

Aortic Aneurysm Disease

Professor Mark E. O'Donnell

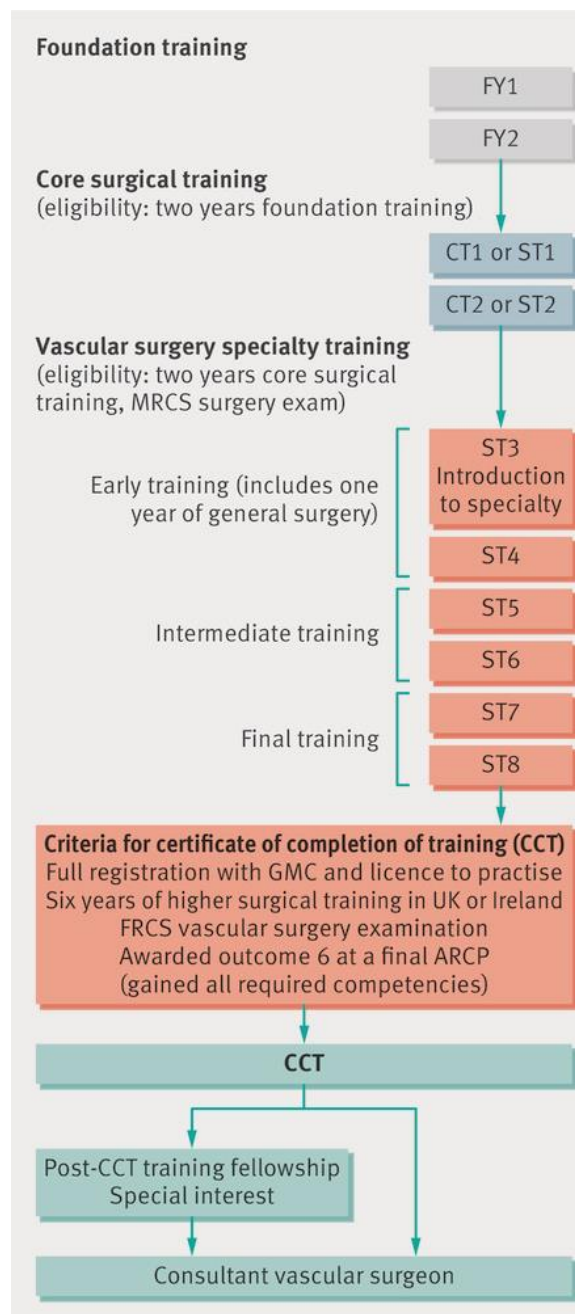
**DipSEM(GB&I) MB BCH BAO(Dist) MFSEM(UK) MFSEM(RCSI&RCPI) MFSTEd MMedSc(Dist) MD ECFMG
RPVI(ARDMS) FRCSEd(Gen&VascSurg) FEBVS(Hon)**

**Consultant Vascular and Endovascular Surgeon, Belfast Health and Social Care Trust and Visiting
Professor, Sport and Exercise Sciences Research Institute, Faculty of Life and Health Sciences, Ulster
University, Northern Ireland.**

Northern Ireland Vascular Trainee Teaching Program

Royal Victoria Hospital – Wednesday 23rd August 2017

Vascular Surgery Training



Early Years of Specialty Training

- To provide a broad based initial training in surgery with attainment of knowledge, skills and professional behaviours relevant to the practice of surgery in any specialist surgical discipline. This is defined within the common component of the syllabus (same as MRCS).
- In addition it will provide early speciality training such that trainees can demonstrate that they have the knowledge, skills and professional behaviours to enter higher specialty training in a surgical specialty. The speciality element in the early years is not tested in the MRCS but through workplace-based assessments (WPBA) in the first instance, and subsequently through the Intercollegiate Specialty FRCS examinations, which are taken towards the end of specialty training.

Early Years of Specialty Training

- To provide a broad based initial training in surgery with attainment of **knowledge, skills and professional behaviours relevant to the practice of surgery** in any specialist surgical discipline. This is defined within the common component of the syllabus (same as MRCS).
- In addition it will provide early speciality training such that trainees can demonstrate that they have the **knowledge, skills and professional behaviours** to enter higher specialty training in a surgical specialty. The speciality element in the early years is not tested in the MRCS but through workplace-based assessments (WPBA) in the first instance, and subsequently through the Intercollegiate Specialty FRCS examinations, which are taken towards the end of specialty training.

Intermediate and Final Years of Specialty Training

- To provide higher specialty training in the specialty with attainment of knowledge, skills and professional behaviours relevant to the practice in the specialty. This is defined within the specialty specific component of the early years syllabus and the intermediate and final stages of the syllabus (same as FRCS).
- Competence to manage patients presenting either acutely or electively with a range of symptoms and conditions as specified in the syllabus.
- Competence to manage an additional range of elective and emergency conditions by virtue of appropriate training and assessment opportunities obtained during training as specified by sub-specialty components of the final stage syllabus. This is tested either by the FRCS and/or by WPBA.
- Professional competences as specified by GMP and GMC.

Intermediate and Final Years of Specialty Training

- To provide higher specialty training in the specialty with attainment of **knowledge, skills and professional behaviours** relevant to the practice in the specialty. This is defined within the specialty specific component of the early years syllabus and the intermediate and final stages of the syllabus (same as FRCS).
- Competence to manage patients presenting either acutely or electively with a range of symptoms and conditions as specified in the syllabus.
- Competence to manage an additional range of elective and emergency conditions by virtue of appropriate training and assessment opportunities obtained during training as specified by sub-specialty components of the final stage syllabus. This is tested either by the FRCS and/or by WPBA.
- Professional competences as specified by GMP and GMC.

Intermediate and Final Years of Specialty Training

- To provide higher specialty training in the specialty with attainment of knowledge, skills and professional behaviours relevant to the practice in the specialty. This is defined within the specialty specific component of the early years syllabus and the intermediate and final stages of the syllabus (same as FRCS).
- Competence to manage patients presenting either acutely or electively with a range of symptoms and conditions as specified in the syllabus.
- Competence to manage an additional range of elective and emergency conditions by virtue of appropriate training and assessment opportunities obtained during training as specified by sub-specialty components of the final stage syllabus. This is tested either by the FRCS and/or by WPBA.
- Professional competences as specified by GMP and GMC.

Aortic Aneurysm Disease - Vascular Surgery Curriculum

- Diagnosis and management including operative management of abdominal and peripheral aortic aneurysms. Have knowledge of both open and endovascular repair of aortic aneurysms.
- Recognise and know the principles of treatment of patients with ruptured abdominal aortic aneurysms.
- Aortic Aneurysm Index Procedures;
 - Elective open repair tube graft.
 - Elective open repair bifurcated graft.
 - Endovascular repair.
 - Ruptured aneurysm repair.

Vascular Surgery Curriculum GMC August 2013

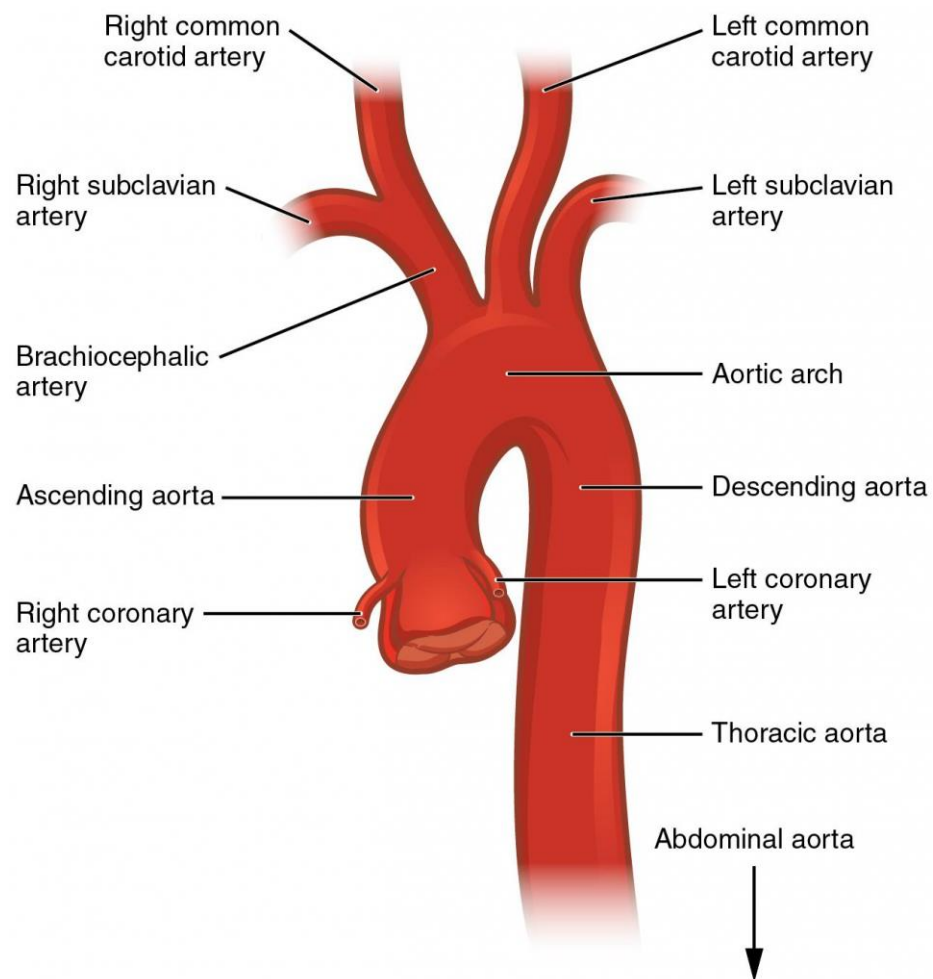
Learning Outcomes

- Background – Why treat?
- Therapeutic Strategies and Treatment Options.
- Operative Technique.
- Clinical Outcomes.
- Current Research.

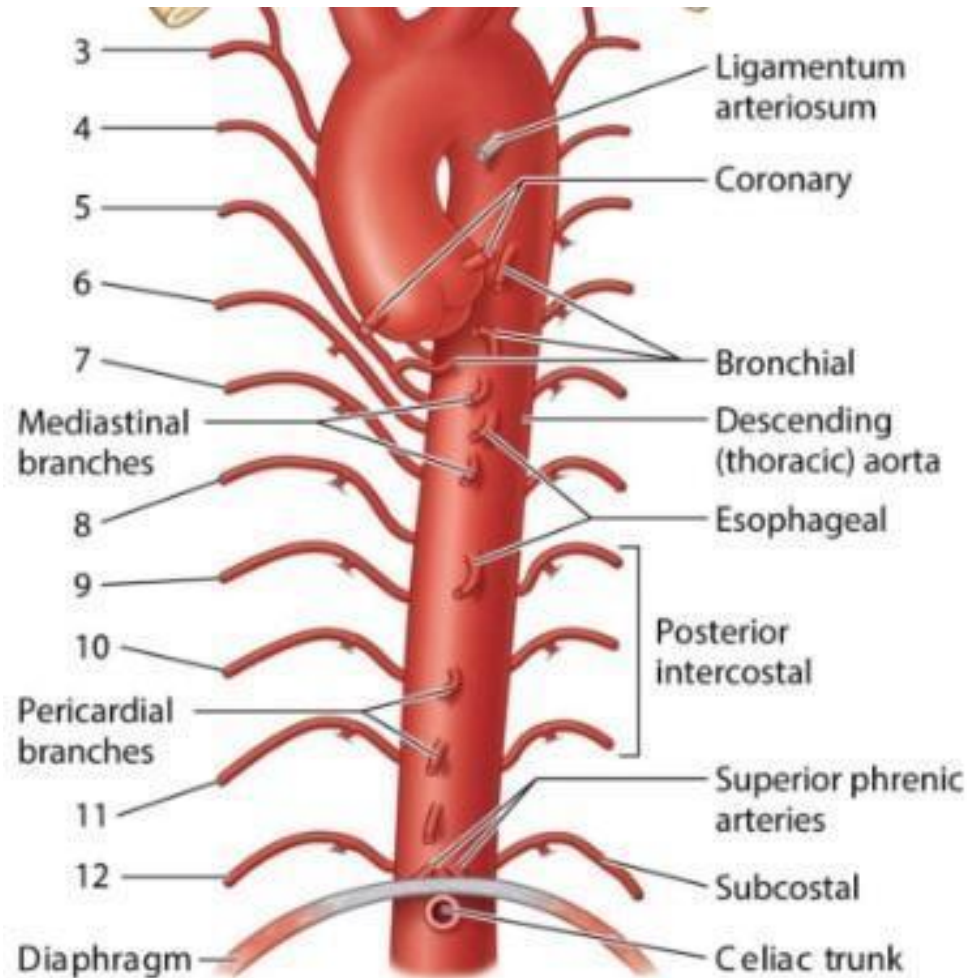
Vascular Surgery Curriculum – Generic Topics

- Anatomy of aorta and adjacent structures.
- Physiology of haemodynamic and haematological indices.
- Epidemiology of aortic aneurysms
- Pathology of aneurysmal degeneration.
- Principles of best medical therapy.
- Radiological investigation.
- Aortic screening programs.
- Open and endovascular techniques for repair of AAA.
- Role of stent / graft surveillance.

Anatomy – Aortic Arch

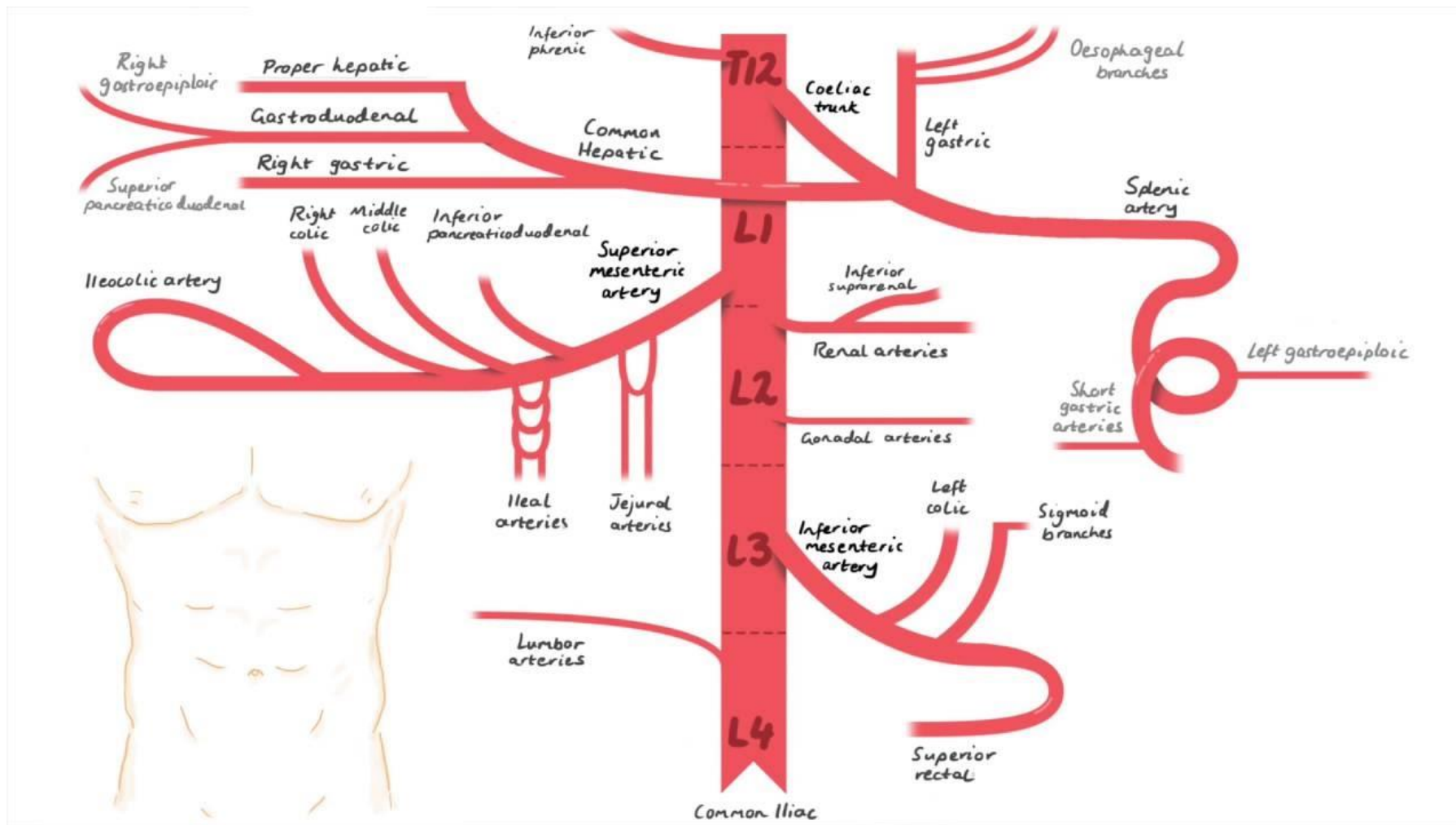


Anatomy – Thoracic Aorta



Anatomy – Abdominal Aorta

Anatomy – Abdominal Aorta

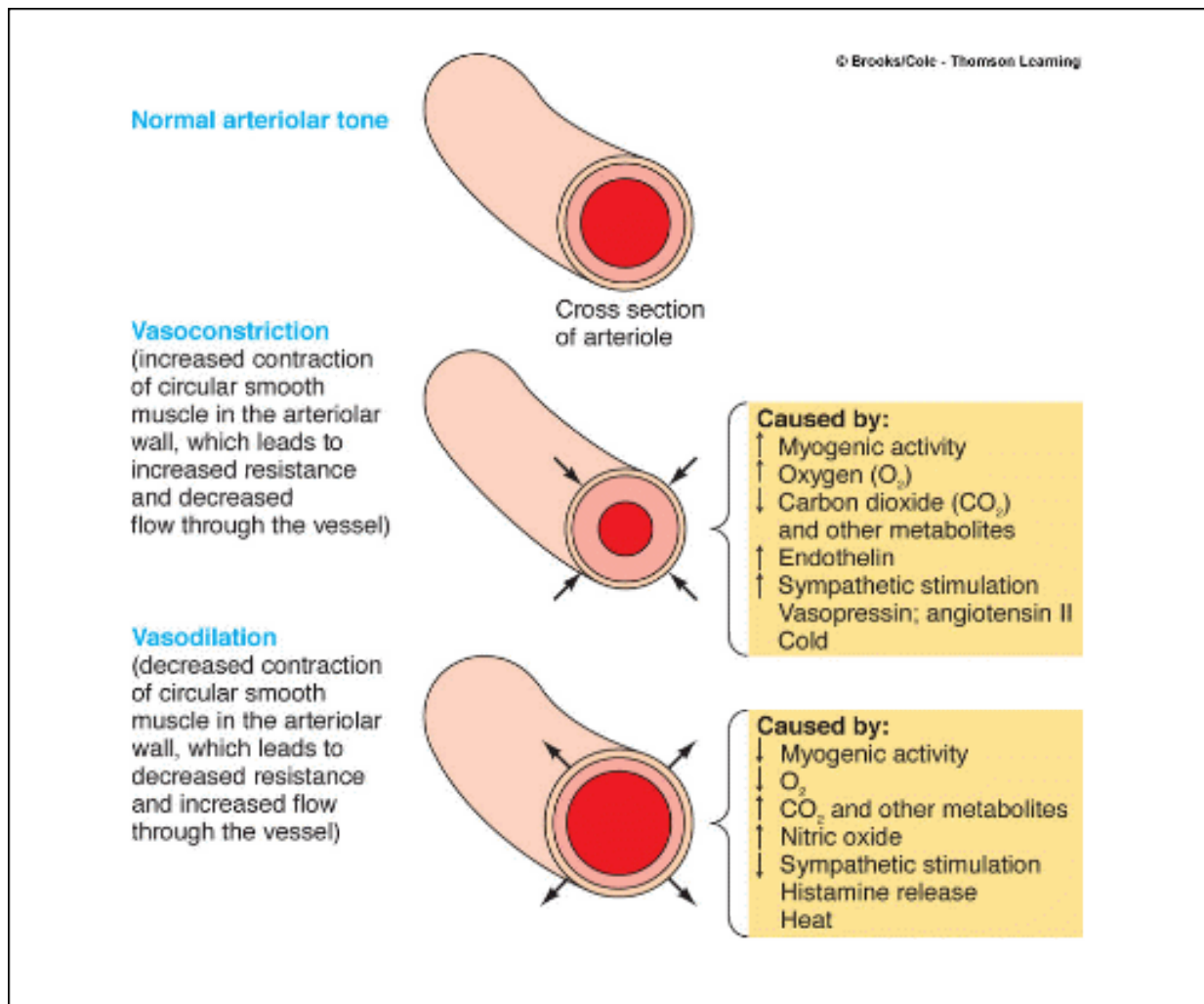


Physiology

Basic Definitions

- **Systolic Pressure:**
 - Measurement of blood pressure when the ventricles contract i.e. the maximum blood pressure in the aorta.
 - Usually around 120mmHg.
- **Diastolic Pressure:**
 - measurement of blood pressure when the ventricles relax i.e. the minimum blood pressure in the aorta.
 - Usually around 80mmHg.
- **Pulse Pressure:**
 - Systolic blood pressure – diastolic blood pressure.
 - Usually $120 - 80 = 40\text{mmHg}$
- **Mean Blood Pressure:**
 - Diastolic blood pressure – $1/3$ pulse pressure.
 - Usually $80 + 13 = 93\text{mmHg}$.
- Mean arterial blood pressure = cardiac output x total peripheral resistance.

Arteriolar Tone Regulation



Poiseuille's Law

Poiseuille's Law

- Describes the relationship between the diameter of the vessel and overall flow where flow increases to the power of 4 as the radius increases.
- When applied to arterial vessel or IV cannula, it states that the flow (Q) of fluid is related to a number of factors: the viscosity (η) of the fluid, the pressure gradient across the tubing (P), and the length (L) and diameter(r) of the tubing.

Q	Flow rate
P	Pressure
r	Radius
η	Fluid viscosity
l	Length of tubing

$$Q = \frac{\pi P r^4}{8 \eta l}$$

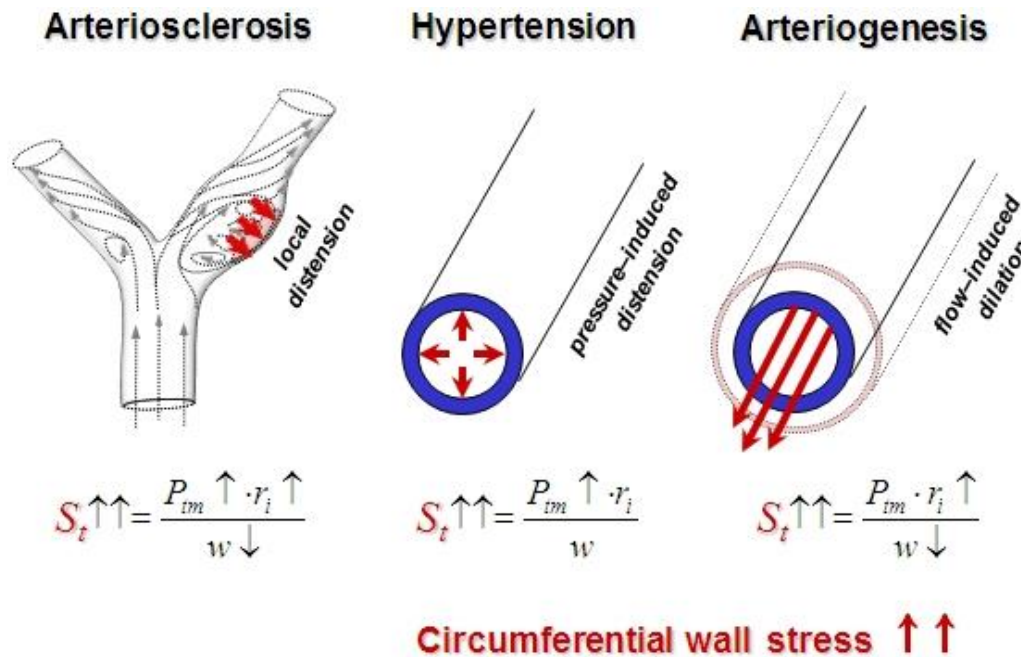
Law of LaPlace

Law of LaPlace

- Describes the pressure-volume relationships of spheres in both the heart and alveoli.
 - $\text{Pressure} = (2 \times \text{Thickness} \times \text{Tension}) / \text{Radius}.$
- This shows how thicker walled vessels usually contain higher pressures.
- If pressure increases, the vessel must react by increasing wall thickness or reducing diameter (the latter will increase resistance).
- An exception is atherosclerosis where the wall thickening has nothing to do with wall stress but simply affects blood flow.

Law of LaPlace

- Describes the pressure-volume relationships of spheres in both the heart and alveoli.



S_t =circumferential wall stress; P_{tm} =transmural pressure difference, r_i =inner radius, w =wall thickness

Definitions

- Arteriomegaly
- Ectasia
- Aneurysm

Definitions

- Arteriomegaly - Diffuse enlargement of an artery but not enough to meet criteria for an aneurysm.
- Ectasia - Diffuse dilatation of an artery with increase in diameter < 50% - Aorta 2cm to 3cm.
- Aneurysm - Increase in diameter of 50% (>1.5x) its normal diameter – Aorta > 3cm.

Aetiology

- Aortic aneurysmal disease is a degenerative process associated with;

Aetiology

- Aortic aneurysmal disease is a **degenerative** process associated with;
 - **Atherosclerosis** – may play a role in the development of degenerative aneurysms but not all degenerative aneurysms are found in patients with atherosclerosis.
 - Dissection.
 - Connective Tissue Disease – Marfan Syndrome, Ehlers-Danlos Syndrome, Loeys-Dietz Syndrome.
 - Infective: Syphilis or distal dissemination - mycotic .
 - Cystic Medial Necrosis.

Aetiology

- Aortic aneurysmal disease is a degenerative process associated with;
 - Atherosclerosis – may play a role in the development of degenerative aneurysms but not all degenerative aneurysms are found in patients with atherosclerosis.
 - Dissection.
 - Connective Tissue Disease – Marfan Syndrome, Ehlers-Danlos Syndrome, Loeys-Dietz Syndrome.
 - Infective: Syphilis or distal dissemination - mycotic .
 - Cystic Medial Necrosis.

- Pseudoaneurysm

Aetiology

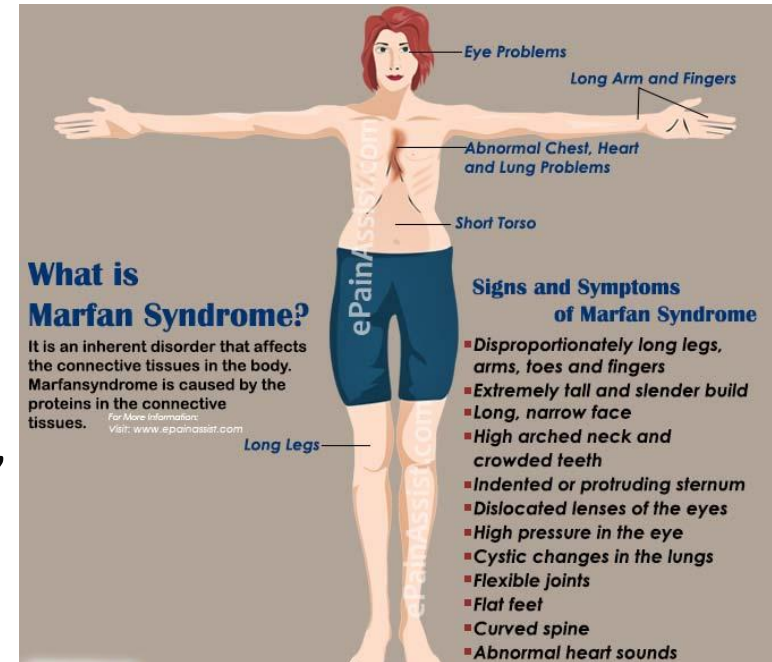
- Aortic aneurysmal disease is a degenerative process associated with;
 - Atherosclerosis – may play a role in the development of degenerative aneurysms but not all degenerative aneurysms are found in patients with atherosclerosis.
 - Dissection.
 - Connective Tissue Disease – Marfan Syndrome, Ehlers-Danlos Syndrome, Loeys-Dietz Syndrome.
 - Infective: Syphilis or distal dissemination - mycotic .
 - Cystic Medial Necrosis.

- Pseudoaneurysm – pulsatile haematoma.

Marfan Syndrome

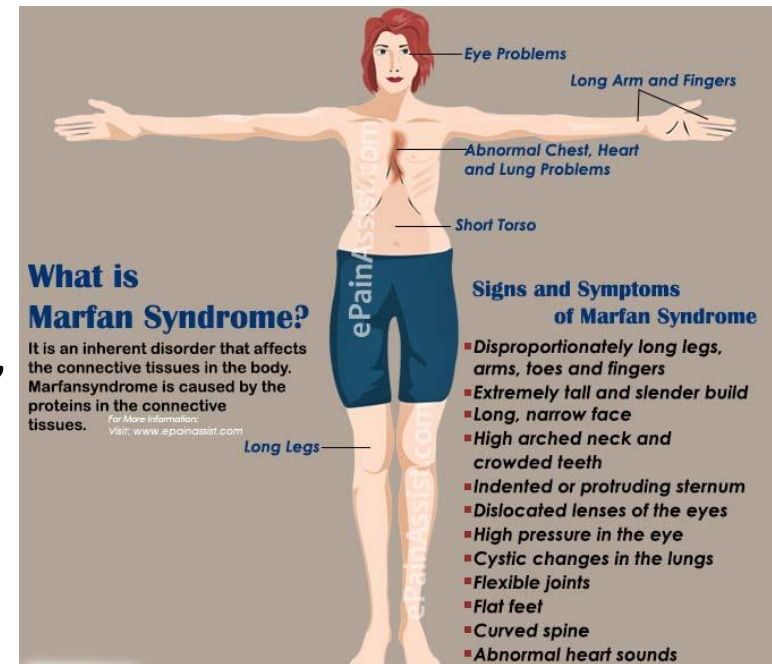
Marfan Syndrome

- Genetic disorder, occurring in about 1 in 5000, that affects the body's connective tissue due to a heterozygous defect (or mutation) in gene encoding for the extracellular matrix protein fibrillin-1.
- This leads to an increase in transforming growth factor beta (TGF- β) most often affecting the heart, blood vessels, bones, joints, and eyes.



Marfan Syndrome

- Genetic disorder, occurring in about 1 in 5000, that affects the body's connective tissue due to a heterozygous defect (or mutation) in gene encoding for the extracellular matrix protein fibrillin-1.
- This leads to an increase in transforming growth factor beta (TGF- β) most often affecting the heart, blood vessels, bones, joints, and eyes.
- Marfan syndrome does not affect intelligence.



Loeys-Dietz Syndrome

Loeys-Dietz Syndrome

- Rare autosomal dominant genetic disorder of the body's connective tissue with some features in common with Marfan syndrome.
- Loeys-Dietz syndrome (type 1-5) is caused by a genetic mutation in one of five genes that encode for the receptors and other molecules in the transforming growth factor-beta (TGF- β) pathway.
 - LDS-1- transforming growth factor beta-receptor 1 (TGF β R1).
 - LDS-2 - transforming growth factor beta-receptor 2 (TGF β R2).
 - LDS-3 - mothers against decapentaplegic homolog (SMAD-3).
 - LDS-4 - transforming growth factor beta-2 ligand (TGF β 2).
 - LDS-5- transforming growth factor beta-3 ligand (TGF β 3).

Types of Collagen

Type	Molecular Formula	Native Polymer	Tissue Distribution
I	$[\alpha 1(I)]_2\alpha 2$	fibril (67 nm)	skin, tendon, bone, dentin, fascia (high tensile strength, 90% of total body collagen)
II	$[\alpha 1(II)]_3$	fibril (67 nm)	cartilage, nucleus pulposus, notochord, vitreous body, cornea
III	$[\alpha 1(III)]_3$	fibril (67 nm)	skin, uterus, blood vessels, reticular fibers, spleen, lymph nodes
IV	$[\alpha 1(IV)]_3$	non-fibrillar (globular)	basal lamina of epithelial cells, kidney glomeruli, lens capsule, etc.
VII	$[\alpha 1(VII)]_3$	small fibers (67 nm)	anchoring fibrils in basal laminae

Ehlers-Danlos syndrome

Name of EDS Subtype	IP*	Genetic Basis	Protein Involved
Classical EDS (cEDS)	AD	Major: <i>COL5A1</i> , <i>COL5A2</i>	Type V collagen
		Rare: <i>COL1A1</i> c.934C>T, p.(Arg312Cys)	Type I collagen
Classical-like EDS (clEDS)	AR	<i>TNXB</i>	Tenascin XB
Cardiac-valvular EDS (cvEDS)	AR	<i>COL1A2</i> (biallelic mutations that lead to <i>COL1A2</i> NMD and absence of pro $\alpha 2(I)$ collagen chains)	Type I collagen
Vascular EDS (vEDS)	AD	Major: <i>COL3A1</i>	Type III collagen
		Rare: <i>COL1A1</i> c.934C>T, p.(Arg312Cys) c.1720C>T, p.(Arg574Cys) c.3227C>T, p.(Arg1093Cys)	Type I collagen
Hypermobile EDS (hEDS)	AD	Unknown	Unknown
Arthrochalasia EDS (aEDS)	AD	<i>COL1A1</i> , <i>COL1A2</i>	Type I collagen
Dermatosparaxis EDS (dEDS)	AR	<i>ADAMTS2</i>	ADAMTS-2
Kyphoscoliotic EDS (kEDS)	AR	<i>PLOD1</i>	LH1
		<i>FKBP14</i>	FKBP22
Brittle cornea syndrome (BCS)	AR	<i>ZNF469</i>	ZNF469
		<i>PRDM5</i>	PRDM5
Spondylodysplastic EDS (spEDS)	AR	<i>B4GALT7</i>	$\beta 4$ GalT7
		<i>B3GALT6</i>	$\beta 3$ GalT6
		<i>SLC39A13</i>	ZIP13
Musculocontractural EDS (mcEDS)	AR	<i>CHST14</i>	D4ST1
		<i>DSE</i>	DSE
Myopathic EDS (mEDS)	AD or AR	<i>COL12A1</i>	Type XII collagen
Periodontal EDS (pEDS)	AD	<i>C1R</i>	C1r

Ehlers-Danlos syndrome

Name of EDS Subtype	IP*	Genetic Basis	Protein Involved
Classical EDS (cEDS)	AD	Major: <i>COL5A1</i> , <i>COL5A2</i>	Type V collagen
		Rare: <i>COL1A1</i> c.934C>T, p.(Arg312Cys)	Type I collagen
Classical-like EDS (clEDS)	AR	<i>TNXB</i>	Tenascin XB
Cardiac-valvular EDS (cvEDS)	AR	<i>COL1A2</i> (biallelic mutations that lead to <i>COL1A2</i> NMD and absence of pro $\alpha 2(I)$ collagen chains)	Type I collagen
Vascular EDS (vEDS)	AD	Major: <i>COL3A1</i>	Type III collagen
		Rare: <i>COL1A1</i> c.934C>T, p.(Arg312Cys) c.1720C>T, p.(Arg574Cys) c.3227C>T, p.(Arg1093Cys)	Type I collagen
Hypermobile EDS (hEDS)	AD	Unknown	Unknown
Arthrochalasia EDS (aEDS)	AD	<i>COL1A1</i> , <i>COL1A2</i>	Type I collagen
Dermatosparaxis EDS (dEDS)	AR	<i>ADAMTS2</i>	ADAMTS-2
Kyphoscoliotic EDS (kEDS)	AR	<i>PLOD1</i>	LH1
		<i>FKBP14</i>	FKBP22
Brittle cornea syndrome (BCS)	AR	<i>ZNF469</i>	ZNF469
		<i>PRDM5</i>	PRDM5
Spondylodysplastic EDS (spEDS)	AR	<i>B4GALT7</i>	$\beta 4$ GalT7
		<i>B3GALT6</i>	$\beta 3$ GalT6
		<i>SLC39A13</i>	ZIP13
Musculocontractural EDS (mcEDS)	AR	<i>CHST14</i>	D4ST1
		<i>DSE</i>	DSE
Myopathic EDS (mEDS)	AD or AR	<i>COL12A1</i>	Type XII collagen
Periodontal EDS (pEDS)	AD	<i>C1R</i>	C1r

AAA Epidemiology

- 30-60 cases per 1000.
- Increasing incidence over past 3 decades.
- 7-8% of patients > 65 years of age.

Incidence of AAA

Autopsy	1.5-3.0%
Ultrasound Screening	3.2%
Patients with coronary artery disease	5.0%
Patients with peripheral arterial disease	10.0%
Patients with femoral / popliteal aneurysms	50.0%

AAA Epidemiology

- 30-60 cases per 1000.
- Increasing incidence over past 3 decades.
- 7-8% of patients > 65 years of age.

Incidence of AAA

Autopsy	1.5-3.0%
Ultrasound Screening	3.2%
Patients with coronary artery disease	5.0%
Patients with peripheral arterial disease	10.0%
Patients with femoral / popliteal aneurysms	50.0%

Approximately 10% of patients with AAA will have a popliteal aneurysm

AAA Risk Factors

- Forsdahl *et al* (2009) completed a seven year ultrasonic follow-up of 4345 patients (m=2035) between ages of 25-82 to identify AAA risk factors which confirmed association between male gender, increased age and smoking history.
- The following variables were significantly associated with increased abdominal aortic aneurysm incidence;
 - Smoking (OR=13.72, 95% CI 6.12 to 30.78, comparing current smokers of ≥ 20 cigarettes/d with never-smokers).
 - Hypertension (OR=1.54, 95% CI 1.03 to 2.30).
 - Hypercholesterolemia (OR=2.11, 95% CI 1.23 to 3.64)
 - In addition, use of statins was associated with increased risk of abdominal aortic aneurysm (OR=3.77, 95% CI 1.45 to 9.81) but this was probably a marker of high risk of cardiovascular diseases.

Forsdahl Circulation 2009; 119: 2202-08

AAA Risk Factors

- Forsdahl *et al* (2009) completed a seven year ultrasonic follow-up of 4345 patients (m=2035) between ages of 25-82 to identify AAA risk factors which confirmed association between male gender, increased age and smoking history.
- The following variables were significantly associated with increased abdominal aortic aneurysm incidence;
 - **Smoking (OR=13.72, 95% CI 6.12 to 30.78**, comparing current smokers of ≥ 20 cigarettes/d with never-smokers).
 - Hypertension (OR=1.54, 95% CI 1.03 to 2.30).
 - Hypercholesterolemia (OR=2.11, 95% CI 1.23 to 3.64)
- In addition, use of statins was associated with increased risk of abdominal aortic aneurysm (OR=3.77, 95% CI 1.45 to 9.81) but this was probably a marker of high risk of cardiovascular diseases.

Forsdahl Circulation 2009; 119: 2202-08

Matrix Metalloproteinases

- Family of digestive enzymes implicated in aneurysmal development.
- Increased levels of MMPs expression and activity have been demonstrated within the aortic wall of AAA, associating with histological alterations.
- An imbalance between MMPs and their inhibitors may tip the equilibrium towards matrix degradation.
 - For TAA, it is most notably MMP-1, -9, -12, and -14 and MMP-2 when a bicuspid aortic valve is present.
 - For AAA, it is MMP-1, -2, -3, -9, -12, and -13.

AAA Pathophysiology

- Elastin degradation due to matrix metalloproteinases (2, 9 and 12) in the aortic media;
 - Increase in the collagenase and elastase activity.
 - Decrease in collagen and elastin in arterial wall.
 - Elastin becomes fragmented leading to arterial elongation and dilatation.

- Law of Laplace - Luminal dilation results in increased wall tension and a cycle of progressive dilation and increased tension.

Clinical Presentation

- Asymptomatic – 75%.

- Symptomatic – 25%:
 - Pain.
 - Collapse.
 - Pain / Fever / Weight loss and raised inflammatory markers suggests an inflammatory aneurysm (up to 10%).

Clinical Presentation

- Asymptomatic

- Symptomatic

- Pain
- Collapse
- Pain +/- flank or testicular pain

**Beware the elderly
medical patient with any
evidence of possible
collapse +/- flank or
testicular pain**

suggests

Radiological Investigation

Plain X-ray

Referral to:	Royal Victoria Hospital	Referral Type:	Out Patient
Specialty:	VASCULAR SURGERY		
Urgency:	URGENT		
Urgency Reason:	no priority reason given		
Electronic Attachment Present?:	Yes		
HCP Name:			
Designation:			

Reason for Referral/ History of Presenting Complaint

Description: AAA

Comment: Complaining of low back pain, XR shows degenerative lumbar changes but also aortic aneurysm, with recommendation from Radiologist; 'Aneurysmal dilatation noted of a calcified infrarenal aorta with a max AP diameter of 4.7 cm. Formal vascular assessment advised' Copy report is attached. Thank you.



Ultrasound – Abdominal Aorta

- Longitudinal images (along the long axis of the vessel):
 - Proximal (below diaphragm, near the celiac artery).
 - Mid (near the level of the renal arteries).
 - Distal (above the iliac bifurcation).
- Transverse images (perpendicular to the long axis of the vessel):
 - Proximal (below diaphragm near the celiac artery);
 - Mid (near the level of the renal arteries);
 - Distal (above the iliac bifurcation).
- Measurements:
 - Anteroposterior measurements of the aorta sufficient to determine if an aortic aneurysm exists.
 - If an aneurysm is present, its greatest dimension should be reported.
 - However, if no aneurysm is identified, the largest diameter of the abdominal aorta should be reported.

Ultrasound – Abdominal Aorta

- Longitudinal images (along the long axis of the vessel):

**FOLLOW-UP SCANS MAY INCLUDE ILIAC
DIMENSIONS**

- T

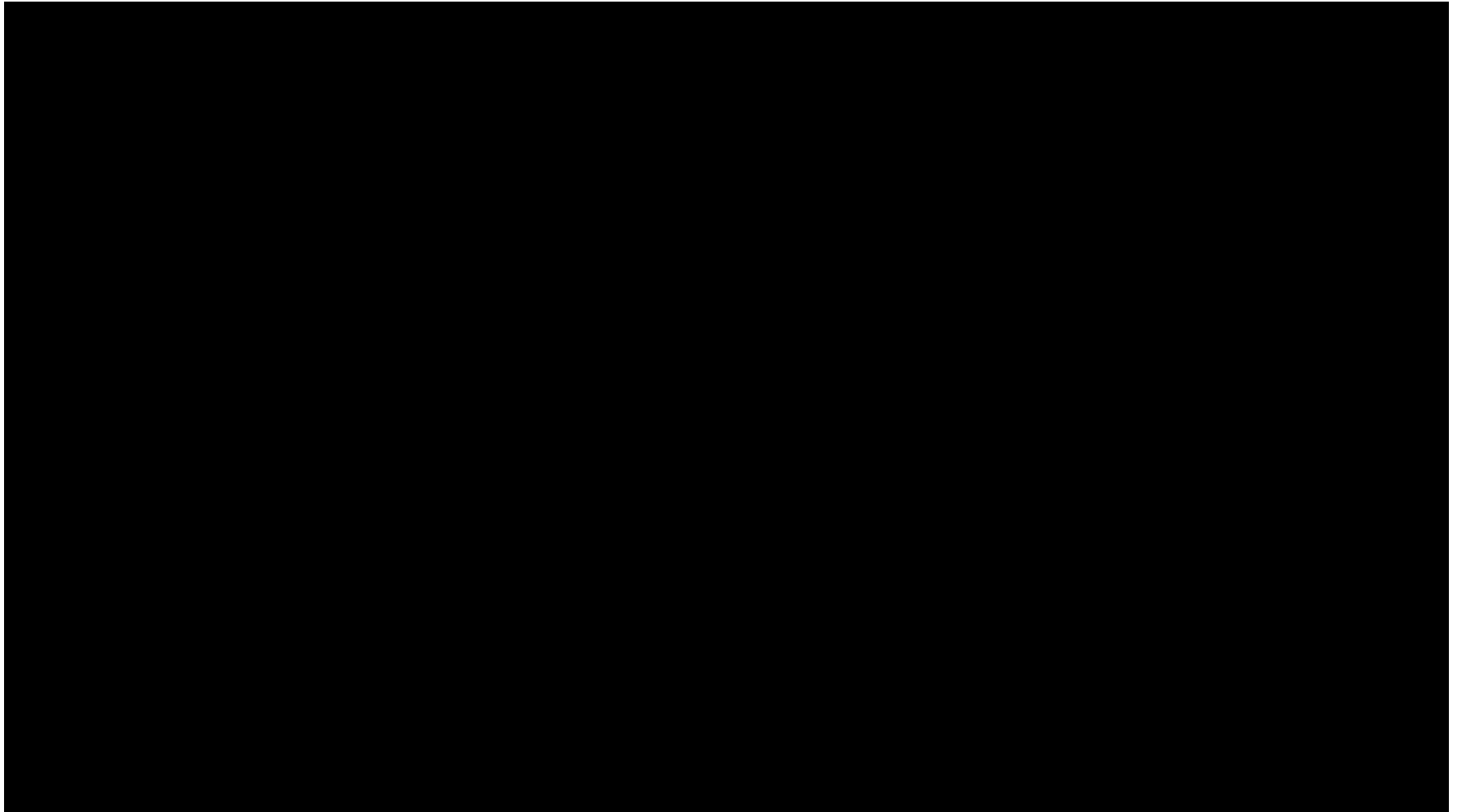
LENGTH IS NEVER REQUIRED

- M **BEWARE OF FAST SCANS FROM THE ED**

...determine
if an aortic aneurysm exists.

- If an aneurysm is present, its greatest dimension should be reported.
- However, if no aneurysm is identified, the largest diameter of the abdominal aorta should be reported.

Ultrasound – Abdominal Aorta

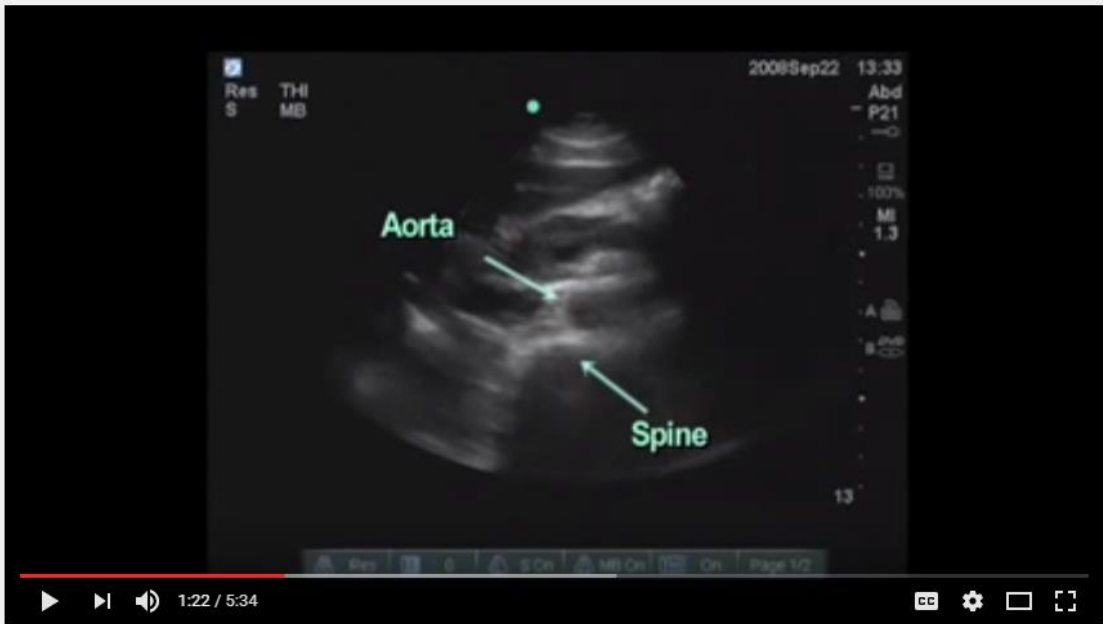


Ultrasound – Abdominal Aorta

Secure | <https://www.youtube.com/watch?v=AqAHzGijNlo>

Apps Diabetes and vascular ResearchGate LinkedIn Mark O'Donnell Vascu Professor Mark O'Donnell

YouTube GB Search



How to Perform Abdominal Aorta Ultrasound Exam - SonoSite Ultrasound

<https://www.youtube.com/watch?v=AqAHzGijNlo>

Ultrasound – Follow-Up Imaging Protocols



- AAAs <4.0 cm require USS every 2 to 3 years.
- In patients with small aneurysms, monitor infra-/juxtarenal AAAs measuring 4.0 to 5.4 cm in diameter with ultrasonography (USS)/CT every 6 to 12 months.
- Consider expansion rates, as some advocate expansion of 4 to 8 mm over 12 months suggests instability

<http://bestpractice.bmj.com/best-practice/monograph/145/follow-up/recommendations.html> 4/5/17

Ultrasound – Follow-Up Imaging Protocols



**FIRST AAA DIAGNOSIS – REPEAT US IN
SIX MONTHS TO EVALUATE GROWTH
POTENTIAL**

**3CM – 4.4CM = ANNUAL US
4.5CM – 4.9CM = 6 MONTH US
5.0CM – 5.4CM = 3 MONTH US
>5.5CM = CTA 2 WEEKS**

<http://>

<http://> 4/5/17

UK AAA Screening program

- Commenced in ?
- Who is Screened ?
- Who is not Screened ?

UK AAA Screening program

- Commenced in 2009.
- Who is Screened ?
 - 65 year old men.
- Who is not Screened ?
 - Women
 - Men under 65
 - People who've already been treated for an AAA
- You can ask for a scan to check for an AAA if you think you might need one but haven't been offered a screening test.

UK AAA Screening Outcomes

- Normal:
 - More than 98% of men screened have a normal result.
 - If your result is normal, you won't need any further scans or treatment because an AAA grows slowly and the chances of you developing one after 65 are very small.

UK AAA Screening Outcomes

- Small AAA:
 - If you have a small AAA, this means your aorta measures 3cm to 4.4cm across.
 - Just over 1% of men screened have a small AAA.
 - You'll also be given advice on how you can stop an AAA getting bigger, such as stopping smoking, eating healthily and exercising regularly.

UK AAA Screening Outcomes

- Medium AAA:
 - If you have a medium AAA, this means your aorta measures 4.5cm to 5.4cm across.
 - About 0.5% of men screened have a medium AAA.
 - You won't need any treatment at this stage as the chance of the AAA bursting is small.
 - You'll be invited back for a scan every three months to check its size. Treatment will usually only be needed if it becomes a large AAA.
 - You'll also be given advice on how you can stop an AAA getting bigger, such as stopping smoking, eating healthily and exercising regularly.

UK AAA Screening Outcomes

- Large AAA:
 - If you have a large AAA, this means your aorta measures 5.5cm or more across.
 - About 0.1% of men screened have a large AAA.
 - As large AAAs are at the highest risk of bursting if left untreated, you'll be referred to a specialist surgeon within two weeks to talk about your treatment options.
 - Most men with a large AAA are advised to have surgery to stop it getting bigger or bursting.
 - While surgery carries a risk of serious complications, this is generally smaller than the risk of not treating a large AAA.

Why Treat ??

Why Treat ??

- Risk of AAA rupture;
 - Below 5cm <2%
 - 5cm to 5.9cm 5%
 - 6cm to 6.9cm 6.6%
 - 7cm to 7.9cm 20%
 - Greater than 8cm 30-50%

Why Treat ??

- Risk of AAA rupture;

■ Below 5cm	<2%
■ 5cm to 5.9cm	5%
■ 6cm to 6.9cm	6.6%
■ 7cm to 7.9cm	20%
■ Greater than 8cm	30-50%

- UK Small Aneurysm Trial;
 - Multicentre RCT across 93 UK hospitals.
 - 1276 patients between 60-76 with AAA between 4.0 and 5.5cm.
 - Safe to monitor AAA up to 5.5cm unless tender or growth rates >1cm per year.

Lancet 1998; 352: 1649-55

Aneurysm Treatment Thresholds ?

Aneurysm Thresholds

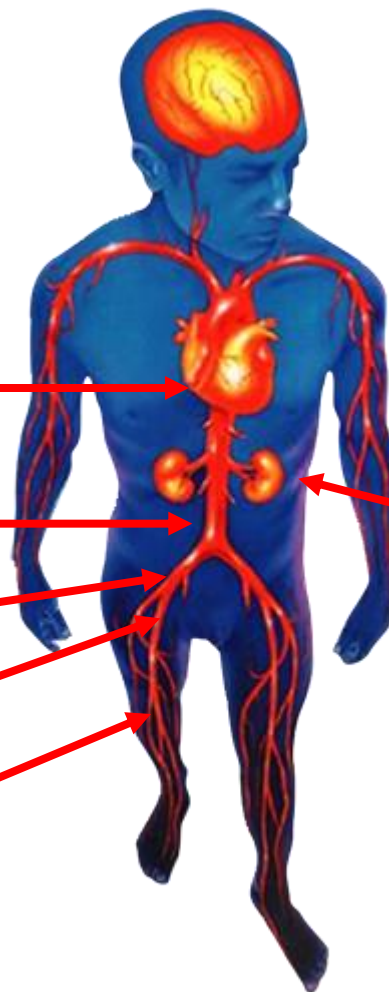
Thoracic Aorta 6cm

Abdominal Aorta 5cm

Iliac 4cm

Femoral 3cm

Popliteal 2cm



2cm Visceral Aneurysms
Splenic
Hepatic
Renal

What's Next ?

CT Angiography



What are you going to consider next?

Medical Optimisation

- Risk Factor Modification;
 - Smoking cessation.
 - Blood pressure.
 - Fasting glucose and lipids.

Medical Optimisation

- Risk Factor Modification;
 - Smoking cessation.
 - Blood pressure.
 - Fasting glucose and lipids.
 - **General health advice and exercise.**

Medical Optimisation

- Risk Factor Modification;
 - Smoking cessation.
 - Blood pressure.
 - Fasting glucose and lipids.

- Best Medical Therapy;
 - Antiplatelets.
 - Lipid lowering medication.

Why prescribe aspirin?

Why prescribe aspirin?

- Antithrombotic Trialists' Collaboration reported that antiplatelet therapy was associated with a 23% reduction in non-fatal MI, non-fatal stroke and vascular death in patients with PAD.
- The clopidogrel versus aspirin in patients at high risk of ischaemic events trial (CAPRIE) showed that clopidogrel reduced the relative risk of major vascular events by 8.7% compared to aspirin.

Antithrombotic Trialists' Collaboration BMJ 2002; 324: 71-86

Why prescribe statins?

Why prescribe statins?

- Medical Research Council (MRC) recommended statin therapy for all patients with PAD where simvastatin 40mg daily resulted in a 22% relative risk reduction in rates of MI, stroke and revascularisation in patients with PAD with a cholesterol level $>3.5\text{mmol/L}$.
- Walking performance improvements.
- Potential reduction in plaque volume.

Heart Protection Study Lancet 2002; 360: 7-22

Pre-Operative Work-Up

- Out-patient clinic visit;
 - End-of-bed-o-gram.
 - Systemic evaluation.
 - Past medical history.

Pre-Operative Work-Up

- Out-patient clinic visit;
 - End-of-bed-o-gram.
 - Systemic evaluation.
 - Past medical history.
 - **How far can you walk ?**

Pre-Operative Work-Up

- Out-patient clinic visit;
 - End-of-bed-o-gram.
 - Systemic evaluation.
 - Past medical history.
 - How far can you walk ?
- Anaesthetic Assessment;
 - Cardiorespiratory function – ECHO / PFT's'.
 - Optimisation – medical / procedural – PCI.
- Role of CPEX testing;
 - There is evidence that CPET can be used for risk stratification.
 - Survival benefit of intervention, critical care length of stay, likelihood of complications, pre-intervention optimisation.
 - Combined with co-morbidity scoring may identify patients unlikely to survive.

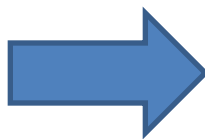
AAA Procedural Consent

- General Local Complications;
 - Pain, Bruising, Bleeding, Wound infection.
- Systemic Complications;
 - Cardiovascular, Respiratory, Thromboembolic.
 - Renal.
- Procedural Specific Complications;
 - Graft sepsis.
 - Graft occlusion and distal ischaemia.
 - Intra-abdominal injury and adhesions .
 - Bowel ischaemia.
 - Post-operative hernia.

What are your patient's options?

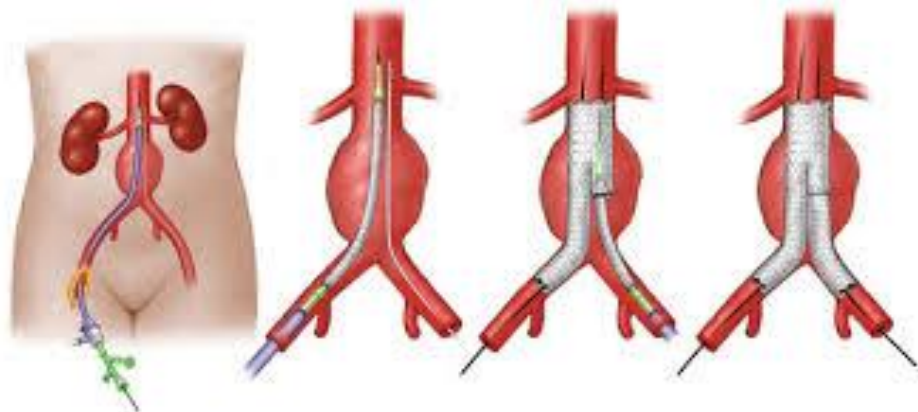
Open AAA Repair

- Replace diseased aorta with artificial artery.
- Requires 7 day hospital stay.
- Recovery time 3-6 months.
- Proven method with good long term results.



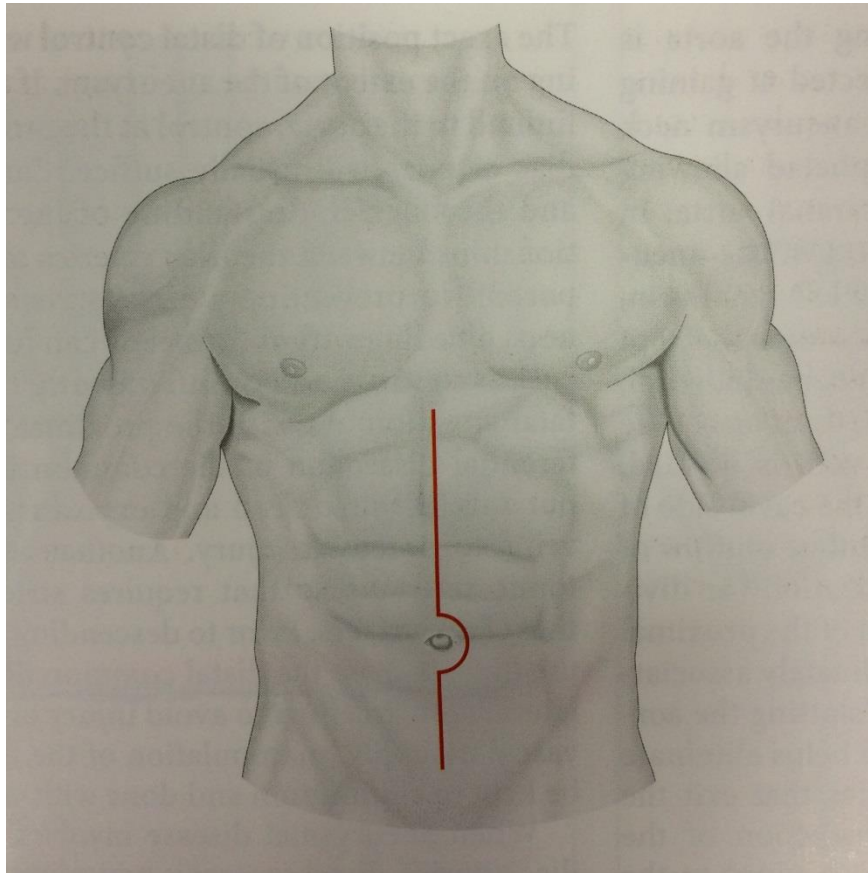
Endovascular Repair

- Repair through an incision in the groin with expandable prosthesis under fluoroscopic guidance
- Requires both surgical and radiological assistance
- Significantly reduced morbidity.
- Long term result unknown
- Hospital stay 2 days, Recovery time 1-2 weeks

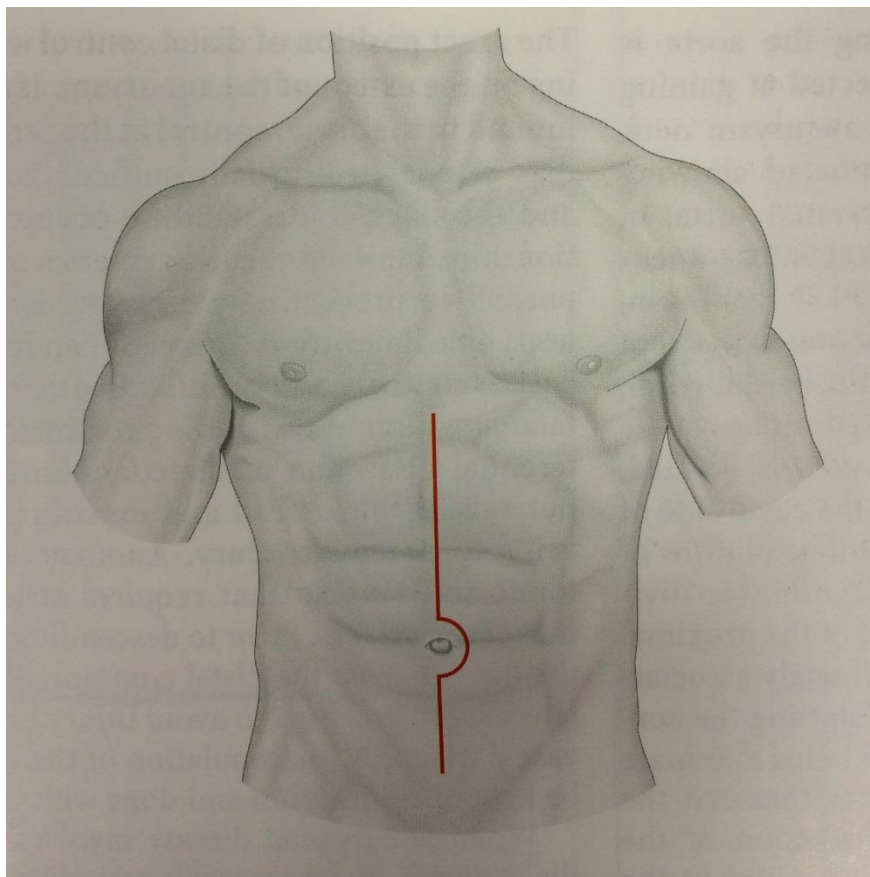


How to perform open AAA repair

1 - Patient Positioning

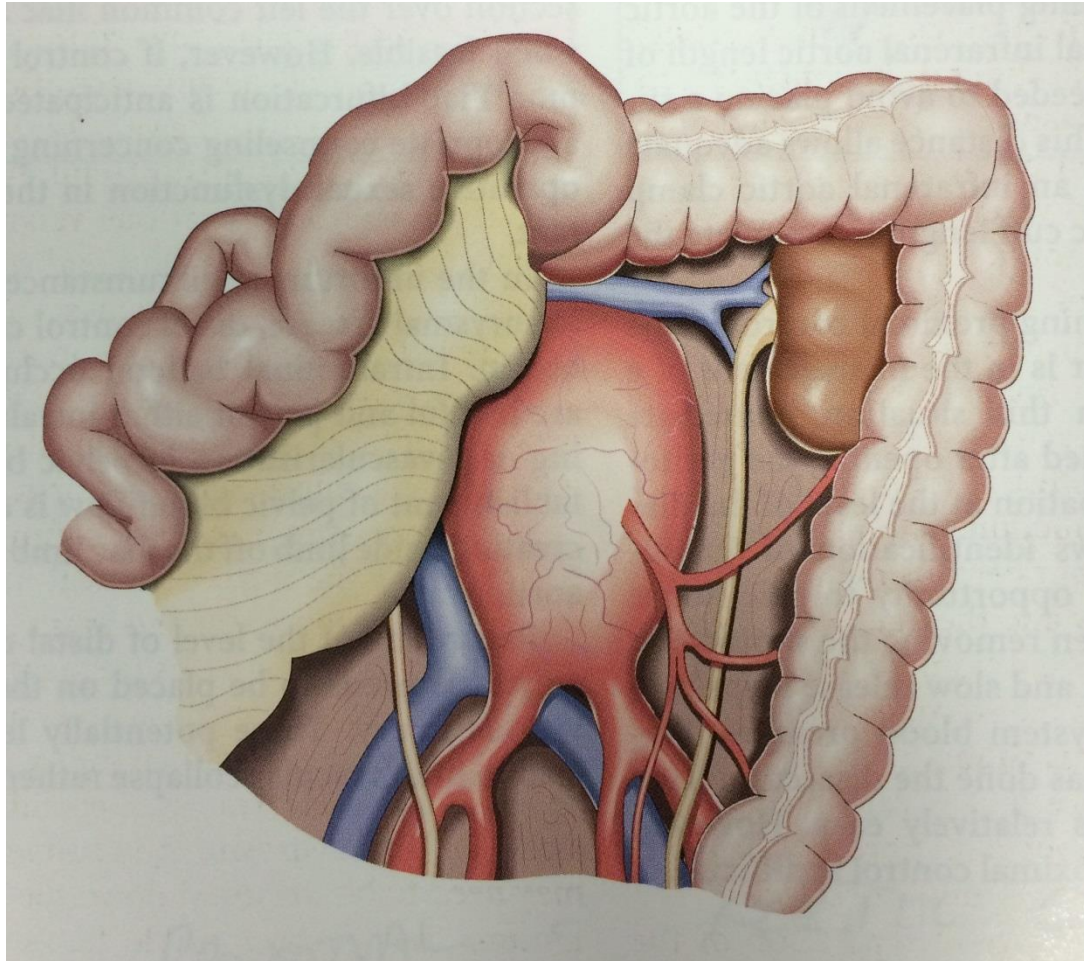


1 - Patient Positioning

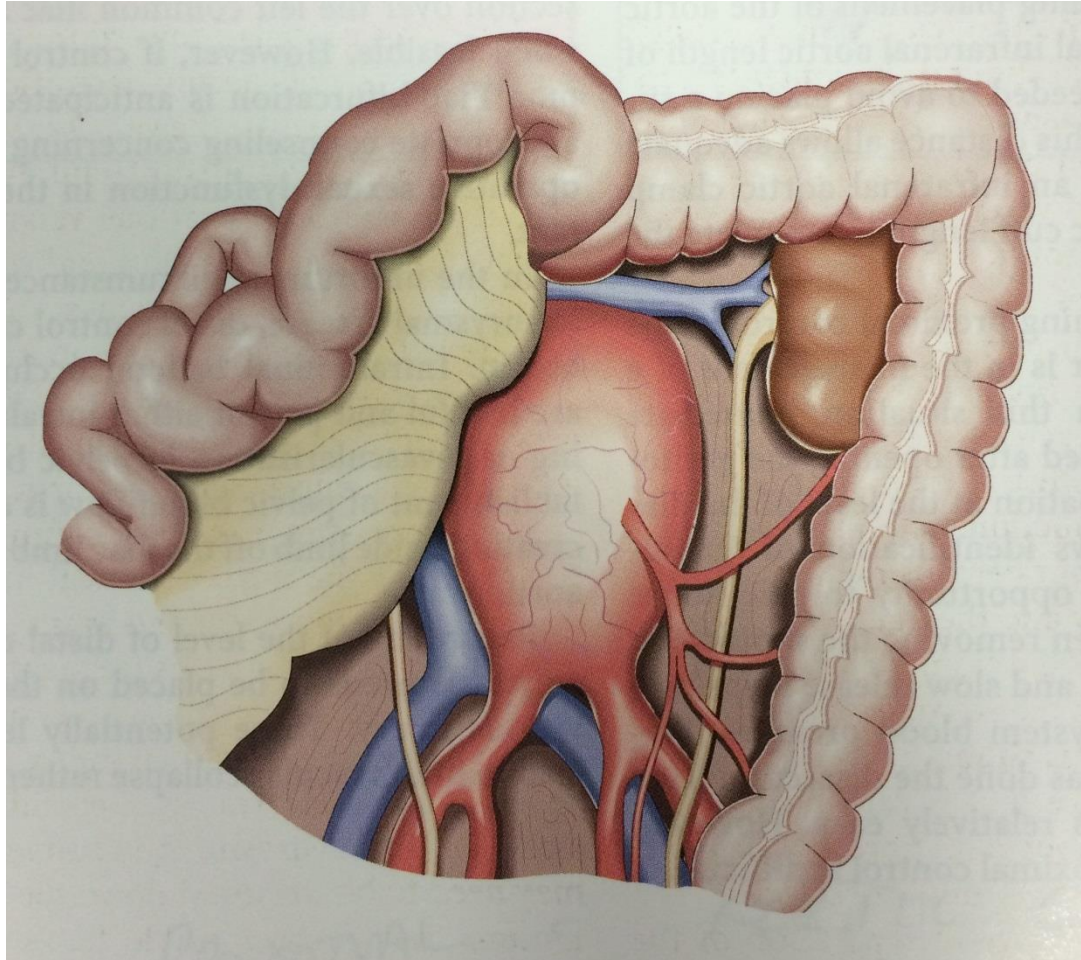


- Clipper.
- Urinary catheter.
- Antibiotics.
- Skin preparation from nipple to knee.
- Groin swab.
- Drapes from just above xiphisternum to proximal thigh.

2 – Dissection down onto aorta

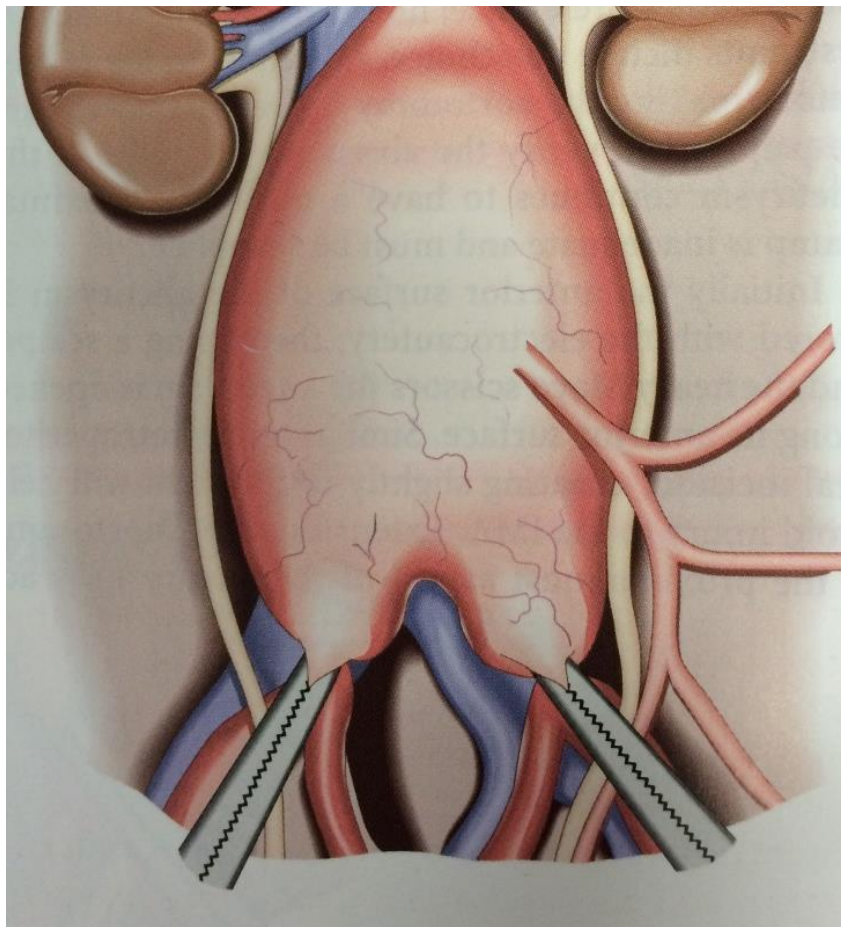


2 – Dissection down onto aorta

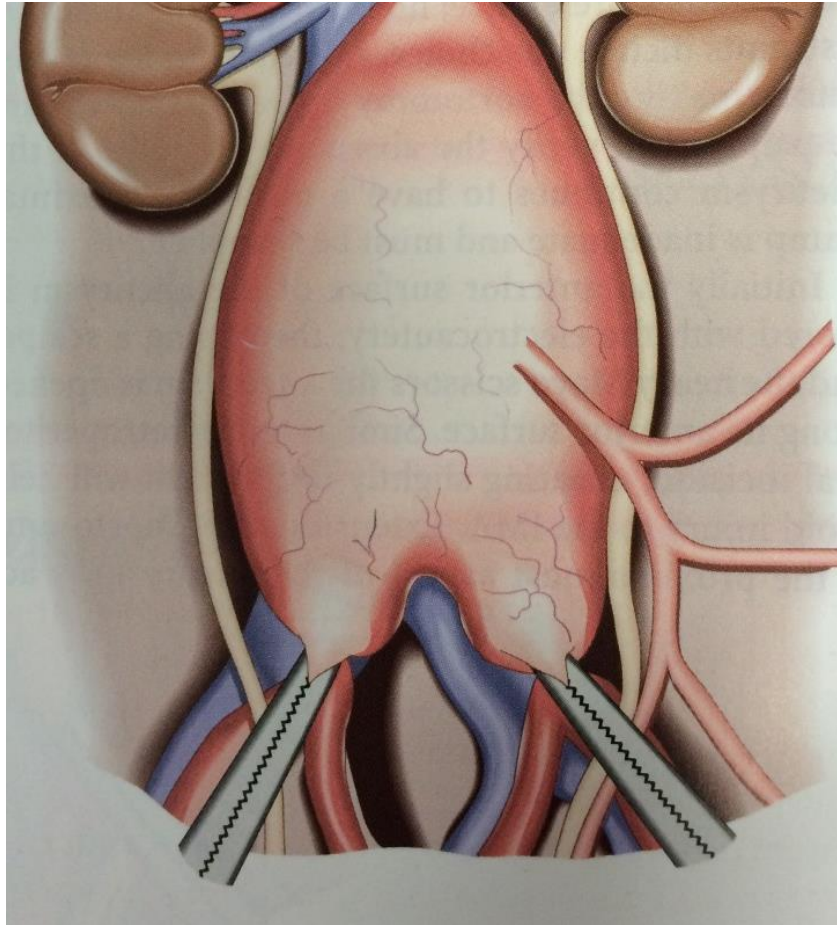


- Mid-line laparotomy.
- Be careful entering peritoneal cavity.
- First retractor transverse colon.
- Second retractor is small bowel to patient's right.
- Kelly retractor gently to RUQ.
- Fingers to splay and mobilise duodenum with forceps / diathermy / scissors.

3 – Clamping of distal vessels

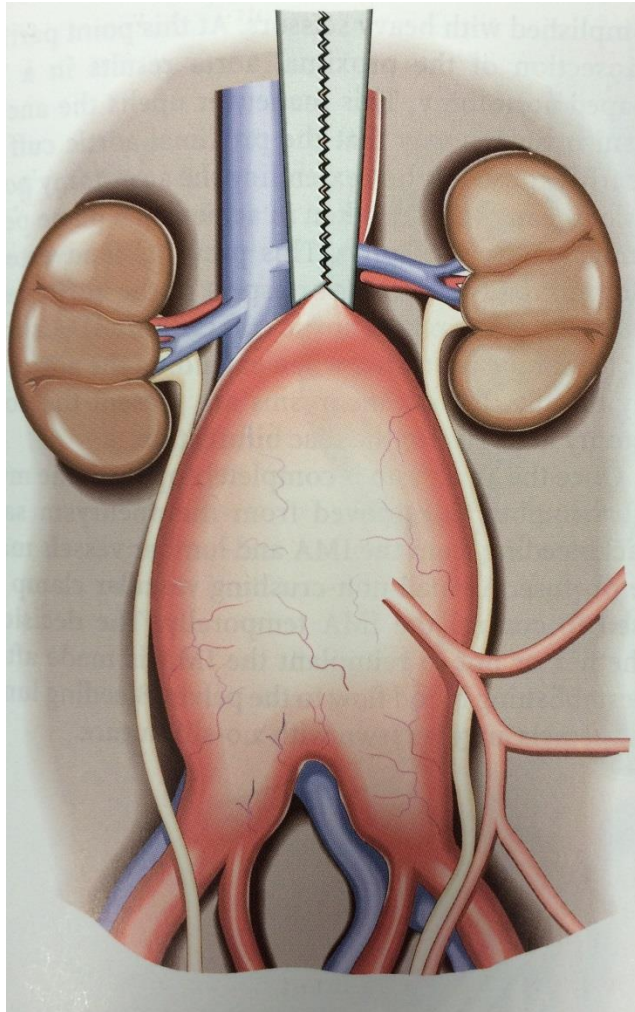


3 – Clamping of distal vessels

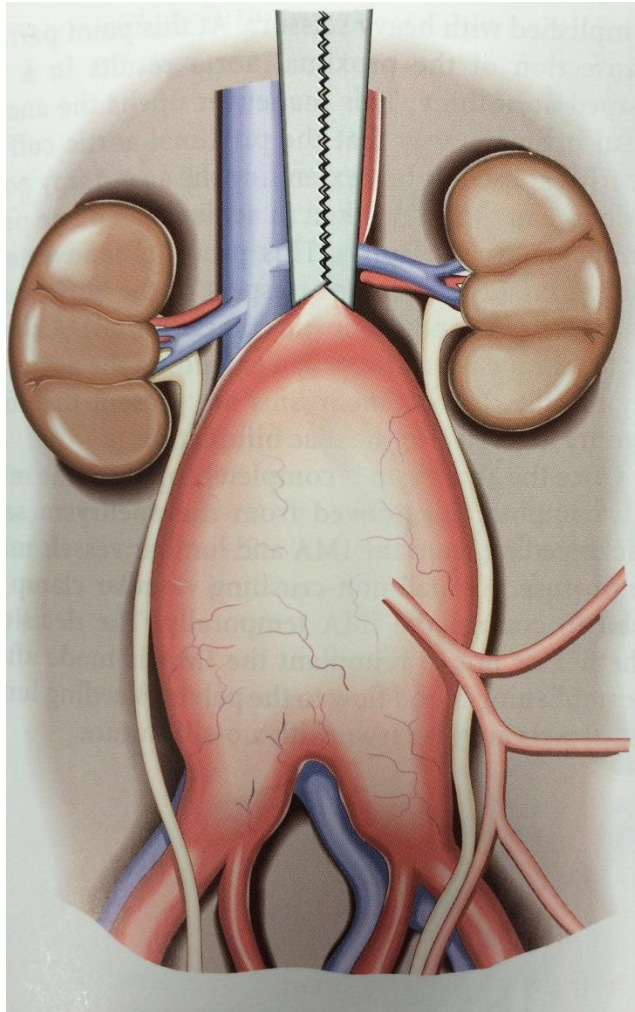


- Once duodenum mobilised and protected dissect through retroperitoneum and move towards proximal neck.
- Identify IMV then left renal vein.
- When on the white of the aorta careful dissection just below renals on either side – sucker or scissors.
- Ensure you can palpate the lumbar vertebrae on either side.
- Insert omnitract.
- Continue dissection down towards iliac arteries gently.
- Administer 5000IU heparin.
- Clamp distal vessels first.

4 – Control of proximal aorta

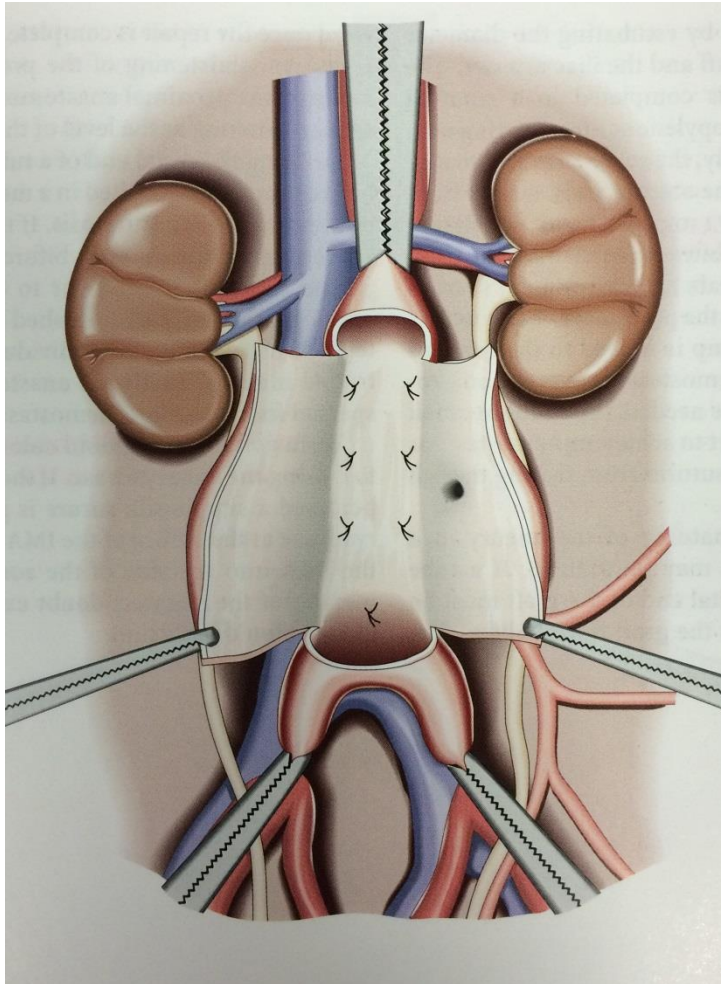


4 – Control of proximal aorta

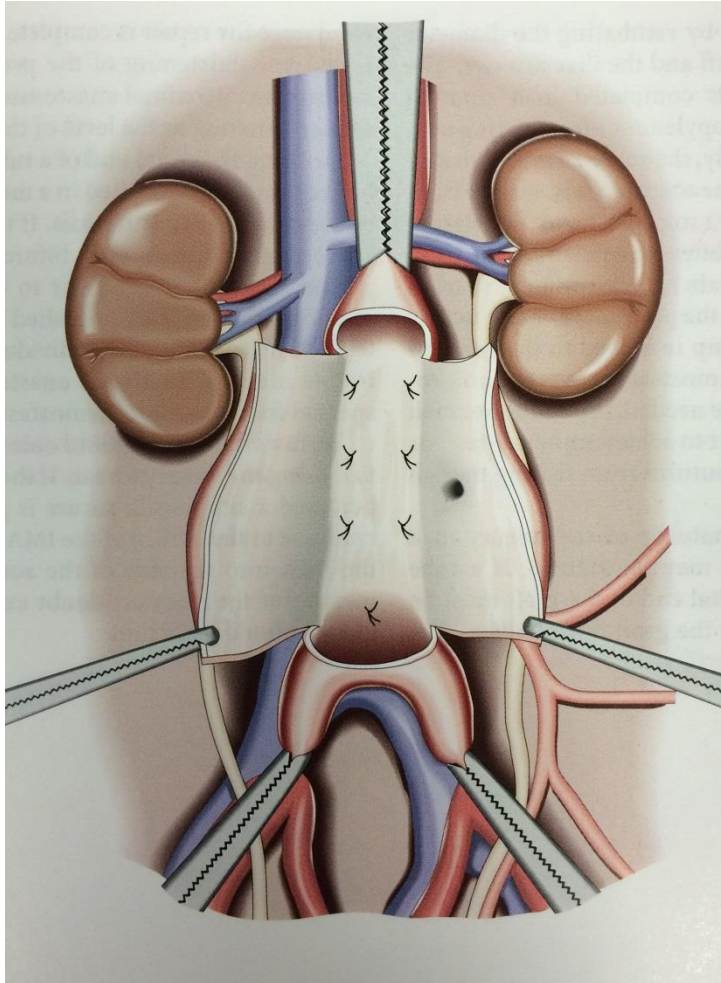


- Remember to position thoracic clamp in previously dissected zones onto vertebrae.
- As you clamp try and arch the clamp upwards to provide more space.
- Apply sloops to clamp.
- Palpate the aorta to make sure pulsation has dissipated.
- Let anaesthetic know you have clamped.

5 – Arteriotomy

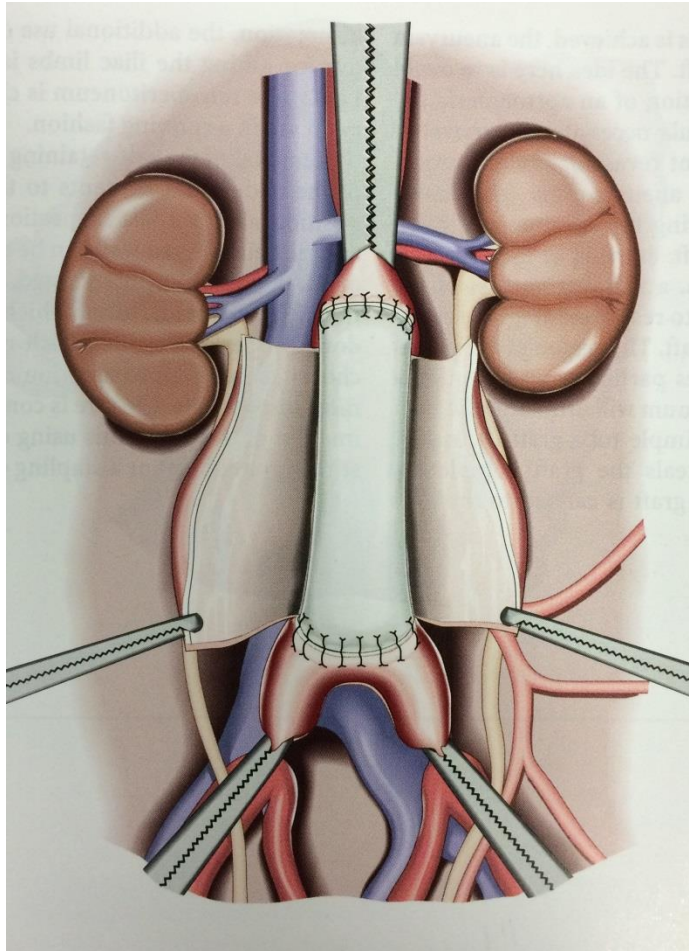


5 – Arteriotomy

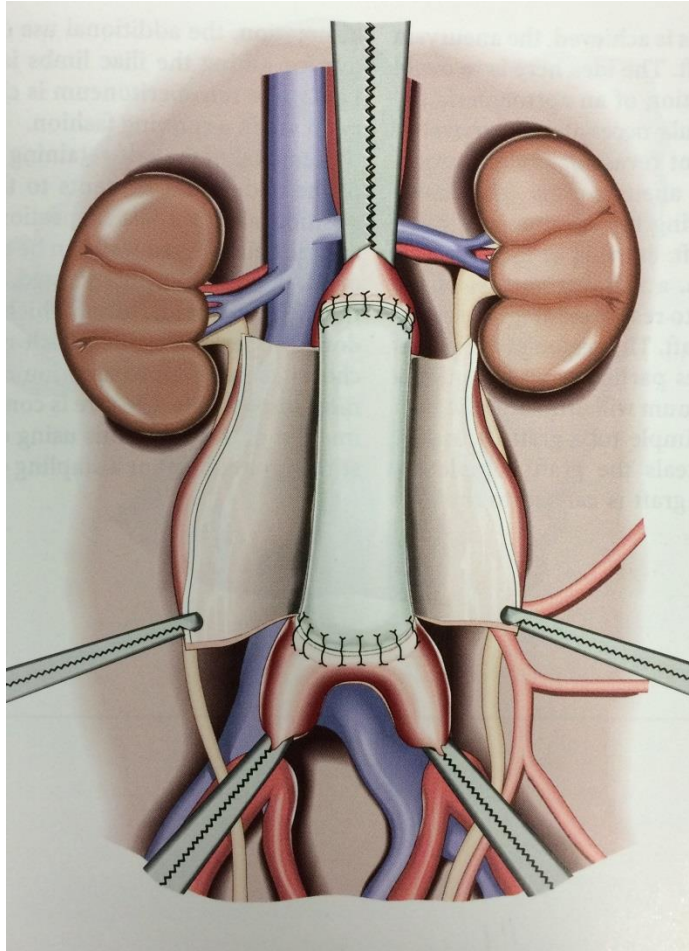


- Tell scrub nurse to have at least 3 x 2/0 prolones ready.
- Tell assistant to switch to cell-salvage only.
- Diathermy length of aorta then enter (use scissors if required for arteriotomy quickly).
- Remember – don't go too high or too low on arteriotomy and "T" both zones.
- Expect a gush of blood.
- If bleeding top end tell assistant to place downward pressure on clamp.
- Insert a small self-retaining retractor.
- "Figure 8" suture all lumbar and IMA as required.

6 – Graft suturing

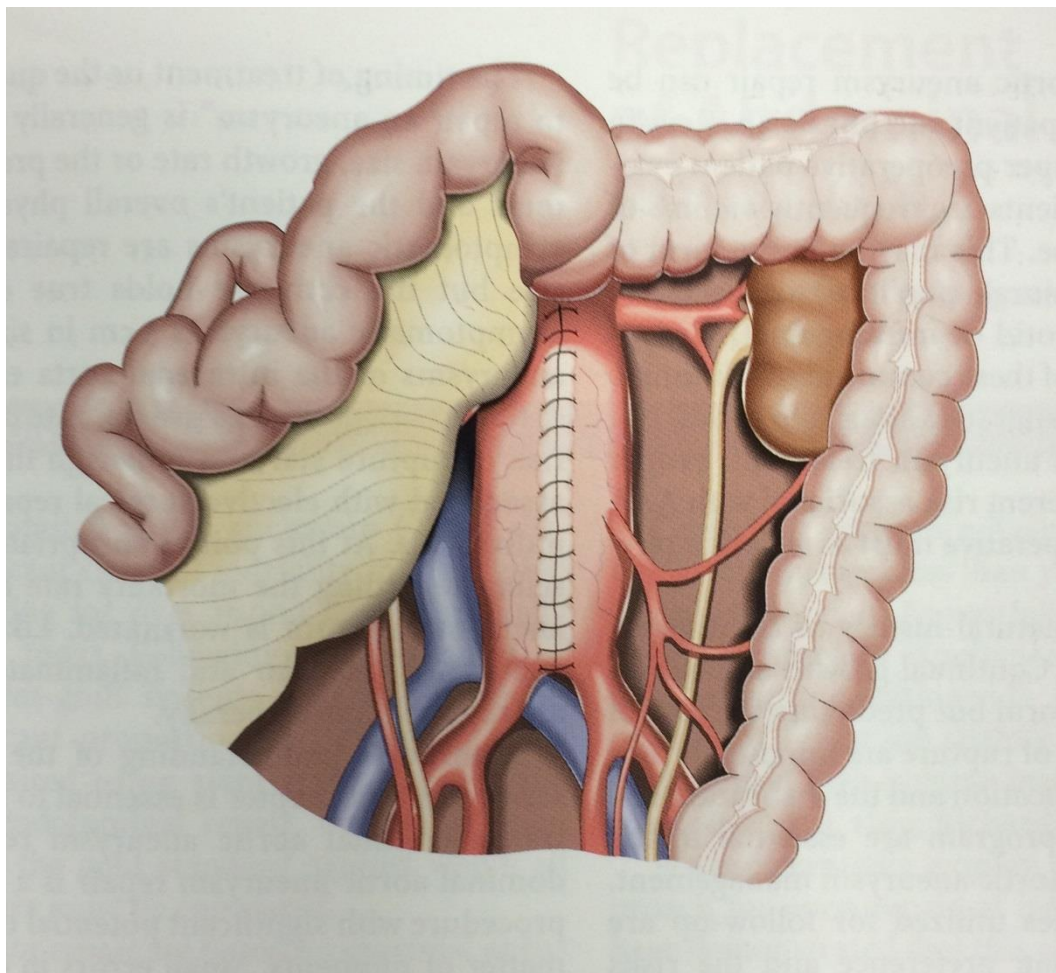


6 – Graft suturing

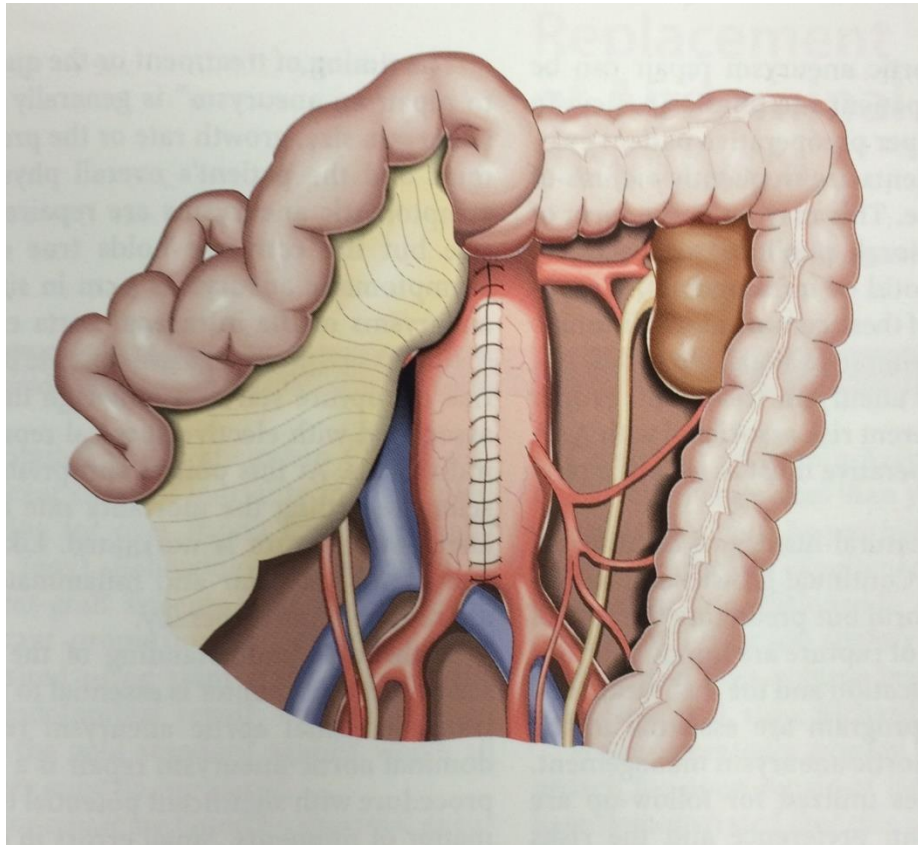


- Dacron type graft.
- Usually 16 or 18mm but use sizers if required.
- Suture top end 2/0 prolene.
- Apply fibrillar and fluffy for test declamp – do not take thoracic clamp out and have DeBakey clamp on graft.
- Complete distal anastomosis with 3/0 prolene.
- Pick your technique of suturing and stick with it.
- Back wall is vital.
- If friable have low threshold for teflon buttress.
- Forward and Backbleed prior to completion of anastomosis.

7 – Aneurysmal sac closure



7 – Aneurysmal sac closure



- Double check haemostasis.
- Close sac 2/0 vicryl.
- Close retroperitoneum 2/0 vicryl.
- Re-position intra-abdominal contents.
- CHECK AND DOCUMENT LEFT COLON STATUS.
- Palpate femorals and check feet.
- En –mass closure.

How to plan and perform endovascular AAA repair

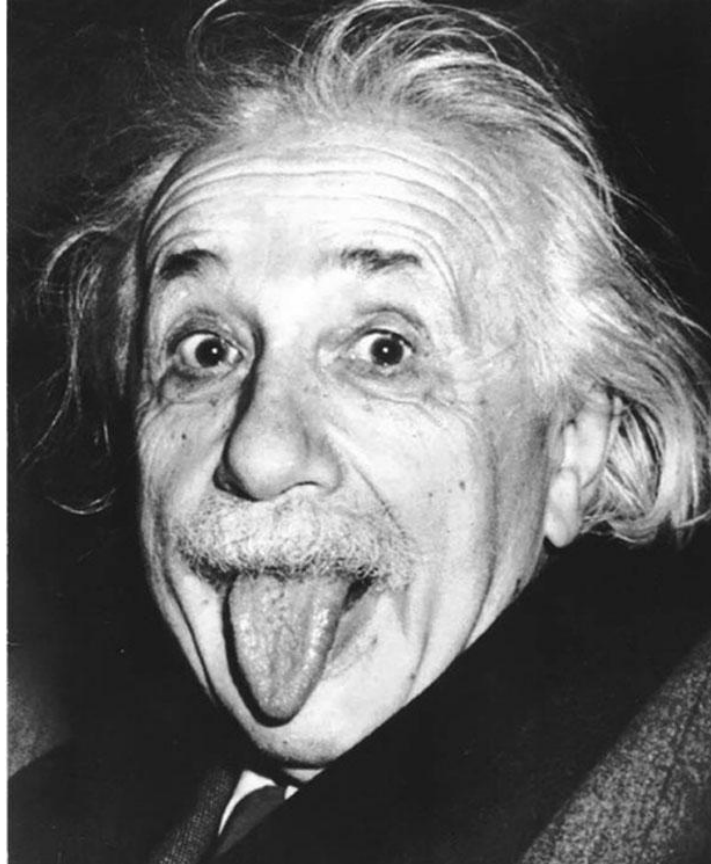


Post-procedural care

- Open AAA repair;
 - Usually POCU / HDU / ICU.
 - Clinical observations.
 - Restart medications – BMT and LMWH.
 - Slow restoration of diet.
 - Out-to-sit, slowly mobilise.
 - Ward transfer and hospital stay 5-10 day

- EVAR;
 - Ward transfer.
 - Restoration of normal diet.
 - Clinical observations / Restart medications.
 - Mobilise and Home day 1-2.

Important Literature



EVAR Trials

- **EVAR I;**
 - UK Endovascular Aneurysm Repair (EVAR) I trial evaluated fit patients.
 - 30-day mortality for EVAR was 1.7% versus 4.7% for open repair.
 - Longer term (4-years) all-cause mortality similar.

- **EVAR II;**
 - Unfit patients.
 - No difference between EVAR stenting and conservative patients.

Lancet 2005; 365: 2179-92

IMPROVE Trial

- 29 UK and 1 Canadian centre recruiting patients with a ruptured AAA;
 - 613 patients randomised to EVAR first strategy or open repair.
 - At one-year all-cause EVAR mortality was 41.1% versus 45.1% for open surgery.
 - EVAR patients had faster discharge with better quality of life.
 - EVAR group more cost effective.

Eur Heart J 2015

Cases

Case 1

- 64 year old male patient brought to emergency department following collapse at local shopping centre.

Case 2

- 76 year old patient referred from colorectal surgery with incidental 6.9cm aortic aneurysm.

Questions ?

