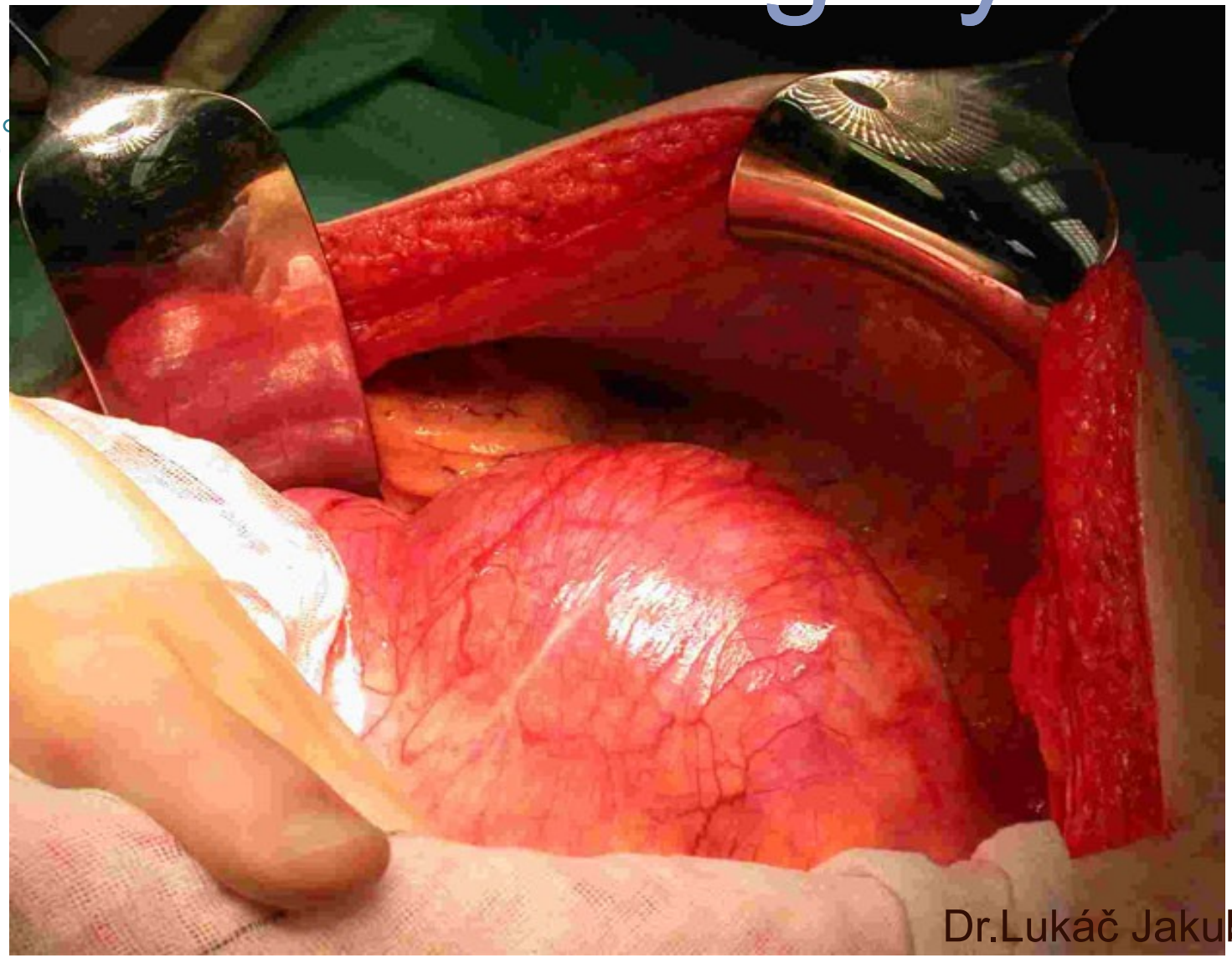
Vascular surgery

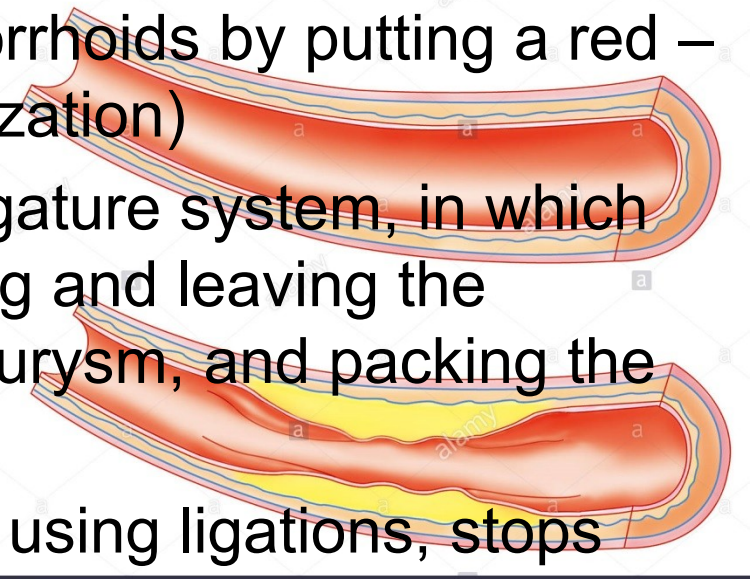


Dr.Lukáč Jakub

FN Brno –Trauma dept.

...little bit of history first

- Studies on egyptian mummies revealed, that people more than 3500 yrs back suffer from atherosclerosis
- Ebers Papyrus (2000 b.c.)-identified peripheral arterial aneurysms, and suggested forms of treatment , e.g. „**treat it with a knife, and burn it with a fire, so it doesnt bleed so much**“ LOL :D
- Hippokrates (400 b.c.) – treated hemorrhoids by putting a red – hot iron in patients anus (first cautherization)
- Antyllus (2 century a.d.)- invented a ligature system, in which he applied ligatures to arteries entering and leaving the aneurysm, then cutting the sac of aneurysm, and packing the cavity
- Ambroise Paré (16th century) – starts using ligations, stops with boiling oil and cautherization



Dark ages treatment of hemorrhoids with hot iron

What is vascular surgery?

Vascular surgery is surgical subspecialty, which is dealing with diseases of vascular system, including lymphatic venous system.

Todays trend is to treat as much as possible conservatively, with medication, or using minimally – invasive procedures.

When need arises, open surgery of vascular reconstruction is done.

Vascular surgeon treats vascular problems,except for heart and brain vacular conditions.

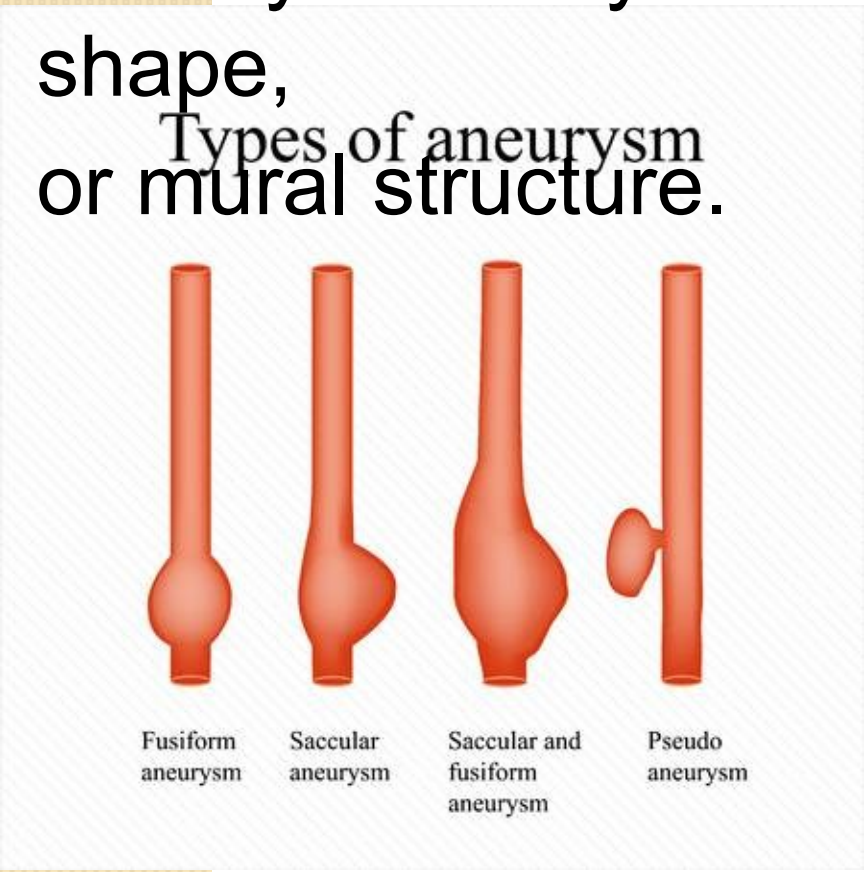
What are the most common vascular diseases?

- **Abdominal aortic aneurysm**
- **Aortic dissection**
- Atherosclerosis
- **Chronic venous insufficiency**
- **Deep venous thrombosis**
- **Peripheral arterial disease**
- **Thoracic aortic aneurysm**
- **Varicose veins**
- Haemorrhoids
- **Vascular trauma**
- Pulmonary embolism
- Lymphedema
- Carotid artery disease
- and other....

Aneurysms

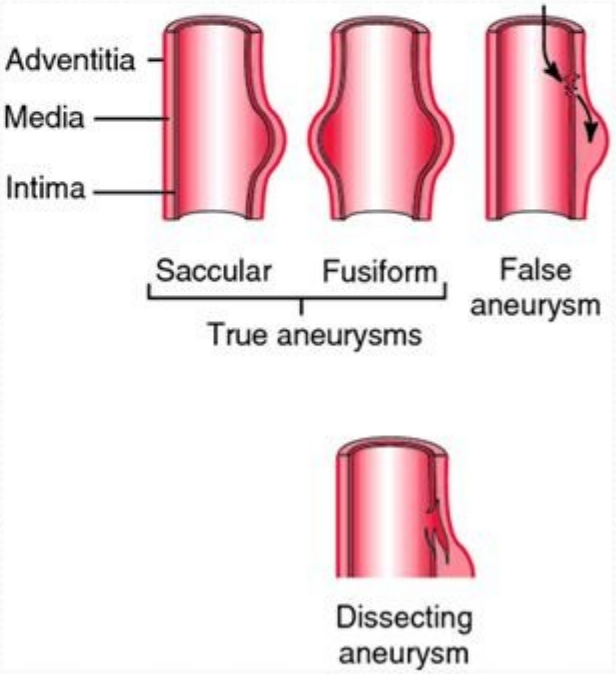
Abnormal, localized weak spot on artery wall, that causes the wall to bulge outward, like a baloon.

Aneurysms may be divided due to localization, shape,
or mural structure.



Types of Aneurysms/ Layers

- **True**: involves all 3 layers of the arterial wall
- **False (pseudo aneurysm)**: presence of blood flow outside of normal layers of arterial wall. Wall of false aneurysm is compose of the compressed, surrounding tissues.
- **Dissecting:** tear in the intema, blood goes to the space between intema and media.



Shapes

Globous

Pear

Beak

Bilobar

Multilobar

Fusiform

Some statistics

-prevalence of AA in older than 65 yrs is 3-4 %

-prevalence in thoracic AA is 6 cases in 100 000 people

-males suffer more often than females

-in last 30.yrs, prevalence of aortic aneurysms has increased

-incidence is increased also

Signs and symptoms-thoracic aneurysm

-in many patients it is discovered incidentally

- depending on the affected part of the aorta

Ascending thoracic aorta aneurysm:

-pain in neck, chest or back

-swelling of head, neck, extremities

-heart failure due to aortic valve regurgitation, distal embolism, rupture

Aortic arch and descending aorta aneurysm:

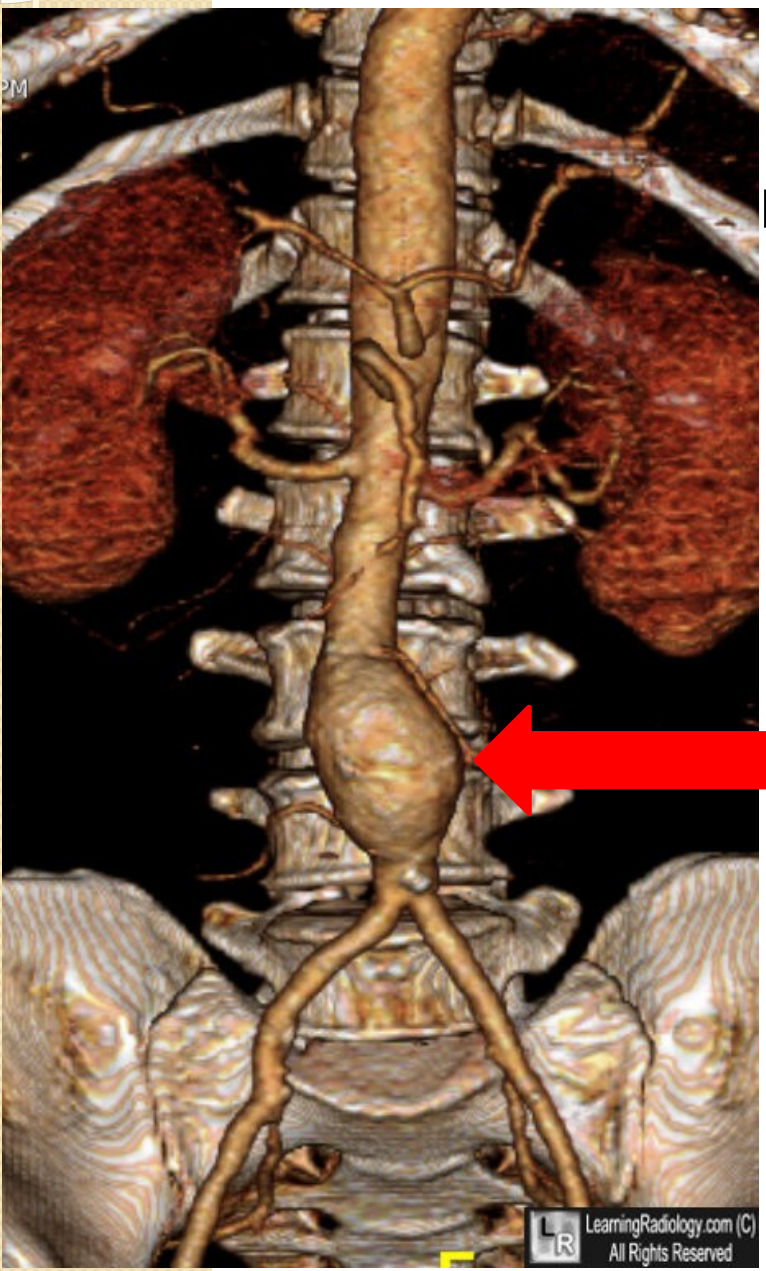
-wheezing,coughing, shortness of breath – trachea compression

-dysphagia, chest pain, hoarseness

other signs and symptoms....

- Heart murmurs
- Paraparesis/paraplegia
- Substantial part of all aortal dilatations remains asymptomatic
- Hemoptysis, hematemesis
- Nausea
- Constipation
- Tachycardia, sweaty skin

Abdominal aorta aneurysm



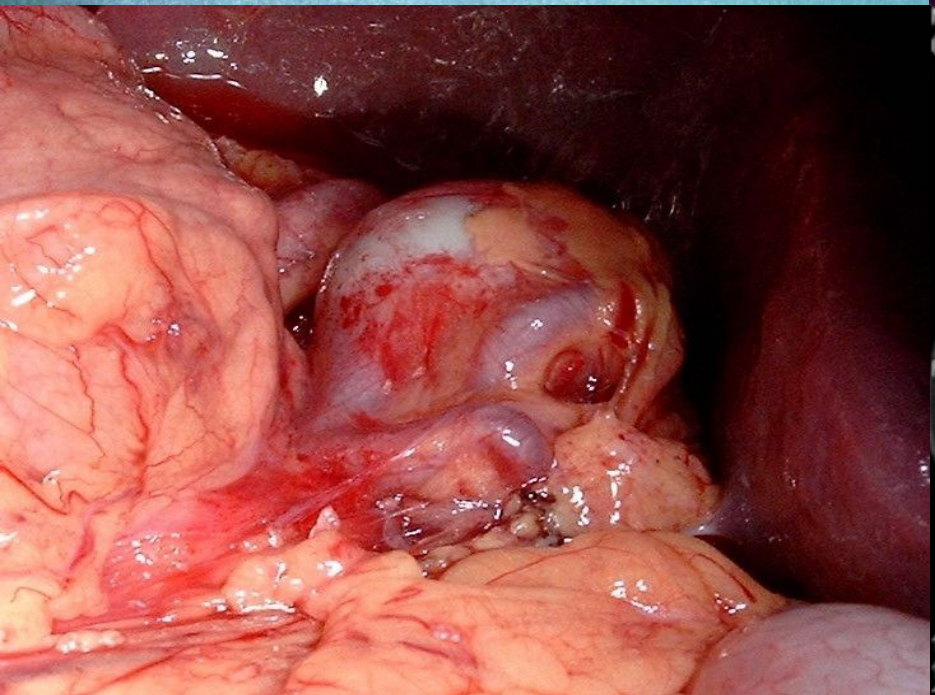
Enlargement of aorta below diaphragm

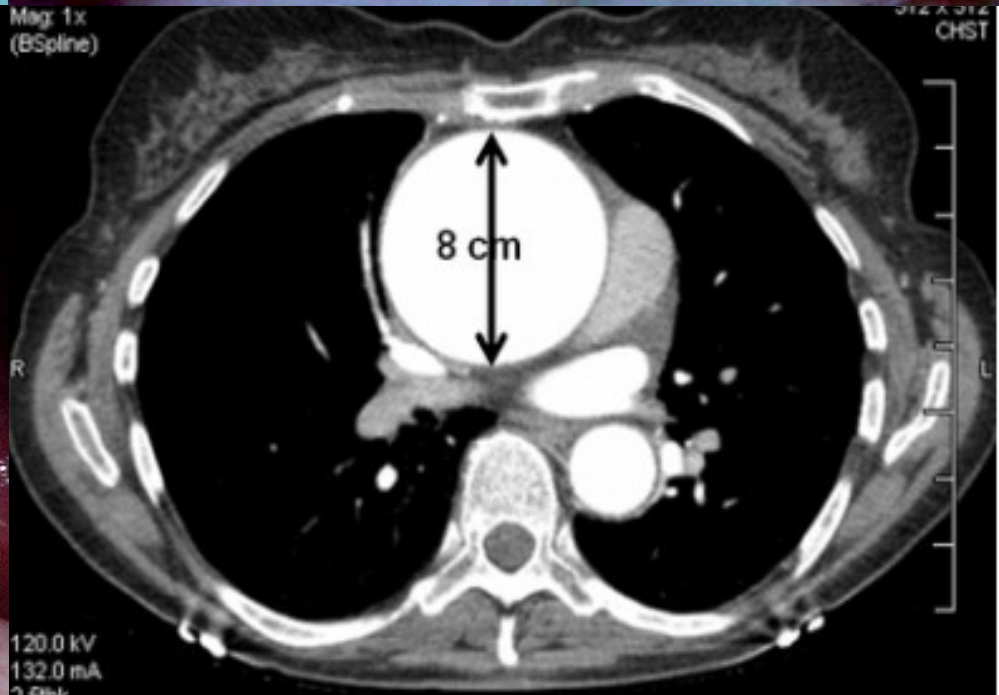
Dilatation over 3 cm is considered an aneurysm

Diagnostics

Anamnesis
Physical examination
Clinical presentation
Laboratory
ECG
Chest and abdominal Xray (according to urgency)
Ultrasound
CT scan (CT angiography)
MRI


Poplitea-Aneurysma




Mag: 1x
(BSpline)
CHST
8 cm
R
120.0 kV
132.0 mA

Conservative approach

In patients with high mortality risk

No improvement of life-expectancy

Less than 5,5 cm in diameter – observation

Growth less than 1 cm per year

Asymptomatic, „small“ aneurysms

Genetics

High predisposition in patients who suffer from some form of connective tissue disorder, e.g.

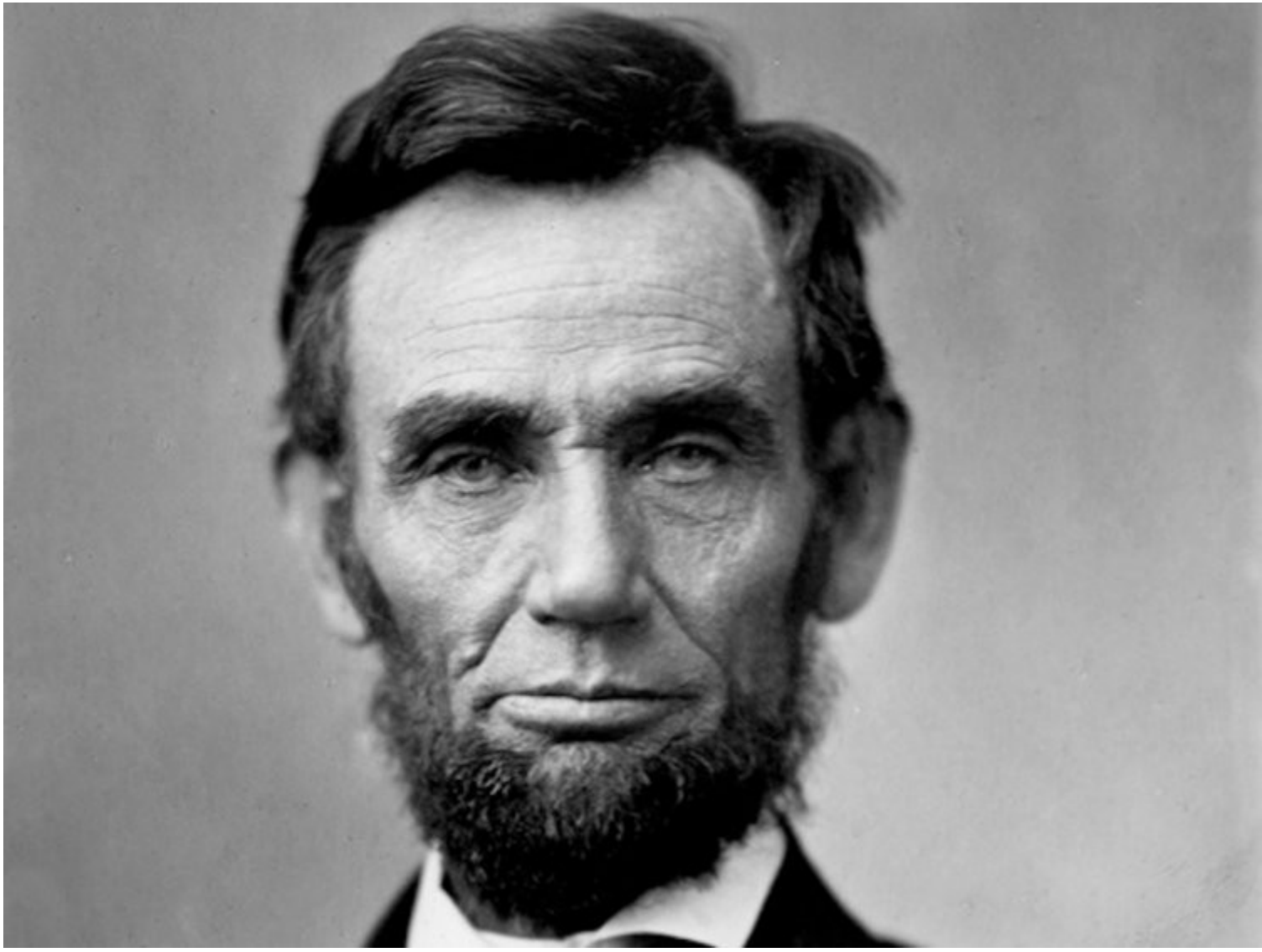
MARFAN SYNDROME

EHLER´S –DANLOS SYNDROME

RELAPSING POLYCHONDRITIS

Characters of past

Marfans syndrome

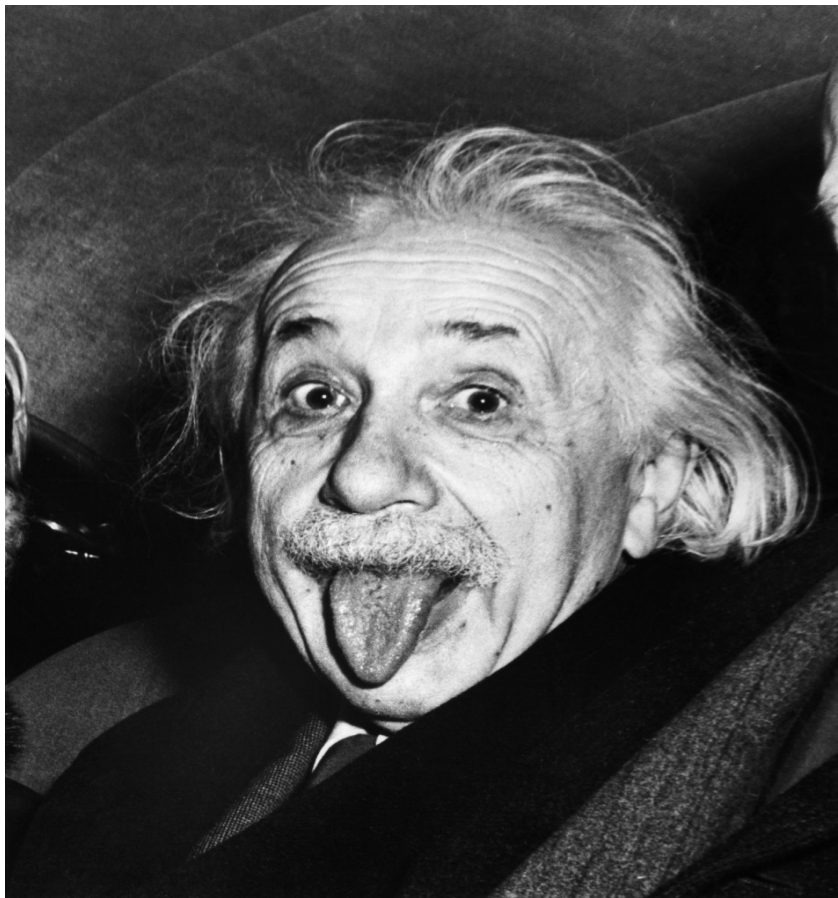


Characters of past

Diagnosed with abdominal aorta aneurysm

-treated by Nissen, who wrapped it in celophane

☺



Prophylaxis

- STOP SMOKING!!! Damn it! –ESSENTIAL FACTOR
- Control your blood pressure
- Correct your diet – less fatty food
- Consume less alcohol
- Regular check-ups with control X ray
- More movement

SURGERY

Open surgery – in younger patiens

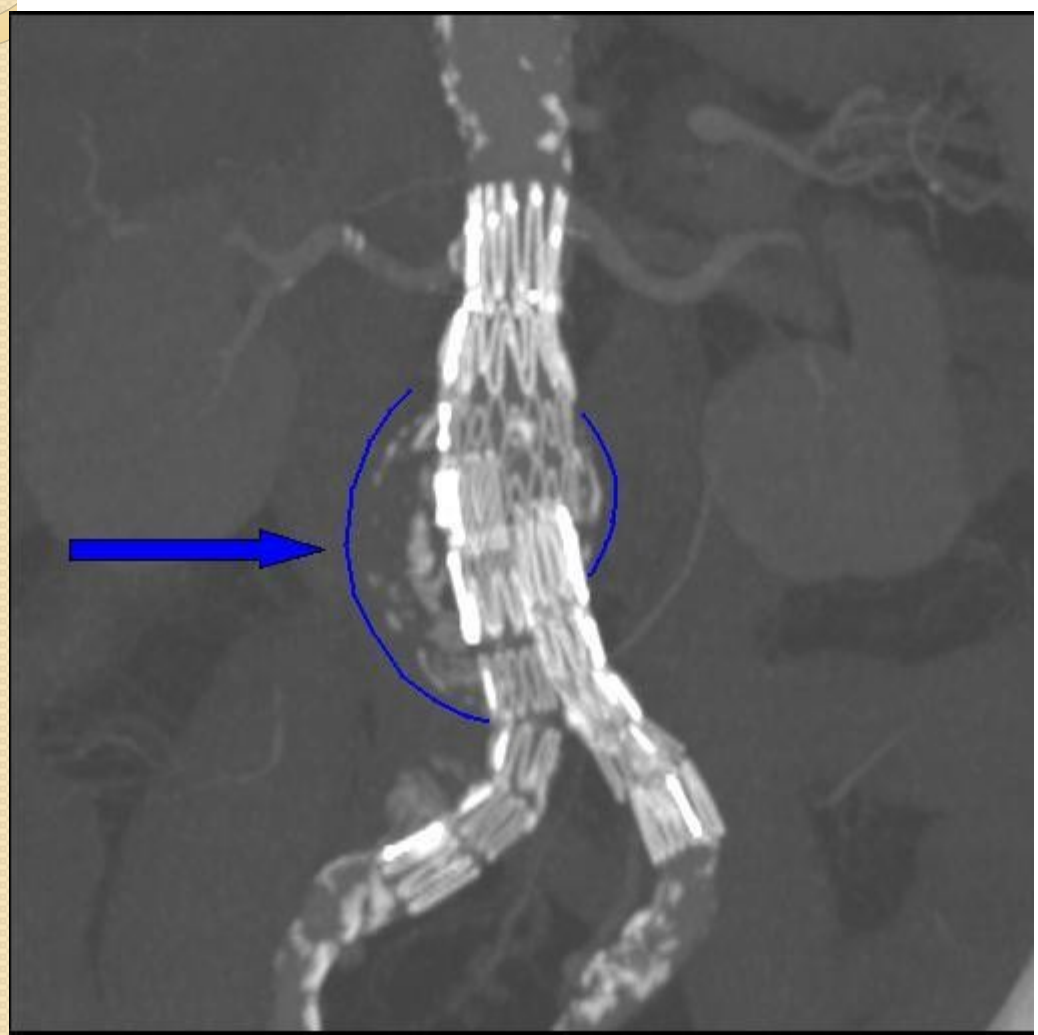
- already ruptured
- symptomatic
- elective

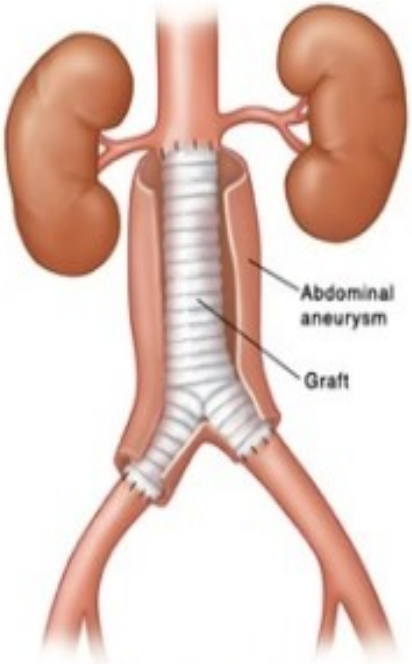
Endovascular repair- older patients

(EVAR)

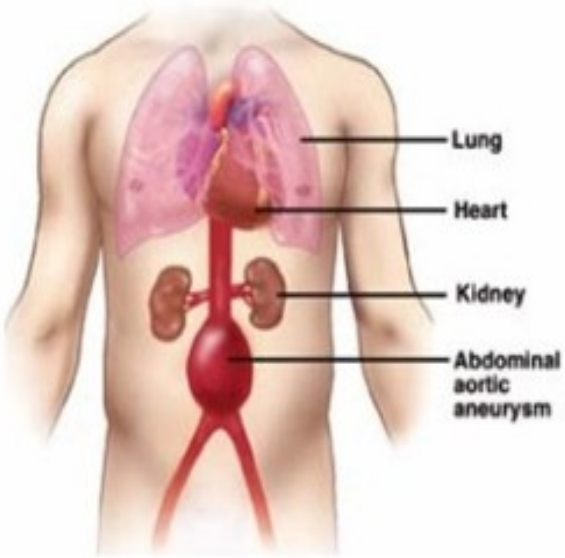
- unfit for open surgery
- feasible for only some types

In general, there is no significant advantage when comparing open end EVAR technique

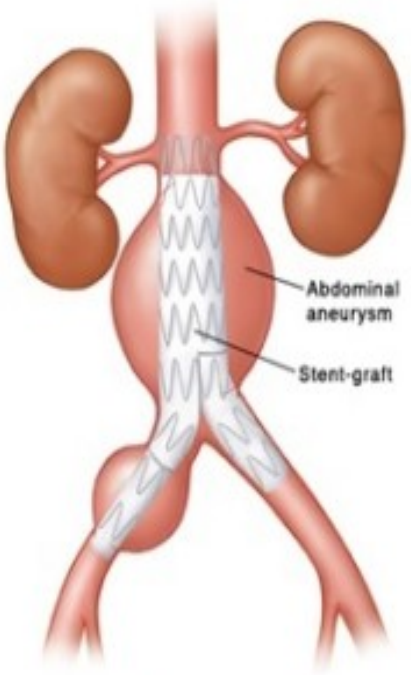




Open surgery for an abdominal aortic aneurysm



Abdominal aortic aneurysm (simple)



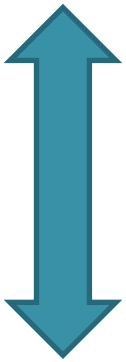
EVAR (endovascular aneurysm repair)

AORTIC DISSECTION

Injury of inner layer of aorta

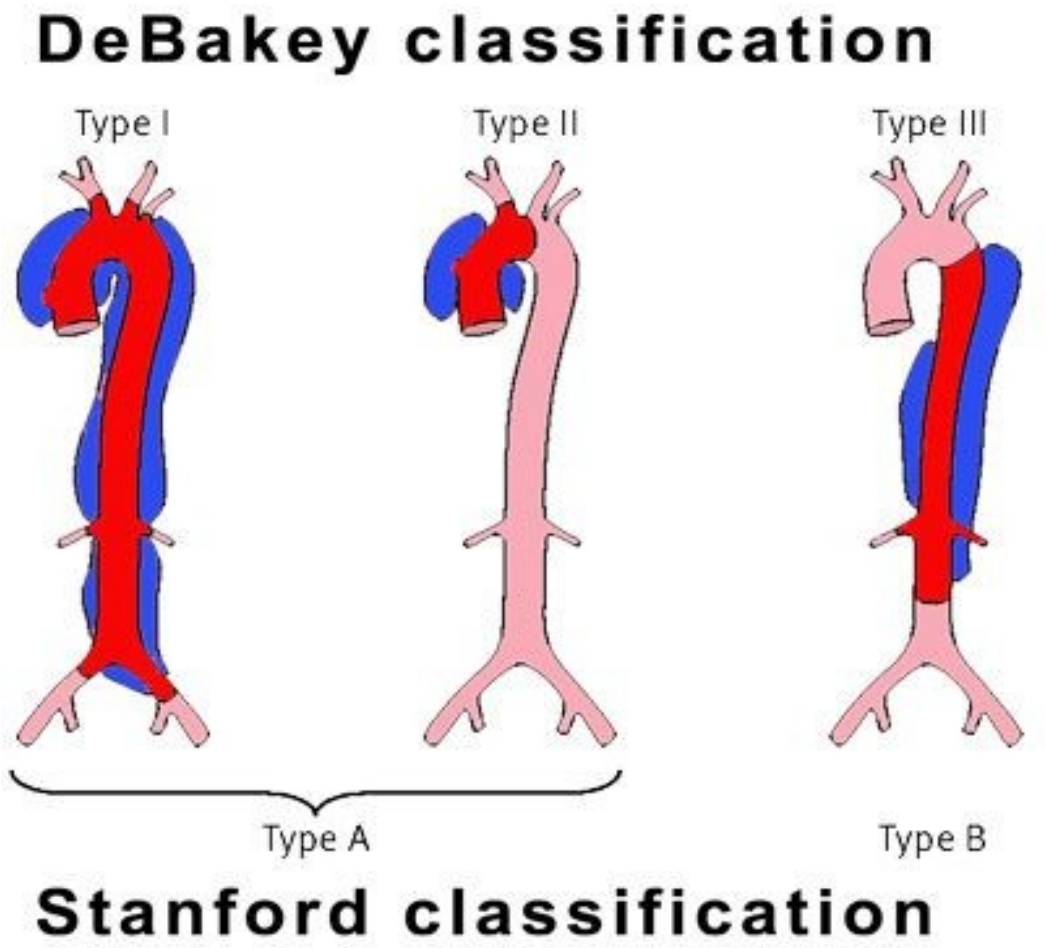
Blood flows through deffect into medial layer of aortic wall, creating FALSE LUMEN

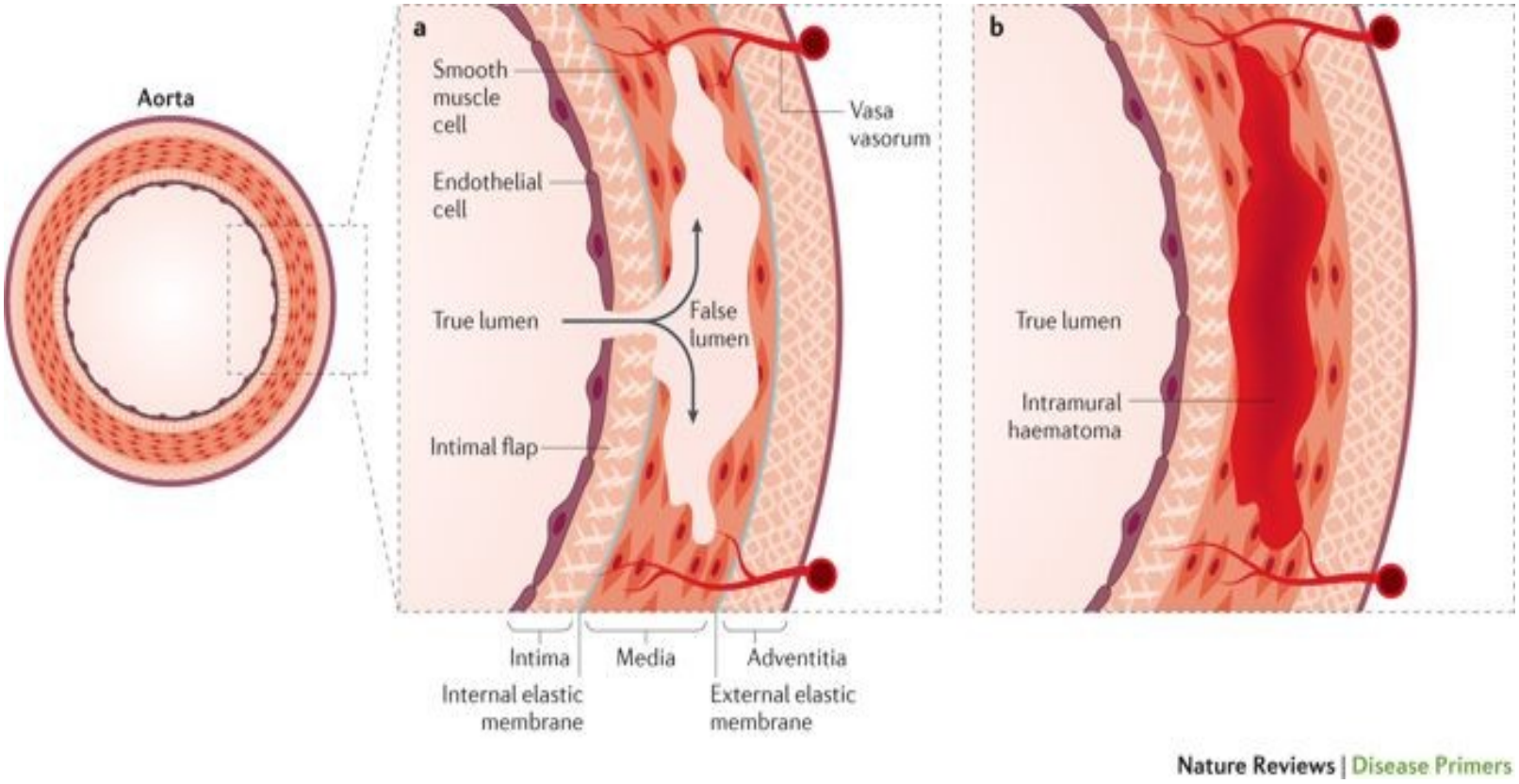
Difference :



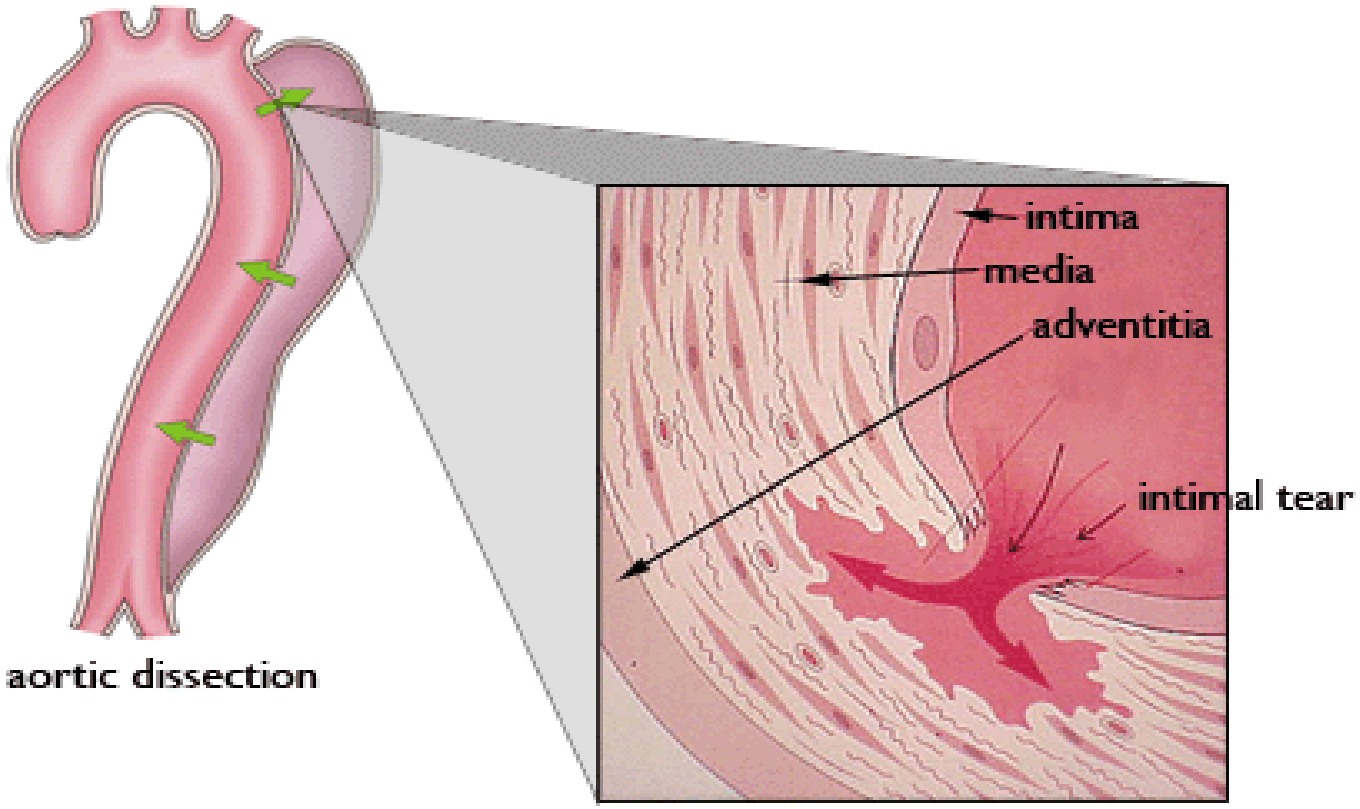
Aortic aneurysms have TRUE LUMEN

Classification





Nature Reviews | Disease Primers

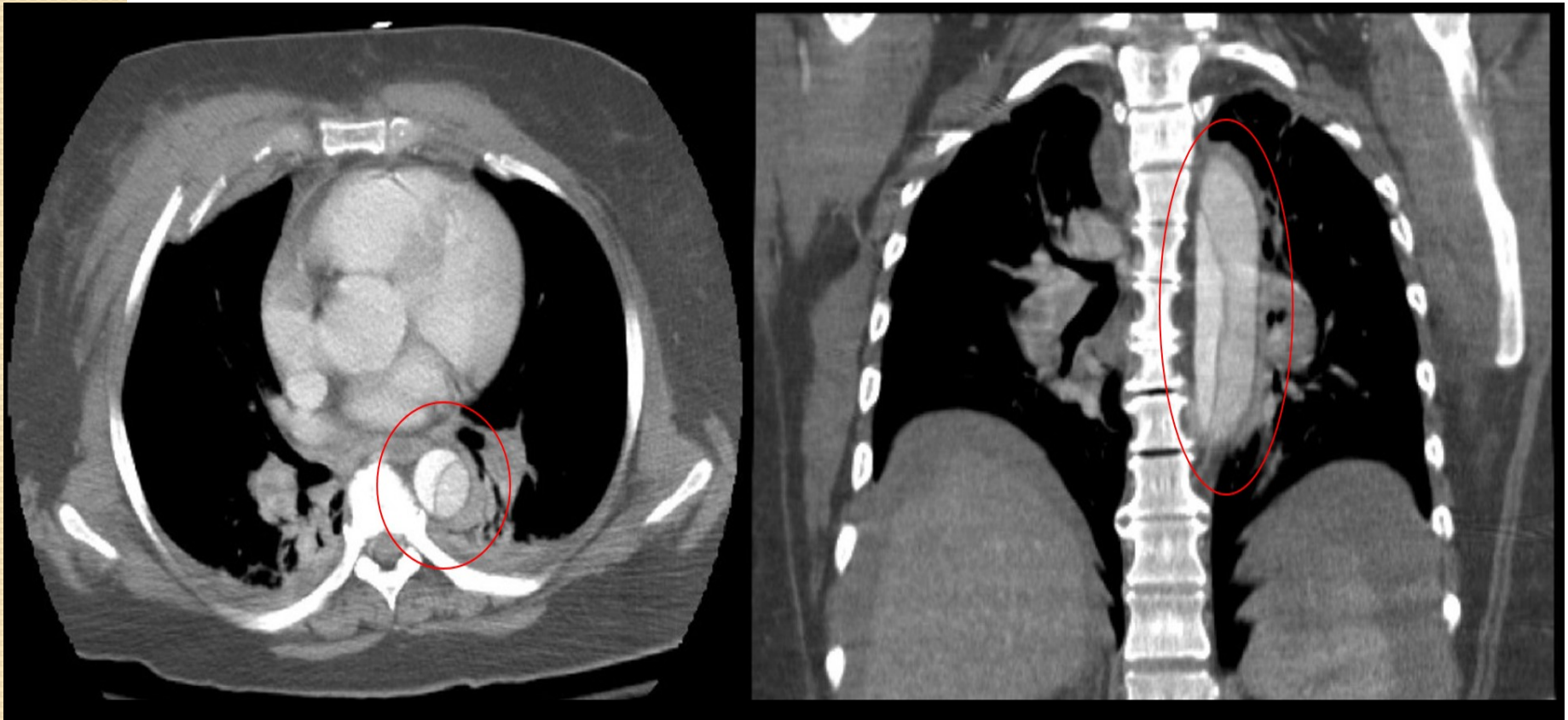

intima
media
adventitia
intimal tear
aortic dissection

Causes

- Atherosclerotic plaque rupture
- In general similar/same as aneurysm
- After trauma

CT – crescent shape in axial plane

Identifiable layering

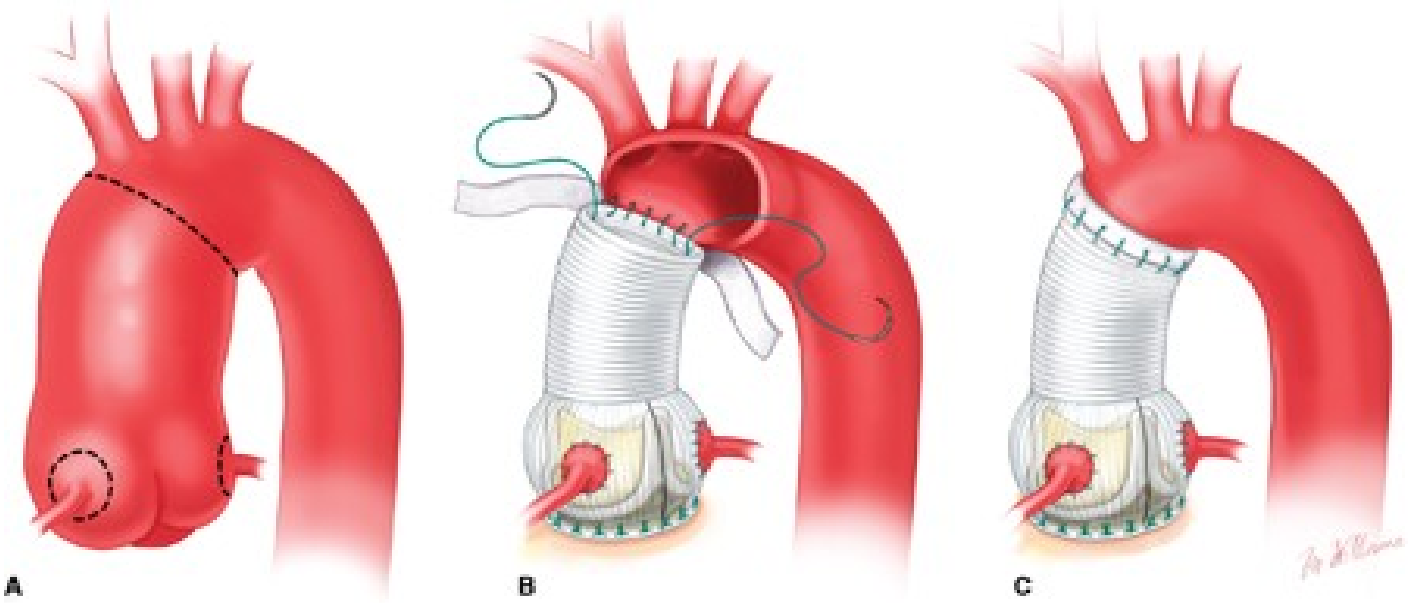


SURGERY

Especially in ascending aorta dissection – surgical approach

SURGERY

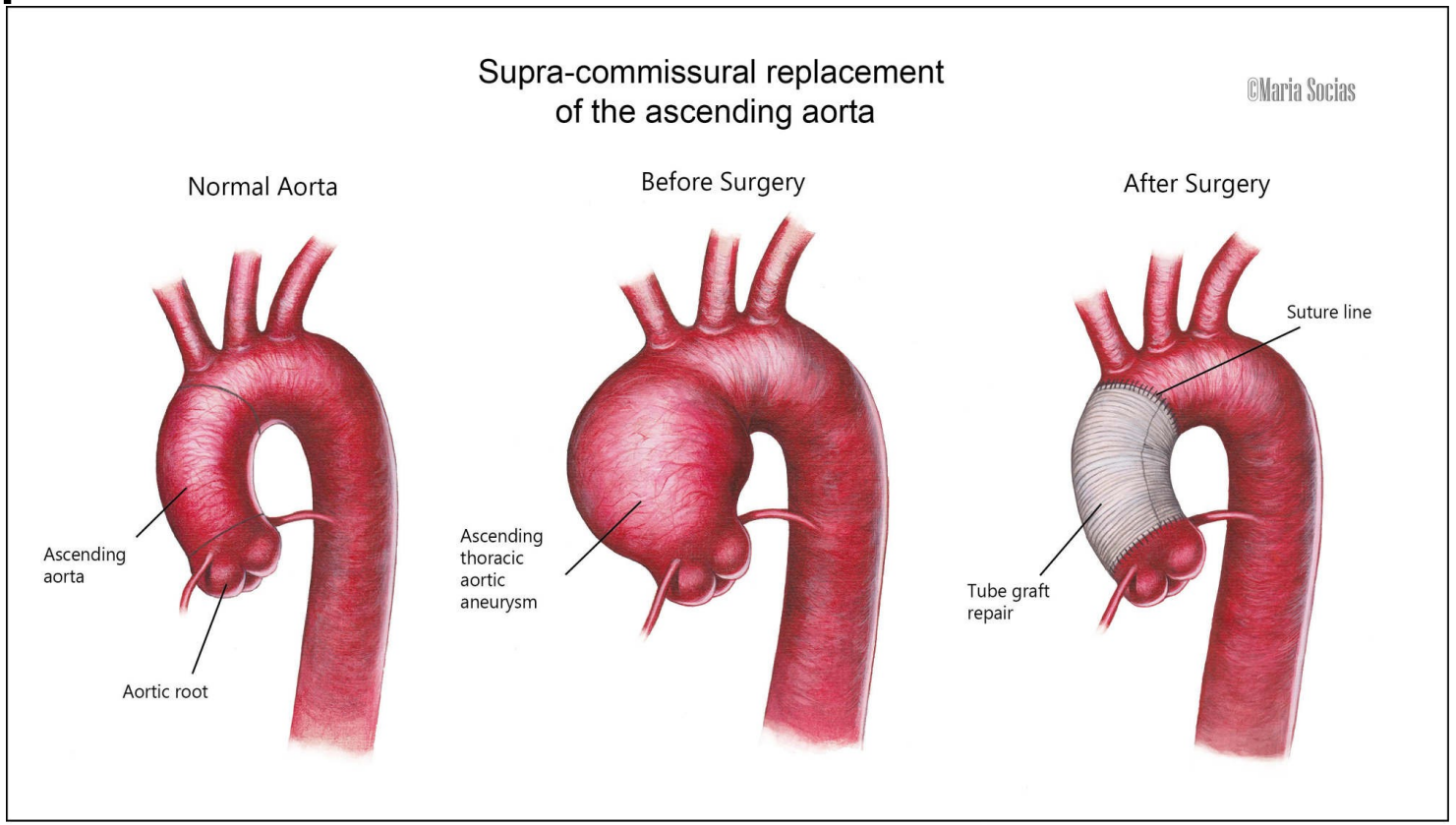
Bentall procedure – graft replacement of ascending aorta, aortic root, with re-implantation of coronary arteries



Source: Cohn LH: *Cardiac Surgery in The Adult, 4th Edition*: www.accesssurgery.com

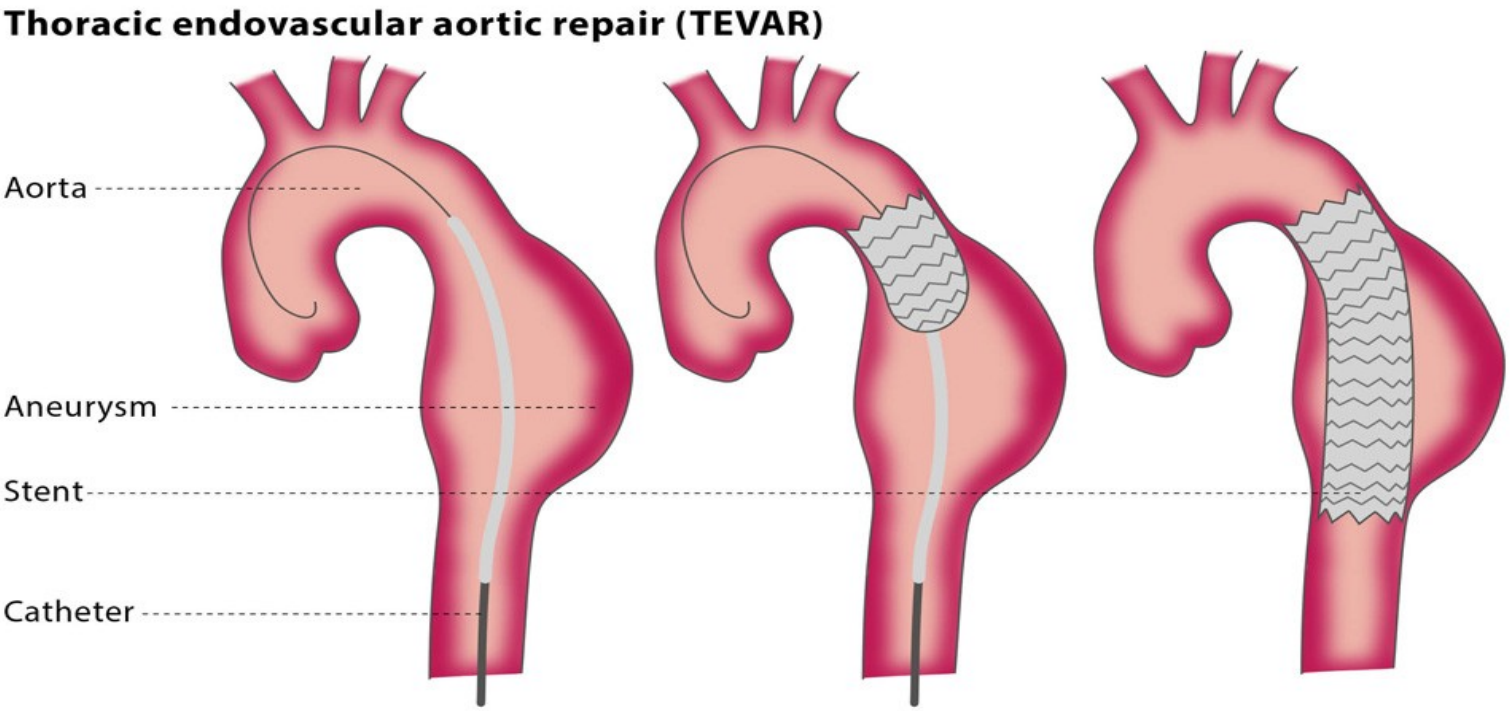
SURGERY

David procedure- valve sparing aortic root replacement

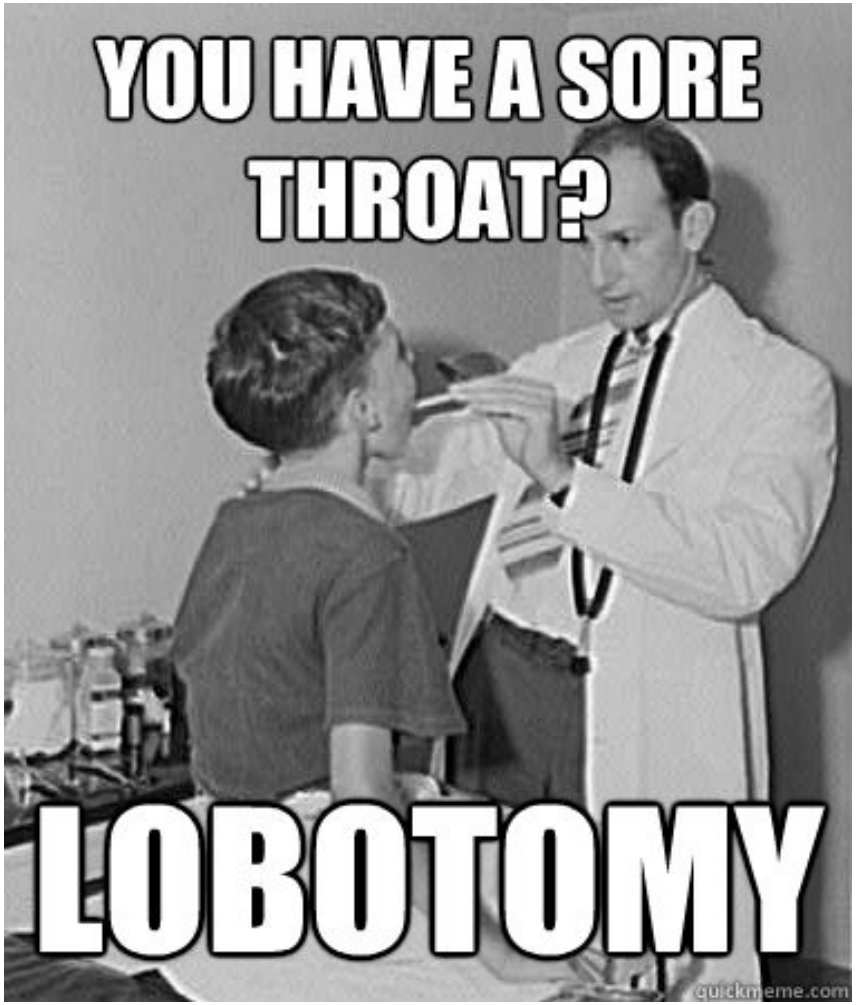


SURGERY

EVAR technique in descending aorta dissection



Short opportunity to take a breather ☺



Peripheral arterial disease (PAD)

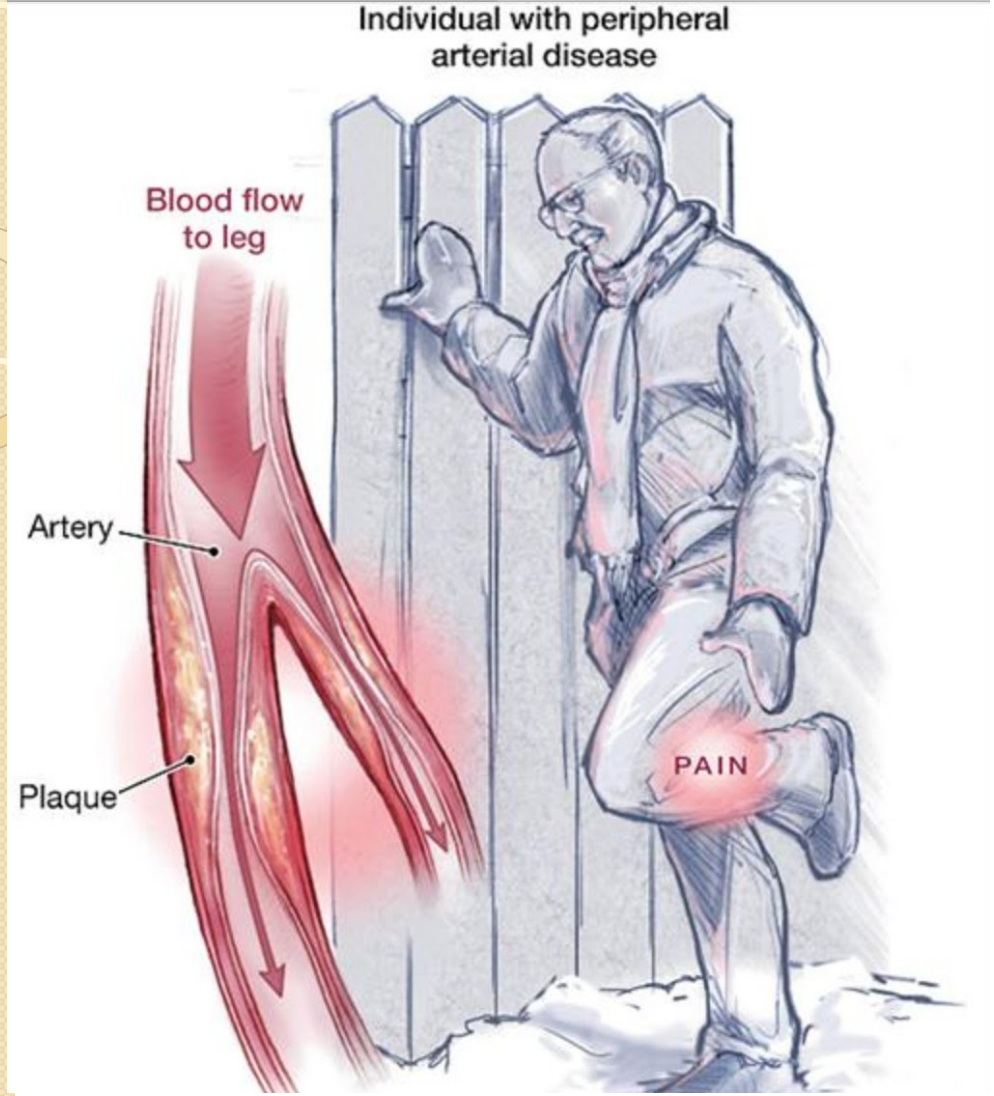
Narrowing of the peripheral arterial system, usually due to atherosclerosis, worsened by other risk factors (diabetes, smoking, hyperlipidemia, artery spasms, hypertension etc...)

Clinical presentation

- Cramping in feets, legs and calfs
- Burning sensations
- Numbness in feet and legs
- Legs/feet cool to touch
- Thick toenails
- Worsened healing

CLAUDICATION!!!!

- pain during physical activity, walk



Patient suffers from pain during walk,and needs a rest. During inactivity, pain dissapear.

Peripheral arterial disease symptoms

Pain in hip, thigh

Leg weakness

Coldness in lower leg

Sores on toes, feet or legs

Change in color of legs

Hair loss

Slower growth of toe nails

Shiny skin on legs

Erectile dysfunction in men

CLASSIFICATION

Fontaine classification

- Stage I Asymptomatic,
- Stage II - Mild claudication pain in limb
  - Stage IIA -Claudication at a distance > 200 m
  - Stage IIB -Claudication at a distance < 200 m
- Stage III - Rest pain, mostly in the feet
- Stage IV -Necrosis and/or gangrene of the limb

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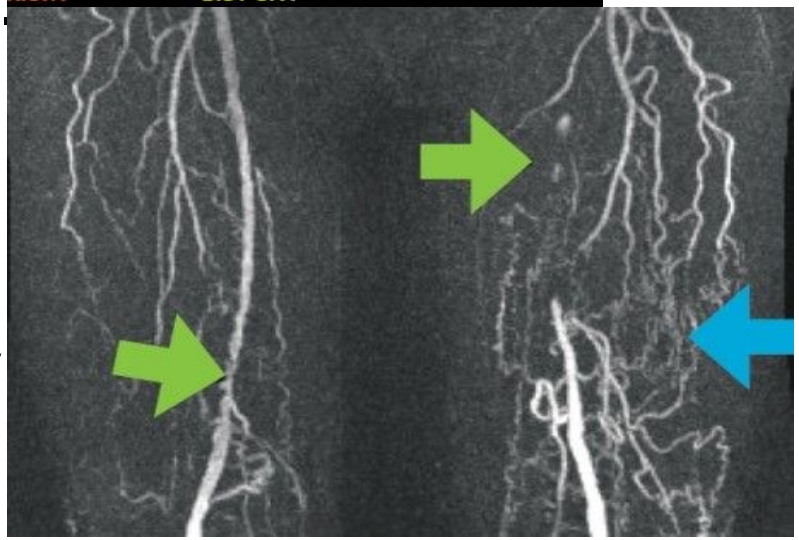
Rutherford classification

Grade	Category	Clinical description
0	0	Asymptomatic
I	1	Mild claudication
I	2	Moderate claudication
I	3	Severe claudication
II	4	Ischemic rest pain
III	5	Minor tissue loss – nonhealing ulcer, focal gangrene with diffuse pedal ischemia
III	6	Major tissue loss – extending above transmetatarsal level, frank gangrene

DIAGNOSIS

Doppler examination

Angiography



Ankle-brachial index

Ankle-Brachial Index (ABI)

Table 3: ABI Value-Based PAD Severity Grade[a]	
ABI Value	**Severity**
>1.30	Poorly compressible
0.91-1.30	Normal
0.70-0.90	Mild
0.40-0.69	Moderate
<0.40	Severe

ABI, ankle-brachial index; **PAD**, peripheral artery disease
[a]Based on the American Diabetes Association guidelines

$$\text{ABI} = \frac{\text{Ankle Systolic Pressure}}{\text{Arm Systolic Pressure}}$$

Recognized as an important indicator of peripheral artery disease (PAD), a common manifestation of atherosclerosis and crucial risk factor for coronary artery and cerebrovascular disease

TREATMENT

According to stage: **mild to moderate**

- change of regimen –smoking, compensation of DM
- Medication (Aspirin, statins, ACE inhib.)
- Walk/exercise with gradual overload – helps to induce angiogenesis, which provides collateral arterial blood flow

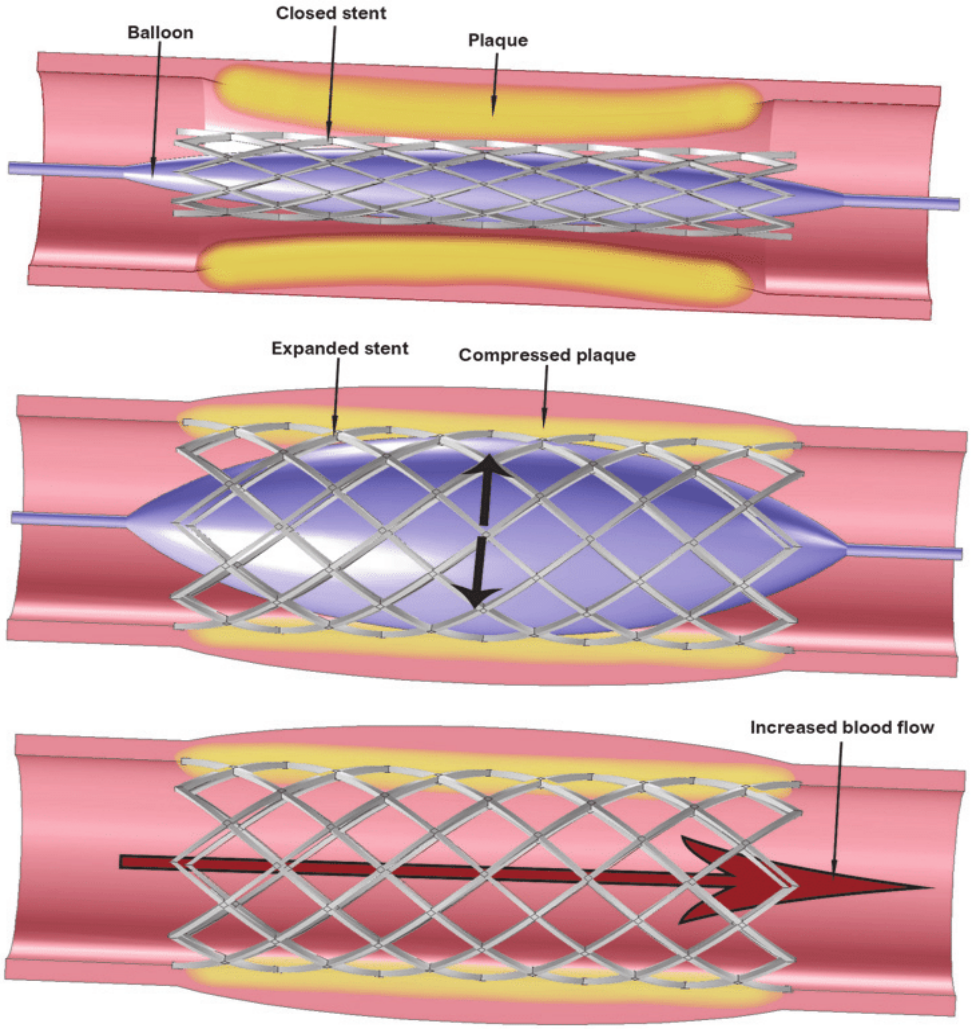
SURGERY

- PTA – Percutaneous Transluminal Angioplasty

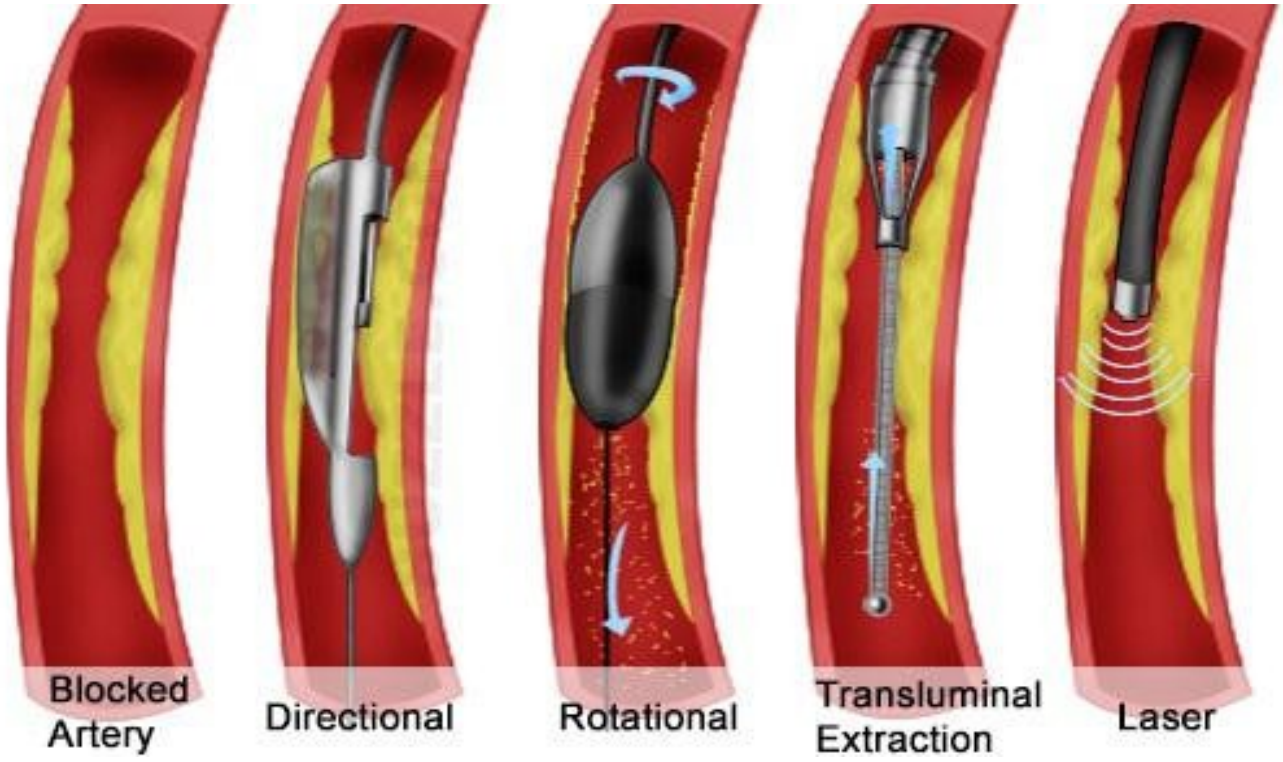
-better for solitary lesions and narrowings, such as in femoral, popliteal or iliac artery

- Atherectomy – atherosclerotic plaque removal from inside the artery-scraping
- Vascular bypass – used to circumvent the diseased area – either using VSM, or PTFE (polytetrafluorethylene) or Gora-tex graft
- Amputation – if gangrene develops
- Thrombectomy, thrombolysis

PTA


Balloon
Closed stent
Plaque
Expanded stent
Compressed plaque
Increased blood flow

Atherectomy – „snow plow with suction



Multiple devices – same effect

Successful recanalisation

Before

After

Amputation

-definite treatment of gangrene, and chronic ulcers, causing septic complications, especially in lower extremities

Gangrene

- Gangrene is a condition that involves the death and decay of tissue, usually in the extremities due to loss of blood supply.

Dry gangrene

- no infection
- little tissue liquefaction
- In early stages, dull, aching pain, extremely painful to palpate, cold, dry and wrinkled.
- In later stages, skin gradually changes in color to
  - dark brown, then
  - dark purplish-blue, then
  - completely black

Wet gangrene

- Bacterial infection
- copious tissue liquefaction
- offensive odor
- swollen, red and warm.
- usually develops rapidly due to blockage of venous and/or arterial blood flow

- Treatment is surgical debridement and amputation.

Transcutaneous oxygen measurement – TcPO2

Non- invasive, objective and realiable method which reflects the saturation of sking and soft-tissues with oxygen.

Helps to objectify ischemia, and to decide whether the extremity (defect) can be healed, or whether amputation should be performed

Transcutaneous oxygen (tcpO_2 / TCOM)

Measures the **local oxygen** tension in the skin deriving from the local capillary (nutritive) blood perfusion.

- Predicts wound healing potential
- Helps define degree of small vessel disease
- Accurately determines amputation level
- Monitors efficacy of patients ongoing therapy
- Establishes candidacy for HBO treatment

Reference values

50-70 mmHg Normal
< 40 mmHg Impaired Wound Healing
< 30 mmHg Critical Limb Ischemia

PERIMED


Recently Amputated
Great toe
Area of spreading necrosis

Vascular bypass – in PAD and in general

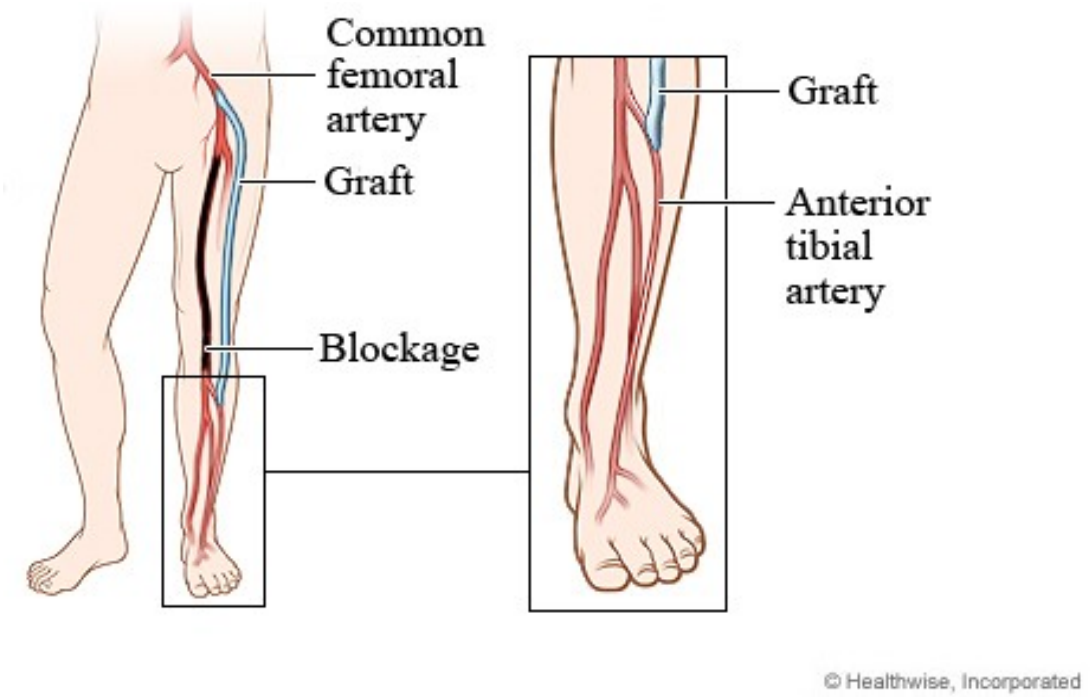
- surgical procedure, allowing the redirection of blood flow from one place to another, using either prosthetic material grafts, allo-grafts or auto-grafts
- there are many possible bypass locations, e.g.

fem-tib, ax-bifem, aorto-bifem, fem-fem

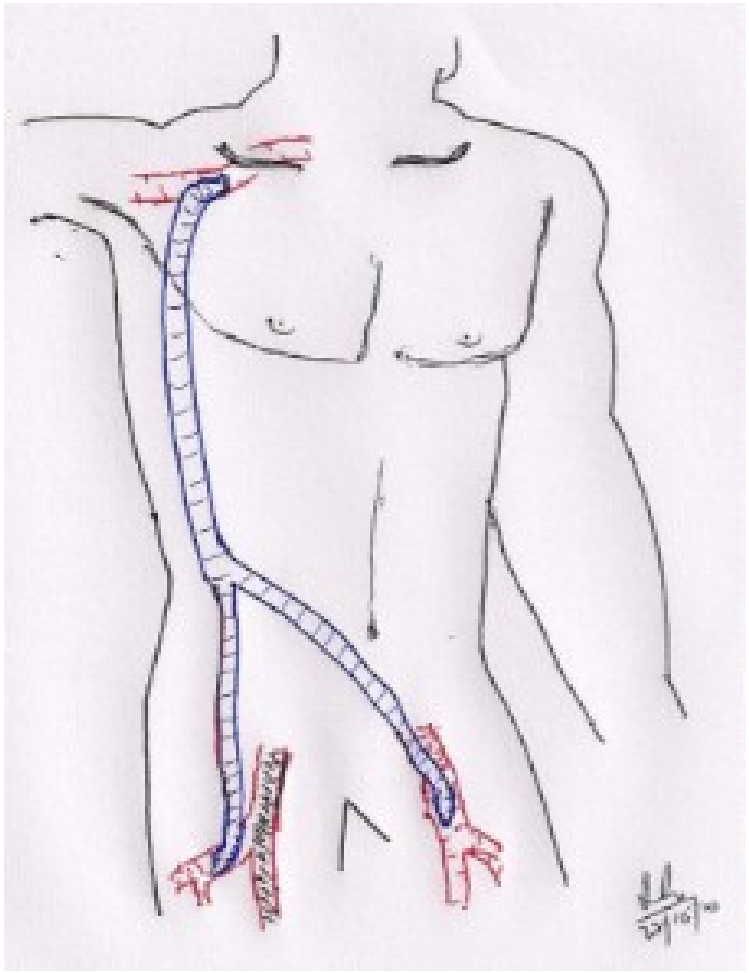
-mimicking physiological path → anatomic

-creating new, unique pathways → extra-anatomic

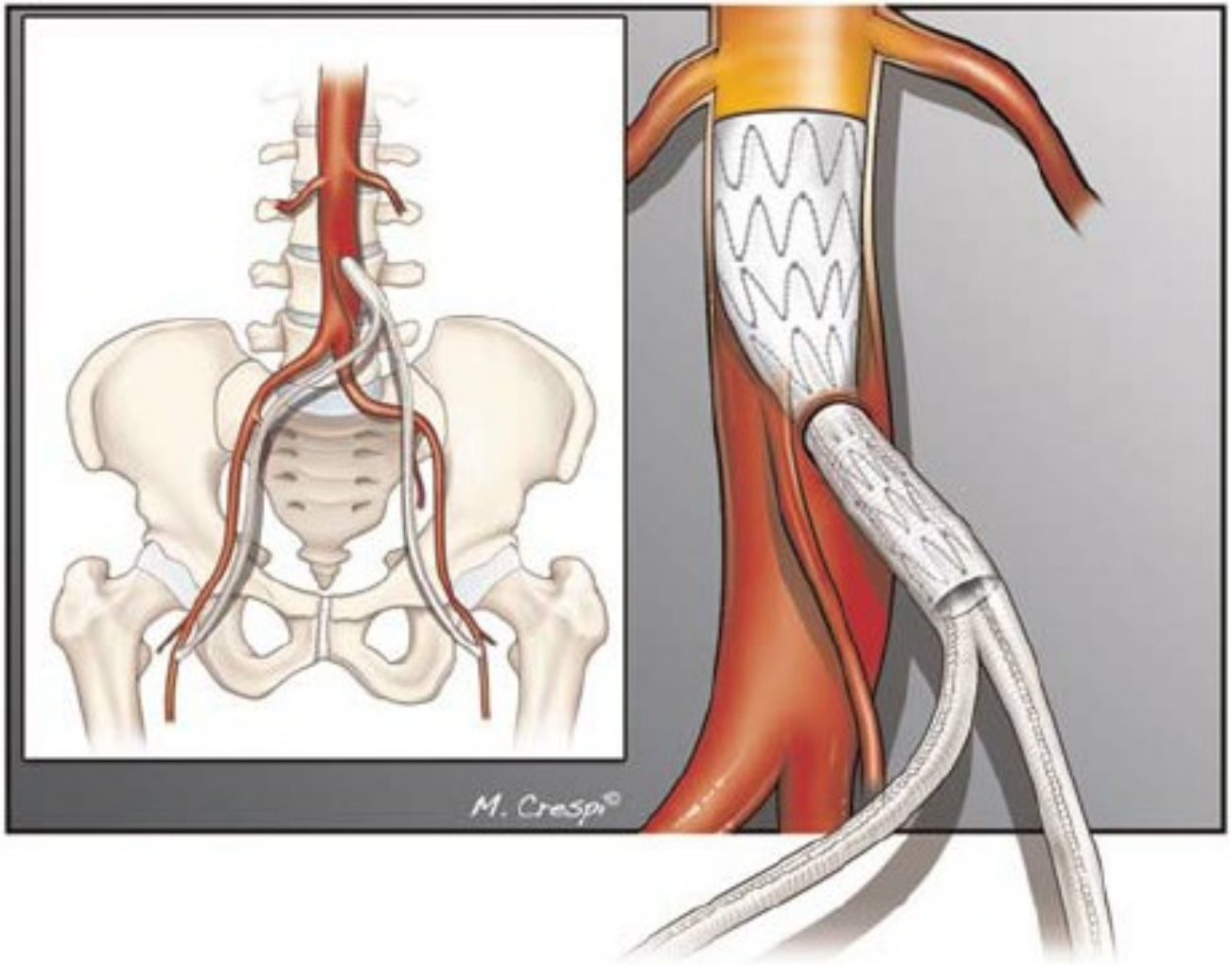
FEM- TIB – anatomic or no?



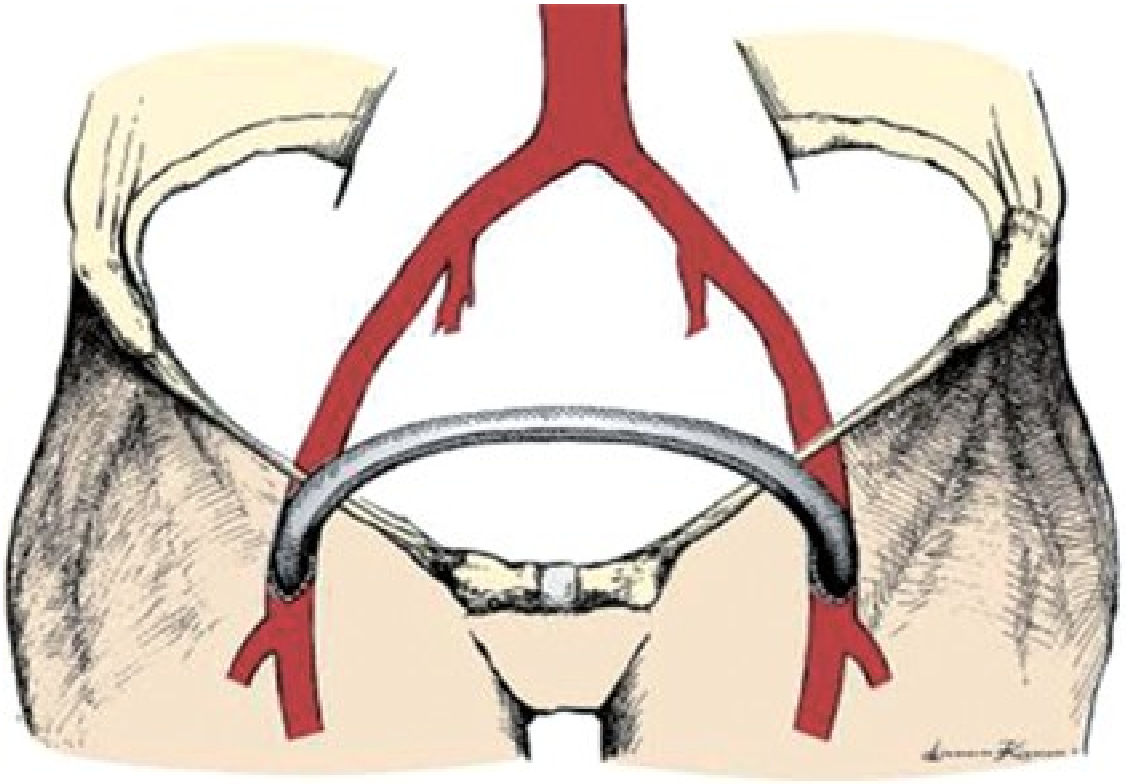
AX-BIFEM



Aorto-bifem

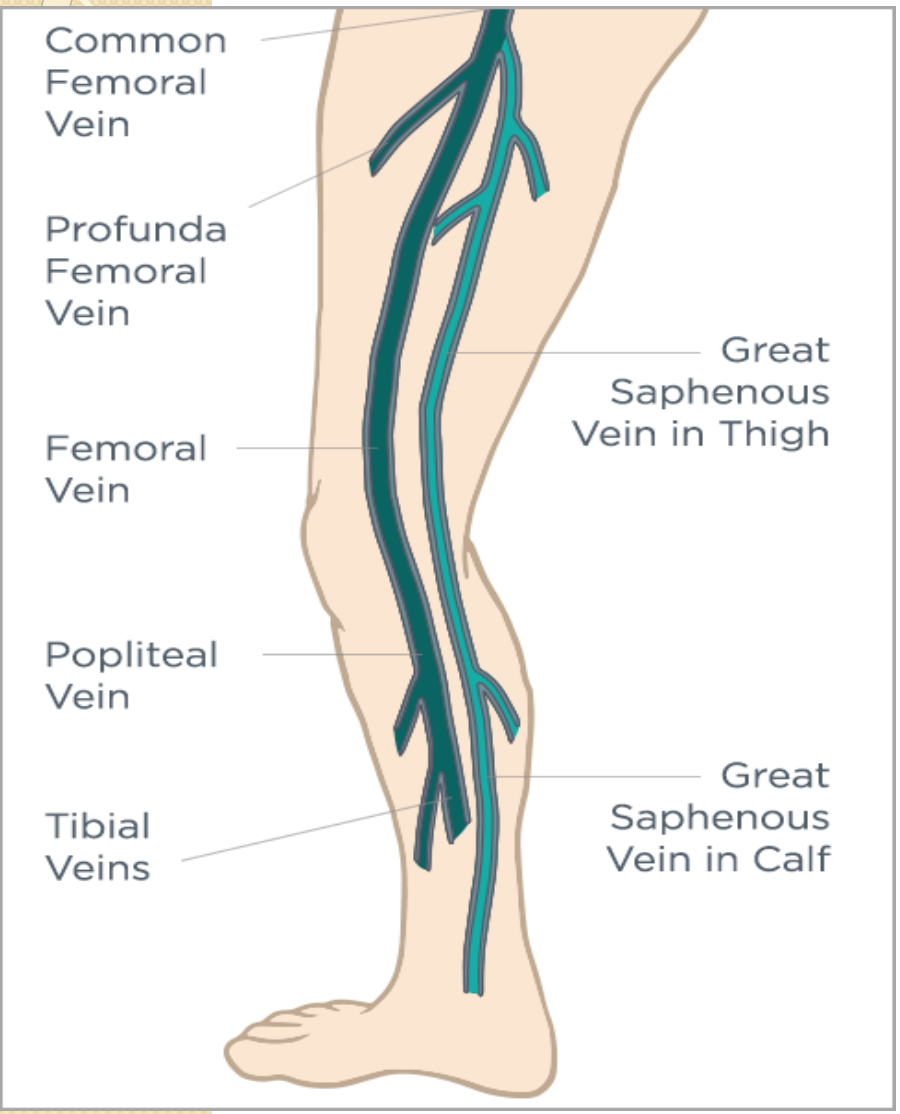


Fem-fem



Source: S. M. Dean, B. Satiani, W. T. Abraham: Color Atlas and Synopsis of Vascular Diseases
www.accesssurgery.com

Great saphenous vein

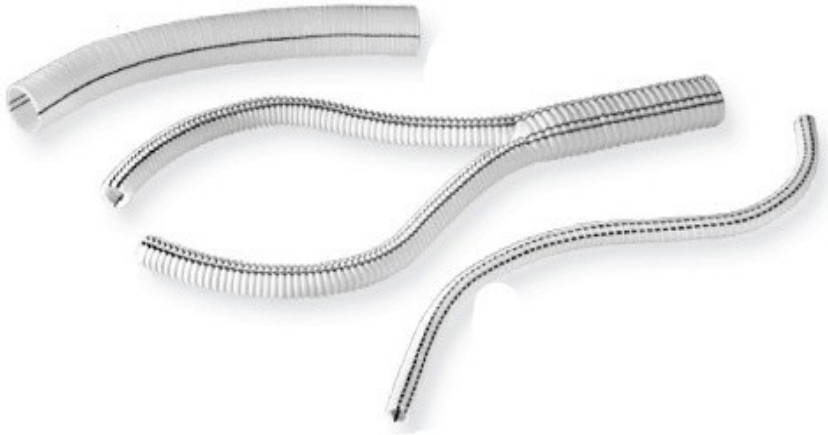


Ideal graft for bypass

Spare part of our body


CyberLink
by PowerDirector

Allo-graft from donor



PTFE (

Short break? No break? Coffee? Juice? Cigarette?

Chronic Venous Insufficiency - Varicose veins

-pooling of blood in veins, straining vein walls

-cause can be found in **venous reflux**, due to inability of vein valves to work properly

Signs and symptoms:

- varices
- swelling
- hyperpigmentation
- pruritus
- ulceration
- phlebitis

Chronic venous insufficiency

The reflux (incompetence) of vein valves can occur due to

Phlebitis - infection of superficial veins, e.g. post-traumatic

Superficial vein thrombosis- this poses little to no danger o pulmonary embolism

Deep-venous thrombosis – blood clot formation in deep venous system, which can result in PE - chronic venous insufficiency is then considered as a part of postthrombotic syndrome

1. Thrombofilic state
2. Trauma
3. Imobility

VIRCHOW´S TRIADE

DIAGNOSIS

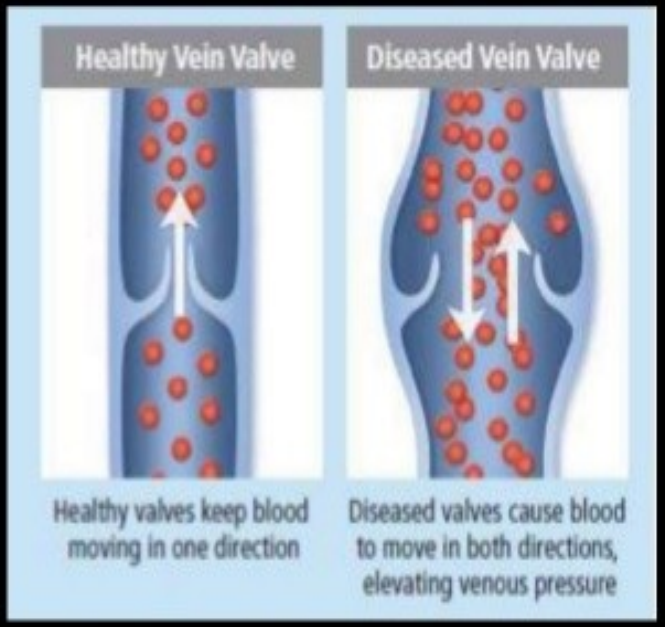
Personal history

Clinical examination

Doppler – ultrasound of venous system

Rule out heart disease and hypervolemic state first!

- Venous valvular incompetence is the main cause in Vericose Veins



DOPPLER EXAMINATION

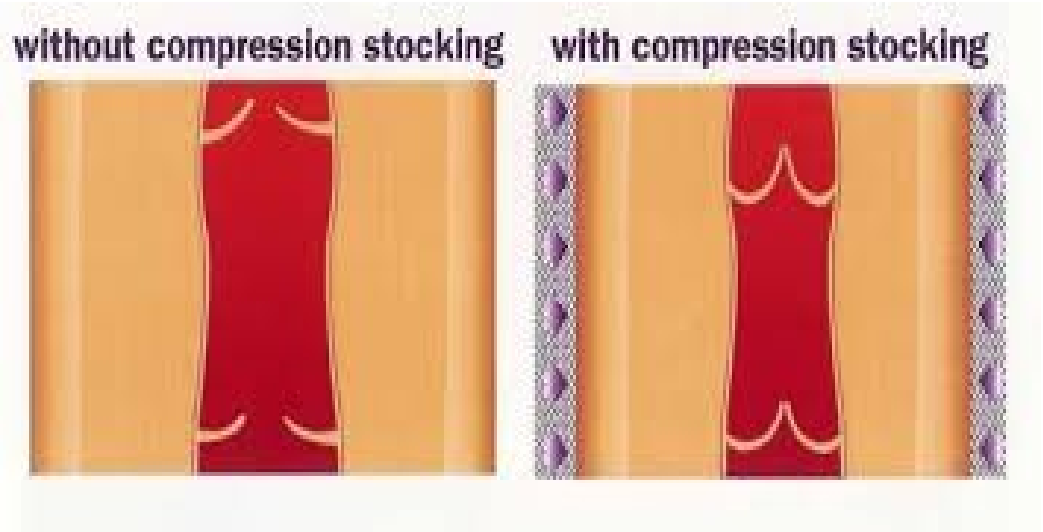
- Patients were examined in standing position.
- Axial scan and continuous scan was performed for superficial and deep venous system.
- The Valsalva maneuver was used to elicit the presence of reflux.

Conservative treatment

Effort to stabilize the condition, and prevent it from worsening

- compression stockings
- venoprotective medication-Detral
- blood pressure maintenance
- elevation of lower extremities

Stockings effect



Surgical therapy

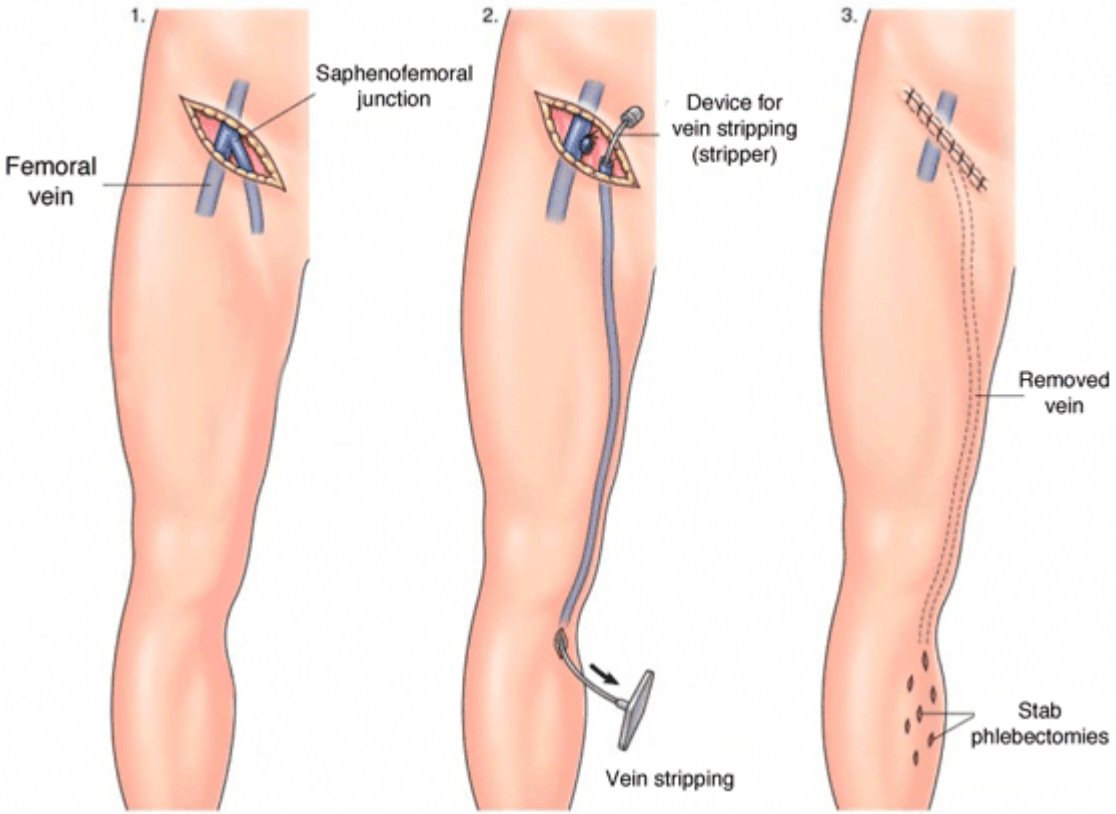
stripping

ligation

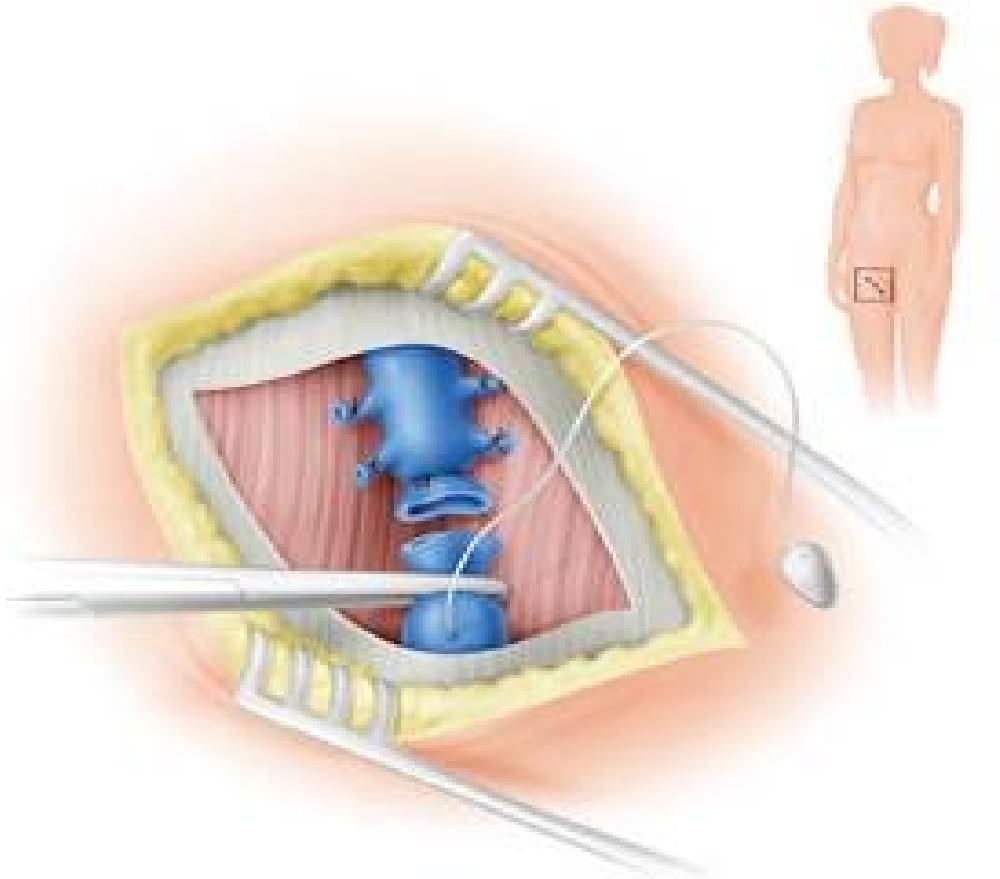
sclerotherapy

endovenous (intravascular) thermal ablation

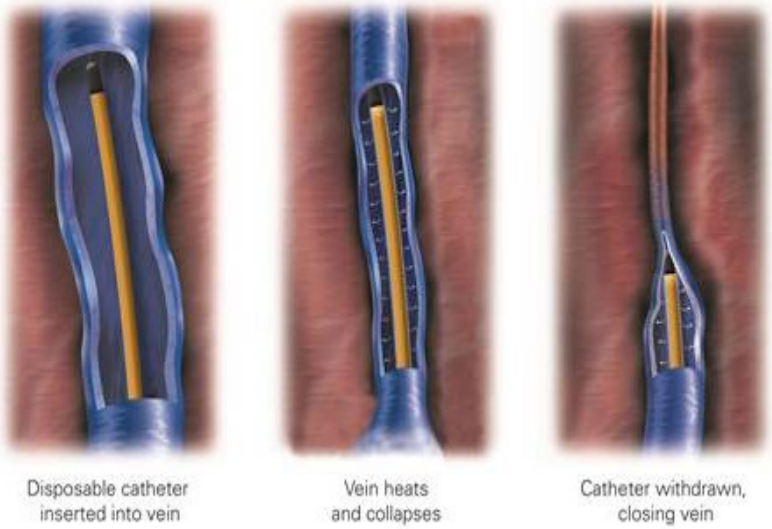
Stripping


1.
Saphenofemoral junction
Femoral vein
2.
Device for vein stripping (stripper)
Vein stripping
3.
Removed vein
Stab phlebectomies

Ligature



Endovenous catether ablation



Sclerotherapy

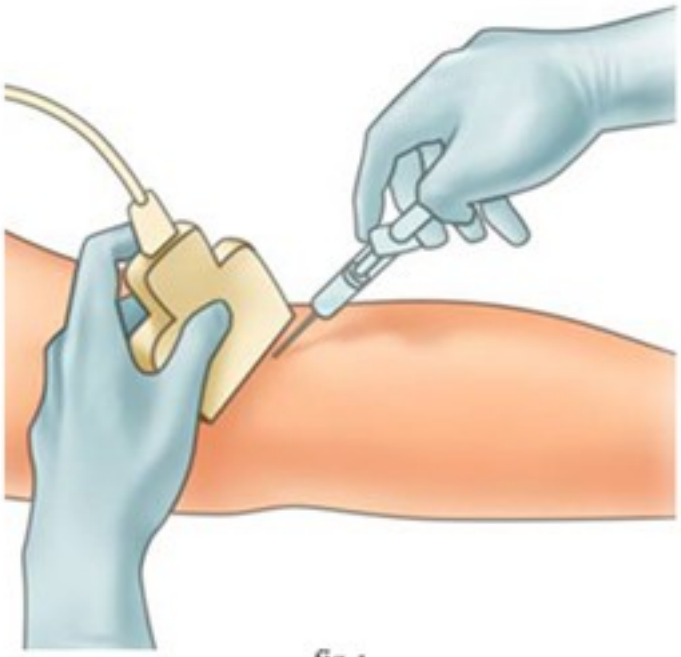


fig.1

FIG 1:

Utilizing ultra sound technology, the varicose vein is located to allow for precision injection of the sclerosant agent.

fig.2

FIG 2:

Once the sclerosant agent is injected into the vein, it causes the varicose vein to collapse.

Deep venous thrombosis – (DVT)

- condition in which blood clot forms in deep venous system of legs (rarely elsewhere)

- untreated may pose a huge health risk
- source of embolism to lungs, or in paradox embolism can cause stroke (venous thromboembolism- VTE)
- many patients remain asymptomatic
- more often in women

Deep venous thrombosis

Deep venous thrombosis

Signs

- pain
- tenderness
- swelling/oedema
- redness (erythema)
- warmth
- discoloration
- may present also with:chest pain, trouble breathing, palpitation,chest dyscomfort, hemoptysis, tachycardia, tachypnoe, ...

Causes and risk factors:

1. Virchow´s triade –post surgery state
2. Smoking
3. Gravidity
4. Contraceptives
5. Older age
6. Medication
7. Genetic predisposition and disorders

Phlegmasia coerulea dolens

- translated as „painful blue oedema“
- form od DVT
- caused by extensive block in outflow venous system
- can present with sudden onset of severe pain, oedema, cyanosis
- sometimes may be a first sign of malignant disease-need further examination

Treatment: cathether directed thrombolysis

Diagnosis of DVT

Clinical presentation, examination, risk factors, labs, doppler ultrasound, venography

Examination

Hommans sign – dorsiflexion of foot, eliciting pain – possibly dangerous

Pratt sign – calf squeeze elicite pain

- positivity does not confirm the diagnosis
- negative result does not rule out DVT

(Chris) Pratt sign

Laboratory and Doppler - essential

D-dimer – fibrin degradation product – concentration gets higher, when blood clot decreases, thanks to fibrinolysis, which is physiological in body

Negative D-dimer concentration – 95% no DVT

Possitive D-dimer concenration – may sign DVT, or other pathological condition

Doppler + D-dimer – sets positive diagnosis

Therapy of DVT

Anticoagulation – LMWH, fondaparinux,UFH

Stockings, walking, check-ups

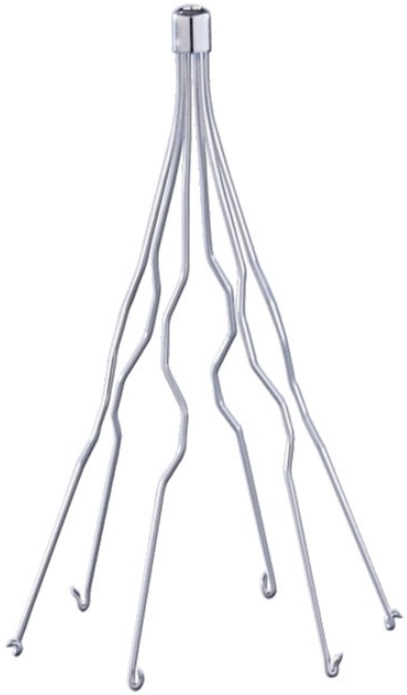
IVC filters

Thrombolysis – direct with catether or indirect- intravenous – using streptokinase, alteplaze, or urokinase (thrombolytics ensymes)

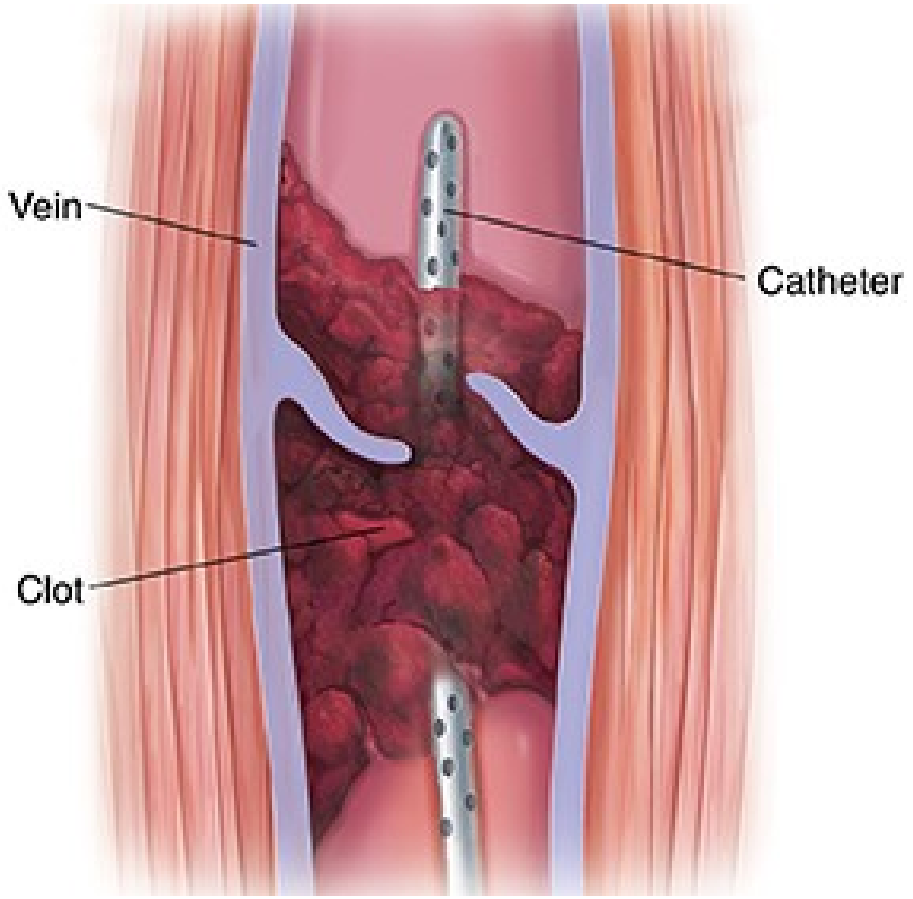
Mechanical thrombectomy – especially in acute, and symptomatic

LMWH – stops growth of blood cloth

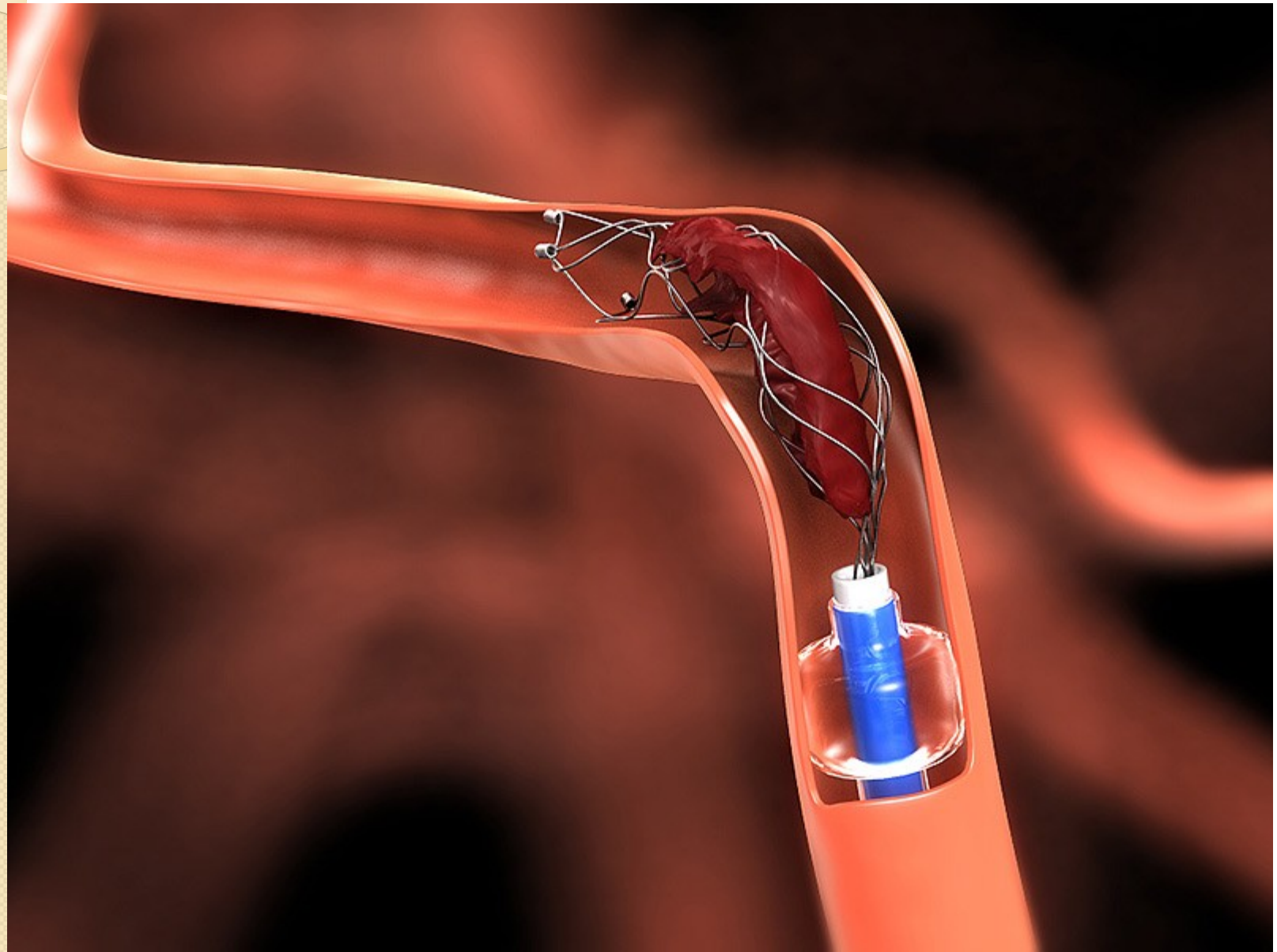
Inferior vena cava filters



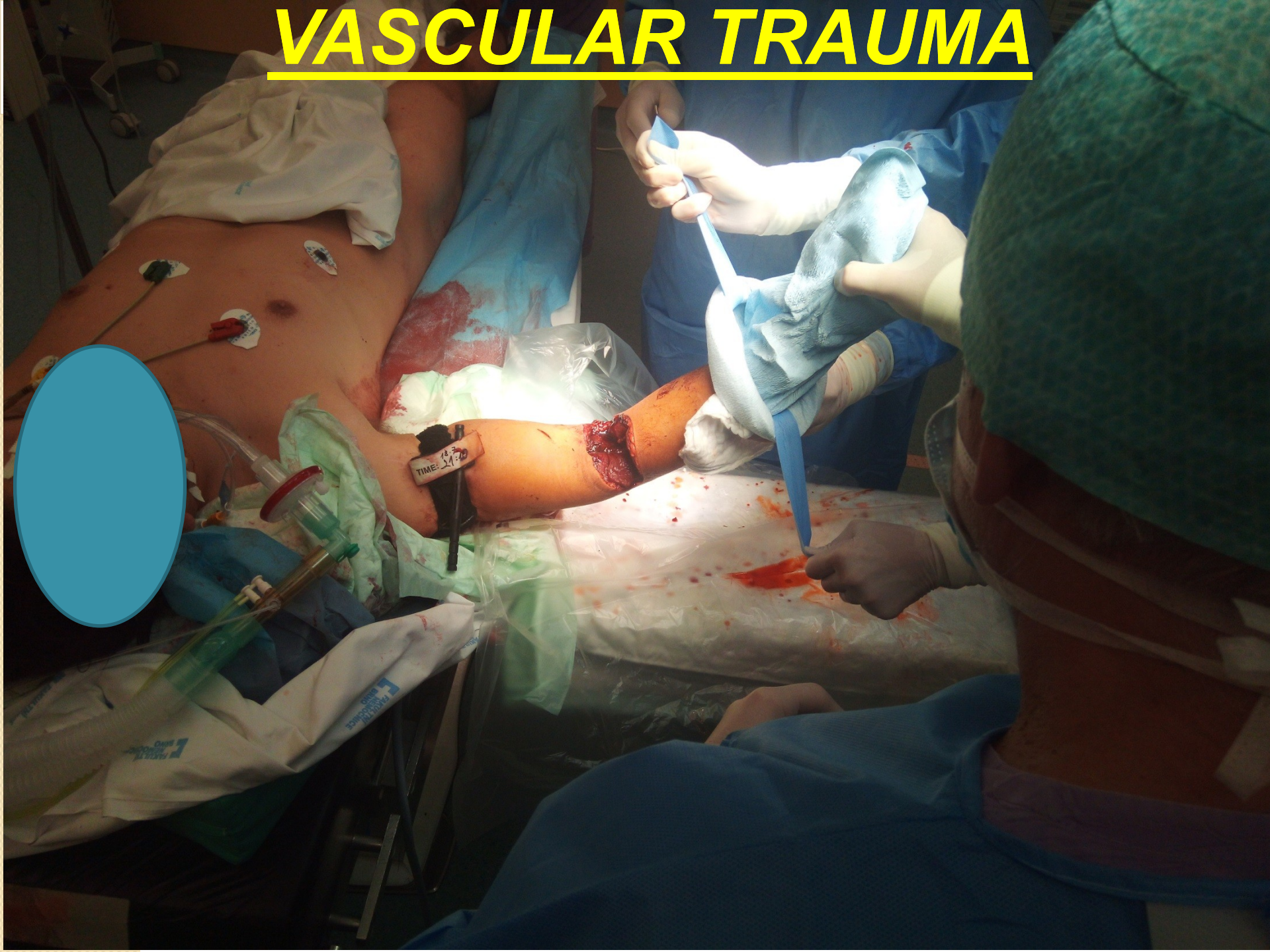
Catether thrombolysis



Mechanical thrombectomy



VASCULAR TRAUMA



Examination

Vascular trauma may be intracorporal or extracorporal (visible)

Check vital signs: Airway

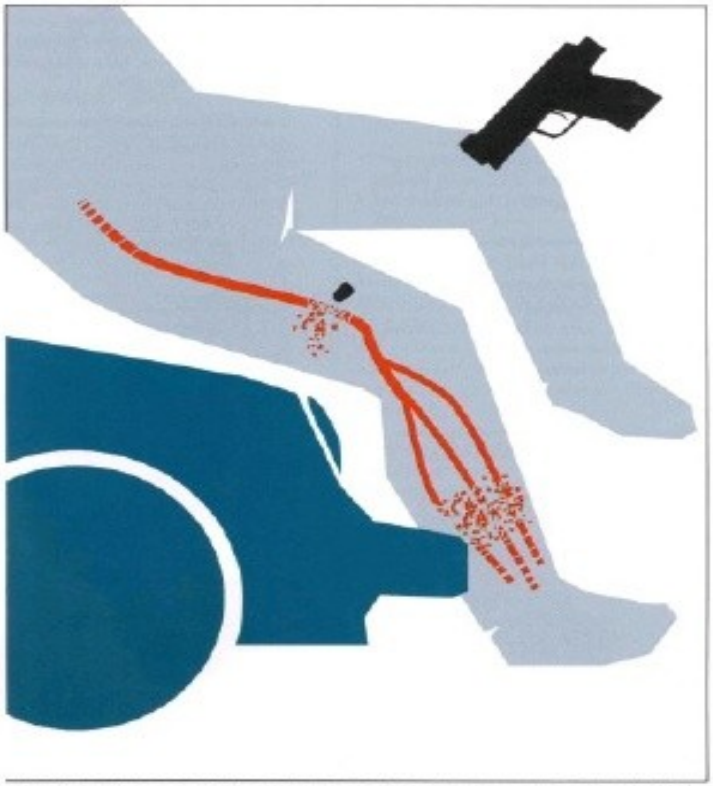
Breathing

Circulation – signs of shock

Anamnesis

Mechanism

www.perfuse.net



- Blunt
  - Orthopaedic #
  - Dislocation (knee)
  - Isolated
- Penetrating
  - High velocity
  - Low velocity
- Iatrogenic

www.amnch.ie

http://www.facs.org/trauma/publications/peripheralvasctrauma.pdf

Pros- usually younger, healthy patients

Check for signs of arterial injury

Hard signs: pulsatile hemorrhage, significant blood loss, acute ischemia

volume resuscitation, tourniquet if possible, and acute repair

Soft signs: minor hemorrhage, small hematoma, associated injury

Aproach to arterial injury

Definitive or damage control therapy

Restore blood perfusion

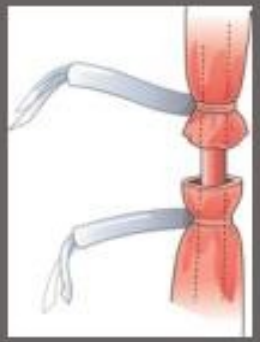
Open vs endovascular treatment

Stable vs unstable patient

Surgery options

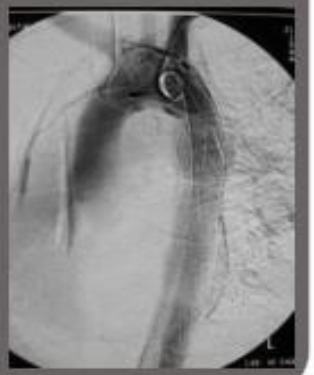
What are your options?

- Observation
- Ligation
  - Ok to ligate external carotid, celiac axis, internal iliac
  - Maintain one major vessel to extremity
- Lateral suture
- End-to-end anastomosis
- Interposition grafts
  - Vein
  - Artery
  - PTFE
  - Dacron
- Extra-anatomic bypass
- Interventional radiology



Endovascular management

- Can be considered in *hemodynamically stable* patient with no *active* bleeding
- Examples:
  - Access to vertebral artery
  - Repair renal artery injury
  - Repair subclavian artery injury
  - Repair of blunt injury to descending thoracic aorta



IN STABLE PATIENTS

Thoracic vascular injury

-take into consideration:

Clinical presentation

Visible penetration of chest wall

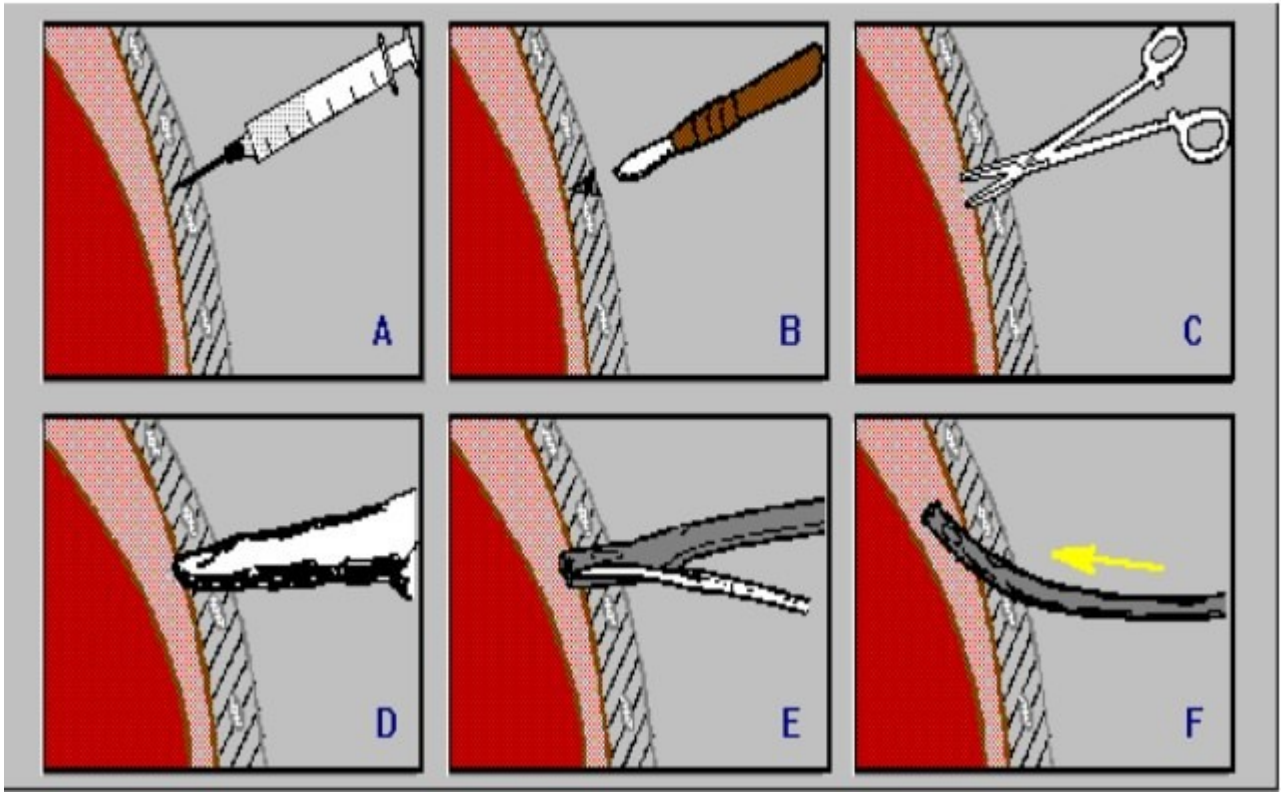
Hemodynamic stability/instability

X-ray, CT, CT-angio finding

Insert chest-tube if needed –chest decompression

Think over procedure in thoracic and abdominal trauma

Insertion of chest tube

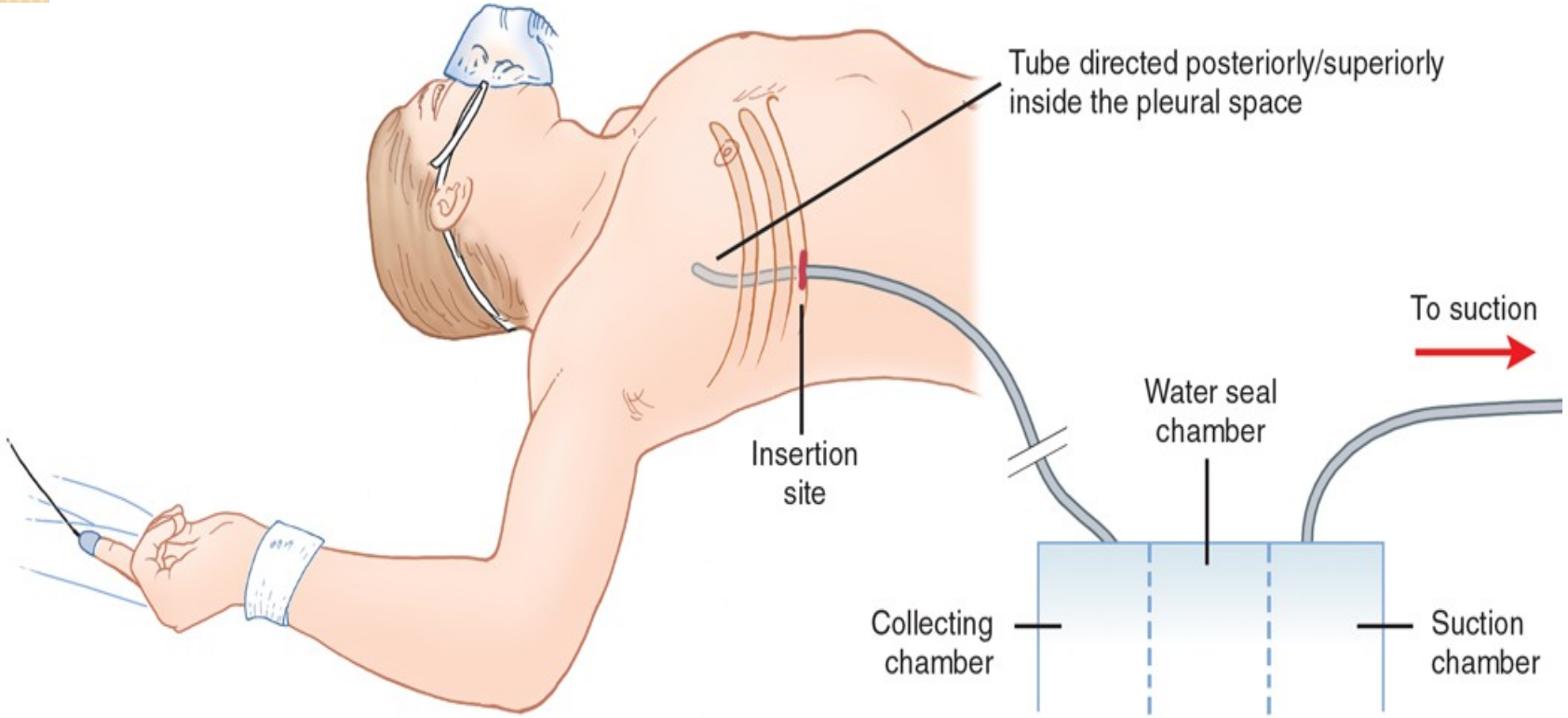


Thoracic vascular injury

Thoracic Vascular Injuries

- Choice of incision
  - If unsure about location of injury, anterolateral thoractomy

Chest tube insertion



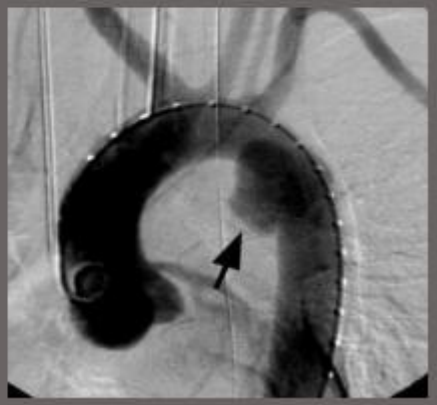
Source: Reichman EF: *Emergency Medicine Procedures, Second Edition:* www.accessemergencymedicine.com

Blunt Thoracic Aortic Injury

- Cause of 10-15% of motor vehicle deaths
- Most commonly seen injury to proximal descending thoracic aorta
- Patients invariably have associated injuries:
  - 50% head
  - 46% rib fxs
  - 38% lung contusions
  - 20-35% orthopedic injuries

Blunt thoracic aortic injuries

- Mechanism is commonly sudden deceleration with shear force between mobile and fixed portion of the thoracic aorta
- A contained injury is almost NEVER the cause of hemodynamic instability – look elsewhere!



Operative management of blunt thoracic aortic injury

- Traditional therapy has always been prompt operative repair
- Consider non operative therapy with severe head injury or multi organ trauma
- Estimated risk for free rupture is 1%/hour
- Control BP and afterload reduction
- Remember follow up imaging when necessary

Abdominal injury

Anamnesis

Clinical presentation

Examination- physical, laboratory

Special:

FAST –focused assessment with sonography for trauma

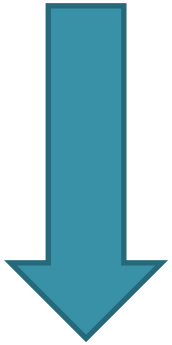
CT

CT-angio

Revision of abdominal cavity

Blunt trauma

- stable patient, no deffance musculaire, no drop in laboratory – Hb,Ery, negative FAST



Observation

Penetrating abdominal injury

Open revision – ALWAYS!!!!

Peripheral vascular trauma

Peripheral Vascular Trauma

- Assess neurologic status of affected extremity
- Look for signs of compartment syndrome
- Traditional window of opportunity ≤ 6 hours
- Hand held Doppler
- Arteriography indicated for any >10 mm Hg difference between extremities

Periferal vascular injury

- open fractures, comminutive fractures, stab wounds, semiamputations, cut wounds, GSW, etc...

Examination?

What to do?


CASE REPORT ☺

Thank you for your attention!!!
Now, run home, save yourselves