok e)			
Any stroke	266/834	211/825	0.08
or death	(31.9%*)	(25.6%*)	

CAROTID STENTING: CURRENT INDICATIONS



- Early recurrent stenosis
- Hostile neck (radiation, radical neck dissection, tracheostomy, infection)
- Surgically inaccessible lesion
- Fibromuscular dysplasia
- High medical risk (hard to define)
- In setting of approved RCT (Not covered by CMS for asymptomatic disease)

SYMPTOMATIC CAROTID PATIENTS



- Stenosis ≥50%
 - CEA + BMM
- Stenosis ≥50% and high perioperative risk
 - CAS (as potential alternative)
- Stenosis <50%
 - BMM
- CEA within 2 weeks of symptoms

ASYMPTOMATIC CAROTID PATIENTS



- Stenosis ≥ 60% and low risk
 - CEA + BMM
- Stenosis < 60%
 - BMM

- Stenosis ≥ 80% and high anatomic risk for CEA
 - CAS (exceptions)



THANK YOU