Vascular Surgery: servicing the body's plumbing

Simon Fraser
Retired Consultant Vascular Surgeon

“Provision of Vascular Services” Report by Vascular Society GBI Sept. 2011

- Prevent death from ruptured Abdominal Aortic Aneurysm
- Prevent stroke due to carotid artery disease
- Preventing leg amputation due to Peripheral Arterial Disease
- Heal leg ulceration

Shaping vascular surgery in last 10 years……

- More elderly patients
- Better information from Randomised Controlled Trials
- Technological advances
- Vascular Service Changes

Main changes in my career……

- Endovascular intervention
- EVAR, AAA Screening
- Urgent CEA
- Advances in imaging
- Endovenous surgery
- 24/7 rotas, consultant led, specialty status coming

Training a Vascular Surgeon……

- 5 years undergraduate at medical school

- 2 years foundation
- 3 years surgery core training
- (2-3 years research)
- 6 years specialty training

Training a Vascular Surgeon 1976-95

- 5 years Edinburgh medical school
- 3 years Edinburgh house officer/ SHO (5 jobs)
- 2 years SHO St Bart's London (4 jobs)
- 18 months Registrar Gen Surg Portsmouth
- 6 months Plastics SHO Billericay
- 3 years research King's College Hospital
- 1 year registrar Gen Surg (liver, GI) KCH
- 3 years Senior Registrar KCH and St Mary's
- Consultant Surgeon (with interest in Vascular Surgery) KCH 1995
- Consultant Vascular Surgeon Edinburgh 2001

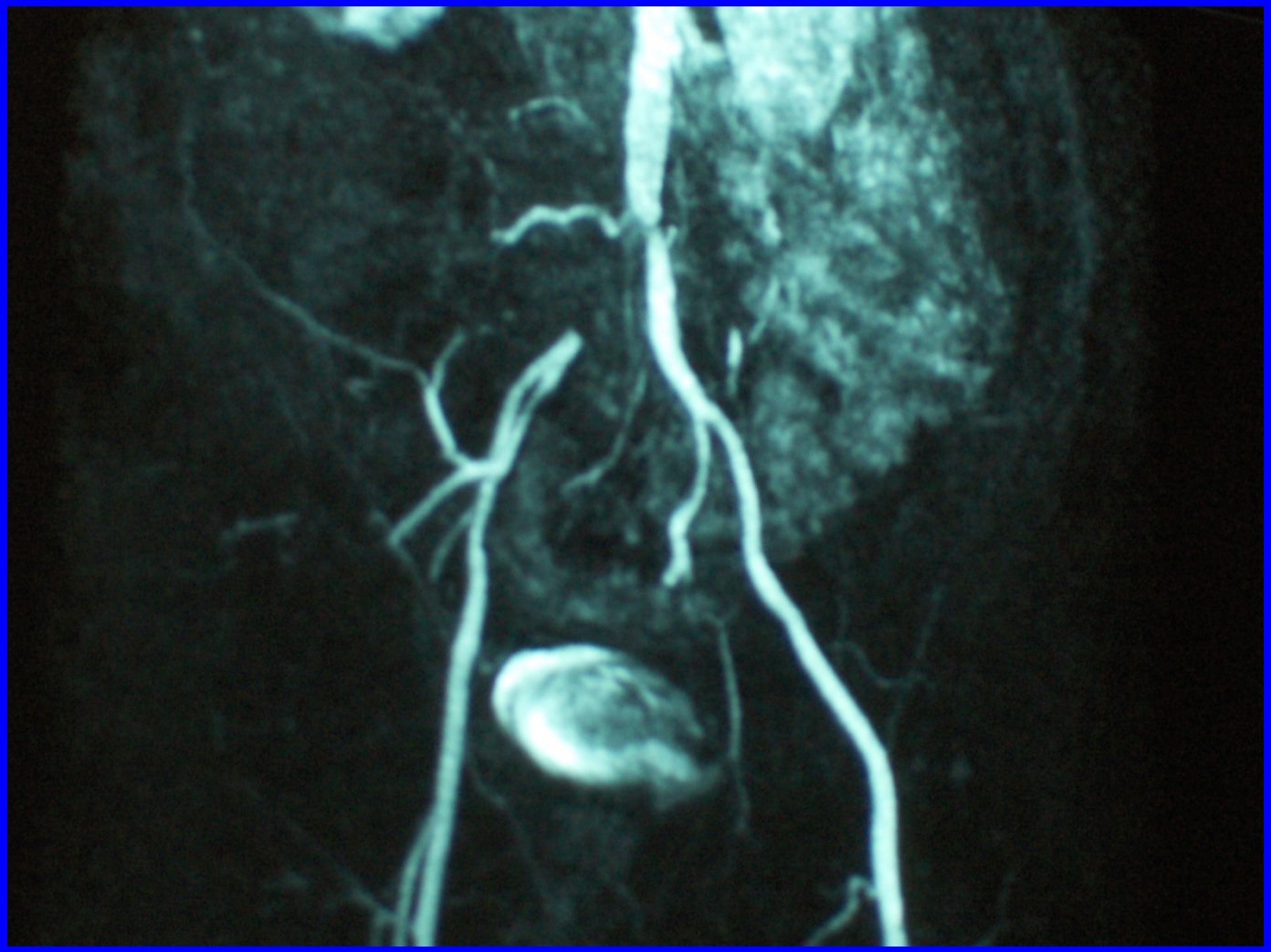
Peripheral arterial disease

- Muscle ischaemia
- Inadequate oxygenated blood
- Lactate accumulation
- Affects muscle group(s) downstream of vascular stenosis/occlusion

Claudication - Causes

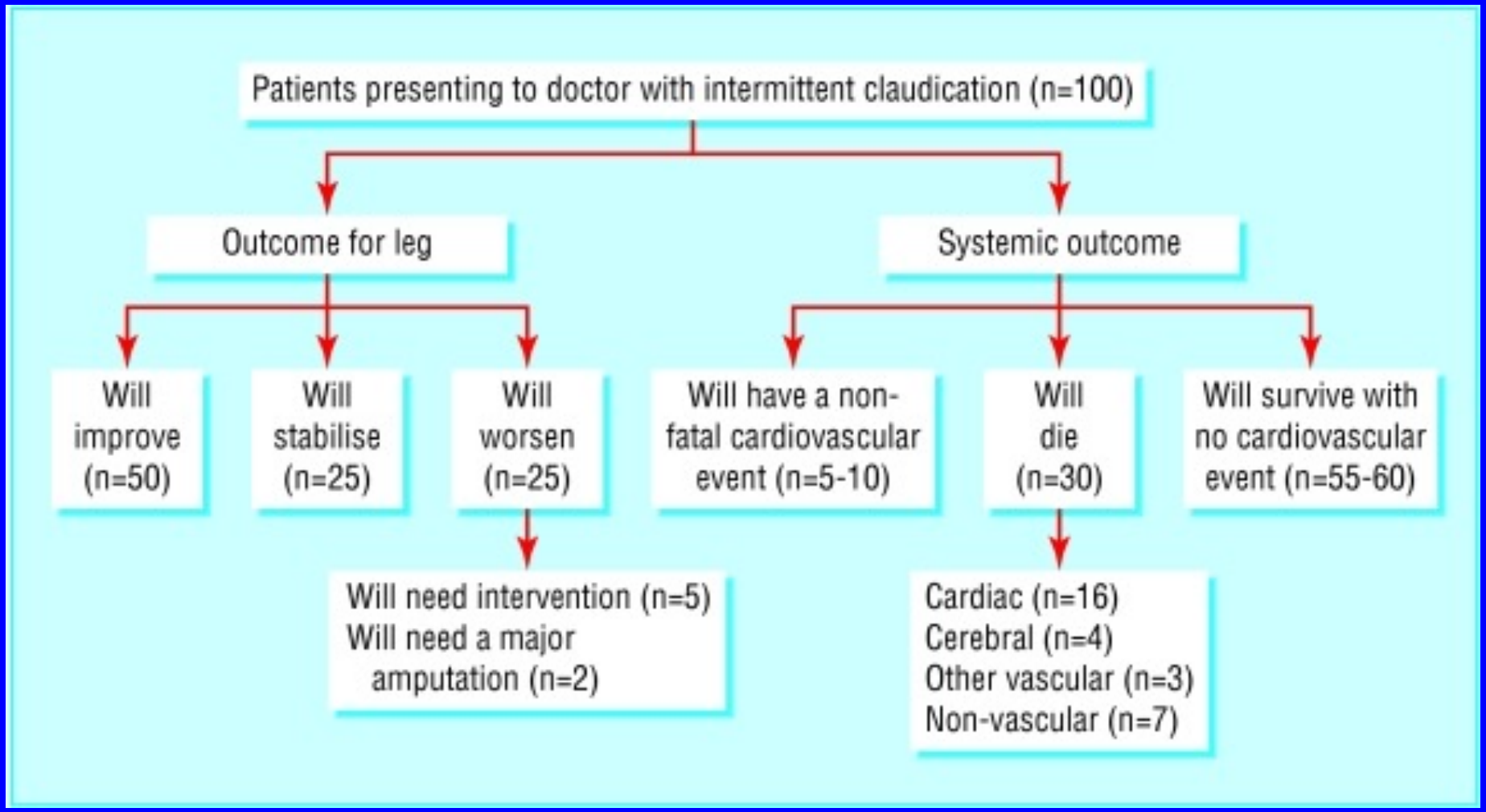
- Atherosclerotic occlusive disease
- Chronic embolic disease
- Cystic medial disease
- Occlusion of (popliteal) aneurysm
- Previous injury
- Kinking or entrapment syndromes

MRA



Ultrasound Mapping

Natural History of Intermittent Claudication



Risk of Amputation <1%, Risk of fatal MI 5-10%/year

Burns *et al , BMJ, 2003,* Management of PAD in primary care

Treatment of claudication

- Control risk factors – stop smoking
- Antiplatelet
- Treat Cholesterol >3.5
- Supervised exercise
- Drugs
- Avoid intervention

Scottish Intercollegiate Guidance Network 89 (2006)

BRITISH MEDICAL JOURNAL

LONDON, SATURDAY 28 MAY 1988

Treating claudication in five words

When the editor asked me to write an article on treating claudication he said it must be "straightforward . . . of direct use to general practitioners" and not over 800 words. I was tempted to say that I could do it in five words—"stop smoking and keep walking"—but even with the modest fee he was offering this would have resulted in an embarrassingly large number of pounds for each word. I have therefore written more, but those short of time may go straight to the final paragraph.

The most important aspect of treatment is talking to the patient. Is the claudication stable or has it worsened recently, indicating a thrombosis of a stenosed artery and thus the prospect of considerable improvement as collateral vessels develop? What effect is the claudication having on the patient's life? Merely asking how far he or she can walk is of little value. Patients grossly underestimate their walking distance, and the disability caused by being able to walk only, say, 250 m depends on their normal activity: the patient who has to walk 2 km to work will be severely disabled, but a retired person with a car is hardly disabled at all. Doctors should ask patients about smoking and about fears of gangrene and amputation. Most patients with stable claudication may be reassured about amputation, particularly if they stop smoking.[1] Stopping smoking may well be the only "treatment" required, and it also increases the walking distance.[2] In my experience nicotine gum raises the rate of those who succeed in stopping from a dismal 5% to a poor 25%.

Exercise increases the distance the patient can walk,[3] presumably by dilating collateral vessels, although recent work[4] has shown that blood viscosity is also reduced by exercise. The form of exercise prescribed must be acceptable to the patient: thrice weekly "treadmill classes" at a hospital five miles away are of no use to workers. Getting off the bus or parking the car 1·5 km from work and thus walking 3 km a day is more likely to be acceptable. Avoiding traumatic chiropody and pressure sores from ill fitting shoes is important in those with very severe claudication that verges on chronic ischaemia.

The many uncontrolled trials of drugs alleging benefit are worthless because many patients improve spontaneously. Even so called "positive" controlled trials often have serious flaws such as large numbers of unexplained drop outs[5] and retrospective analysis of subgroups ("data dredging")[6,7]; and even then the usual claim of 20-30% increase in walking distance may be statistically significant but it is unimportant clinically. Finally, the reluctance of journals to publish negative studies results in a positive reporting bias.

Antiplatelet drugs may delay progression of atherosclerosis,[8] but this requires confirmation. I use them in patients with a clinically obvious stenosis and no contraindications. Hyperlipidaemia is probably worth treating in patients under 60, but I am not convinced that there is benefit in treating older patients. Whether β blockers adversely affect walking distances is still controversial, but it seems likely.[9] Patients with appreciable claudication should thus be switched to vasodilator drugs such as nifedipine or captopril. Haemodilution produces benefit[10] but is logistically difficult and in my experience few patients persist with it. In patients with appreciable claudication and a bruit over the abdominal aorta or the iliac or superficial femoral arteries balloon angioplasty may be worthwhile.[11] Steptokinase has only a limited success rate with serious complications[12] and is not recommended.

Finally, we come to surgery. To get benefit from an operation patients have to clear three hurdles. Their disability must be bad enough for them to be able to say after the operation "That was rough but worth it—it has made a big difference to my life." Next they must not have other diseases that will increase the risk and limit the benefits of operation—for example, angina or osteoarthritis. Angina is the most serious contraindication as it limits both benefit and life expectancy. Finally, an angiogram must show a lesion that can be bypassed with a good chance of long term benefit.

Thus, in summary, the treatment of intermittent claudication is reassurance that gangrene and amputation are most unlikely, advice to stop smoking and keep walking, treatment of hypercholesterolaemia in those under 60, and referral to a specialist if there is evidence of an arterial stenosis or if the claudication is severely disabling. In practice therefore the treatment for most patients is "stop smoking and keep walking."

E HOUSLEY

Consultant Physician,
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Royal Infirmary of Edinburgh,
Edinburgh EH3 9YW

Stop smoking and keep walking

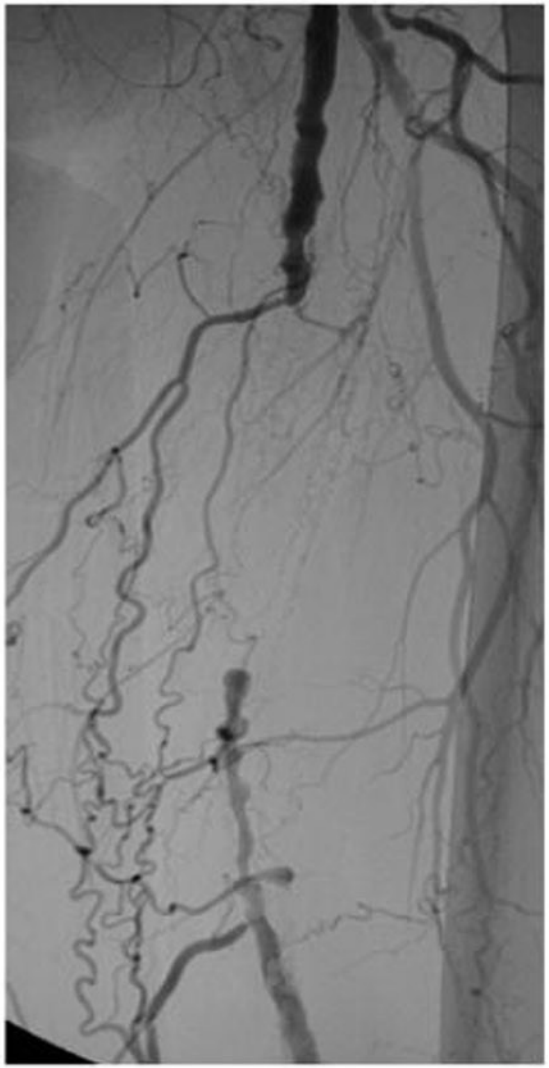
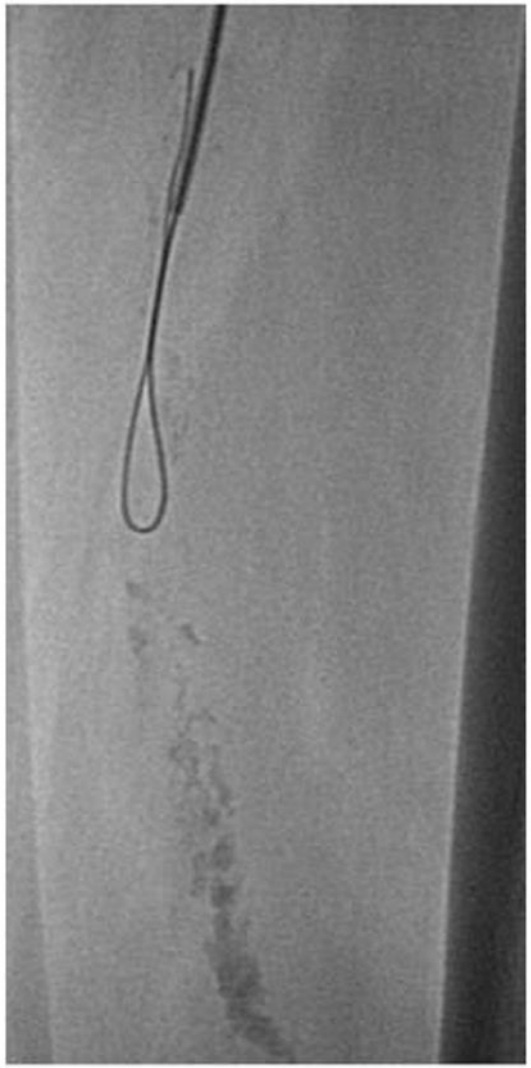
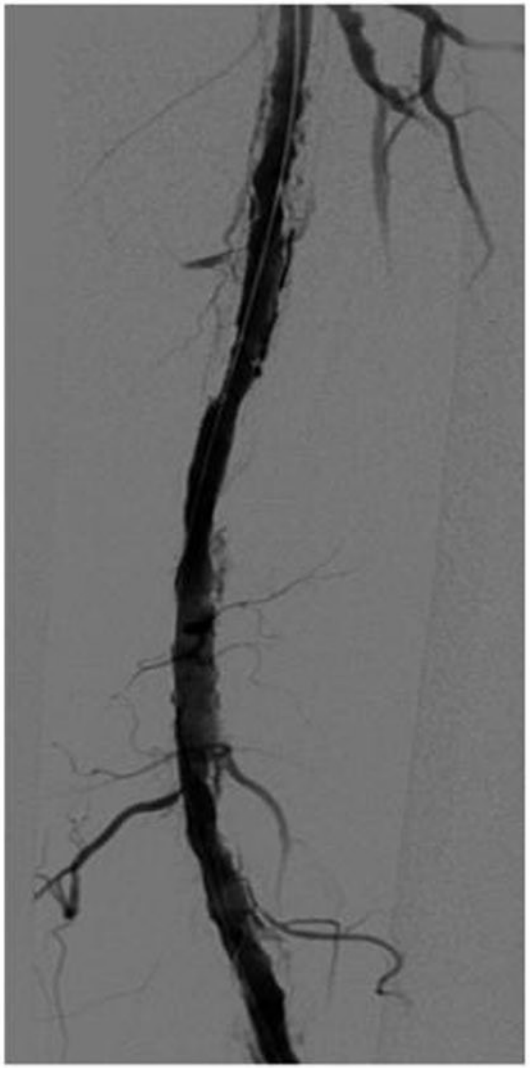
Treatments

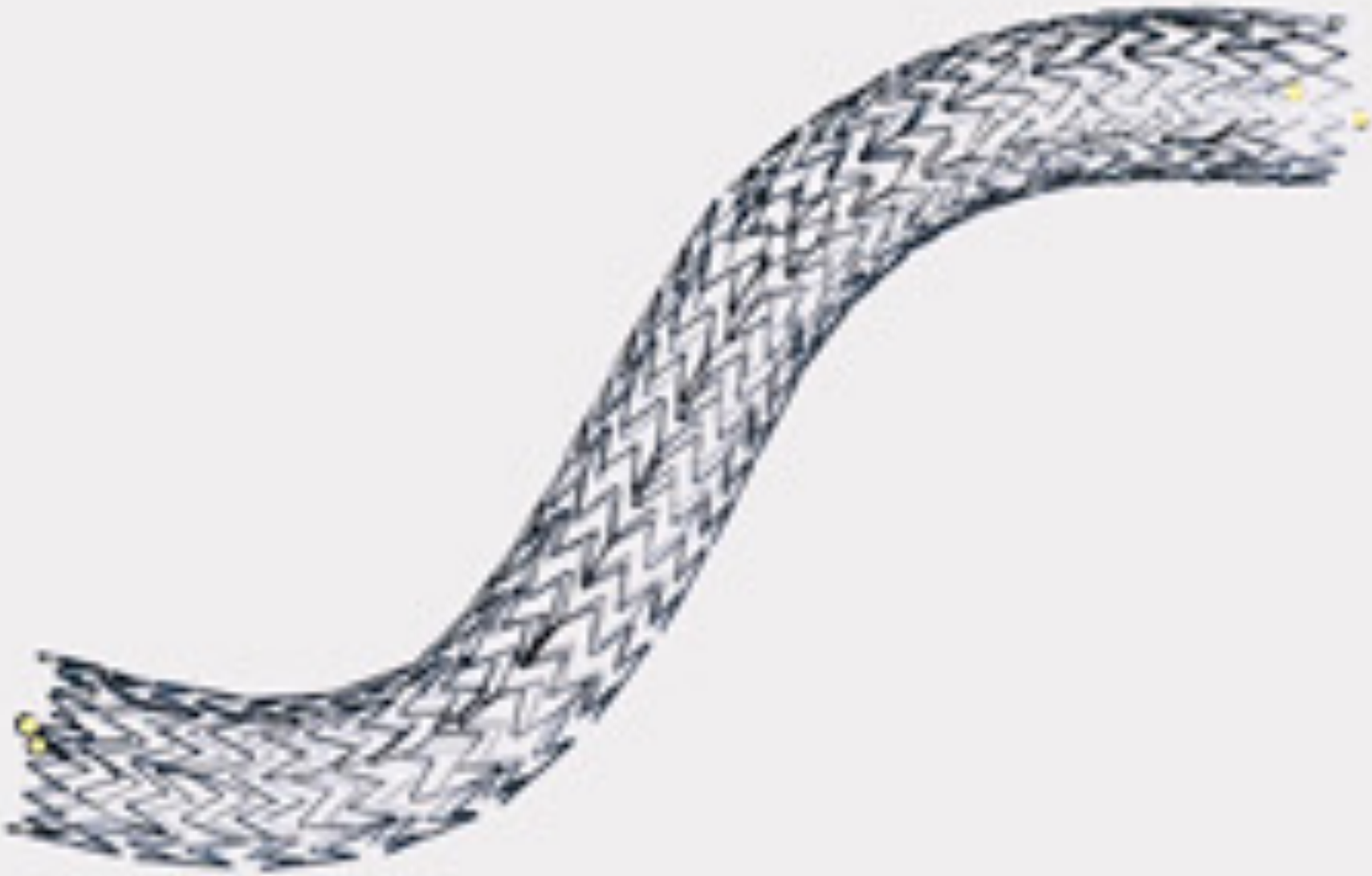
- Femoropopliteal bypass 1960s
- Drugs 1970s
- Angioplasty 1980s
- Stents 1990s

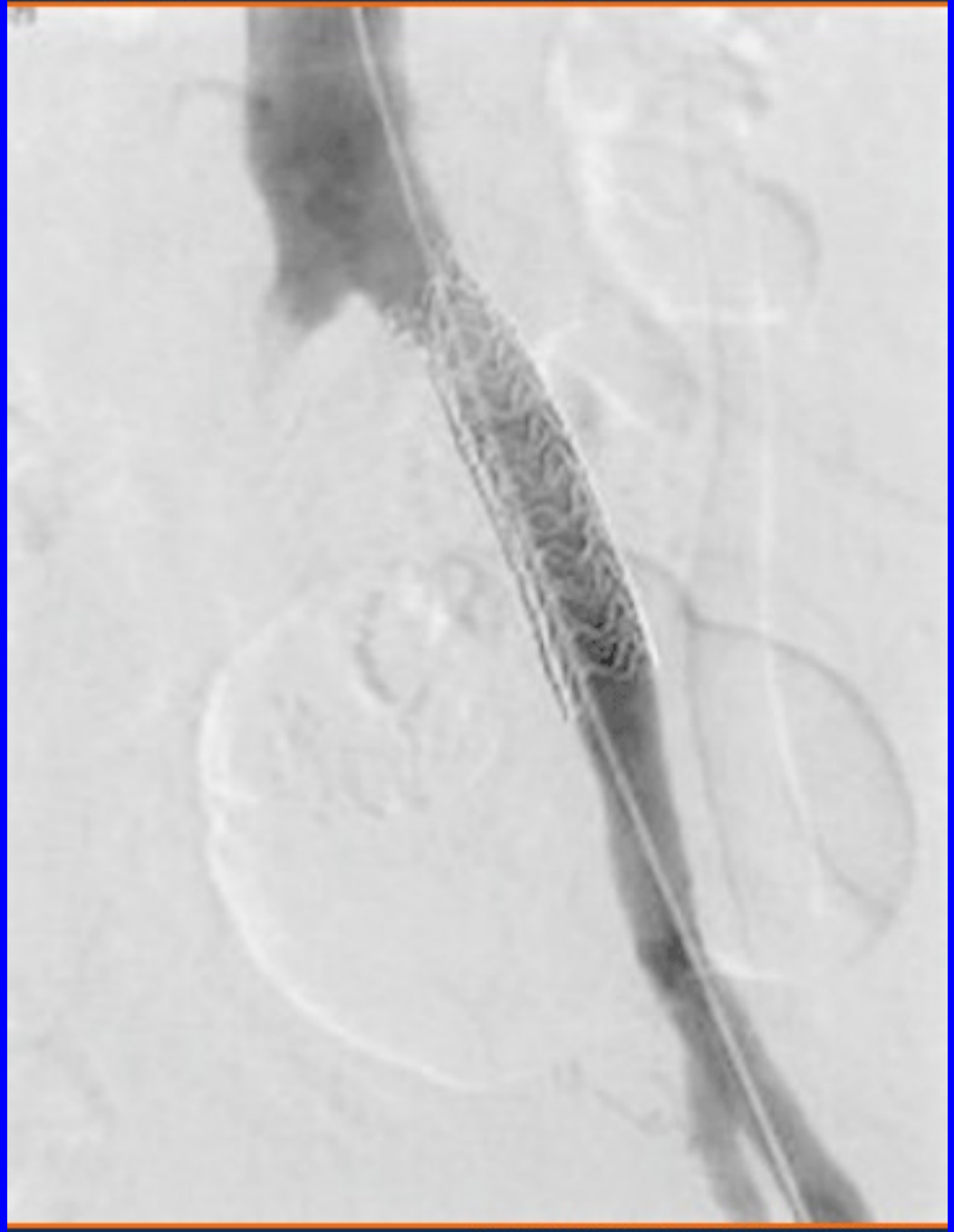
- Remember: 15 minutes' conversation in clinic can save 3 hours in the OR

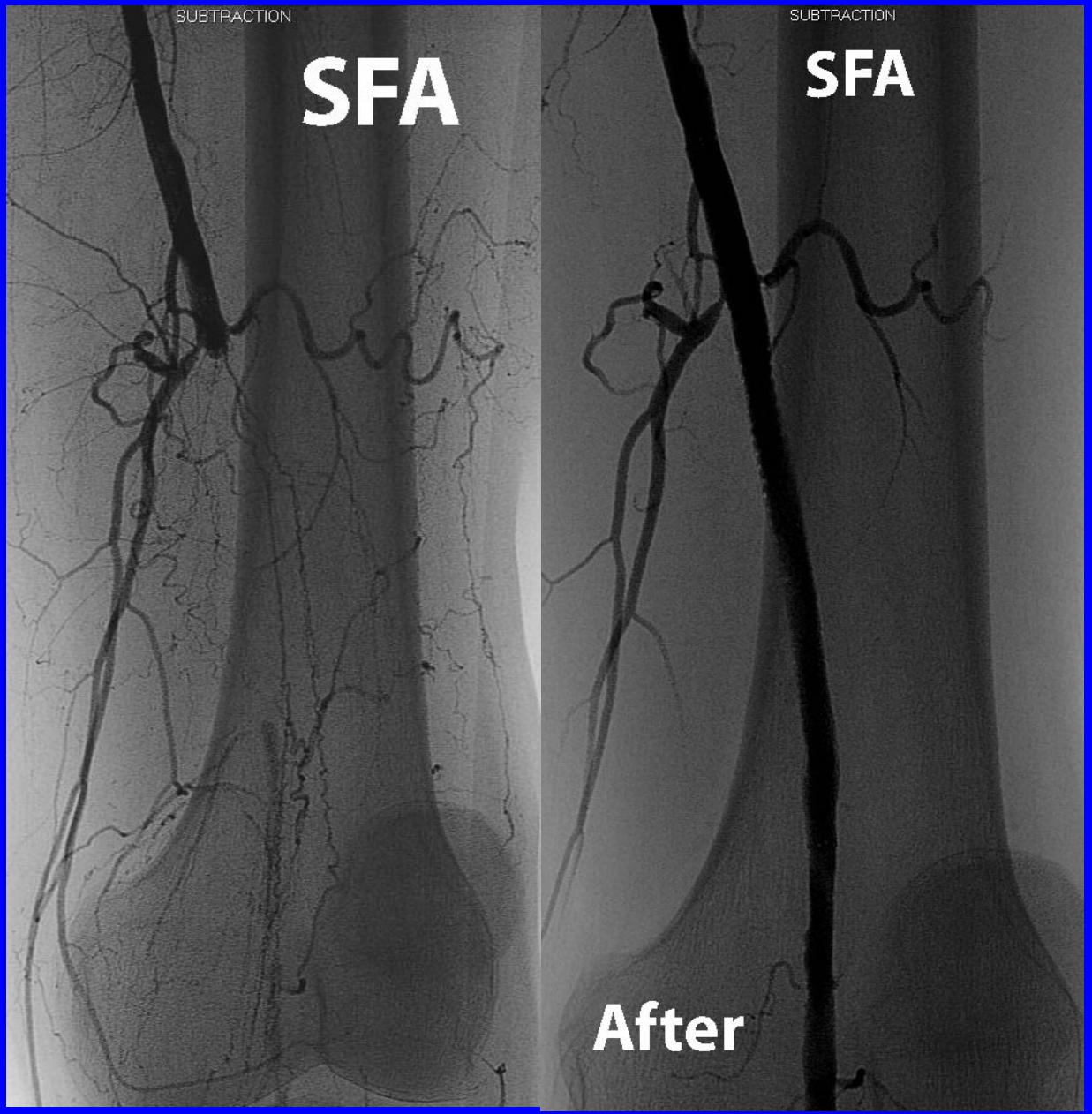
SFA occlusion


Cor>Sag 20
>Tra -6
A





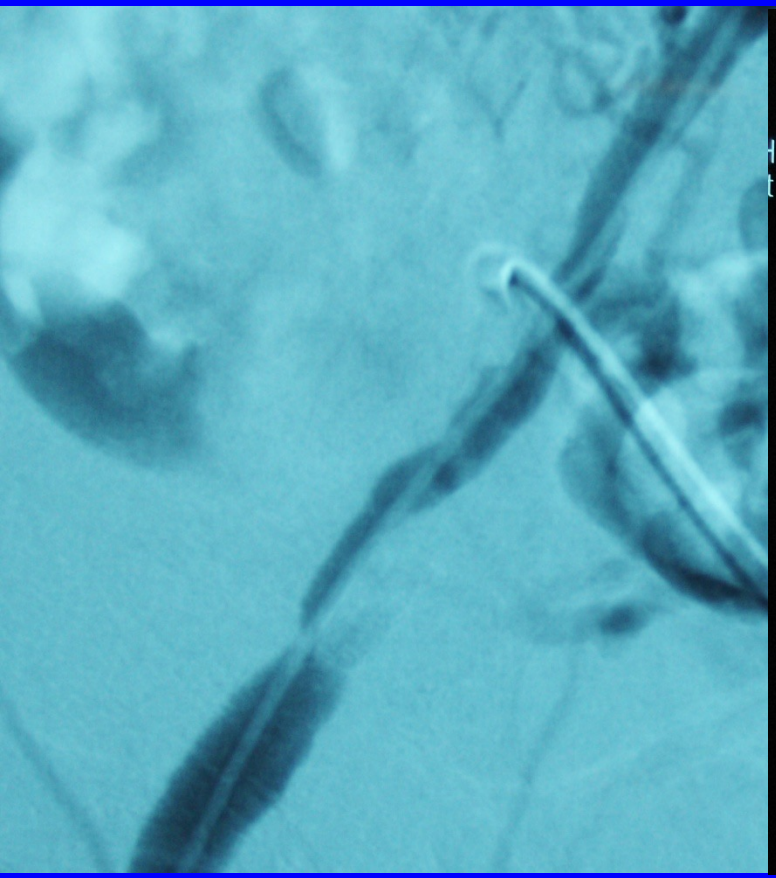

SUBTRACTION
SFA
SUBTRACTION
SFA
After

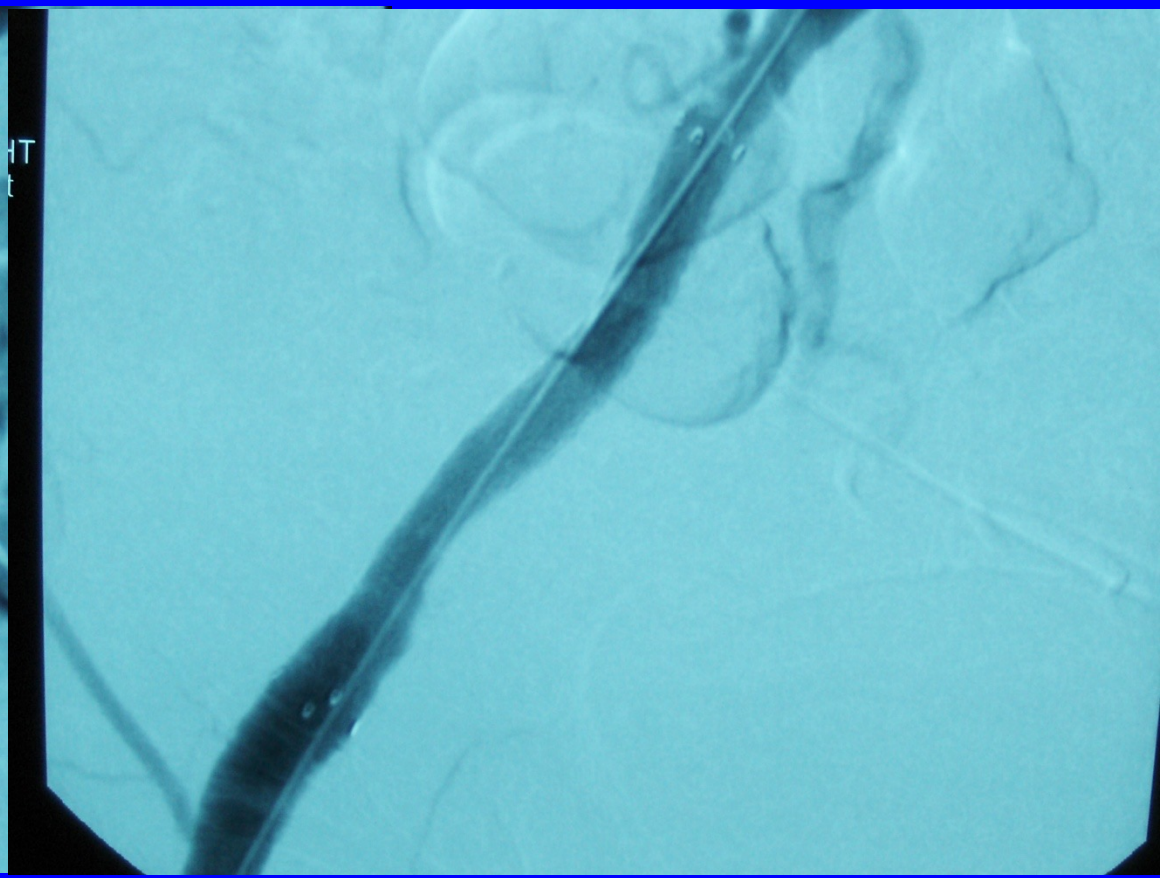
MRA

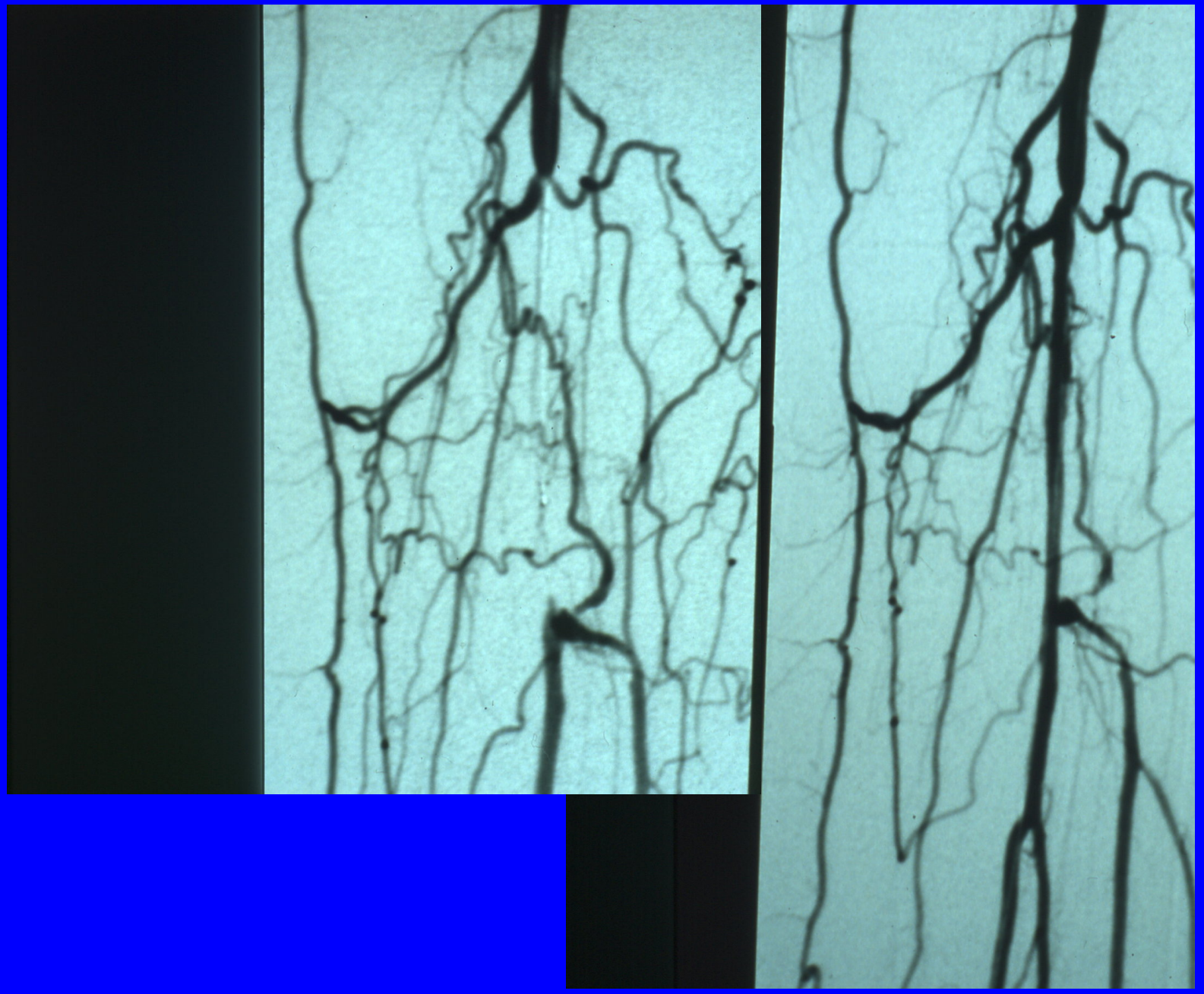
Conclusions

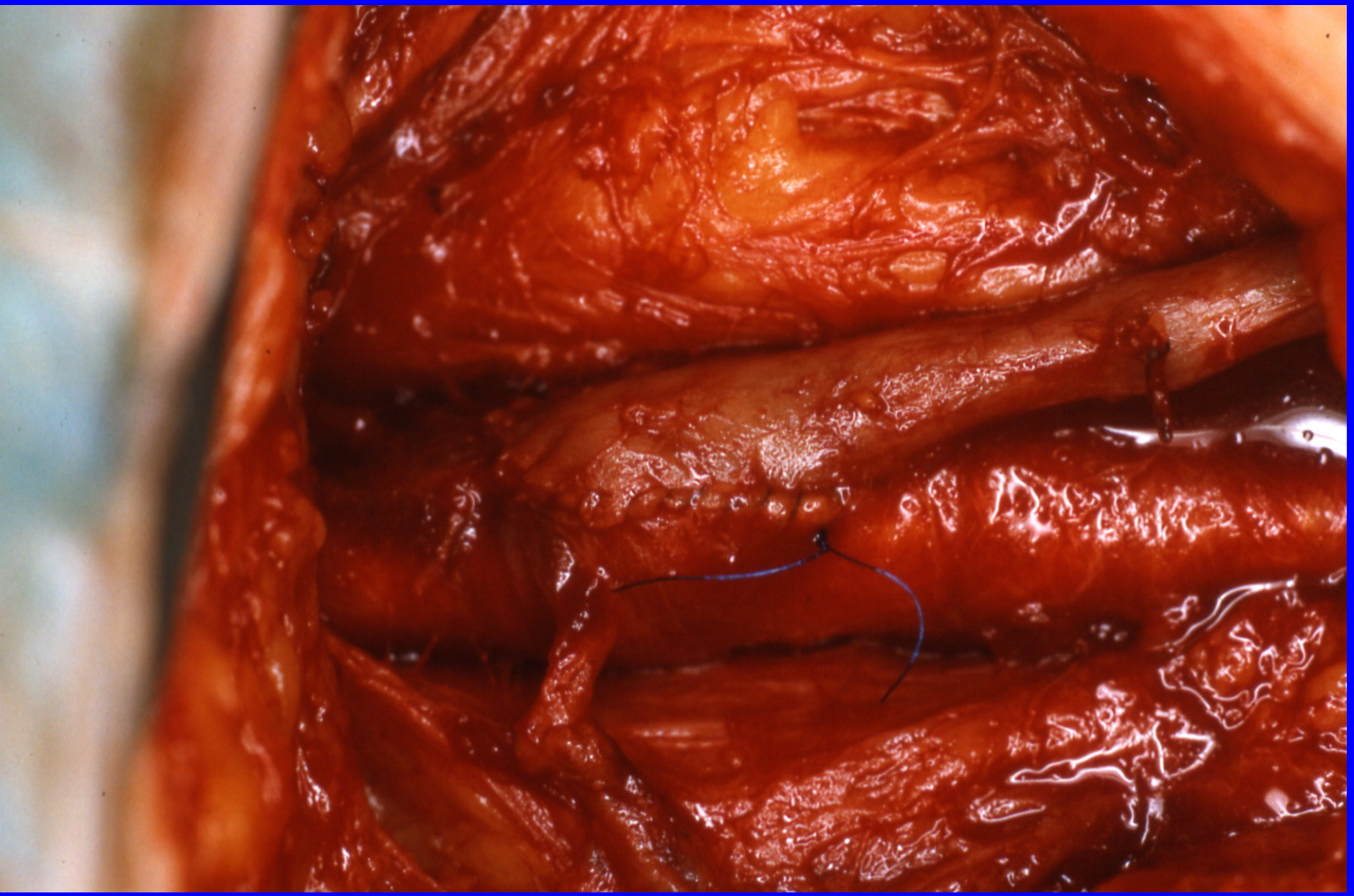
- Claudication implies atherosclerotic load
- Risk factor management
- Patient information
- Interventions have improved
- The indications for intervention haven't changed

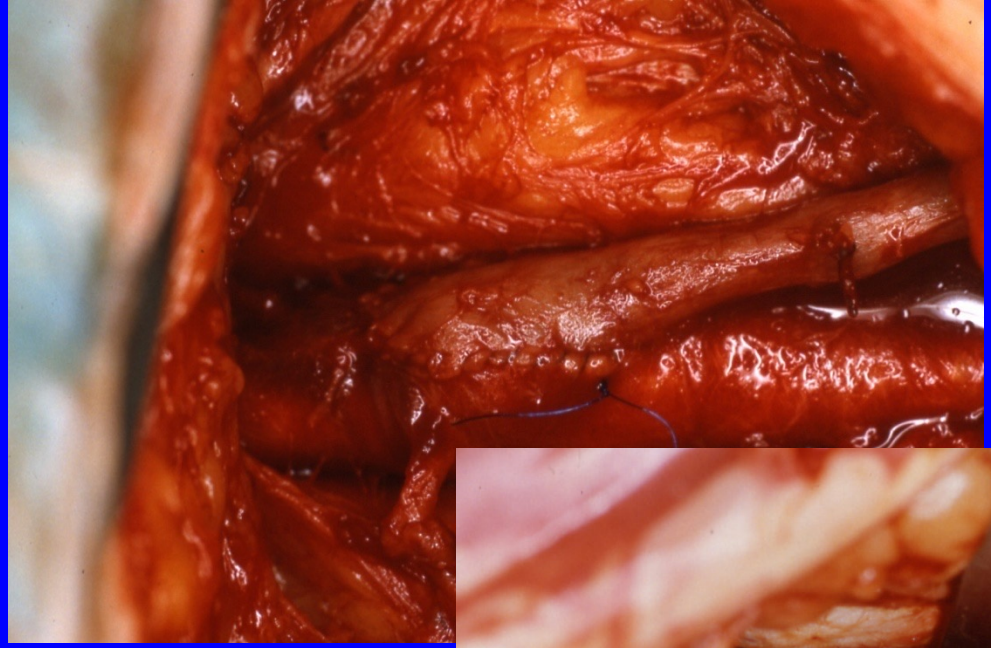
Arterial reconstruction

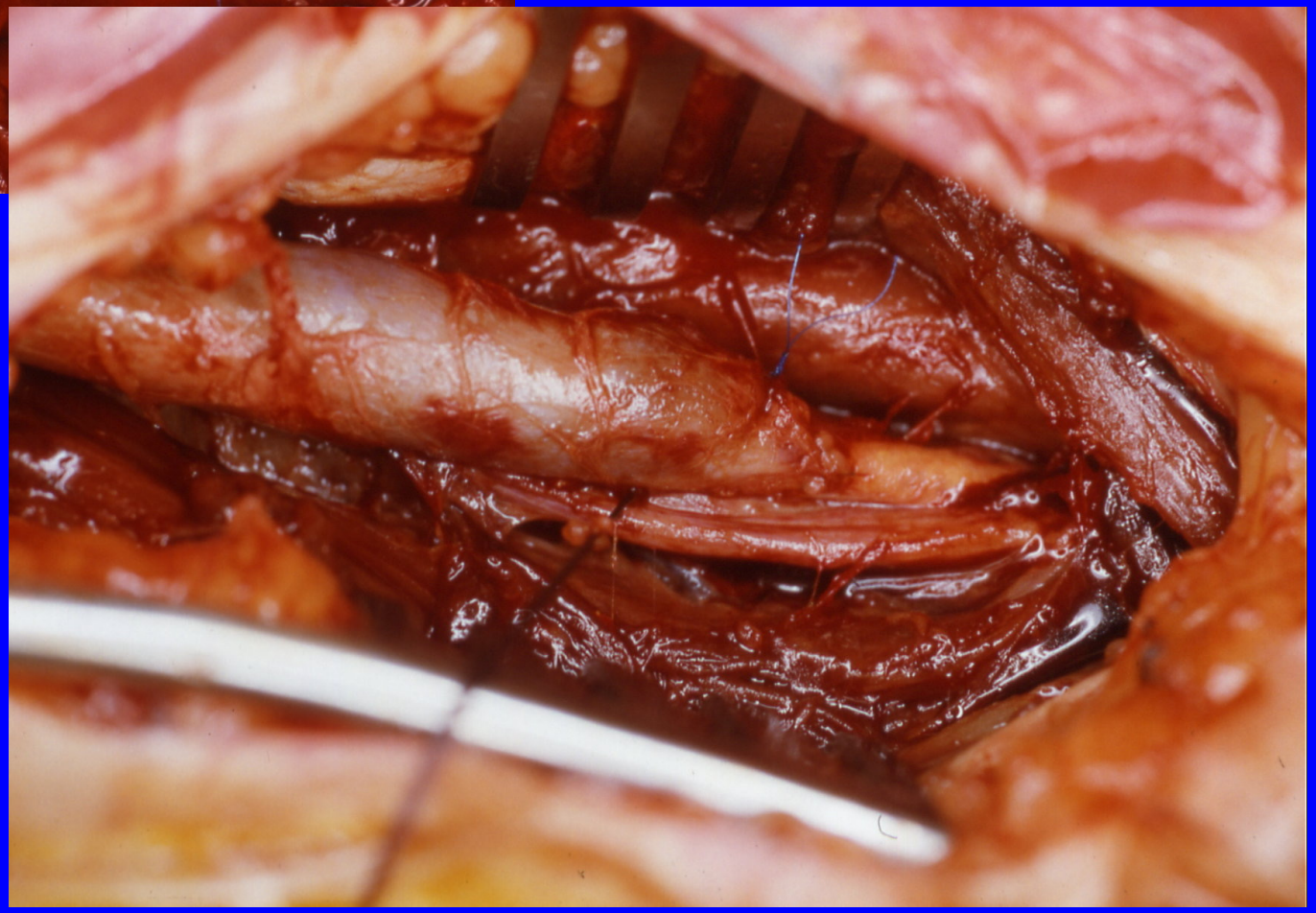


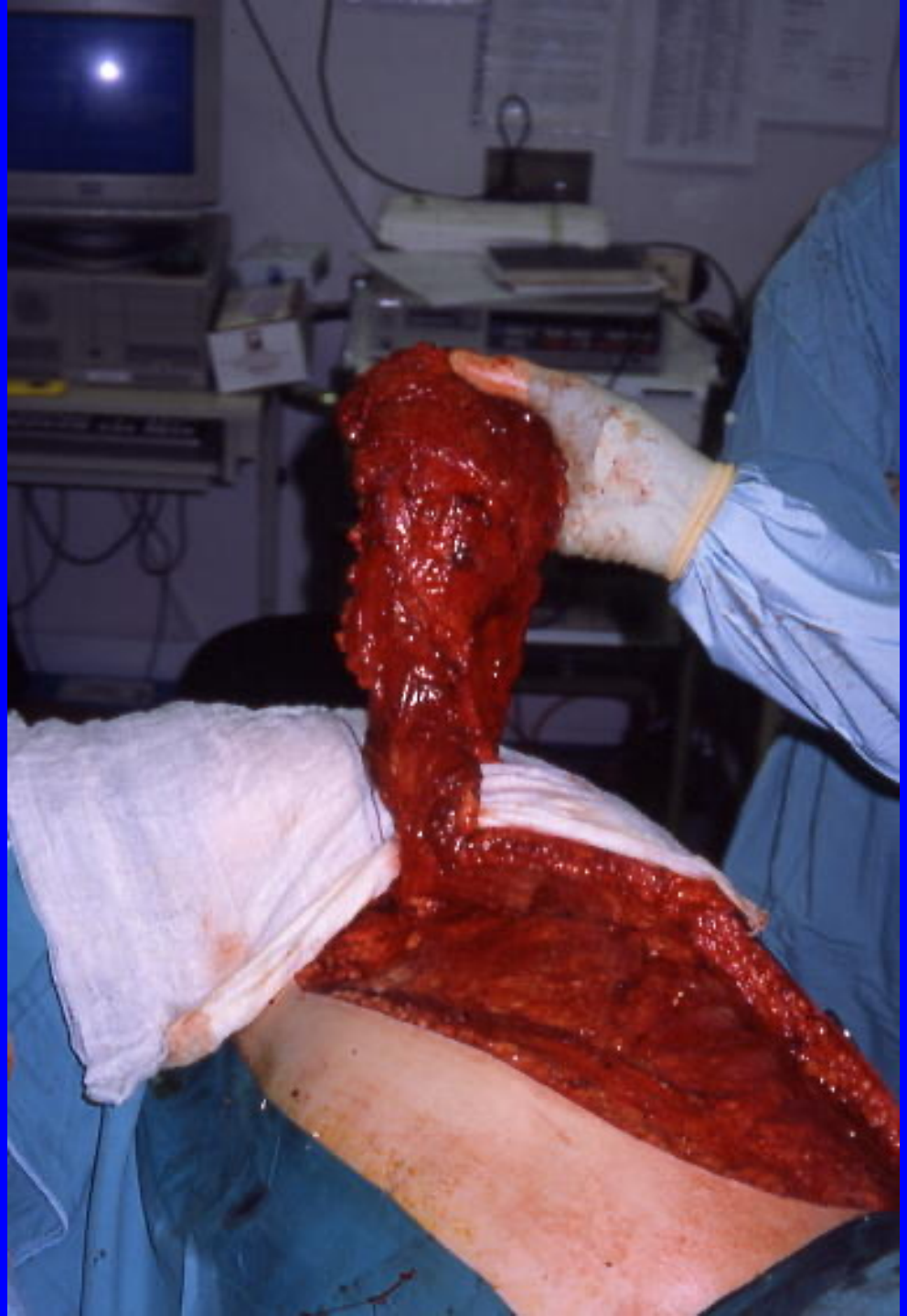












Diabetic ulcer

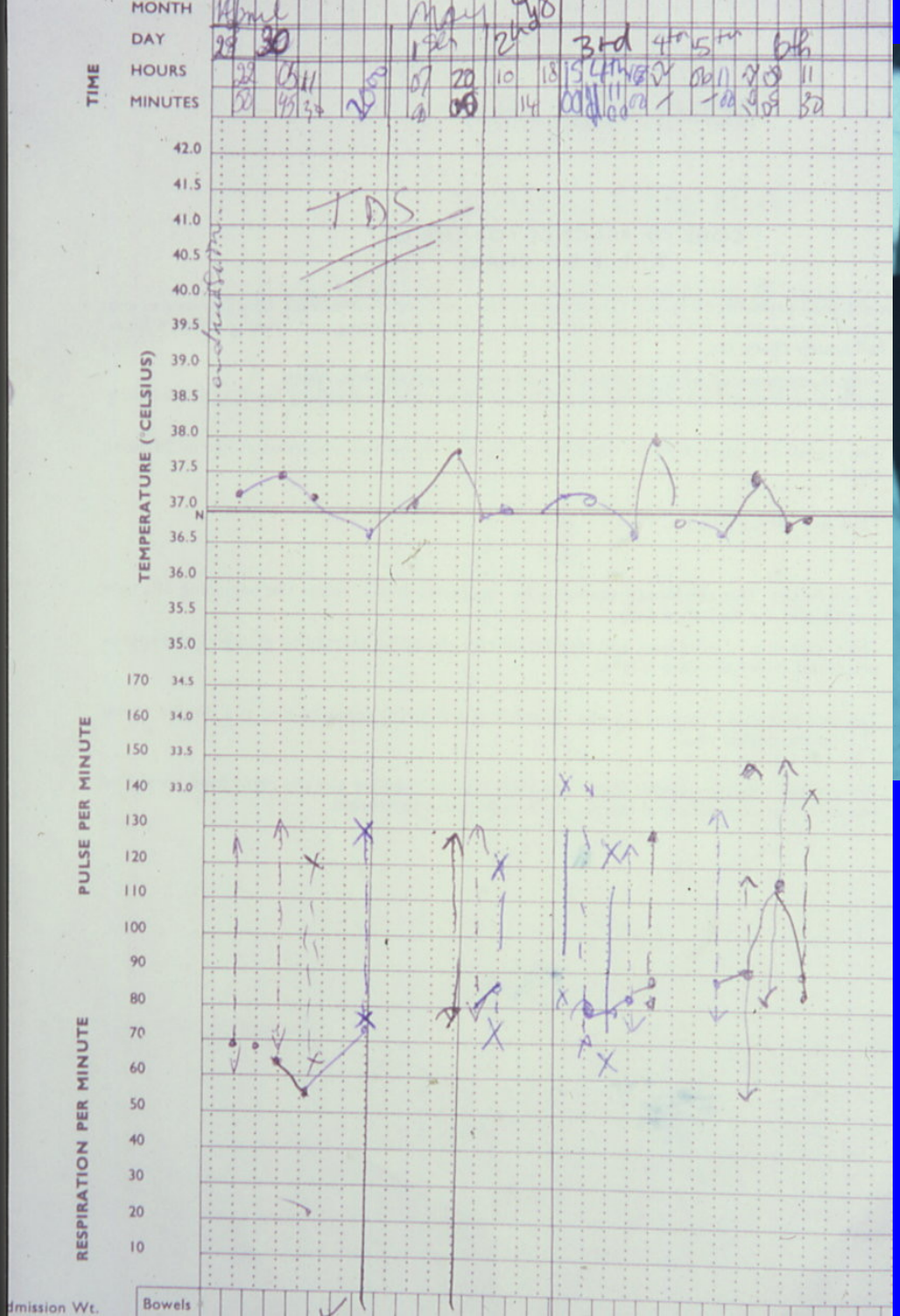
- Early intervention saves legs

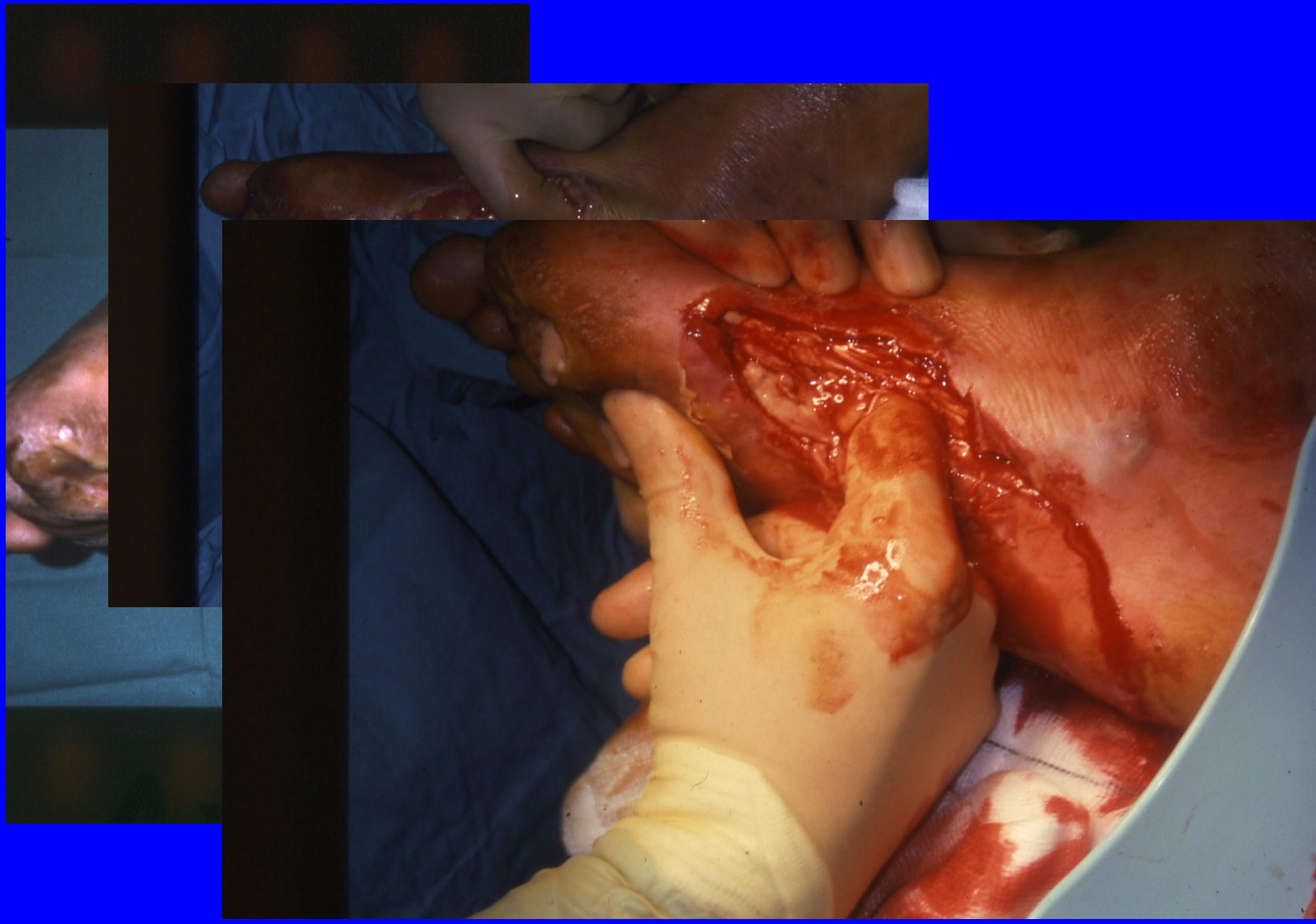
Diabetic Ulcers

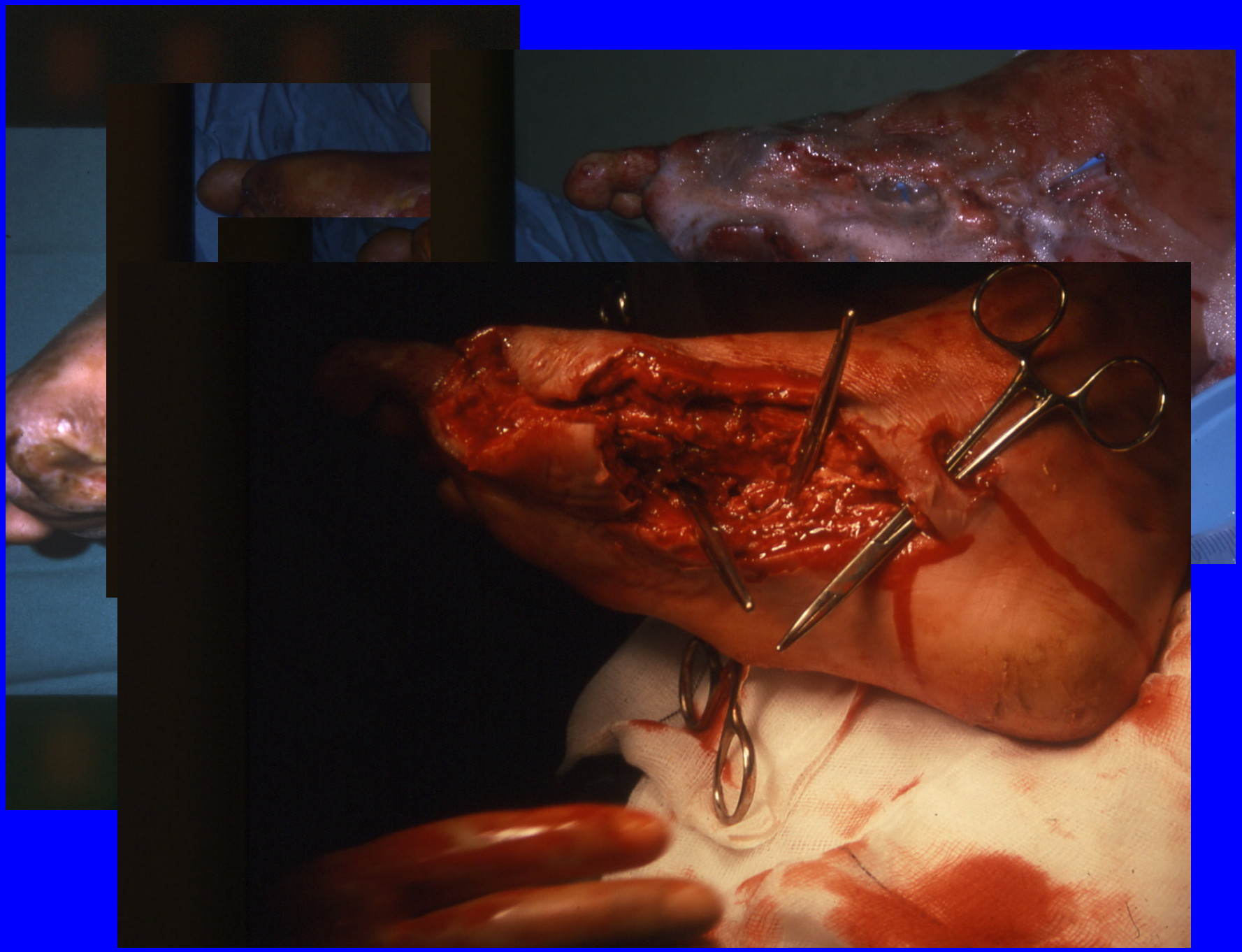
- 15% diabetics during lifetime
- 15% of admissions
- 5% prevalence
- 3% incidence
- Precede 85% of amputations
- 8 fold increased risk of amputation

2 year Mortality after BKA

36%

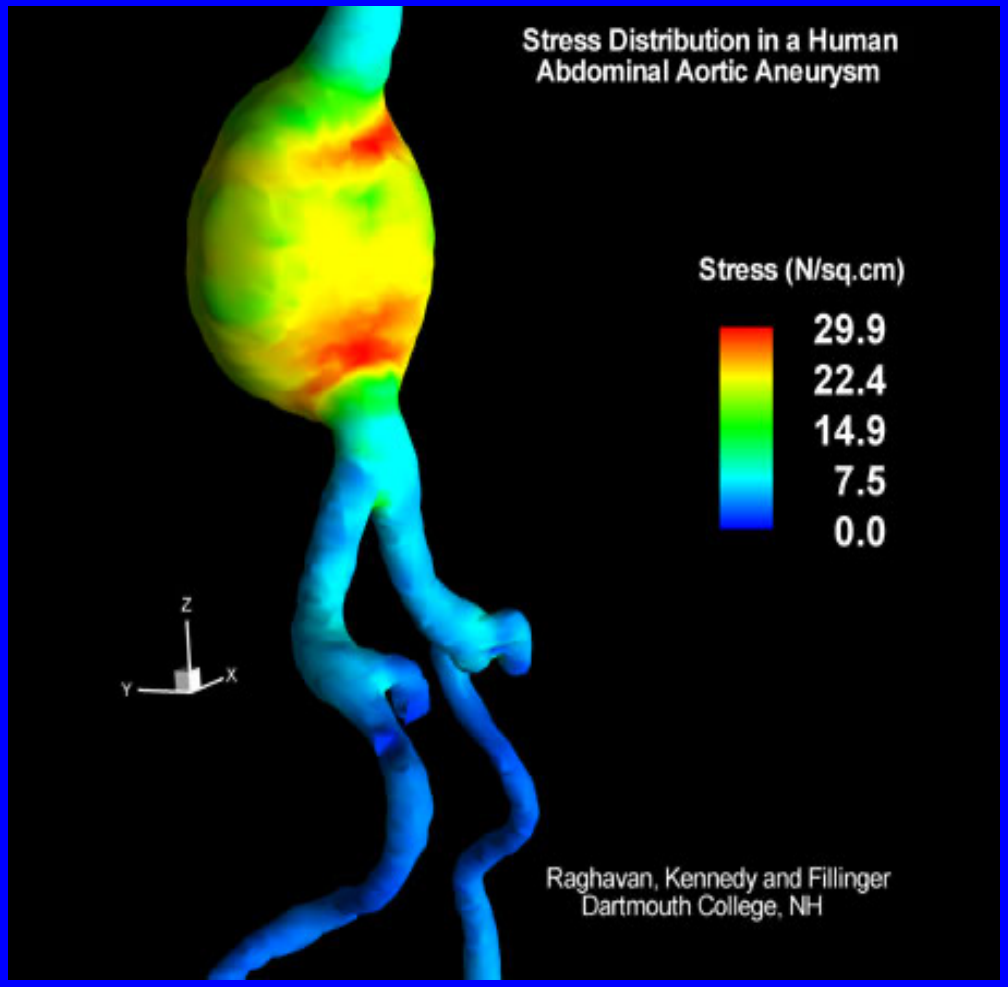

TIME
MONTH
DAY
HOURS
MINUTES
TDS
TEMPERATURE (°CELSIUS)
PULSE PER MINUTE
RESPIRATION PER MINUTE
Bowels





Abdominal Aortic Aneurysm

AAA - Pathology



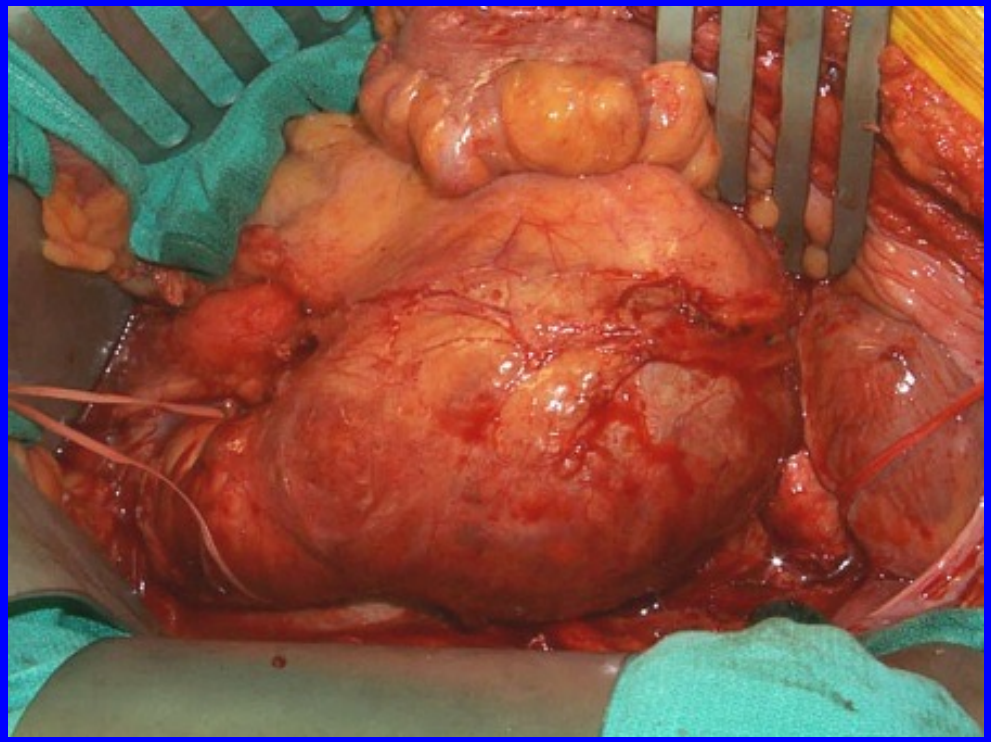
Risk factors

- Elderly
- Male x4
- Hypertension
- PVD
- Family Hx

Collagen

Infection?

AAA - mortality



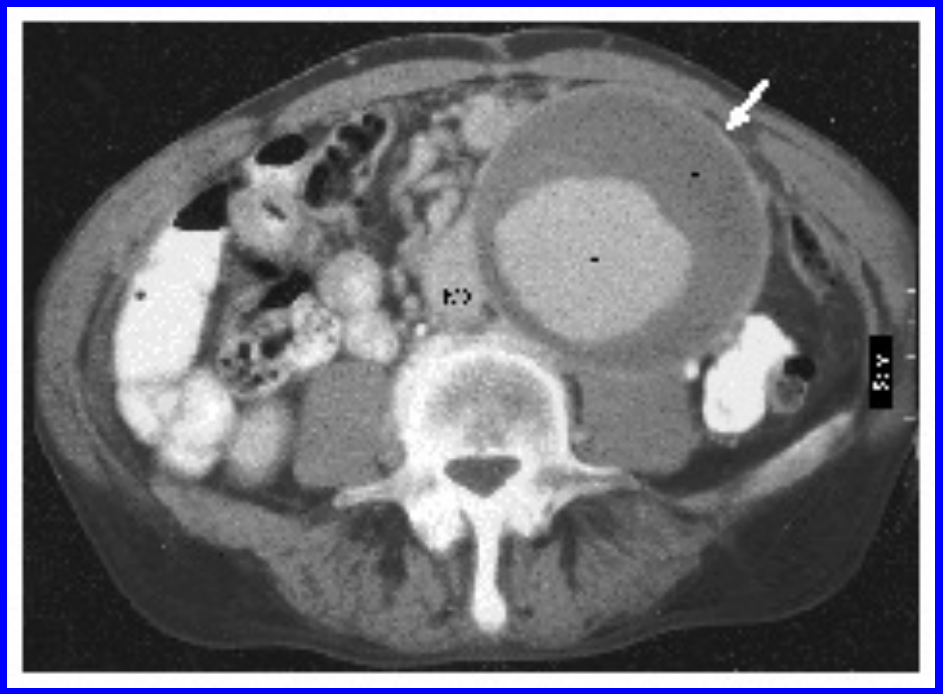
2% male deaths >55

3000 elective and 1500 emergency ops / year

Rupture op. mortality 45% (90% overall)

Elective mortality 8%

AAA - clinical course



Diameter 10% p.a.

Only 15% rupture

85% die of something else

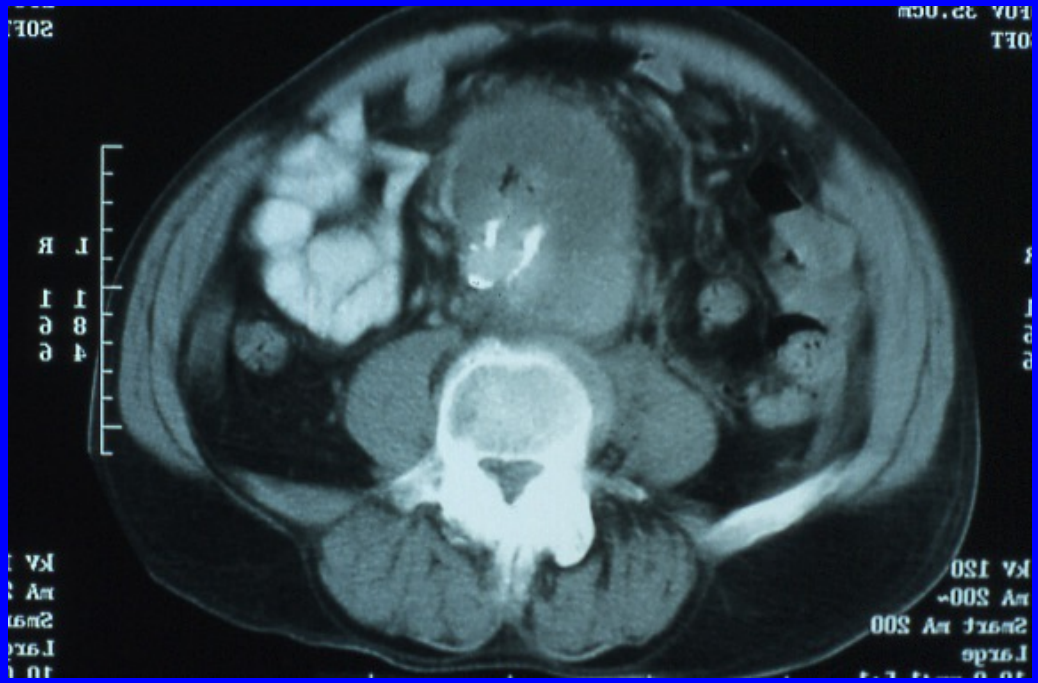
5 year rupture risk

- 5-5.9cm - 25%
- 6-6.9cm - 35%
- >7cm - 75%

AAA - clinical features

75% asymptomatic
- Epigastric pain
- Back pain
- Malaise + weight loss

Rupture
- Severe back pain
- Circulatory shock



Indications for surgery

Diameter >6cm

Rupture

Symptomatic

Expansion >1cm/year

UK small aneurysm trial: no advantage operating on 4-5.5cm aneurysms

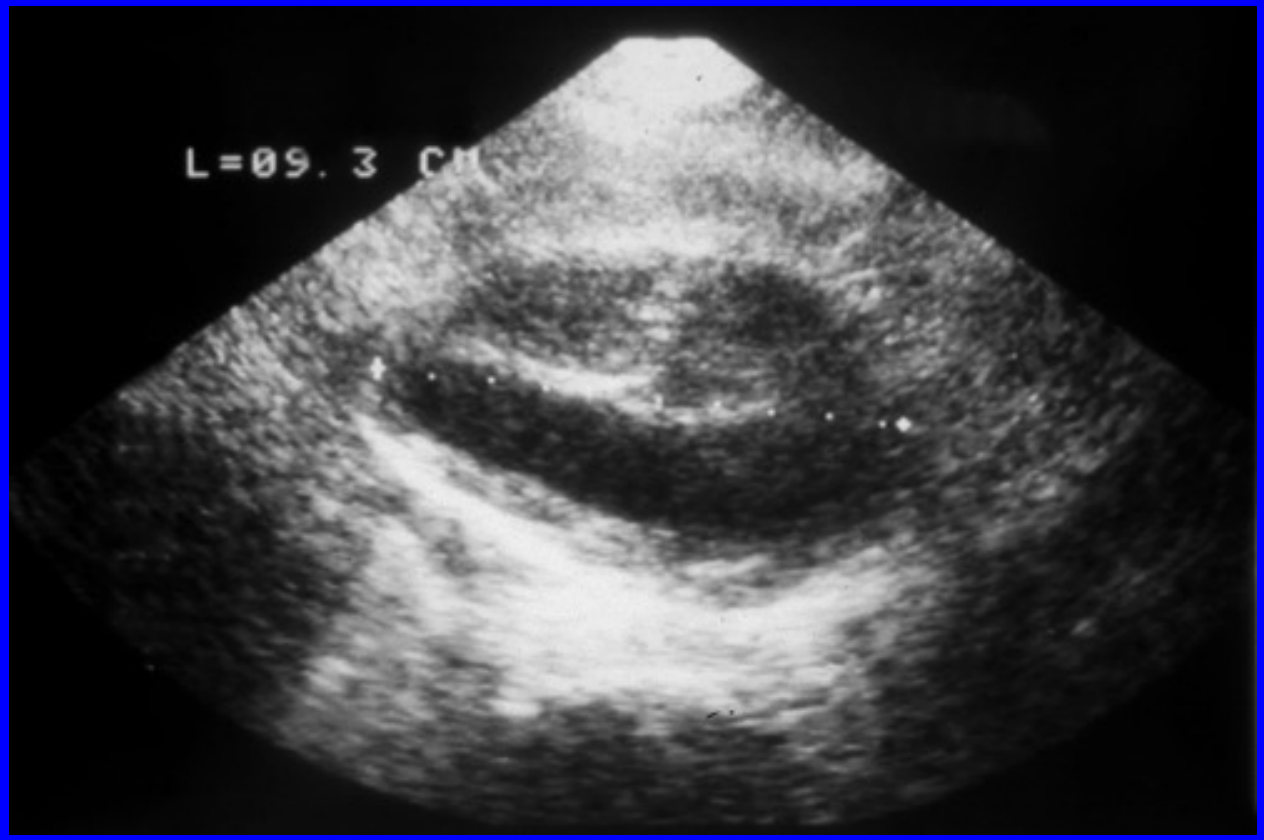
Surveillence of AAA <5.5cm

3 months

6 months

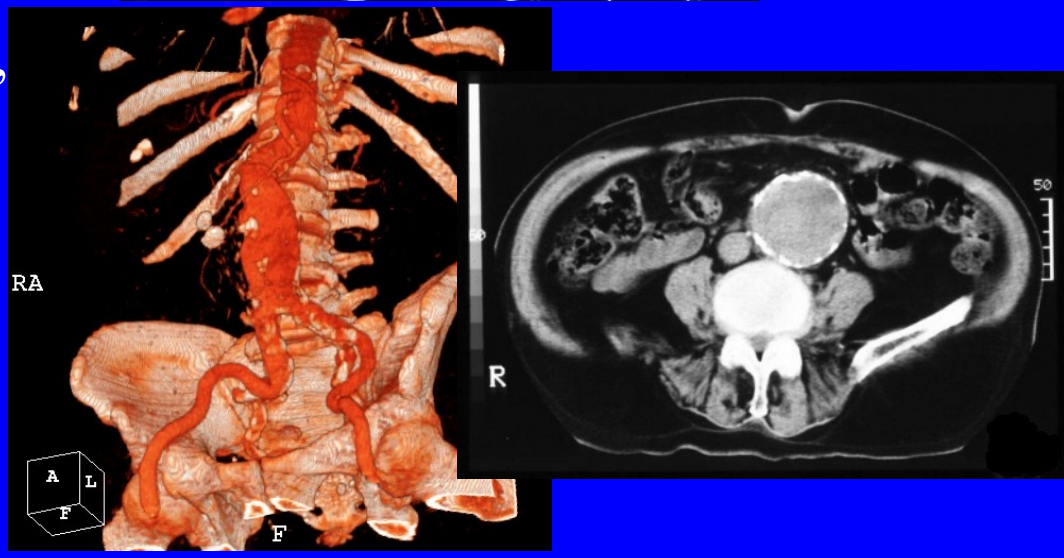
12 months

Yearly



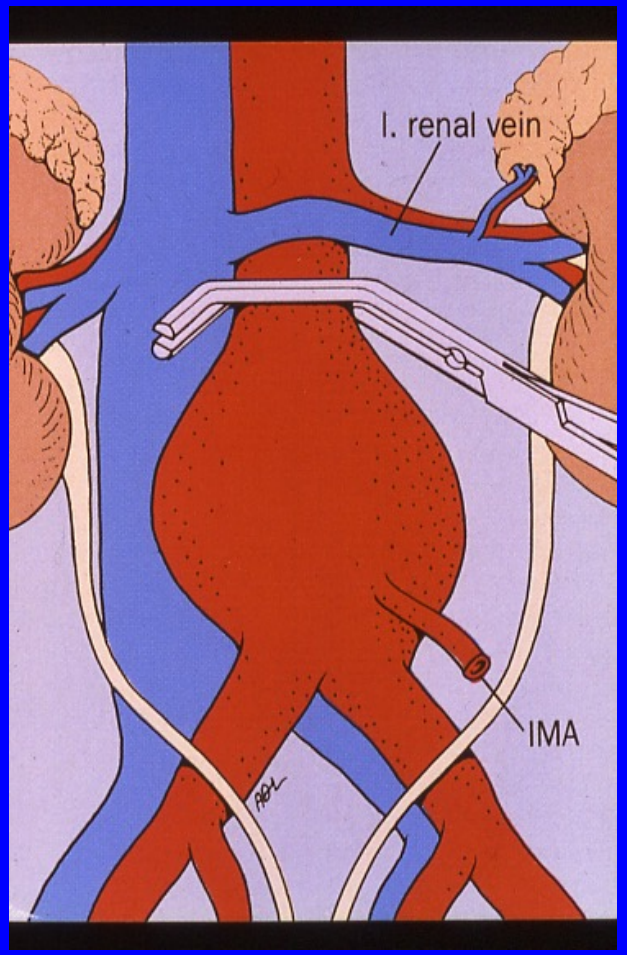
Preoperative investigation

- Extent of aneurysm
  - CT scan to show anatomy
- Fitness for anaesthesia
  - Renal function: Cr
  - Cardiac function: ECG, Echo/stress, angio?
  - Respiratory function: LungFTs, exercise



Operation

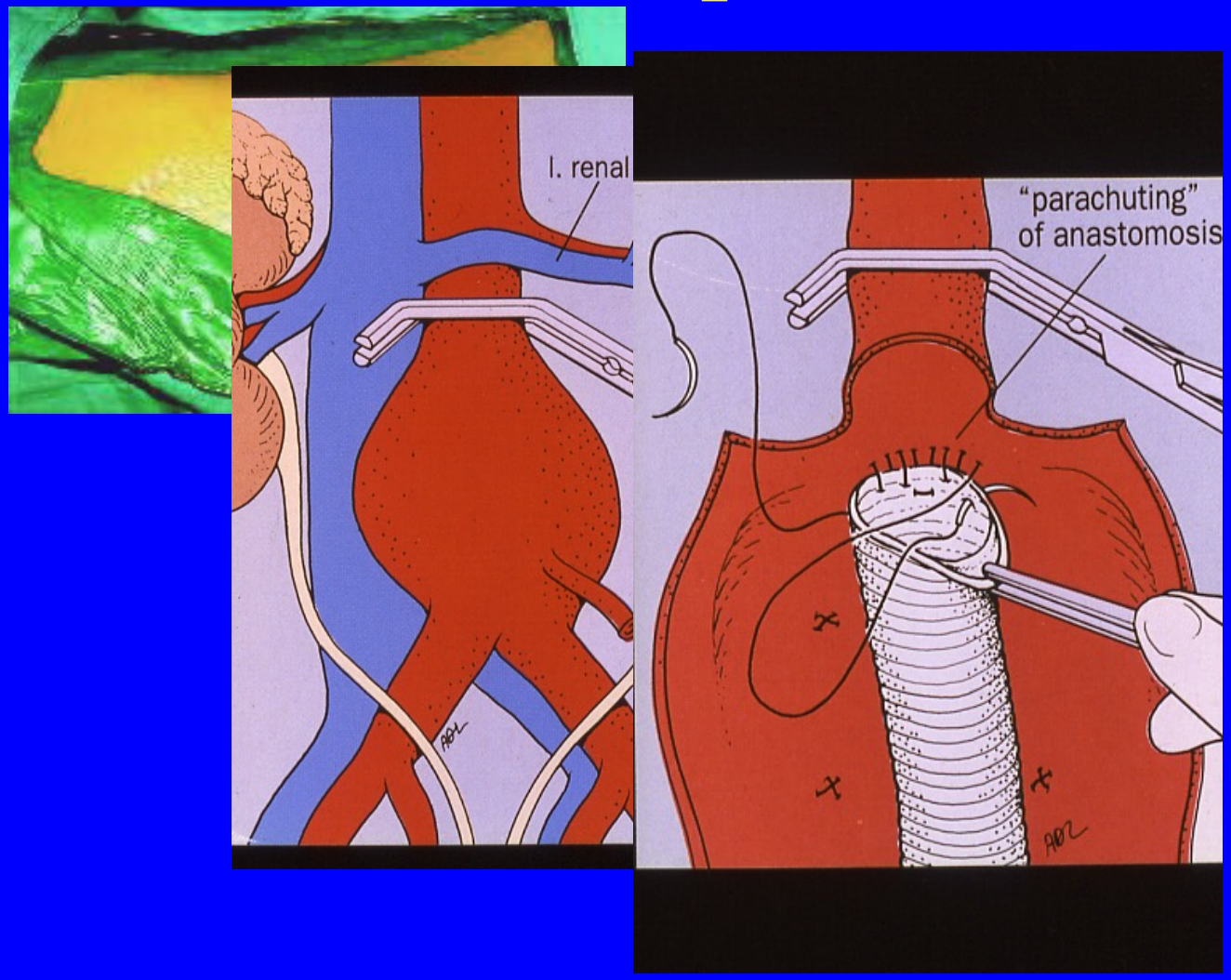
Operation



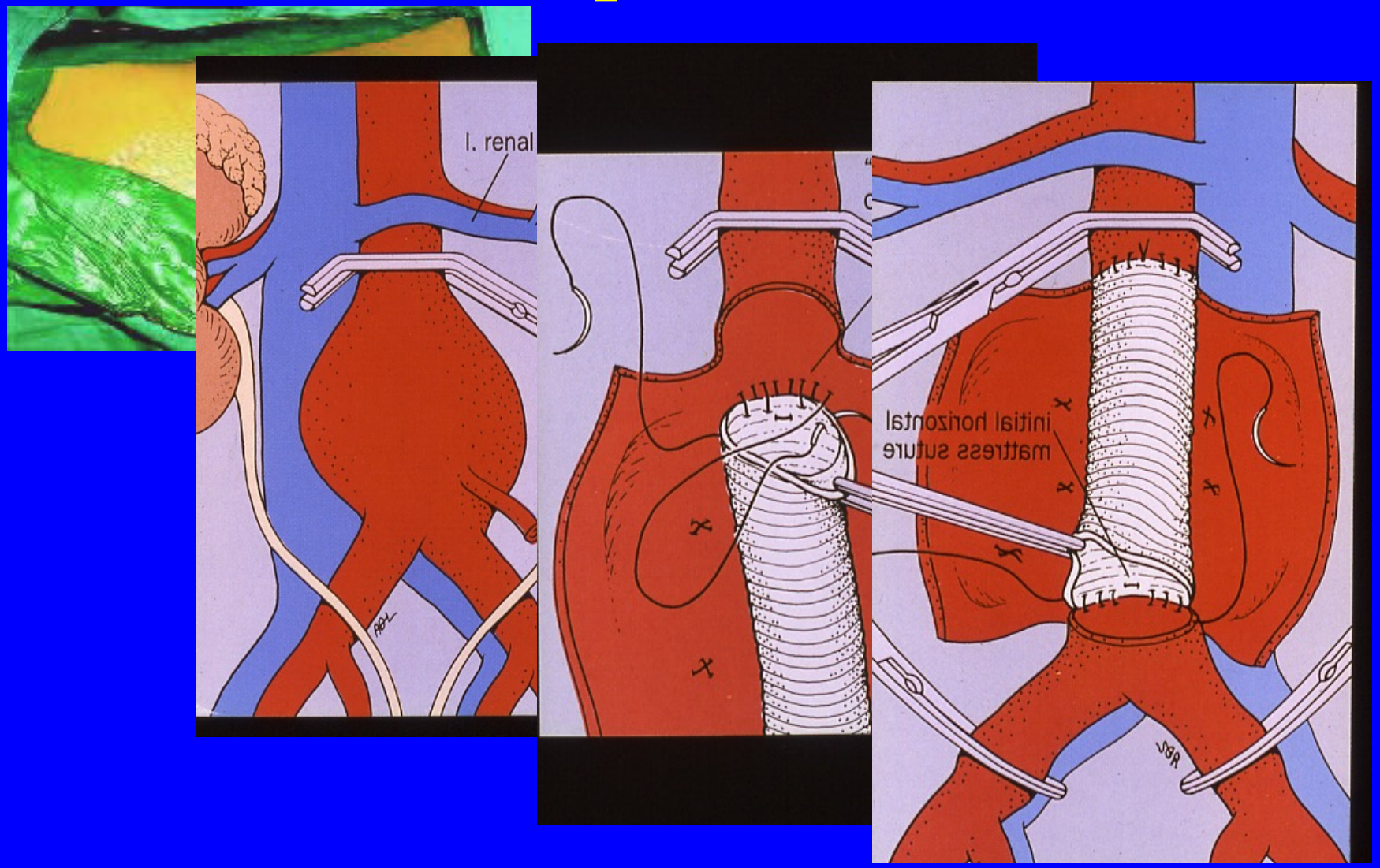
Operation

Operation

Operation



Operation



Postoperative care

- HDU
  - Epidural
  - Fluid intake
  - ECG
  - Resp function
  - GI
  - Peripheral circulation

Aneurysms elsewhere

Popliteal

Aneurysms elsewhere

Popliteal

Carotid

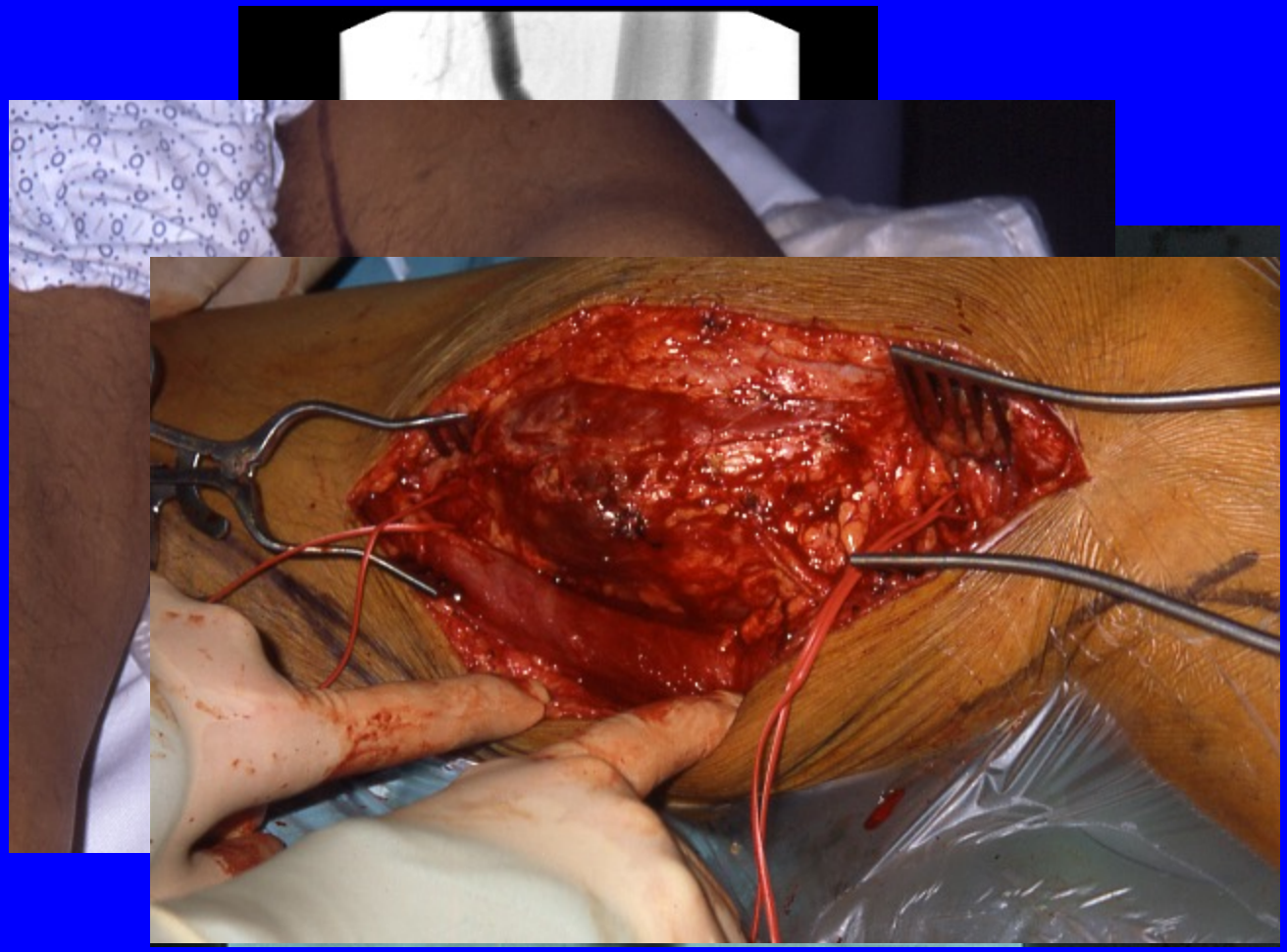
SFA

Aneurysms elsewhere

Popliteal

Carotid

SFA



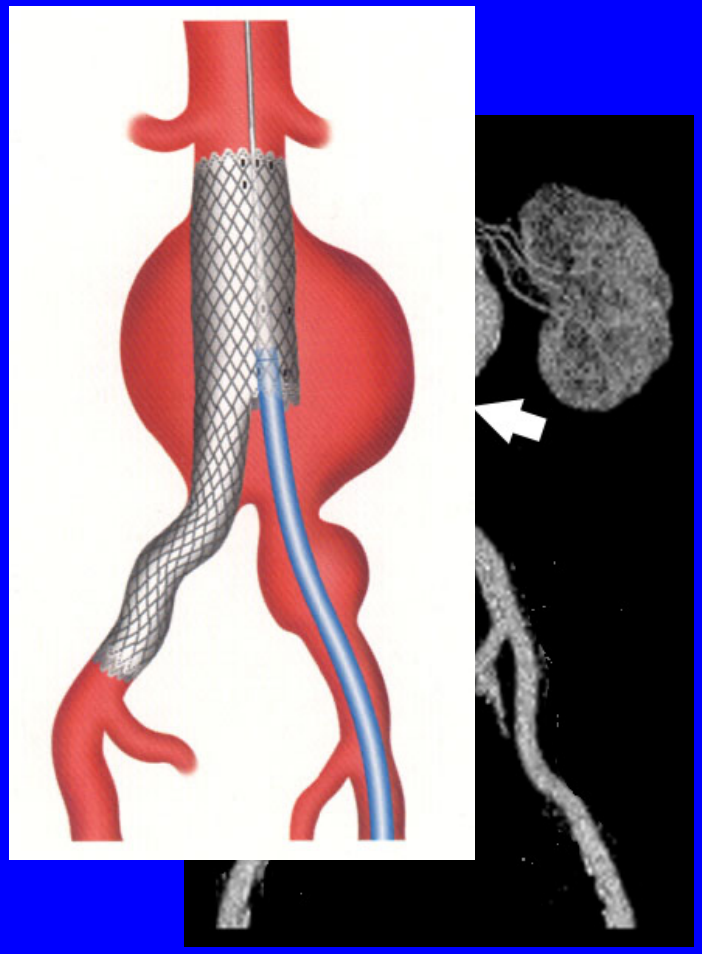
Aneurysms elsewhere

Popliteal

Carotid

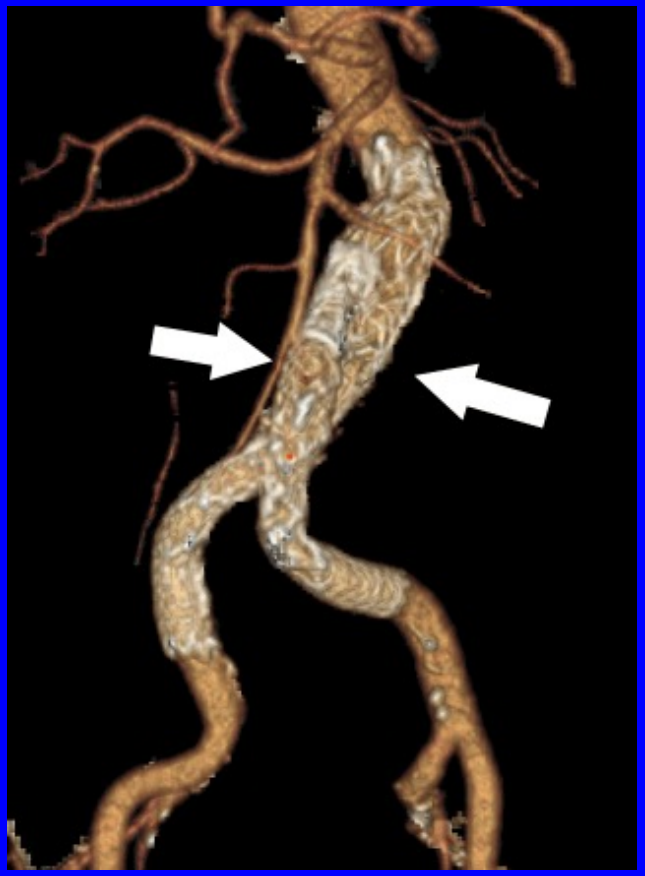
SFA

Endovascular Aneurysm Repair



- Reintervention
  - EVAR 9.8%
  - Open 5.8%

EVAR 1, *Lancet*, 2005



- 4 years median
- All-cause mortality 28% both
  - EVAR 4%
  - Open 7%
- 4yr Hospital Costs
  - EVAR £13257
  - Open £9946

EVAR 2, *Lancet* 2005

	EVAR	Medical Rx
n	166	172
30 day Mortality	9%	9 ruptures per 100 person years
Cost	£13632	£4983
4 Year follow-up	Overall mortality	64% both groups

Multicentre Aneurysm Screening Study (MASS), *BMJ* 2002

- *67800 men 65-74*
- *47 fewer aneurysm deaths*
- *Cost £28000 per life year gained*
- *Falling to £8000 at 10 years*

Stroke

20-30% attributable to carotid artery disease

Symptoms of carotid bifurcation disease

Hemispheric event – TIA

Hemispheric event – completed stroke

Monocular event – amaurosis fugax or blindness

Face, Arm, Speech, Time

Carotid surgery to prevent stroke

Eastcott 1954

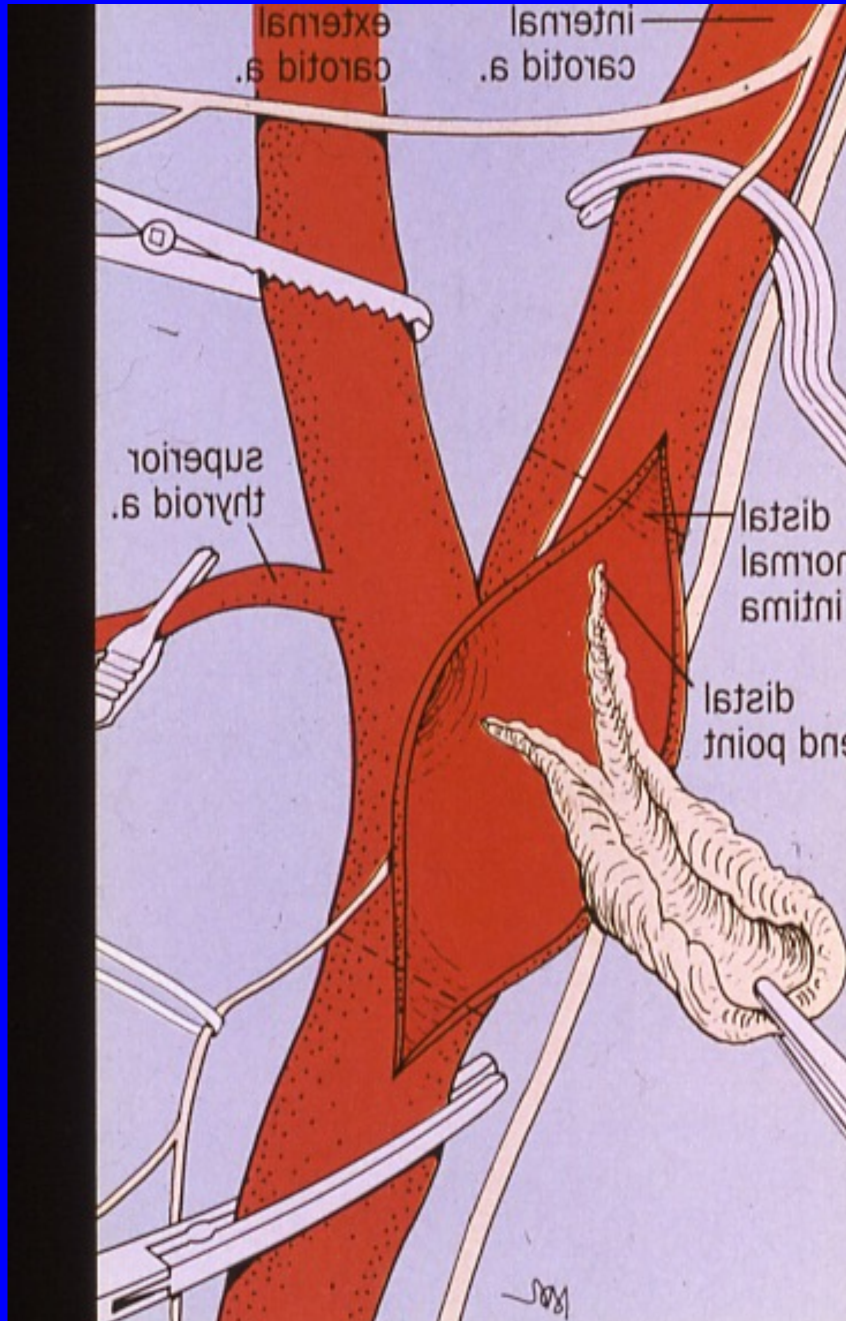
(?Debakey 1953)

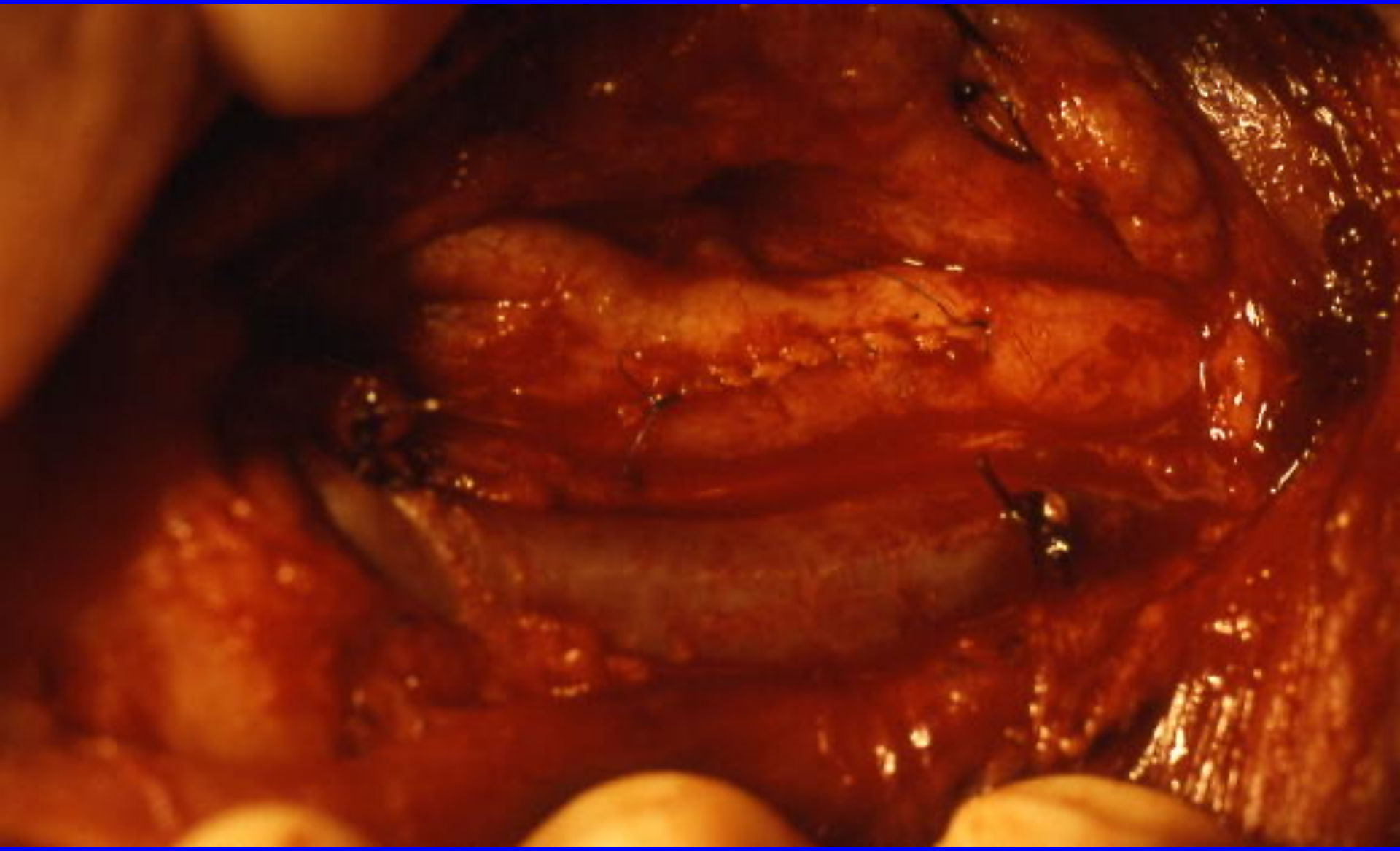
3-year death and stroke rate, >70% stenosis

BMT	26.5%
CEA	14.9%

30 day death and stroke after CEA 7%.

Symptomatic disease, > 70% ipsilateral stenosis, NNT = 7
Asymptomatic disease, > 70% ipsilateral stenosis, NNT = 50.


external carotid a.
internal carotid a.
superior thyroid a.
distal normal intima
distal end point



Key steps in carotid intervention

- Ischaemic event
- Presents to primary care
- Prompt imaging – ultrasound, CT brain
- Stroke service - ?thrombolysis
- Refer vascular intervention team
- Intervention

Determinants of outcome

- Patient selection
- Best medical therapy
- Timing of intervention
- Quality of intervention

Stroke risk after TIA

48 hours	6.7%
7 days	10.4%
30 days	13.4%

Rothwell, 2007

Timing

- Rothwell 2004
  - <2 weeks 1 stroke saved for 5 CEAs
  - >12 weeks 1 stroke saved for 125 CEAs

Improved outcome in carotid intervention

- Public education
- Shorten event to intervention time
- Adequate capacity in intervention service
- High volume = better outcomes

Acute management

Recognise symptoms early

CT Scan

Refer to stroke team

Thrombolyse?

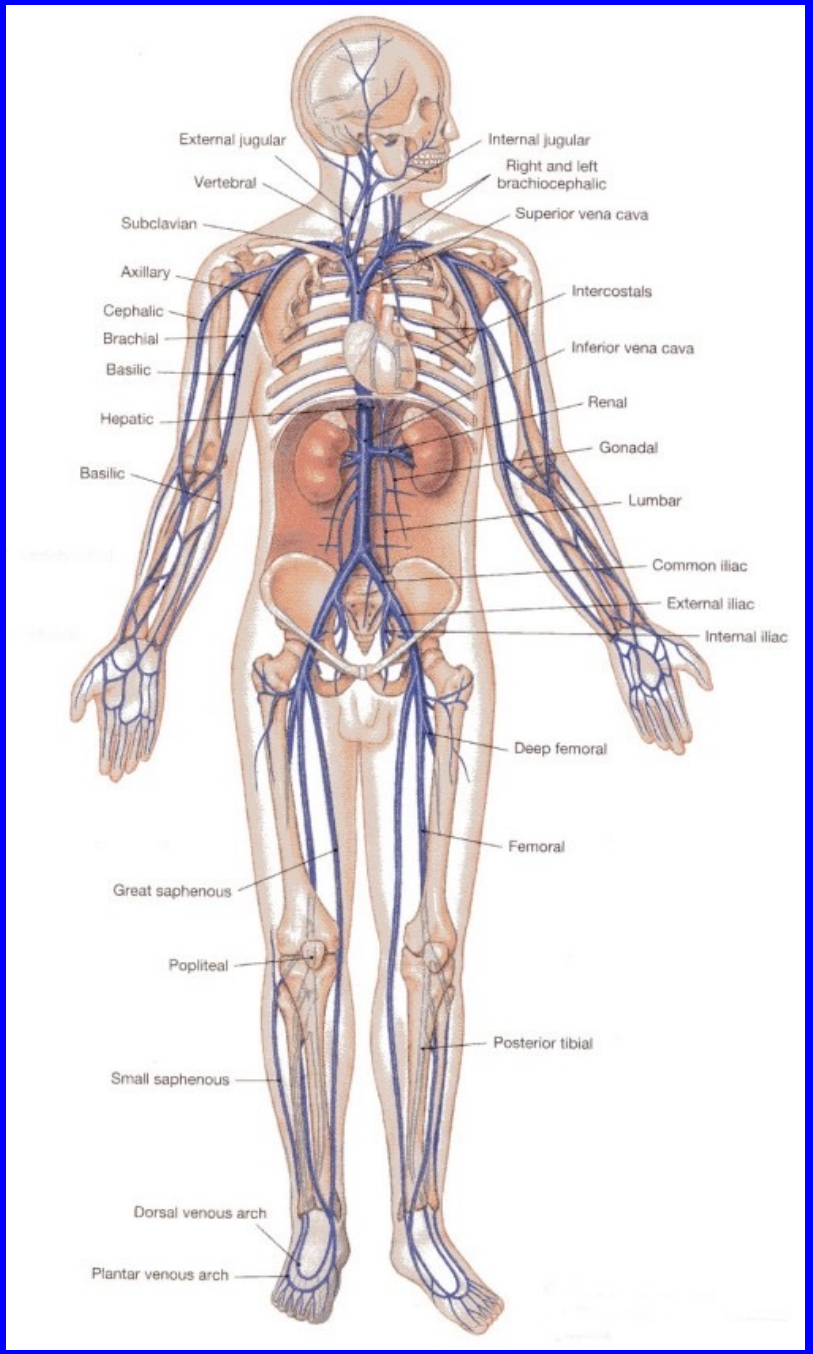
Duplex ultrasound of carotids

Refer to Vascular Surgery

Venous Disease

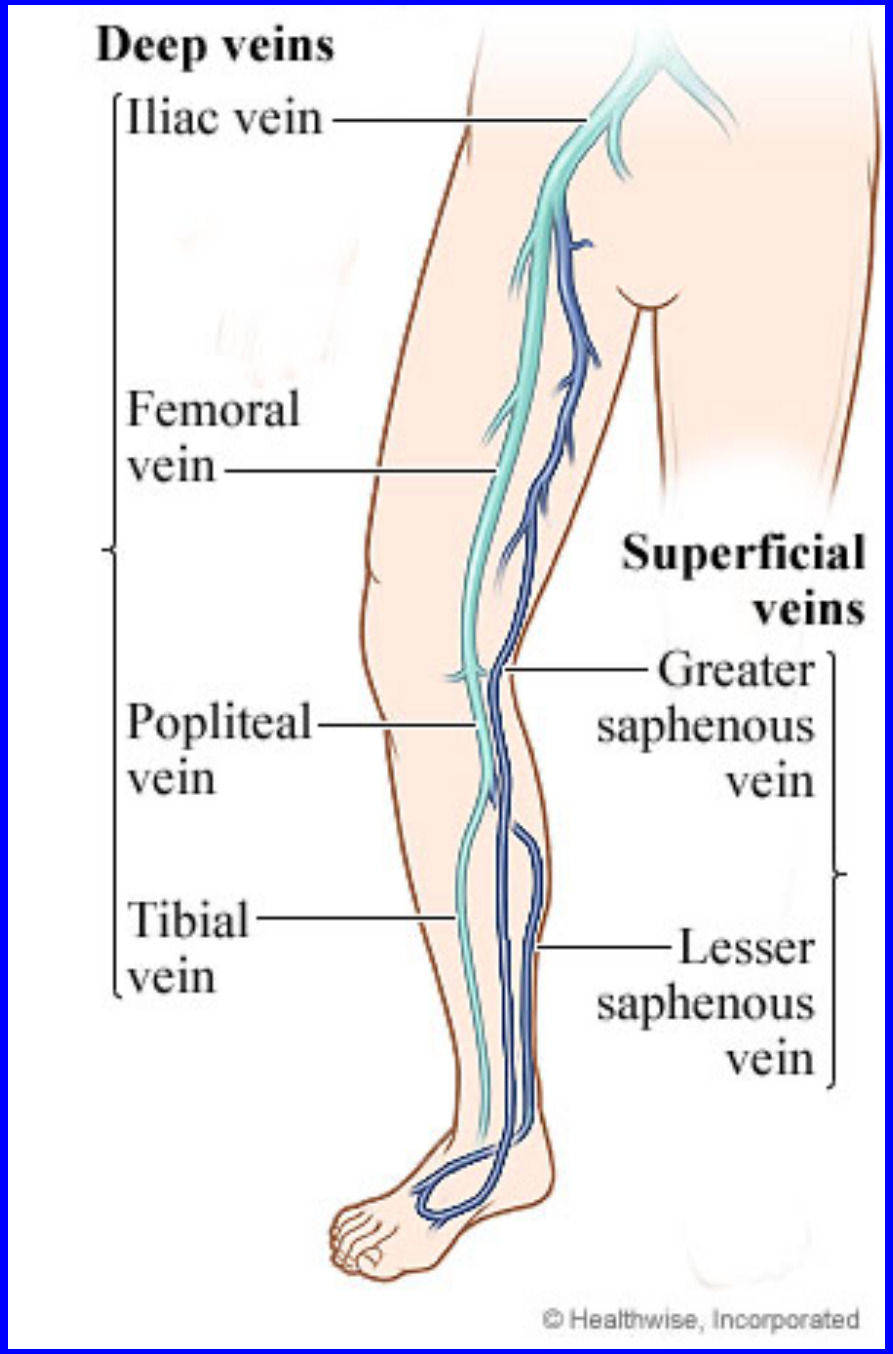
Function of Veins

- Return Oxygen and nutrient *depleted* blood
- Reservoir - 2/3 blood volume
- Passive flow: depends upon
  - Calf muscle pump
  - Respiration
  - Blood volume
  - Distance from heart



Deep Veins

- **Branch like arteries**
- **Close to arteries**
- **Do the main job of returning blood from limbs and organs**

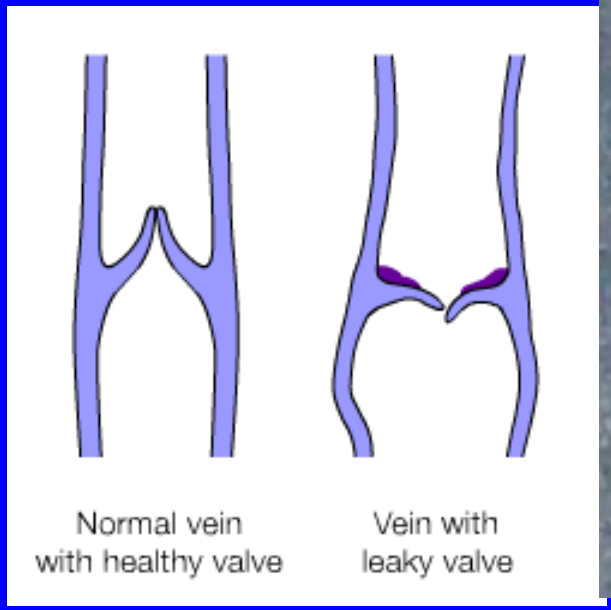


Surface veins

- **Do not follow arteries**
- **Can be removed without consequence**
- **Often used for bypass procedures**
- **Can become varicose**

Structure

- **Thin wall**
- **Distensible**
- **Valves**




Valves prevent backflow

Ultrasound Mapping

Diseases of Veins

- Deep Vein Thrombosis
  - Acute DVT
  - Pulmonary embolus
  - Chronic Post-DVT syndrome
- Superficial Venous Reflux (varicose veins)

DVT

- Risk factors
- Complications
- Prevention
- Treatment
- Complications and sequelae

Superficial venous incompetence

- Varicose veins

Superficial venous incompetence

- Varicose veins

Indications for treatment of Superficial Venous Reflux

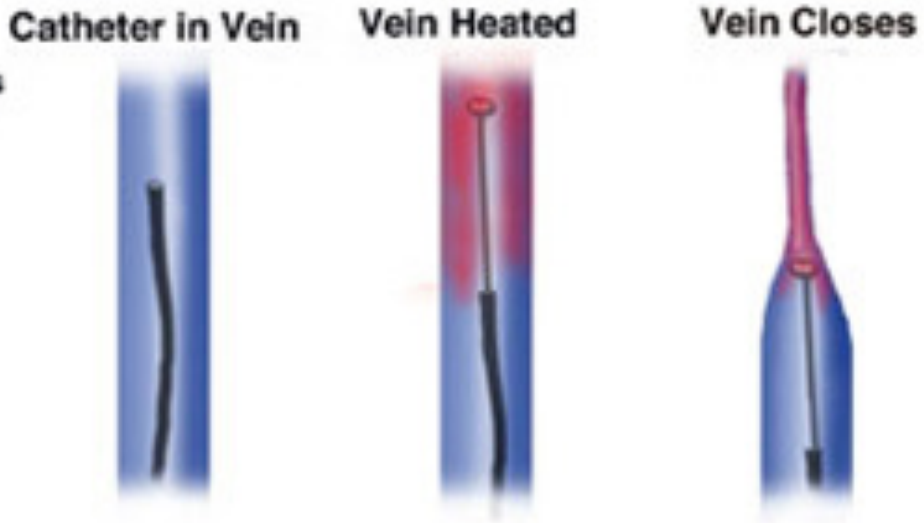
- Absolute:
  - Ulcer
  - Eczema
  - Bleeding
  - Superficial vein thrombosis
- Relative:
  - Pain
  - Cosmesis
  - Swelling

Superficial Venous Reflux: Treatment Options

- Graduated compression stockings
- High tie and strip
- Radiofrequency ablation
- Phlebectomies
- Foam sclerotherapy
- Novel Therapies

Stripping

Vein Ablation

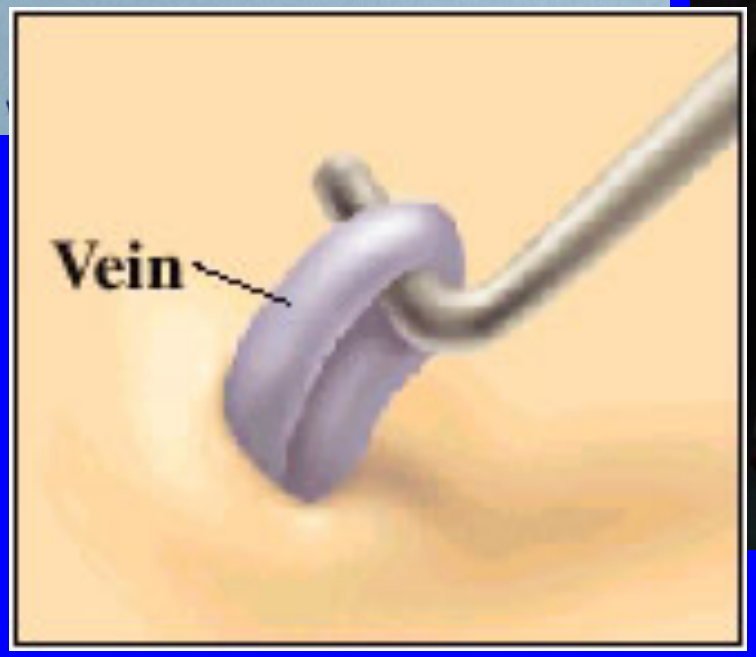


Laser therapy – similar in effect to radiofrequency ablation

BEFORE

AFTER

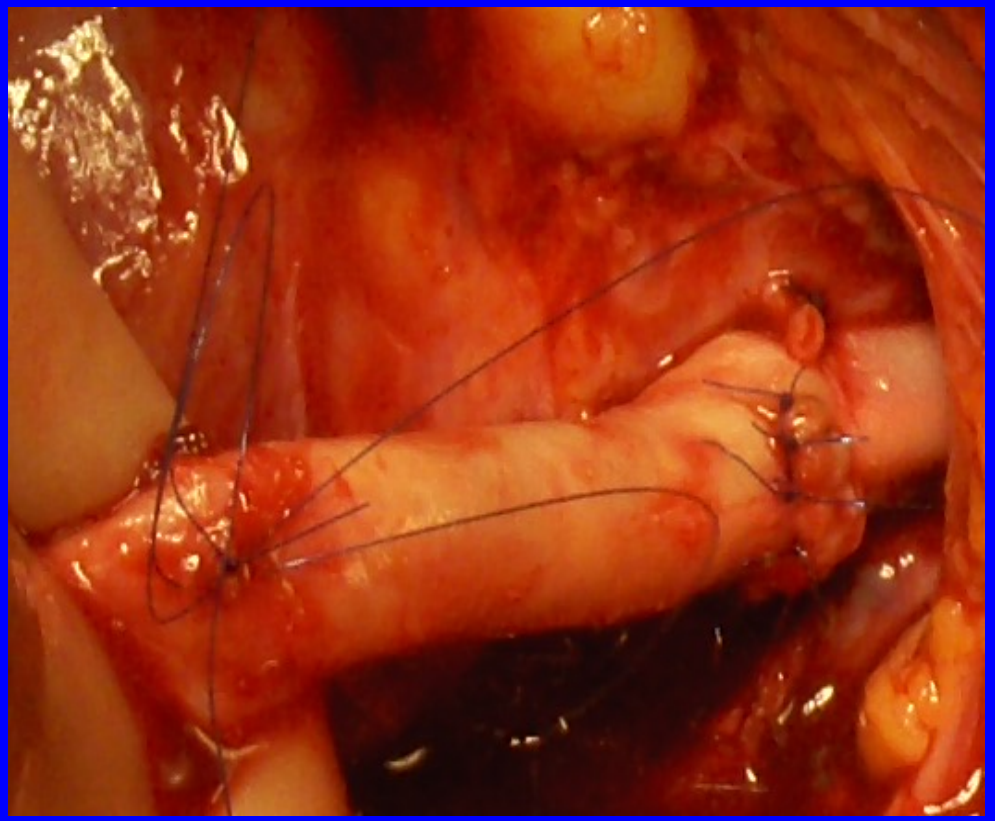
Phlebectomies



Foam sclerotherapy

Remember – veins can be used as arteries!

- Harvest from superficial vein in leg or arm
- Vein wall “arterialises”
- Good long term patency



Chronic venous incompetence

Chronic Venous Ulcer

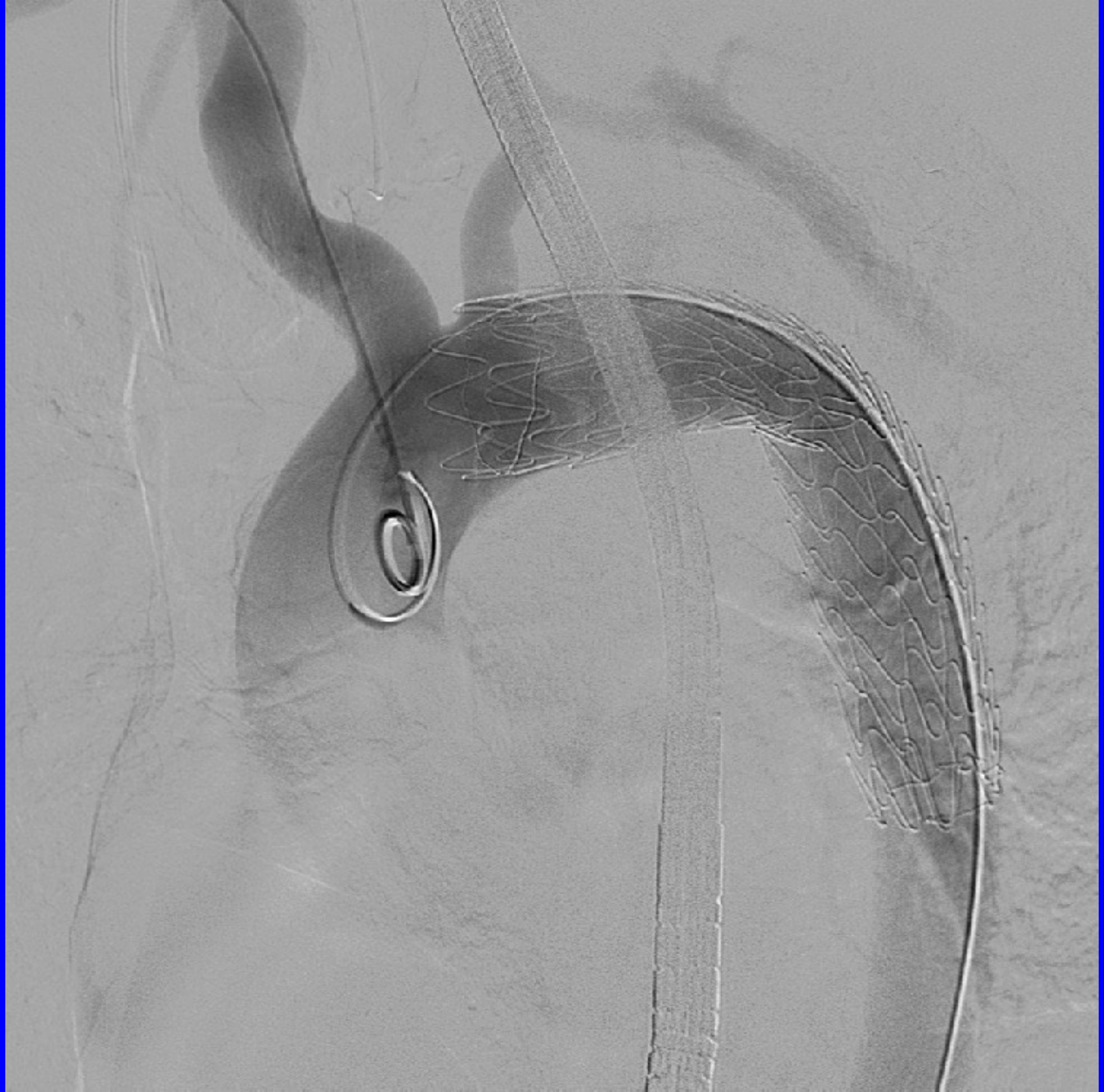
Vascular Trauma

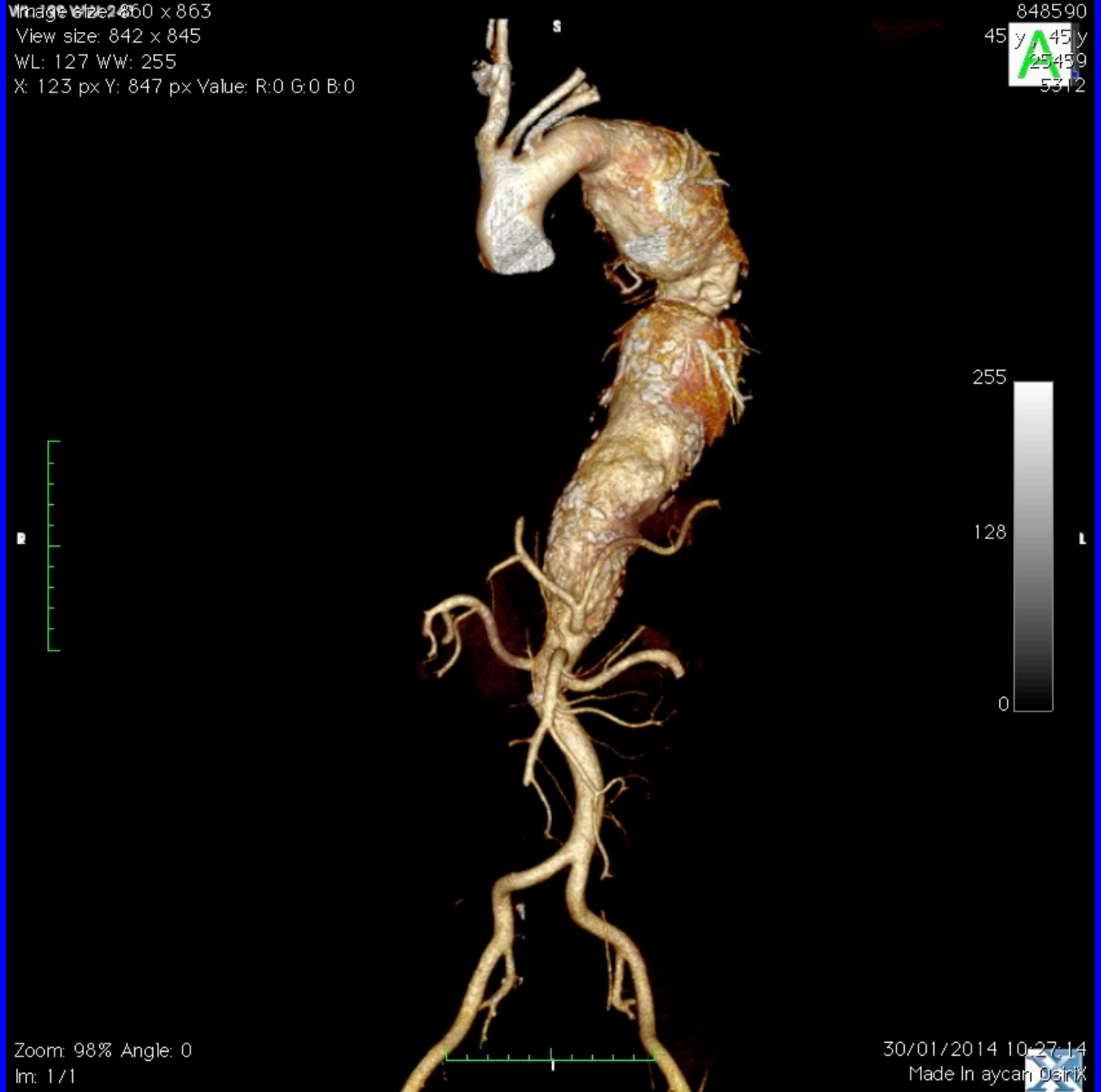
Blunt

Penetrating (sharp)

Iatrogenic

CT Angio

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SAM
SPLINT