MODERN RADIOLOGY eBook

Vascular Imaging

ESR EUROPEAN SOCIETY OF RADIOLOGY

Preface

Modern Radiology is a free educational resource for radiology published online by the European Society of Radiology (ESR). The title of this second, rebranded version reflects the novel didactic concept of the *ESR eBook* with its unique blend of text, images, and schematics in the form of succinct pages, supplemented by clinical imaging cases, Q&A sections and hyperlinks allowing to switch quickly between the different sections of organ-based and more technical chapters, summaries and references.

Its chapters are based on the contributions of over 100 recognised European experts, referring to both general technical and organ-based clinical imaging topics. The new graphical look showing Asklepios with fashionable glasses, symbolises the combination of classical medical teaching with contemporary style education.

Although the initial version of the *ESR eBook* was created to provide basic knowledge for medical students and teachers of undergraduate courses, it has gradually expanded its scope to include more advanced knowledge for readers who wish to 'dig deeper'. As a result, *Modern Radiology* covers also topics of the postgraduate levels of the *European Training Curriculum for Radiology*, thus addressing postgraduate educational needs of residents. In addition, it reflects feedback from medical professionals worldwide who wish to update their knowledge in specific areas of medical imaging and who have already appreciated the depth and clarity of the *ESR eBook* across the basic and more advanced educational levels.

I would like to express my heartfelt thanks to all authors who contributed their time and expertise to this voluntary, non-profit endeavour as well as Carlo Catalano, Andrea Laghi and András Palkó, who had the initial idea to create an *ESR eBook*, and - finally - to the ESR Office for their technical and administrative support.

Modern Radiology embodies a collaborative spirit and unwavering commitment to this fascinating medical discipline which is indispensable for modern patient care. I hope that this *educational* tool may encourage curiosity and critical thinking, contributing to the appreciation of the art and science of radiology across Europe and beyond.

Minerva Becker, Editor
Professor of Radiology, University of Geneva, Switzerland

Vascular Imaging

CHAPTER OUTLINE:

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MODERN RADIOLOGY eBook

Based on the ESR Curriculum for Radiological Education

Vascular Imaging

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Introduction

CHAPTER OUTLINE:

/ Introduction

Advances made over the last decade in vascular imaging have enabled us to uncover some of the underlying mechanisms of vascular diseases. Many efforts have been made to establish the evaluation of atherosclerotic plaque progression and vascular inflammatory changes in addition to other biomarkers and clinical manifestations.

Non-invasive cross-sectional imaging techniques play a crucial role in the assessment of the varied manifestations of vascular disease and intervention planning.

For general anatomical, histological and physiological information regarding the vascular system, please refer to your knowledge obtained during your studies in previous years.

This chapter addresses basic concepts of vascular imaging and the most relevant pathologies. Some vascular pathologies and their imaging aspects are already included in other e book chapters, e.g., cardiac imaging, small and large bowel imaging, central nervous system imaging, emergency radiology or chest imaging.

CHAPTER OUTLINE:

<∞> REFERENCE

> see respective eBook chapters

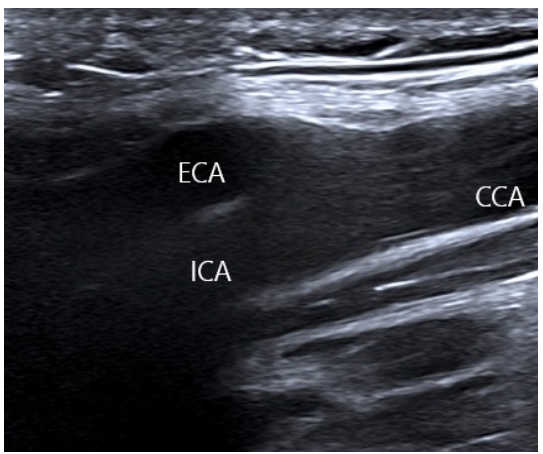
Vascular Imaging Techniques

CHAPTER OUTLINE:

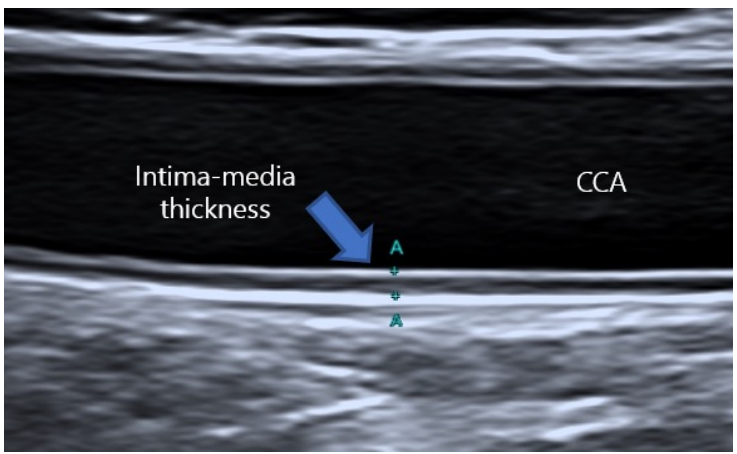
/ Ultrasound (US)

Ultrasound (US) is often the initial screening test for the evaluation of the peripheral vascular system and of the vasculature of some visceral organs, e.g., liver and kidneys.

The examination usually starts with the **B-mode or grayscale mode** - the "normal mode" which allows us to identify the vessel of interest, evaluate its walls, the presence of plaques and vessel narrowing/stenosis.



Sagittal grey-scale B-mode US image showing the common carotid artery (CCA) branching into the internal (ICA) and external carotid artery (ECA).



Sagittal grey-scale B mode US showing the left common carotid artery with a normal intima-media complex measuring 0.8 mm (> 1 mm is taken as abnormal).

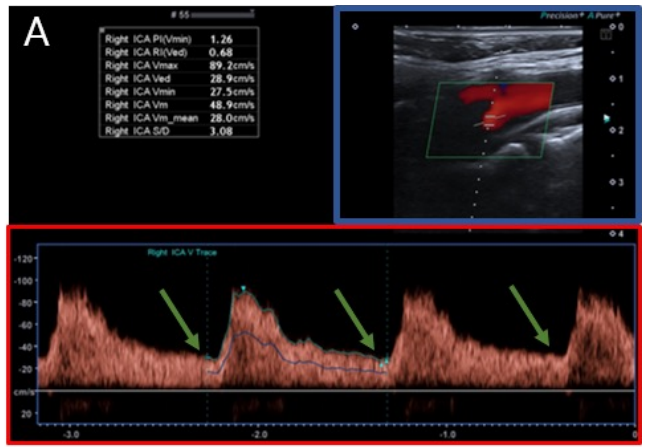
CHAPTER OUTLINE:

/ Doppler Ultrasound (Doppler-US)

To evaluate if the stenosis (i.e. due to an atherosclerotic plaque) is hemodynamically significant we employ more complex US modes like **Doppler imaging**.

Doppler imaging includes:

- **Colour Doppler – blue** (changes colour inside blood vessels depending on flow and speed of blood)
- **Spectral Doppler – red** (blood flow information is represented in a graph, as a waveform, where the quantitative values can be derived)



Triplex Doppler (M-Mode, Colour Doppler and Spectral Doppler) of the internal carotid artery (ICA) (A) and of the external carotid artery (ECA) (B). The ICA (A) demonstrates a low-resistance pattern with robust diastolic flow (arrows) because it is supplying the brain (which needs constant blood flow). The ECA (B) demonstrates a **high-resistance waveform** and a **low diastolic flow** (arrows).

/ Vascular Imaging

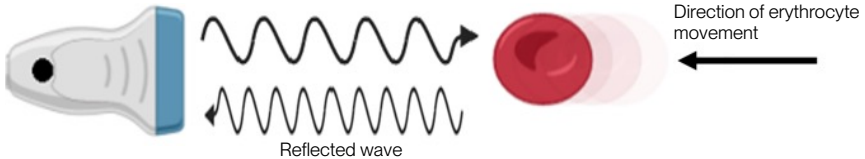
CHAPTER OUTLINE:

When sound waves hit an object, some of the sound waves are reflected to the sound source. If the reflector is **stationary**, the **reflected sound waves** will **have the same frequency as the sound waves emitted by the sound source**.

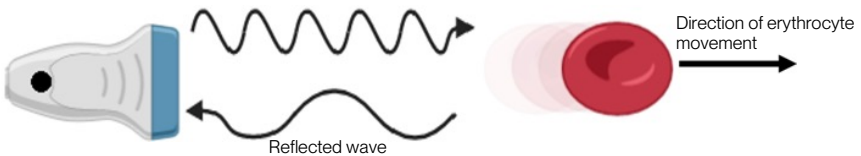
If the reflector is in **motion** - like **red blood cells (RBC)** inside the vessels - the **frequency of the reflected sound waves** will differ from the original emitted sound waves (by our probe).

This change in frequency is also known as the **Doppler effect**.

This change in frequency is then used to calculate the velocity and the direction of flow.



When the RBC are moving towards the probe, the reflected sound waves are compressed, which leads to shortening of the wavelength and thus an increase in frequency.



On the contrary, when the RBC are moving away from the probe, the reflected sound waves are stretched out, leading to an increase in wavelength and decreased frequency.

>=< FURTHER KNOWLEDGE

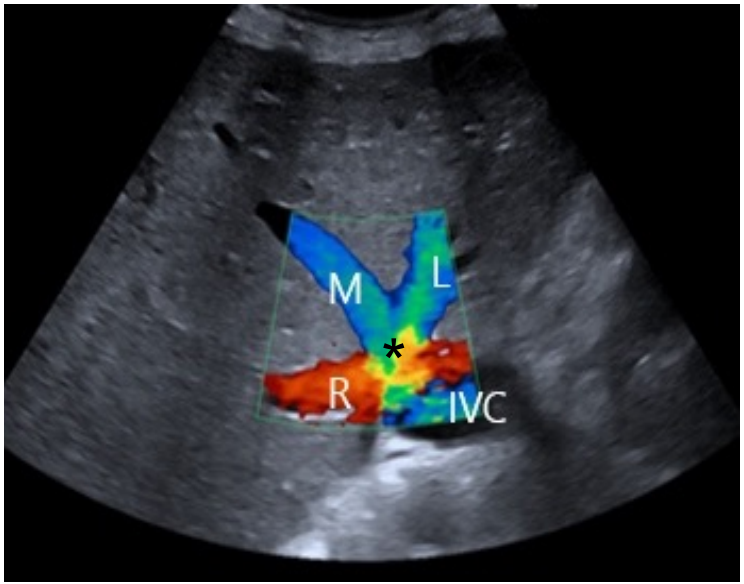
Christian Andreas Doppler (1803-1853) – the Austrian physicist who formulated the principle of the Doppler Effect

This information can be colour coded into a normal ultrasound image. In colour coded Doppler ultrasound, colour type and intensity indicate the direction and speed of the blood flow. By convention:

- Blood flow running away from the probe is depicted in blue
- Blood flow running towards the probe is depicted in red

<!> ATTENTION

If you accidentally rotate the scanner probe by 180 degrees, the colours will switch! And as your probe approaches a 90-angle relative to the vessel, your Doppler signal vanishes altogether!



Hepatic vein sonography: Transverse view of the three main hepatic vein trunks (Right – R; Middle - M; Left – L) as they enter the inferior vena cava (IVC).

Blood inside the hepatic veins usually runs towards the IVC and - depending on the position of the probe - flow will go away from the probe (depicted in blue in the left and middle hepatic vein) or towards the probe (depicted in red in the right hepatic vein).

At the level of convergence of the three veins, the blood flow is turbulent – as seen by the area of aliasing effect (*). Aliasing results in the inability to record the direction and the velocity of blood flow accurately.

CHAPTER OUTLINE:

Using Spectral Doppler, we can assess the **peak systolic velocity (PSV)** throughout a vessel of interest.

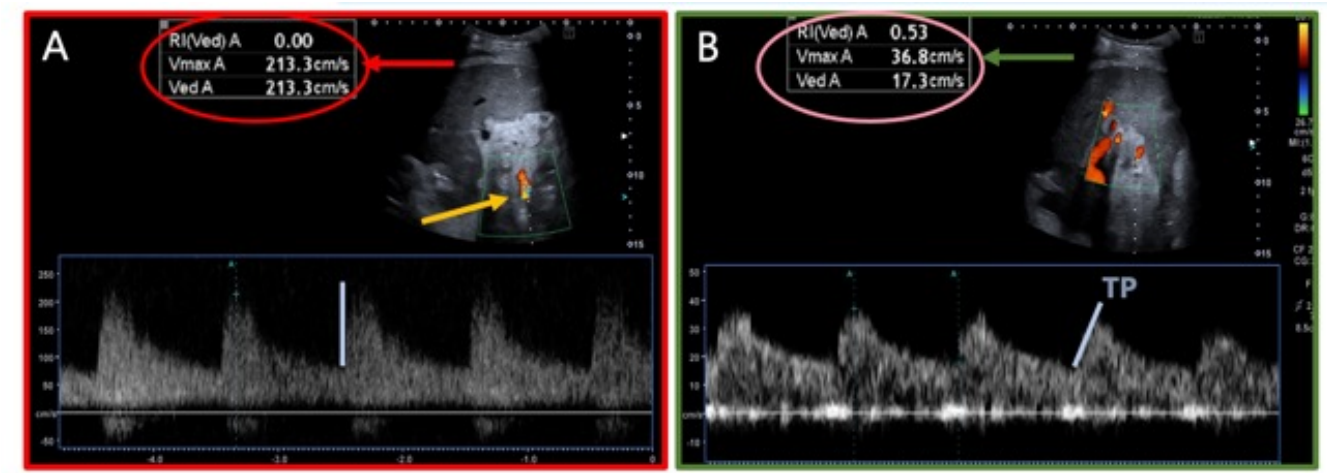
The table on the right shows the reference PSV values in different vessels.

However, more important than the reference PSV values is the variation of velocities (i.e., when assessing a site for a possible stenosis).

BLOOD VESSEL	PSV (CM/S)
Abdominal aorta	100-150
Iliac arteries	100-120
Fermoral artery	80-110
Popliteal artery	50-80
FInternal carotid artery	80 -120
Vertebral artery	25-40
Vena cava	10-45

<!> ATTENTION

Keep in mind that numerous other reasons can account for discrepancies regarding the measured PSV values. Therefore, the proper examination technique, correct patient positioning and proper interpretation of results should be taken in account!



Usually the PSV:

- Before the stenosis → is slightly higher (yellow).
- At the site of stenosis → is significantly higher (red).
- After stenosis → is lower (green).

There are a few other "clues" we use to confirm that a site of stenosis is hemodynamically significant besides the variation in PSV:

The two images bellow exemplify a Triplex Doppler in a patient with a recent liver transplant:

- On **A**, the probe is located just above the stenosis; note the aliasing artifact in a segment of the hepatic artery. Spectral Doppler shows a significant elevation of the PSV (arrow)
- Downstream of the stenotic segment (**B**) we have a reduced PSV velocity, with a "Tardus Parvus" (TP) waveform (= prolonged systolic acceleration and small systolic amplitude with rounding of the peak) and a decreased Resistive Index (RI)

RI = (PSV- EDV)/ PSV; EDV = End Diastolic Velocity. The normal RI values vary from one artery to another as they depend on the target organs, which have different flow requirements.

Vascular Imaging

CHAPTER OUTLINE:

Indications for DSA (diagnostic and/or treatment):

- / Aneurysms
- / Thrombosis
- / Vascular abnormalities
- / Arteriovenous malformations and/or fistulas
- / Haemorrhage
- / Complications post-transplant
- / Tumours

>|< COMPARE

ADVANTAGES OF DSA:

- \+ Minimally invasive
- \+ Can be performed on an outpatient basis
- \+ Real time observation
- \+ DSA resolution is superior to the resolution of CTA and MRA
- \+ Ability to perform concurrent endovascular treatment of many pathologies

DISADVANTAGES AND COMPLICATIONS OF DSA:

- – Exposure to X-rays
- – Contrast-related allergic reactions
- – Acute kidney Injury (due to contrast media)
- – Haematoma, infection, thrombus, pseudoaneurysm at the puncture site
- – Vessel dissection (at the puncture site or in a distant location)

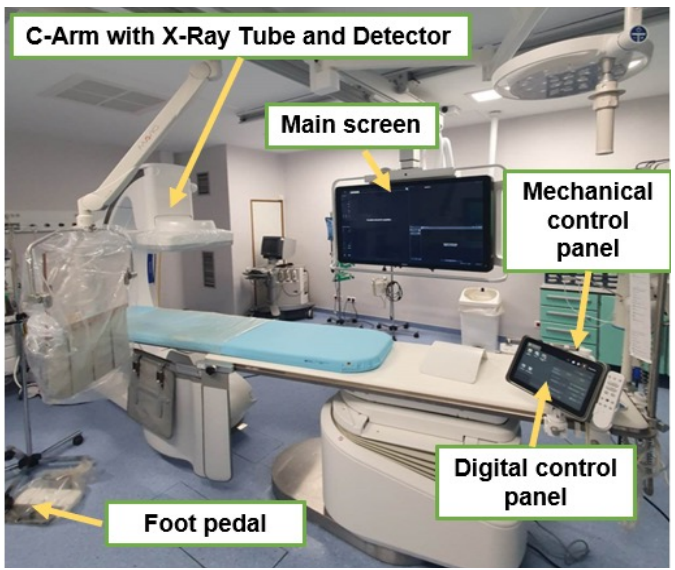
/ Digital Subtraction Angiography (DSA)

Digital Subtraction Angiography (DSA) is an imaging technique, where a catheter is inserted into a blood vessel (for arteriography usually the femoral artery) after which contrast material is injected through the catheter under fluoroscopic guidance. Local anaesthesia is administered at the puncture site.

The **goal** is to visualise blood vessels for diagnostic or radiologic interventional procedures.

First, a **mask** (non-contrast image) is obtained. Then, **consecutive images** of the area to be investigated are acquired at a set rate **during** the injection of contrast material. The mask is subtracted from these images to better visualise the filled vessels (by removing the distracting bony structures or other dense structures). The subtracted images can be seen in **real-time**.

After the DSA, haemostasis is applied at the puncture site and the immobilised patient is carefully observed during usually 4-6 h.

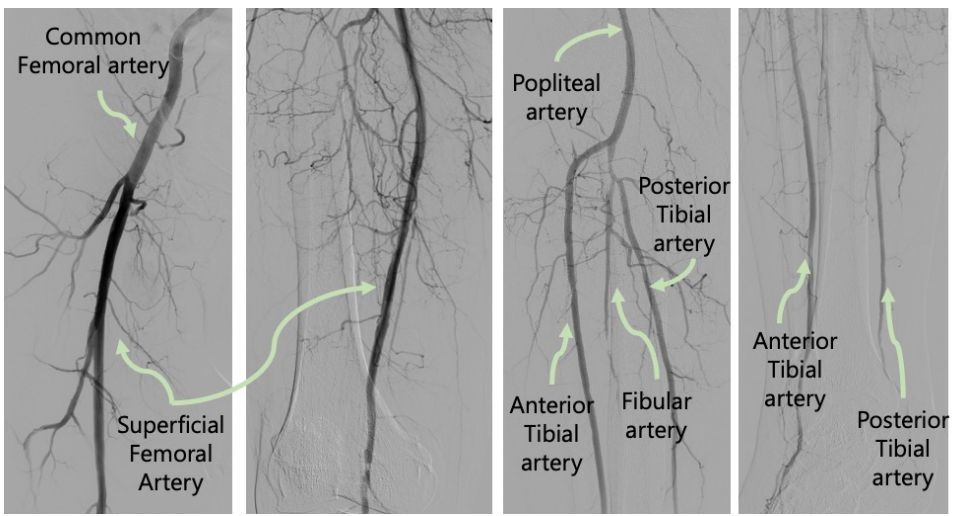


Angio-suite at Coimbra Hospital and University Centre

Before performing a DSA, several things must be considered:

- Arterial vs. venous
- Access site (arm, leg, neck etc.)
- Access side (right or left)
- Direction of puncture (antegrade or retrograde)

Access choice depends on the procedure whether it is **diagnostic angiography** or **interventional treatment**, and which is the target site (cerebral blood vessels, peripheral blood vessels, visceral blood vessels, etc.).



Lower Limb Angiography

Femoral artery angiography
↓
Distal femoral and popliteal angiography
↓
Popliteal and proximal tibial angiography
↓
Distal tibial and foot angiography

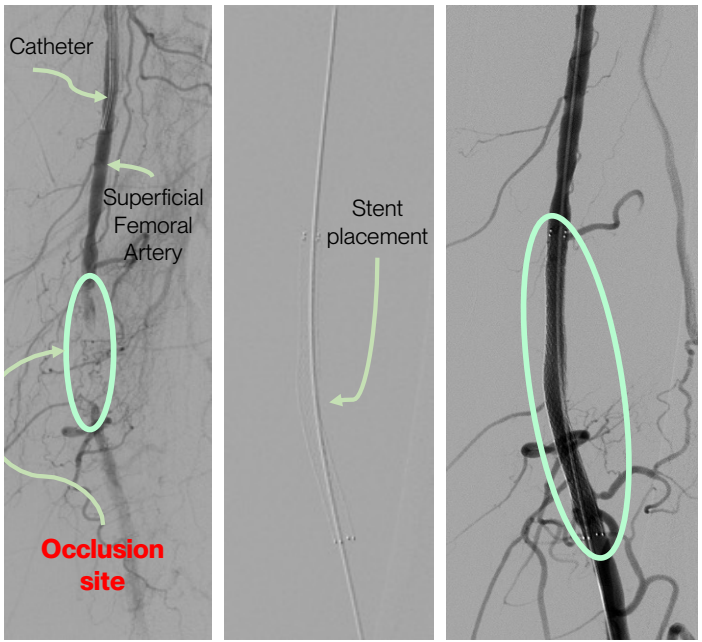
Vascular Imaging

CHAPTER OUTLINE:

Endovascular interventional procedures are performed by first getting access to a vessel, usually in the groin (most commonly the femoral artery).

After recanalisation of the stenotic or occluded segment, the artery is **dilated with a balloon** and, if necessary, recanalisation is followed by **stenting**.

Newer developments include **drug-coated balloons** to improve vessel patency and **atherectomy** to debulk calcifications.



Superficial Femoral Artery Stenosis → Stenting→ Post-stenting Result

>=< FURTHER KNOWLEDGE

Sven Ivar Seldinger (1921-1998) - the Swedish interventional radiologist who pioneered the Seldinger Technique (= gold standard method for vessel catheterisation)

https://www.sciencedirect.com/topics/medicine-and-dentistry/seldinger-technique

Image from: https://www.jvir.org/article/S1051-0443%2821%2901195-7/pdf

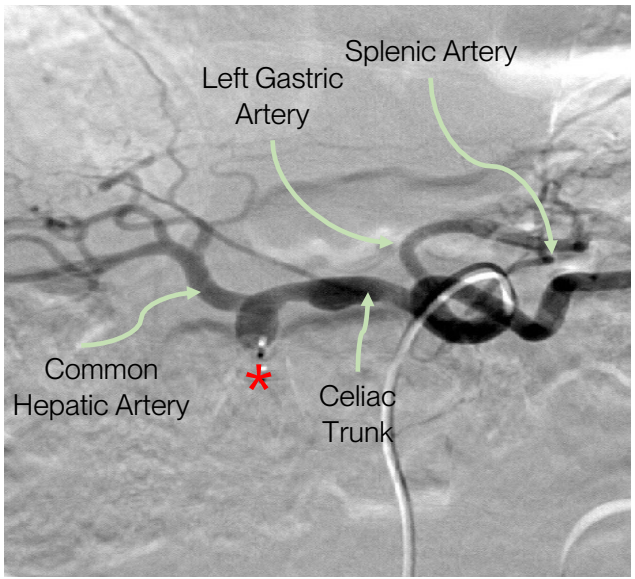
/ Vascular Imaging

CHAPTER OUTLINE:

/ Visceral Angiography

It's also possible to access visceral organ arteries and perform DSA. To do so it is necessary to insert the catheter in the femoral artery and then "navigate" through the aorta until the catheter is placed in the desired artery and contrast media is injected.

During visceral angiography one may also perform therapeutic interventions, like stenting, embolisation (with coils, «glue» or other materials).



DSA of the celiac trunk and its branches:

- Common hepatic artery
- Left gastric artery
- Splenic artery

<?> QUESTION

In this case shown on the left the patient had upper gastrointestinal bleeding treated by the deployment of a "metallic plug".

Can you identify the vessel where the plug was inserted?

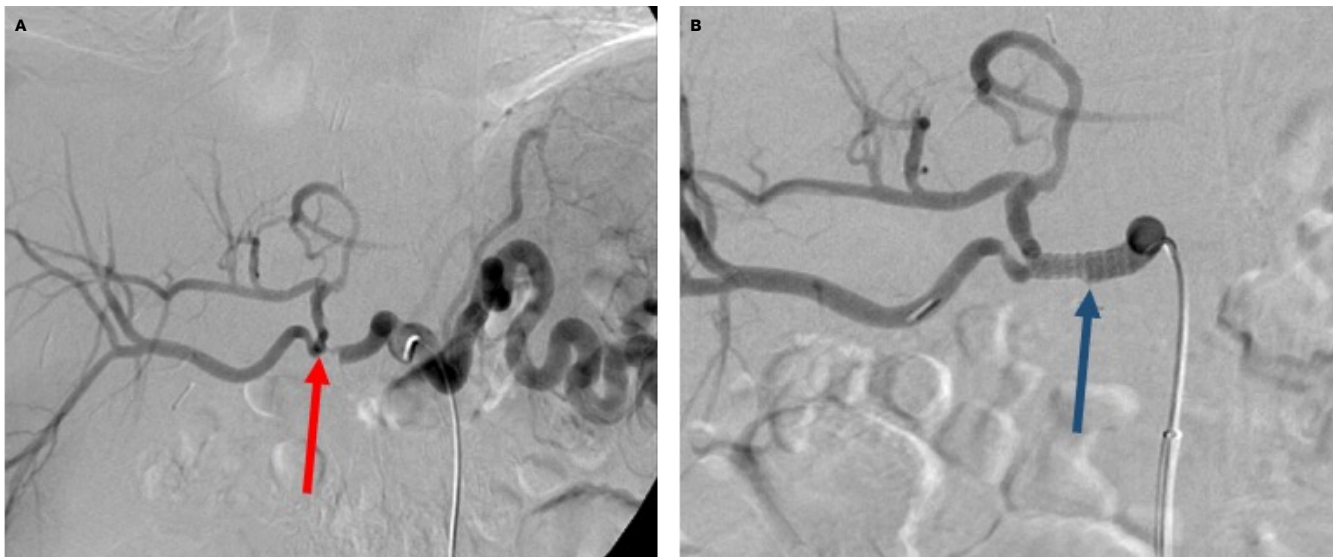
> **Answer:** Gastroduodenal artery (*)

<∞> REFERENCE

> see eBook chapter Interventional Radiology

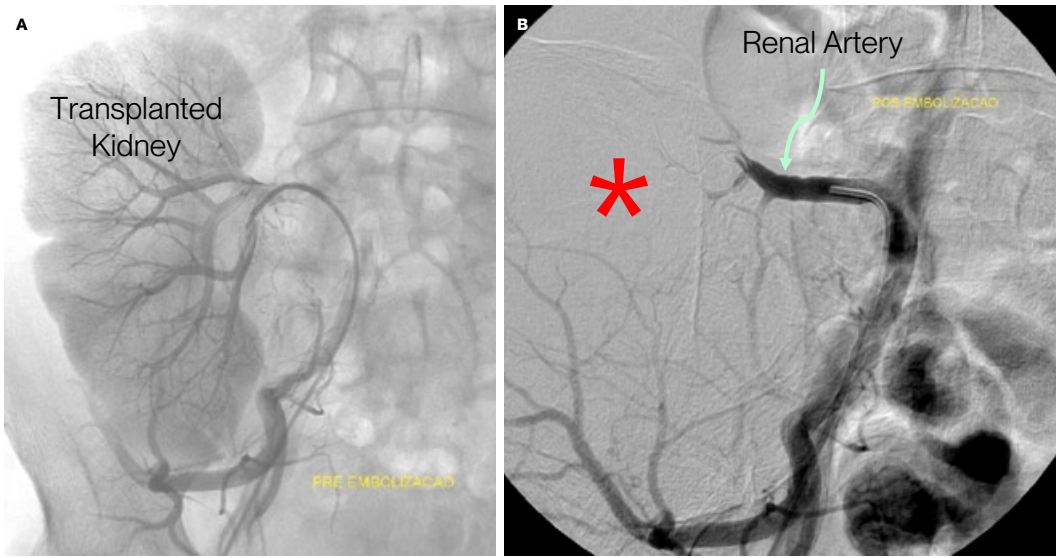
/ Vascular Imaging

CHAPTER OUTLINE:



DSA of the celiac trunk **showing a stenosis of the proper hepatic artery**, right before it branches into the left and right hepatic arteries (**A**).

On the right you can see an angioplasty of the hepatic artery with **stent implantation** (**B**).



Right renal angiography catheter access route:

Right femoral artery
↓
External iliac artery
↓
Common iliac artery
↓
Catheter in the right renal artery

DSA of the renal artery of a non-functioning **transplanted kidney** (**A**). On **B** the kidney is no longer visible (*) as it was excluded by embolisation of the **renal artery** (arrow).

/ **Vascular** Imaging

CHAPTER OUTLINE:

DSA allows not only access to arteries, but we can also access veins!

By performing venography keep in mind in which direction the blood flows.

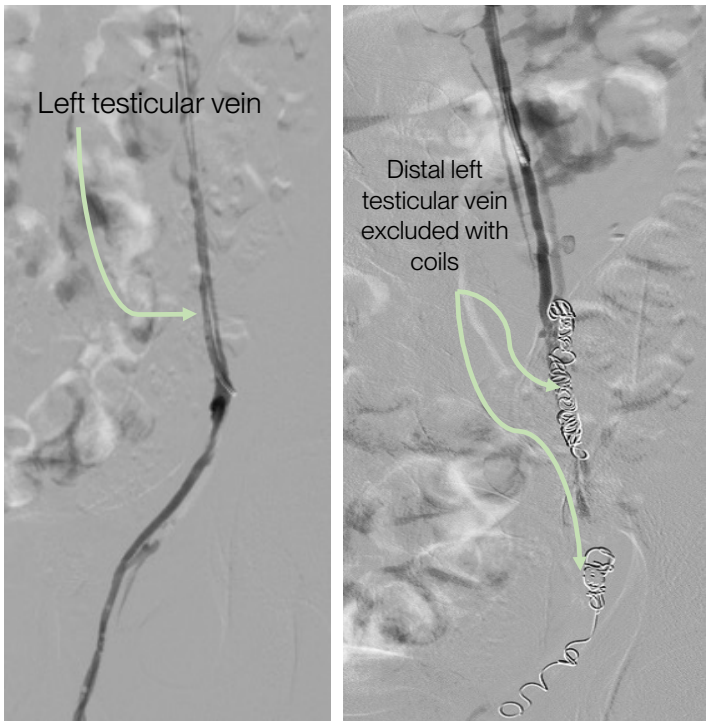
For lower limb and pelvic venography, access is gained through the popliteal vein.

It is also possible to perform interventions, like balloon dilatation, stent implantation, venous filter placement or embolisation.

On the images on the right, we have an example of a left varicocele embolisation via the right femoral vein with coiling of the left testicular vein.

<!> ATTENTION

Isolated right sided varicoceles should be further evaluated for the presence of retroperitoneal disease (e.g., renal cancer) as the right testicular vein drains directly into the inferior vena cava (IVC)!



Access route: Right femoral vein > Inferior vena cava > Left renal vein > Left testicular vein embolisation (coiling)

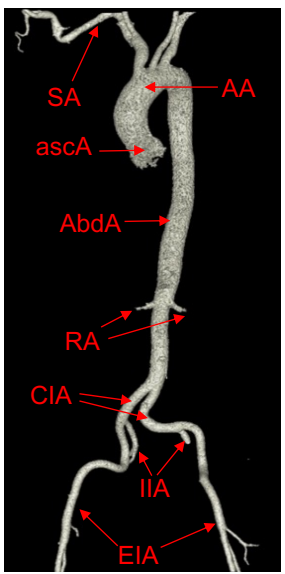
/ Computed Tomography Angiography (CTA)

Computed tomography (CT) angiography (CTA) is a powerful CT scanning technique used for the visualisation of **arteries and veins** (CT venography, CTV) following intravenous contrast injection. It is routinely used for the assessment of the cerebral and neck vessels, chest vessels, coronary arteries, splanchnic, pelvic and peripheral vessels. CTA is a cost-effective, widely available technique, which is less invasive than DSA.

>=< FURTHER KNOWLEDGE

The actual procedure depends on **institutional protocols and guidelines**, as well as on the **area to be examined**. Nevertheless, the following applies for all different CTA examinations:

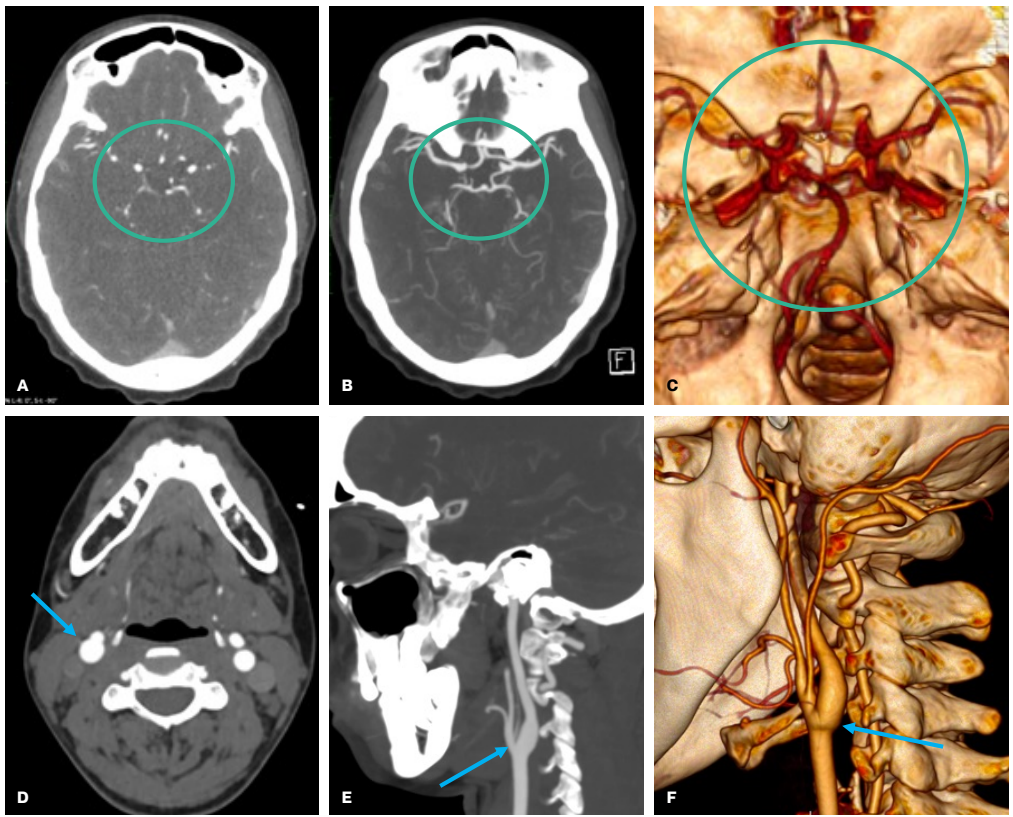
- The patient is in the supine position
- Iodinated contrast material is injected in an antecubital vein; the injection rate is about 4-5 ml/s for arterial imaging
- ECG gating can be used in some instances (coronary and chest CTA)
- Arterial phase images are obtained either by monitoring arrival of contrast bolus in the region of interest (bolus tracking) or by administering a test bolus to calculate the optimal scan delay; alternatively pre-defined scan intervals can be used (especially for splanchnic CTA)
- In addition to the axial acquired images, two-dimensional (2D) multiplanar reconstructions (MPR), Maximum Intensity Projections (MIP) and volume rendering (VR) or surface shaded displays (SSD) are used for data analysis



CTA with 3D SSD reconstruction of the entire aorta with the ascending aorta (ascA), aortic arch (AA), subclavian artery (SA), abdominal aorta (AbdA), renal arteries (RA), common iliac arteries (CIA), internal iliac arteries (IIA), external iliac arteries (EIA).

CHAPTER OUTLINE:

Vascular Imaging



Different types of CTA reconstructions can be obtained from the same contrast-enhanced CT acquisition. In this figure, reconstructions of the polygon of Willis (green circle, **A-C**) and of the right carotid bifurcation (blue arrow, **D-F**) are shown. Both the polygon and the carotid arteries are normal.

The goal in this CTA examination is to achieve maximum contrast enhancement in the arteries with minimal or no enhancement in the venous system.

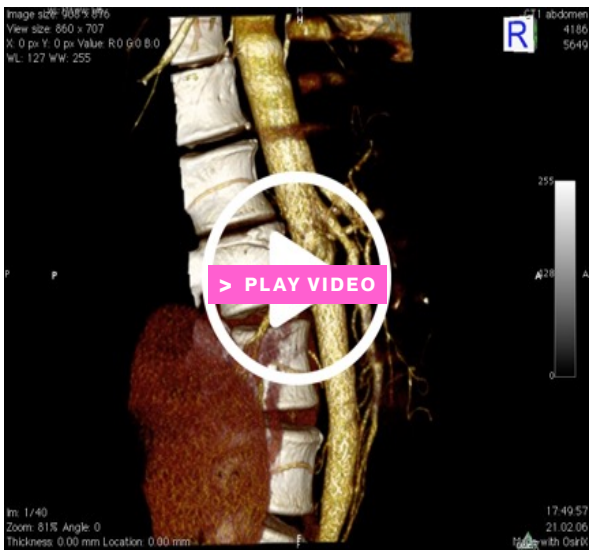
A and **D**: axial contrast enhanced CT images obtained in the arterial phase (0.75 mm slice thickness).

Axial (**B**) and sagittal (**E**) MIP

3D VR view of the polygon of Willis (green circle) seen from above (**C**) and lateral view of the right carotid bifurcation (blue arrow, **F**).

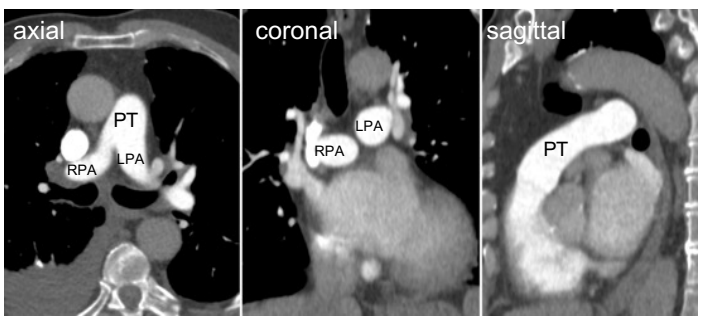
CHAPTER OUTLINE:

Figures illustrating normal CTA reconstructions from different anatomical regions, as used in clinical routine.



CTA 3D VR of the aorta, celiac trunk, superior mesenteric and renal arteries.

Click to Play Video in Browser (External)



Normal CT pulmonary angiogram with 2D MPR in the axial (**A**), coronal (**B**) and sagittal planes (**C**). This examination is done to exclude pulmonary emboli. The goal is to opacify the pulmonary artery and its branches. Pulmonary trunk (PT); right pulmonary artery (RPA); left pulmonary artery (LPA).

Vascular Imaging

CHAPTER OUTLINE:

<!> ATTENTION

Vascular variants are common and can be seen in the aorta, superior and inferior vena cava and intracranially.

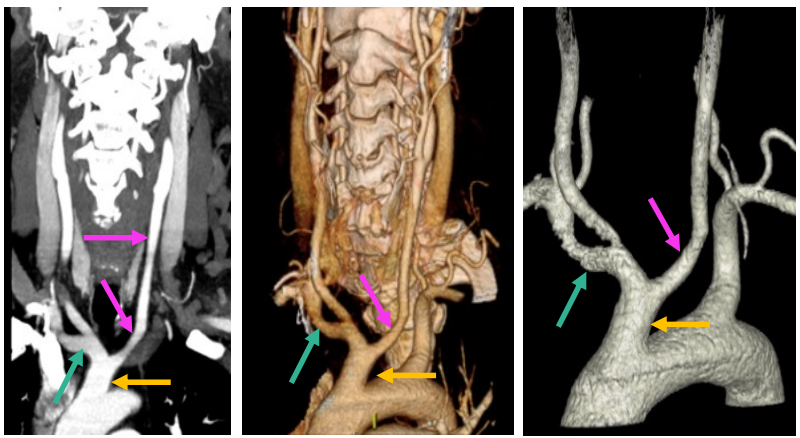
Even if patients are asymptomatic, it is important to recognise and precisely mention vascular variants in the radiologic report because of the following reasons:

- To avoid confusion with vascular pathology
- To adequately plan interventional procedures and surgery
- To suggest the presence of other associated abnormalities

>=< FURTHER KNOWLEDGE

Aortic arch branching patterns
https://www.sciencedirect.com/science/article/pii/S002252231500152X#fig1

Figure illustrating CTA reconstructions of vascular variants.



The bovine arch is the most common variant of the aortic arch in which the innominate artery (green) has a common origin (orange) as the left common carotid artery (pink). It is present in about 15% of the population and it is mostly asymptomatic. It can be associated with an aberrant left subclavian artery (also called arteria lusoria), which can cause dysphagia by compressing the oesophagus (dysphagia lusoria).

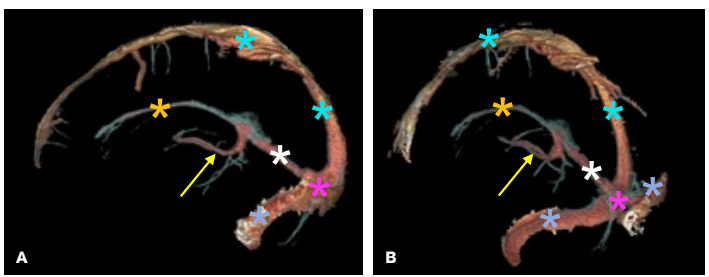
CHAPTER OUTLINE:

/ CT Venography (CTV)

CT venography (CTV) of the head (also called cerebral CTV) is performed to visualise the cerebral veins and venous sinuses filled with contrast.

>=< FURTHER KNOWLEDGE <!> ATTENTION

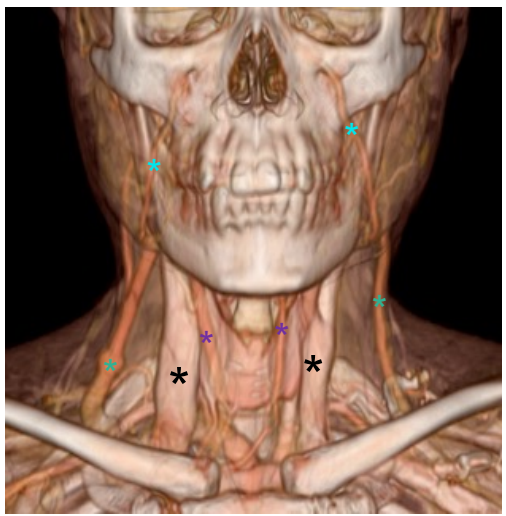
In CTV, the scan delay must be adapted (later acquisition compared to CTA). Usually, the scan delay is about ≥ 45 s after injection. Reconstructions are similar as for CTA (see previously).



Sagittal (**A**) and sagittal oblique (**B**) 3D VR of a normal cerebral CTV. Superior sagittal sinus (turquoise asterisk); Inferior sagittal sinus (orange asterisk); Straight sinus (white asterisk); Confluence of sinuses (pink asterisk); Transverse sinus (blue asterisk); Great cerebral vein (yellow arrow).

The goal is to assess their patency in suspected cerebral venous thrombosis and to assess the venous anatomy prior to cranial surgery.

CTV is also done to assess other venous vascular structures in the body, e.g., the internal jugular veins, subclavian or iliac veins.



CTA 3D VR of the venous system of the neck. Internal jugular veins (*); external jugular veins (green asterisk); anterior jugular veins (purple asterisk); facial veins (turquois asterisk).

/ Main indications

<!> ATTENTION

Main indications for CTA are:

- / Vessel stenosis
- / Thrombosis / occlusion
- / Detection of ongoing bleeding
- / Aneurysms
- / Dissection
- / Vascular malformations
- / Vascular anomalies & variants
- / Trauma
- / Vascular tumours and non-invasive assessment of feeding arteries of tumours prior to their resection
- / Guide interventional radiologists and surgeons prior to stent placing or other surgical interventions
- / Evaluation of vessel patency after treatment

/ Magnetic Resonance Angiography (MRA)

Magnetic resonance angiography (MRA) is an imaging method that uses a powerful magnetic field and radio waves to produce detailed images of blood vessels in the body.

Unlike traditional CTA or DSA, which use X-rays, **MRA does not use ionising radiation**.

Also, it's possible to obtain MR angiography **with** or **without** contrast media, depending on indications.

MRA is used to visualise blood vessels in almost any part of the body, including the brain, heart, kidneys, and leg blood vessels, although some limitations occur, and technical parameters have to be adjusted to perform such examinations.

<!> ATTENTION

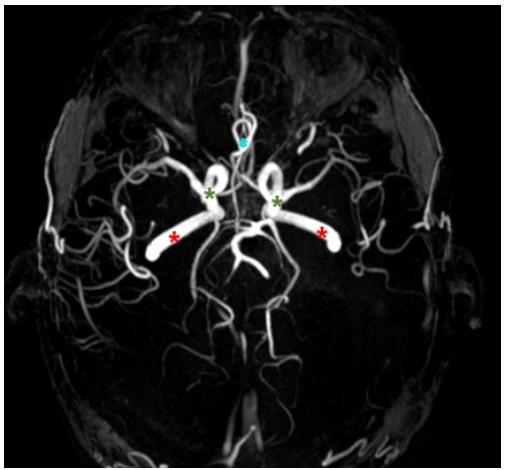
Always make sure that there are no contraindications for MRI!

Due to the strong magnet field, MRI cannot be always performed, for example in patients with:

- Certain types of pacemakers
- Certain intracranial clips
- Certain types of cochlear implants
- Any type of ferromagnetic metal implants

<∞> REFERENCE

> see eBook chapter on MRI



MRA using a 3D TOF sequence **WITHOUT contrast media** showing the intracranial blood vessels.

Internal carotid arteries (red asterisk); Middle cerebral arteries (green asterisk); anterior cerebral arteries (turquoise asterisk).

>=< FURTHER KNOWLEDGE

Anatomy of the Polygon of Willis and Common Variants
https://doi.org/10.53347/rID-51777

/ Without Contrast Media

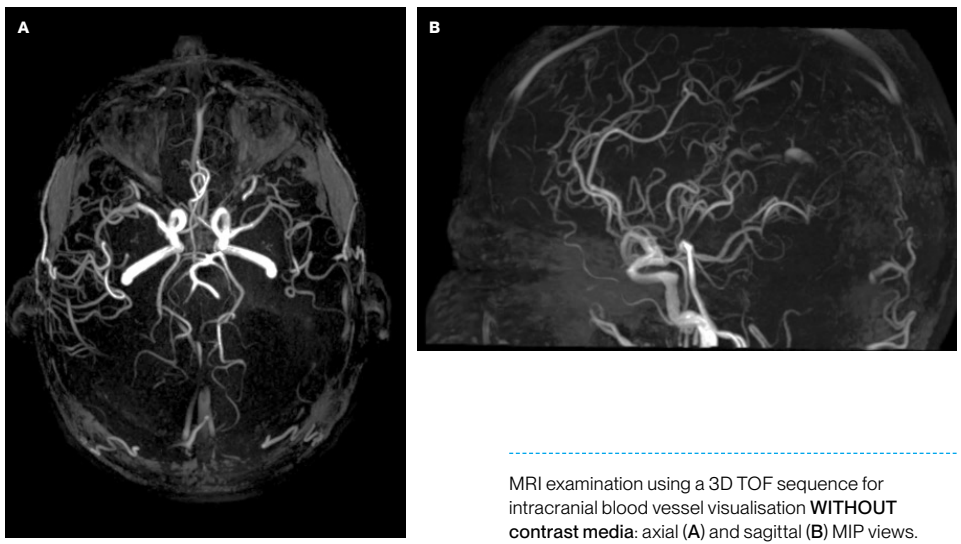
The most commonly used MRI sequence to assess blood vessels without contrast media is a **3-dimensional (3D) time-of flight (TOF) sequence**. This technique assesses the differences between stationary tissues and blood flow.

It is important to keep in mind that with this sequence we don't really see the intraluminal changes, but we see **blood flow changes inside the blood vessels**.

<!> ATTENTION

Blood flow intracranially can be influenced by numerous factors:

- Carotid artery stenosis
- Low ejection fraction in heart
- Competitive flow from other blood supply regions
- Anatomical variations of Circle of Willis



MRI examination using a 3D TOF sequence for intracranial blood vessel visualisation **WITHOUT contrast media**: axial (**A**) and sagittal (**B**) MIP views.

/ Without Contrast Media

Contrast enhanced MRA (CE-MRA) is in many ways, like CTA, but a gadolinium-based contrast agent (instead of iodinated contrast) is used.

CE-MRA is an excellent alternative to CTA for vascular evaluation and follow-up without requiring iodinated contrast material and ionising radiation.

Indications for MRA:

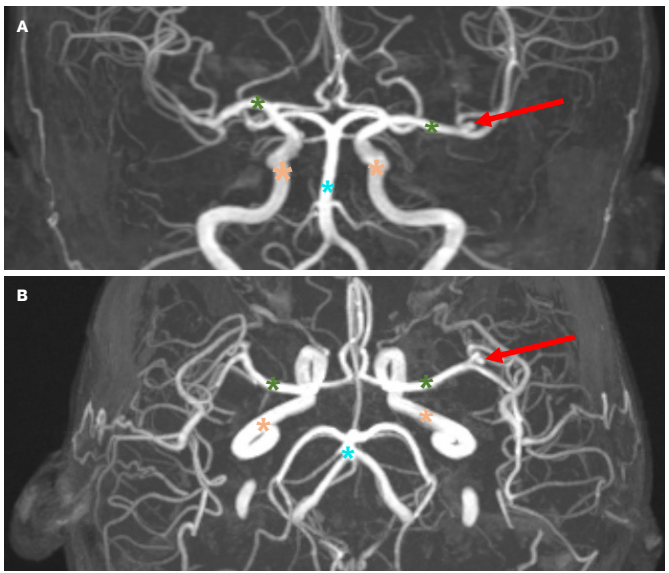
- Aneurysms
- Vasculitis
- Cerebral artery occlusion or stenosis (non-acute setting)
- Vascular malformations (AVM)
- Neurovascular conflict assessment
- Vascular anatomical variations, that could cause clinical symptoms

<!> ATTENTION

MRA represents a great imaging option in patients with allergy to iodine-based contrast material!

<∞> REFERENCE

> see eBook chapter on Contrast Media



MRA WITH contrast media (MIP reconstruction) is particularly good for the detection of aneurysms. Frontal (**A**) and axial (**B**) views. Note focal dilatation of the left MCA (green asterisk) representing an aneurysm (arrow).

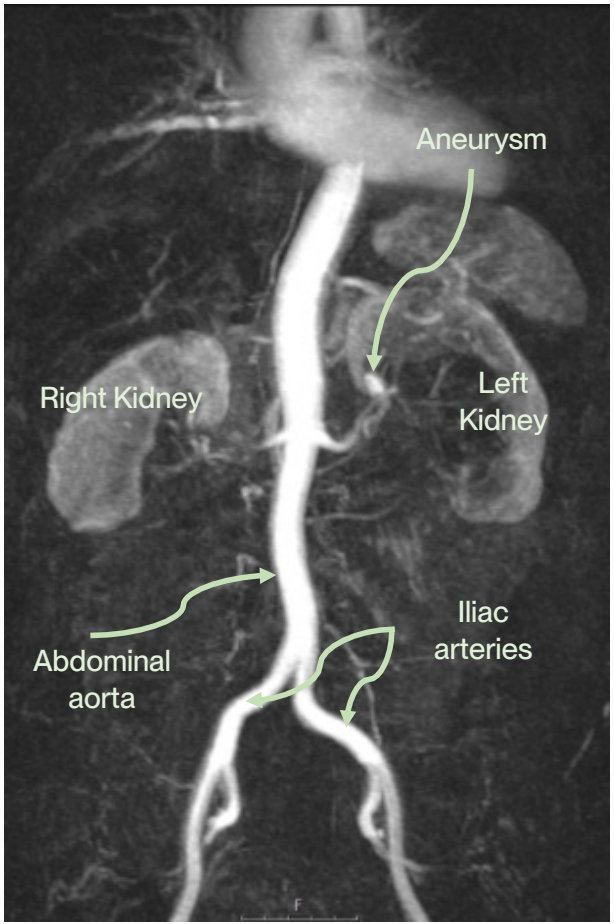
Internal carotid arteries (orange asterisk);
Middle cerebral arteries, MCAs (green asterisk);
Basilar artery (turquoise asterisk).

/ With Contrast Media

Rapid gradient echo sequences are particularly useful when **swift image capture is needed** (scenarios where movement might compromise image quality, such as dynamic studies).

This type of quick MR sequences has various clinical applications, including:

- / Angiography
- / Cardiac imaging
- / Abdominal imaging (i.e., liver, kidneys, bowel)



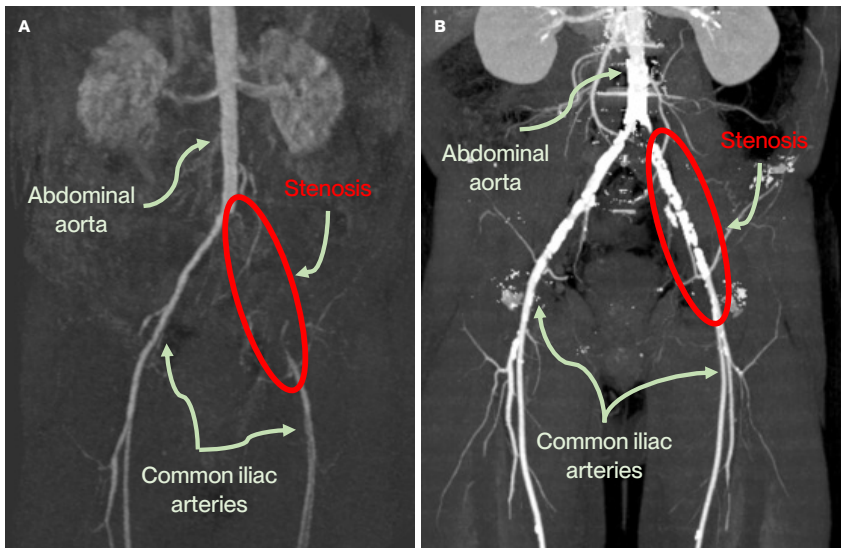
MRA renal 3D reconstruction depicting a **left renal artery aneurysm**

MRA provides a 3D alternative to CTA for the evaluation of peripheral arterial disease (PAD). Although CTA has a high diagnostic performance to detect arterial stenoses and occlusion, calcifications and metallic stents can hinder the evaluation of vessel patency. **> see figure on the right.**

Newer technical developments, e.g., image subtraction and/or energy subtraction, as well as ultra-high-resolution CT are increasingly used to overcome the current limitations of conventional CTA.

MRA can also better evaluate run-off vessels because of its ability to detect blood flow with lower velocities than other imaging modalities.

MRA is equally considered the gold standard in inflammatory or degenerative changes of vessel walls.



MRA of the proximal part of the lower limbs (**A**) versus conventional CTA (**B**) of the same region in the same patient. Note improved visualisation of a long segment stenosis of the left common iliac artery on the MRA in comparison to the conventional CTA. Extensive calcifications on the left impair assessment of vessel patency on the conventional CTA image.

/ Positron Emission Tomography Computed Tomography (PET CT)

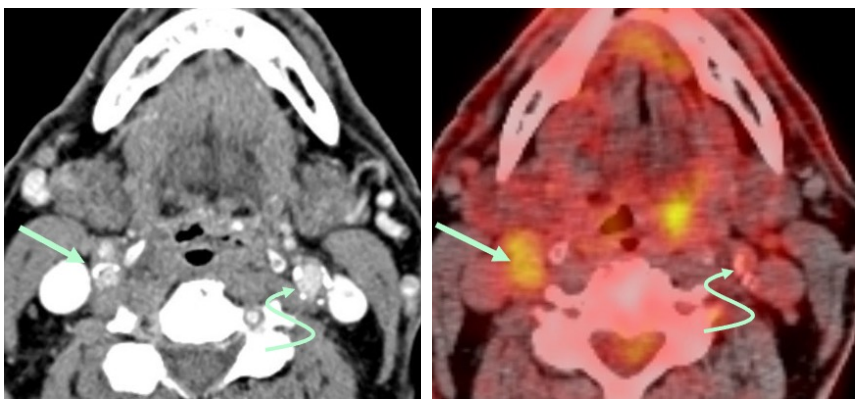
FDG PET CT (> see chapter on nuclear medicine) is a well-established hybrid imaging technique mainly used for oncologic indications. Nevertheless, PET CT can also be used to image inflammatory and infectious conditions.

The **main indications for FDG PET CT in vascular diseases** include atherosclerosis, vasculitis and complications of vascular grafts.

Because an atheromatous plaque represents an area of dynamic inflammation (see next pages), FDG PET CT can characterise the inflammatory state of a plaque. Vulnerable active plaques with a high risk of rupture accumulate FDG whereas calcified inactive plaques do not.

<!> ATTENTION

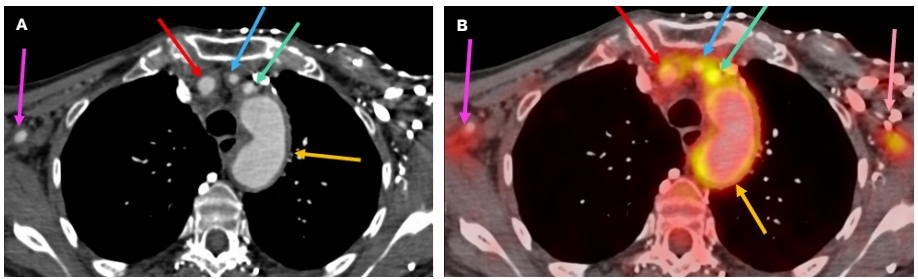
Higher FDG uptake in carotid plaques is associated with a higher risk of stroke.

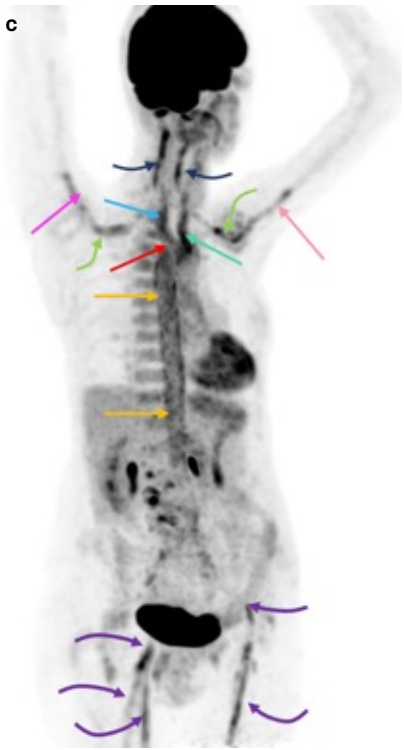


Focal, patchy FDG uptake in an active atherosclerotic plaque at the right carotid bifurcation (straight arrows) seen on a PET CT scan performed for follow-up purposes of a head and neck squamous cell carcinoma patient. Note that although both carotid bifurcations have mixed plaques, FDG uptake is seen only on the right. Mixed inactive plaque (curved arrows) on the left.

FDG PET CT can detect vessel wall inflammation in **Large Vessel Vasculitis** (LVV) before the development of obvious morphologic vessel wall changes. Typically, FDG PET CT in LVV reveals smooth, continuous and circumferential FDG uptake of the large vessel walls as opposed to the focal and discontinuous uptake in atherosclerosis.

Infection of vascular prosthetic grafts can be difficult to diagnose especially in cases with low-grade infection. FDG PET CT is very useful in this clinical situation as infected grafts typically show intense FDG uptake, either focal or focal-on-diffuse.





FDG PET CT in LVV. Contrast-enhanced CT (**A**) and corresponding fused PET CT image (**B**): regular thickening (3.9 mm) and smooth and linear FDG uptake of the wall of the aorta (orange arrow) and its branches (left common carotid artery (turquoise arrow), right common carotid artery (blue arrow), brachiocephalic trunk (red arrow)), right axillary (magenta arrow) and left axillary (light pink arrow) arteries. PET Maximum Intensity Projection (MIP) image (**C**): characteristic smooth linear increased FDG uptake (SUV max = 5.7) of the aorta and its branches. Note also FDG uptake of the subclavian (green arrow), vertebral (dark blue arrow) and common, superficial and profound femoral arteries (curved purple arrows) on this MIP.

Vascular Imaging

CHAPTER OUTLINE:

Arterial Disease

CHAPTER OUTLINE:

/ Atherosclerosis

Atherosclerosis is a progressive disease characterised by the accumulation of lipids and fibrous elements in large arteries. Atheromatous plaques **begin as fatty streaks composed of lipid-laden macrophages** (foam cells). This process begins in childhood but not all fatty streaks progress to plaques.

A **plaque is a raised focal lesion within the intima**. It consists of a necrotic core (lipids, foam cells and debris) surrounded by inflammatory cells, smooth muscle cells and neovascularisation can also be present. All of it is covered by a fibrous plaque. Plaques often undergo **calcification**.

The underlying pathogenesis is believed to involve **chronic endothelial injury (intima)** which results in an **inflammatory response**, accumulation of **lipids**, platelet aggregation and activation of smooth muscle cells.

Plaques form most commonly in large elastic arteries (e.g., aorta, carotid and iliac arteries), and large and medium-sized muscular arteries (e.g., coronary, renal, lower limb, mesenteric and cerebral vessels). They are most prominent at branching points and at ostia of major branches.

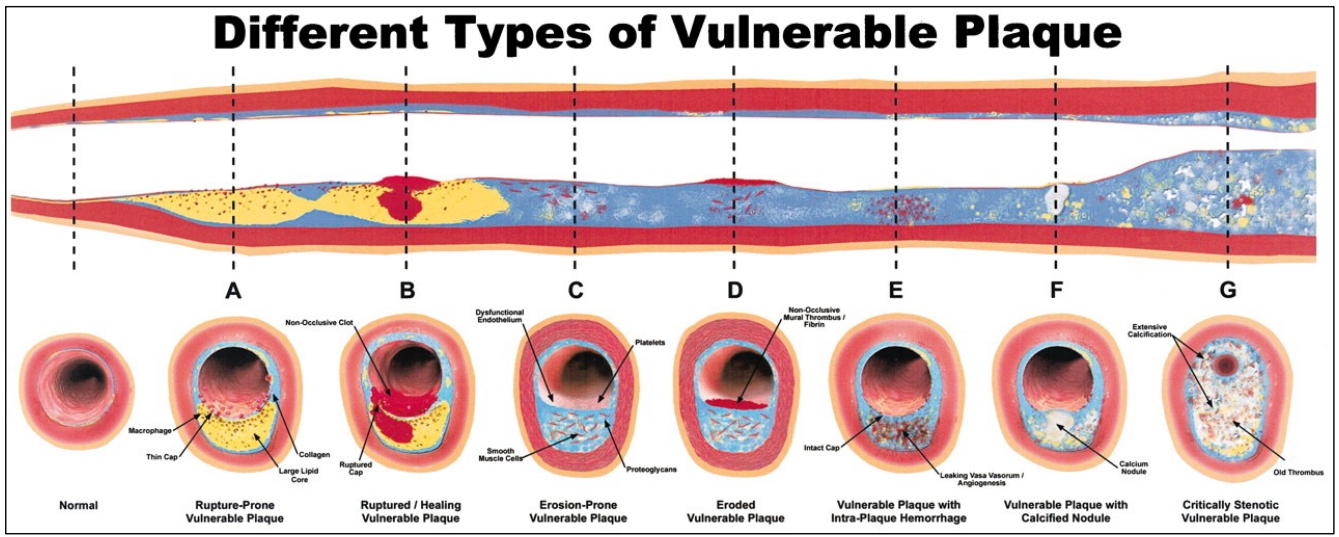
<!> ATTENTION

Vulnerable plaque = plaque susceptible to complications

Alternative acceptable terms include high-risk plaque, dangerous plaque, unstable plaque.

Plaque rupture is responsible for 70% of fatal acute myocardial infarction and/or sudden coronary death.

The **different types of vulnerable plaques** are shown in the figure below.



A. Rupture-prone plaque with large lipid core and thin fibrous cap infiltrated by macrophages. B. Ruptured plaque with subocclusive thrombus and early organisation. C. Erosion-prone plaque with proteoglycan matrix in a smooth muscle cell-rich plaque. D. Eroded plaque with sub-occlusive thrombus. E. Intraplaque haemorrhage secondary to leaking vasa vasorum. F. Calcific nodule protruding into the vessel lumen. G. Chronically stenotic plaque with severe calcification, old thrombus and eccentric lumen.

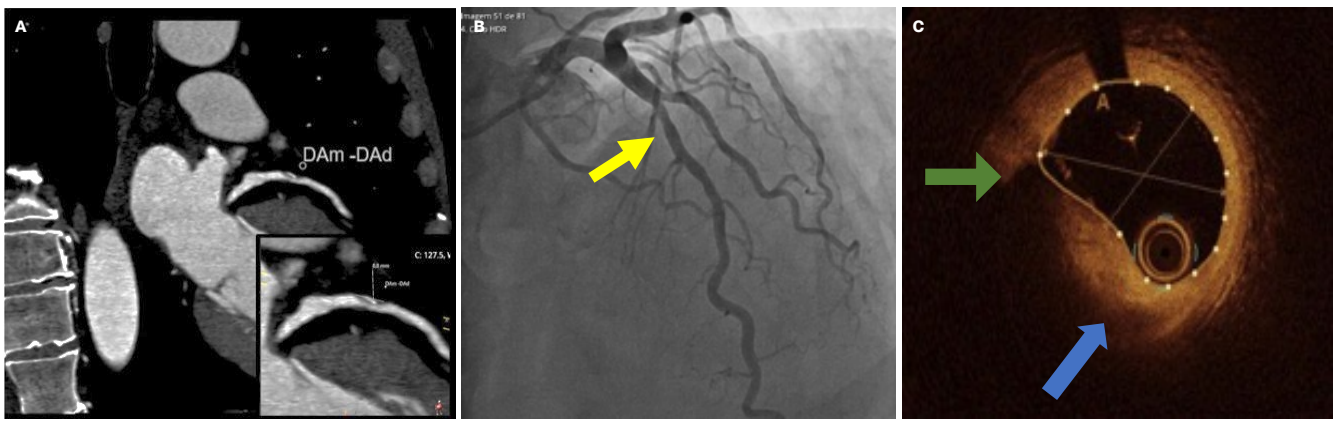
<∞> REFERENCE

Figure reproduced from: Morteza Naghavi. From Vulnerable Plaque to Vulnerable Patient. Circulation 108 (14); 1664-1672
DOI: (10.1161/01.CIR.0000087480.94275.97)

Vascular Imaging

CHAPTER OUTLINE:

CT, MRI, ultrasound (including **intravascular ultrasound**) and - more recently - **optical coherence tomography (OCT)** are used to analyse the atherosclerotic plaque structure, thus playing an important role in therapeutic decisions.



A. CT angiography of a patient with ECG changes (Biphasic T waves in precordial leads, and T wave inversion in precordial leads) showing mid left descending artery (LAD) moderate plaque lesion with unstable features with a 50-69% reduction of vessel calibre. **B.** Coronarography demonstrating a non-significant 40% stenotic segment of the LAD (yellow arrow). **C.** OCT at the stenotic segment showing a thin cap fibroatheroma (blue arrow) with a small rupture area (green arrow) and an overall 70% stenosis area.

<∞> REFERENCE

The role of optical coherence tomography in coronary intervention. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3295975/Terashima M, Kaneda H, Suzuki T. Korean J Intern Med. 2012 Mar;27(1):1-12. doi: 10.3904/kjim.2012.27.1.1 Epub 2012 Feb 28. PMID: 22403493; PMCID: PMC3295975.

A plaque can gradually enlarge, it can rupture and it can facilitate thrombus formation. The consequences are:

- **Critical stenosis** (= severe arterial narrowing with a significantly reduced maximal flow capacity in the distal vascular bed; typically, 60%-75% reduction in diameter of a large artery)
- **Ischaemia** (= insufficient blood flow to provide adequate oxygenation); ischaemia leads to tissue hypoxia or anoxia. Ischaemia can manifest with pain (e.g., angina, intermittent claudication) and/or loss of function (e.g., neurologic dysfunction)
- **Aneurysm formation** due to atrophy of the underlying media by the enlarging plaque

Atherosclerosis of the large intracranial arteries (typically affecting the middle cerebral artery, basilar artery, anterior or posterior cerebral arteries and the internal carotid artery) can cause a **transient ischaemic attack**, an **ischaemic stroke** or cognitive impairment due to **chronic white matter ischaemia**.

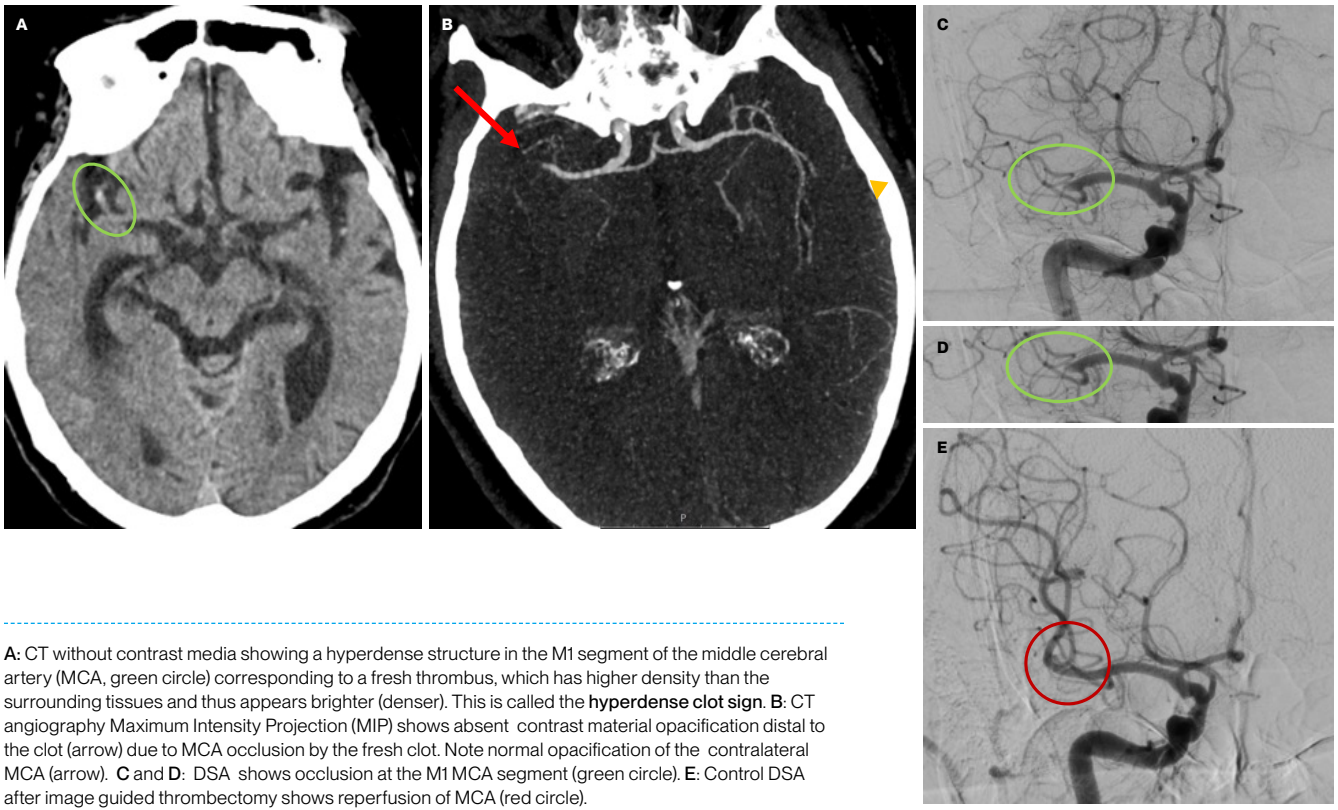
The role of **CTA in the acute setting** is:

- to identify the thrombus within a vessel, thus guiding intra-arterial thrombolysis or clot retrieval
- exclude intracranial haemorrhage
- identify the core infarct (i.e., the infarct part which does not recover despite recanalisation therapy) and the penumbra (potentially salvaged infarct zone)
- assess the status of collateral vessels (which are reliable predictors of clinical outcomes after endovascular interventional clot removal)

<∞> REFERENCE

> see eBook chapter on Central Nervous System

/ Middle Cerebral Artery (MCA) Occlusion



A: CT without contrast media showing a hyperdense structure in the M1 segment of the middle cerebral artery (MCA, green circle) corresponding to a fresh thrombus, which has higher density than the surrounding tissues and thus appears brighter (denser). This is called the **hyperdense clot sign**. **B**: CT angiography Maximum Intensity Projection (MIP) shows absent contrast material opacification distal to the clot (arrow) due to MCA occlusion by the fresh clot. Note normal opacification of the contralateral MCA (arrow). **C** and **D**: DSA shows occlusion at the M1 MCA segment (green circle). **E**: Control DSA after image guided thrombectomy shows reperfusion of MCA (red circle).

Peripheral arterial disease (PAD) is a common condition caused by atherosclerosis. It manifests with stenosis and/or thrombus formation or complete occlusion.

PAD can lead to limb ischaemia, which can present clinically with intermittent claudication in mild to moderate PAD (ischaemic pain during exercise due to tissue hypoxia caused by higher oxygen demand), with rest pain, ischaemic ulcers or gangrene.

Typically, the femoral and popliteal arteries are most often affected. Calcified atherosclerotic plaques along the vessels are common.

Stenoses and occlusions can be single, multiple, with or without calcification, with or without collateral vessels.

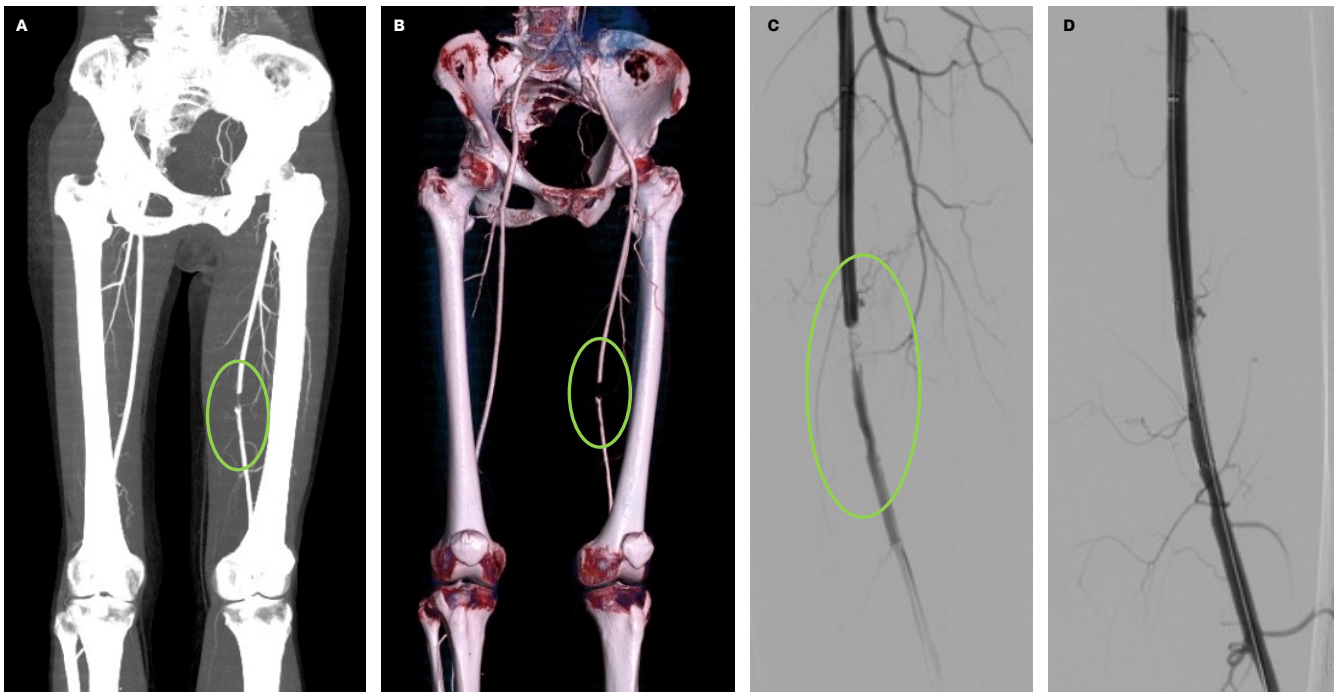
<!> ATTENTION

Both CTA and MRA can be used to assess PAD manifestations (stenosis, occlusion and collateral vessels).

However, MRA tends to sometime overestimate stenosis severity.

DSA is considered the gold standard for assessing PAD manifestations. In addition to its diagnostic value, DSA also allows radiological interventional endovascular therapy.

/ Superficial Femoral Artery Stenosis and Interventional Endovascular Radiologic Treatment



A and **B:** CTA with MIP and Volume rendering Technique (VRT) reconstruction depicting a left superficial artery stenosis (green circle). **C** and **D:** Lower limb DSA showing how interventional radiologic vascular treatment is performed with stenting (first passage of a guide wire – image **C**) and then stent placement (in **D**).

/ Pulmonary Embolism (PE)

Pulmonary embolism (PE) is defined as occlusion of a pulmonary artery or its branches. Most often it is caused by a clot traveling from the deep venous system (DVT emboli). Other rarer causes include fat, amniotic fluid, air or septic emboli.

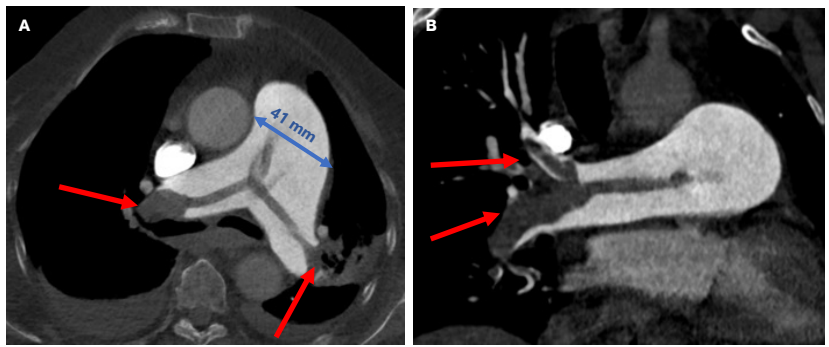
One should always suspect acute PE in a patient with a history of:

- Sudden onset dyspnea
- Pleuritic chest pain
- Tachycardia
- Hypoxia (proven by arterial blood gas test)

<!> ATTENTION

PE has a broad spectrum of symptoms. Several "scores" exist to stratify the probability of having PE (e.g., Well's score, Geneva Score).

CTA is the gold standard for the diagnosis of a suspected PE.



A (axial CT) and **B** (coronal CT image): CTA showing saddle pulmonary embolism (arrows) affecting the left and right pulmonary artery and segmental branches. Note enlargement of the pulmonary artery trunk (usually < 30 mm).

<!> ATTENTION **>=< FURTHER KNOWLEDGE**

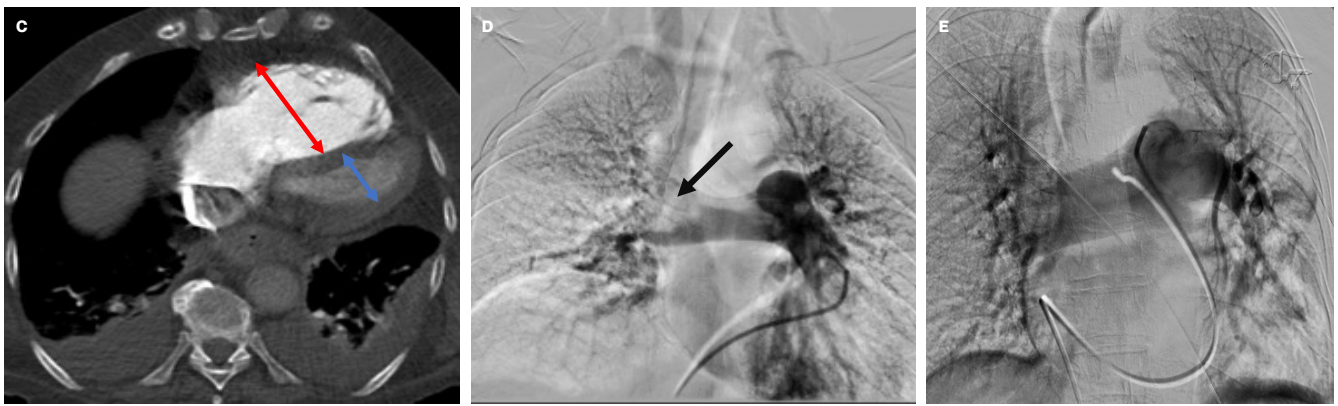
PE is stratified based on the haemodynamic burden to guide treatment and need for surgical intervention. PE is divided into 3 main severity groups:

- **High risk**-PE in the presence of hypotension (not caused by arrythmia)
- **Intermediate risk**-PE without hypotension but with signs of myocardial damage or right ventricle (RV) dysfunction
- **Low risk**-PE without hypotension or signs of myocardial damage or RV dysfunction

<!> ATTENTION

PE treatment depends on risk stratification:

- **High risk**- usually treated with anti-coagulation therapy
- **Intermediate risk**- can be treated with anti-coagulation alone or interventions (catheter guided thrombolysis and aspiration thrombectomy) depending on co-morbidities, evolution and other risk factors
- **Low risk**- systemic thrombolysis or local interventions (aspiration thrombectomy / open surgical embolectomy



C: CTA (continuation of the figure shown on the previous slide). Note enlargement of the right ventricle (RV) with interventricular septal deviation towards the left ventricle (LV) – indicative of a **RV/LV ratio** > 1, indicative of **right ventricle dysfunction**. This is a case of an intermediate risk PE (also known as sub-massive). **D**: This patient underwent catheter guided thrombectomy of the bilateral thrombi (note filling defect of the right pulmonary artery – arrow). **E**: final result after thrombectomy with complete opacification of the bilateral pulmonary arteries by the injected contrast material.

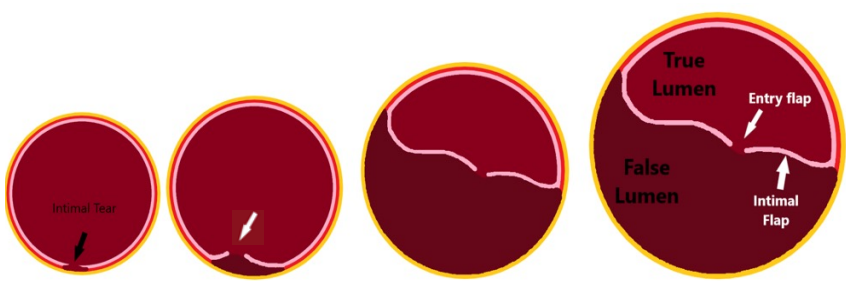
/ Dissection

Aortic dissection is defined as a **tear in the inner wall of the aorta**, with further inner layer separation (or dissection) **leading to the formation of a second blood-filled channel within the wall of the aorta**.

This can lead to a loss of blood flow to vital organs and can be life-threatening if not treated promptly.

Signs and symptoms of aortic dissection may include:

- / Sudden chest or back pain
- / Difficulty breathing
- / Dizziness/loss of consciousness
- / Blood pressure difference between both arms ≥ 20 mmHg



Pathogenesis and stages of aortic dissection:

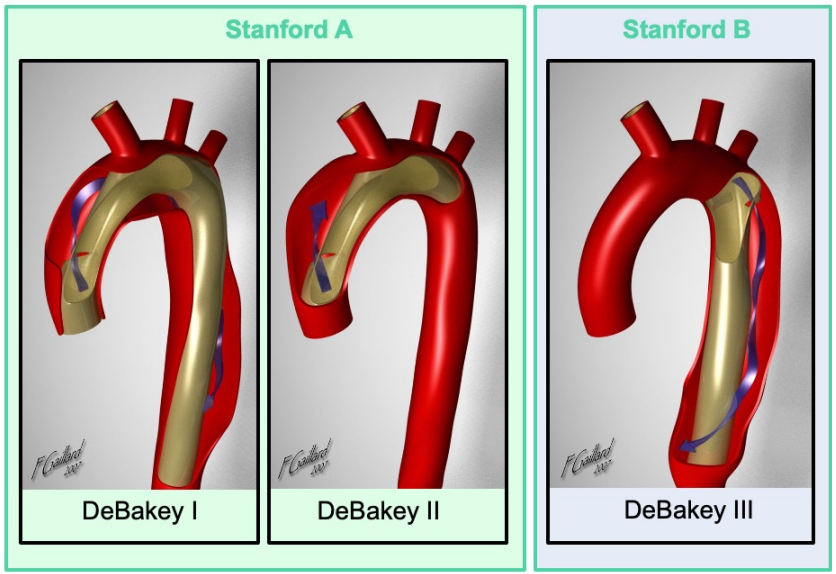
1. Aortic dissection starts from an intimal tear within the aorta
2. Blood enters and infiltrates the media (arrow)
3. A false channel or lumen is created separating the intima from the rest of the aortic wall
4. The displaced intimal flap may cause obstruction of a branch vessel which may result in end-organ hypo-perfusion

/ Aortic Arch Dissection Classification

The most common aortic dissection classification systems are Standford and DeBakey.

Stanford classification is based on the location of the intimal tear:

- **Type A:** involves any part of the aorta proximal to the origin of the left subclavian artery (ascending aorta)
- **Type B:** Dissection involving the descending aorta (with proximal tear distal to the origin of left subclavian artery)



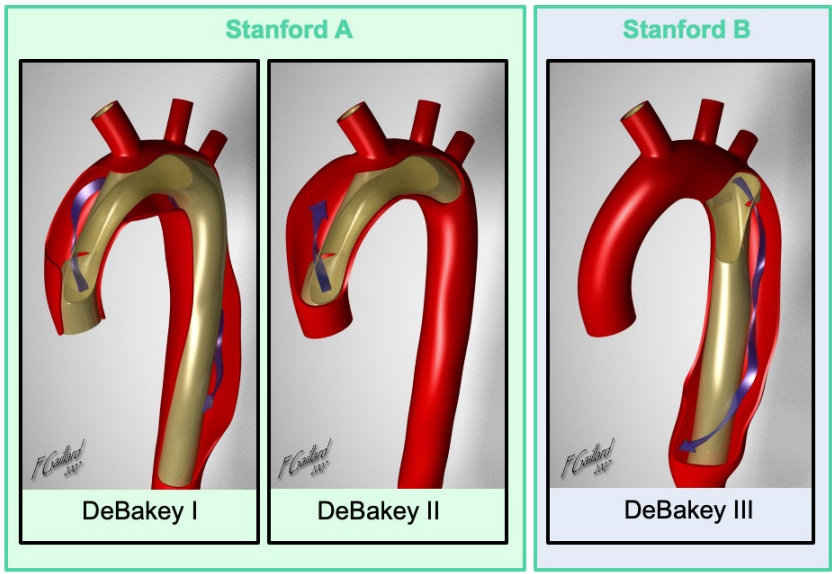
>=< FURTHER KNOWLEDGE

Consensus statement from society of Vascular surgery and Society of Thoracic Surgeons
https://www.sciencedirect.com/science/article/pii/S000349751931687X?via%3Dihub

Figures: Case courtesy of Frank Gaillard, Radiopaedia.org, rID: 7640

DeBakey classification (Type I-III):

- **Type** I: Ascending and Descending aorta (= Stanford A)
- **Type II:** involves ascending aorta only (= Stanford A)
- **Type III:** involves descending aorta only, starting after branching of the left subclavian artery (= Stanford B)



Figures: Case courtesy of Frank Gaillard, Radiopaedia.org, rID: 7640

<!> ATTENTION

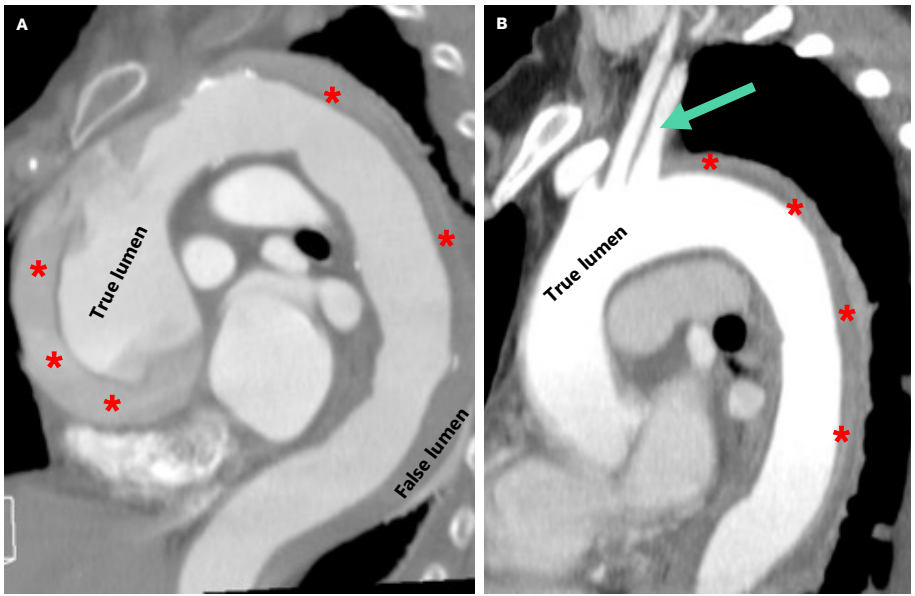
The division between Type A and Type B dissection is very important as it usually guides treatment:

Type A dissections can lead to thrombosis of the coronary arteries and have an increased risk of rupture and cardiac tamponade = **Urgent surgical intervention!**

Type B dissections are usually **treated conservatively** with blood pressure control.

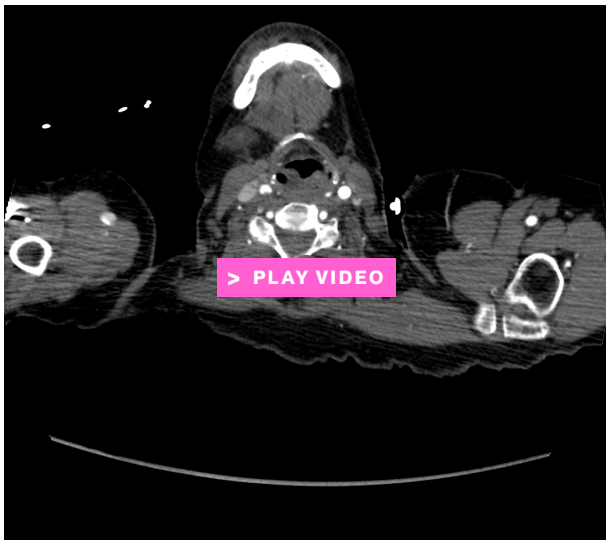
Stanford **Type A** dissection

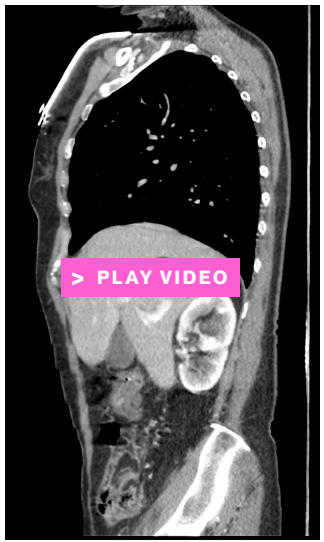
Stanford **Type B** dissection



CTA of the aorta in arterial phase: Oblique sagittal CTA reconstruction (**A**) showing dissection (*) of the ascending and descending aorta - Stanford Type A dissection (DeBakey I). Sagittal CTA reconstruction in a different patient showing dissection of the descending aorta, distal to the branching of the left subclavian artery (arrow)– Stanford Type B dissection (DeBakey III)

Stanford **Type A** dissection

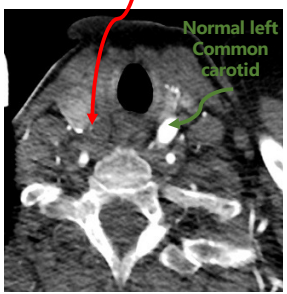




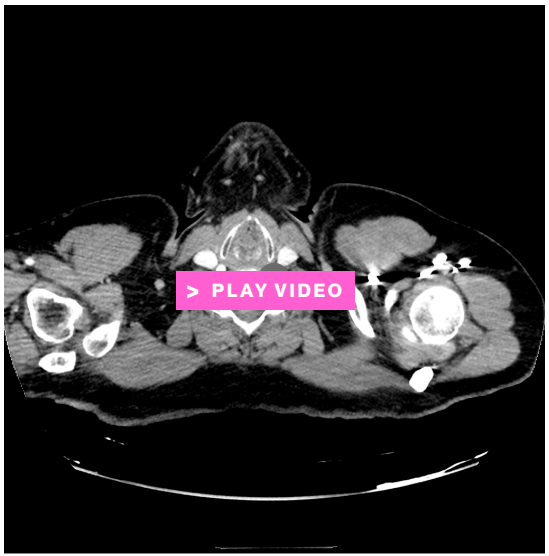
CTA of the aorta (videos): Axial and sagittal reconstructions showing the dissection starting in the ascending aorta and extending all the way to the suprarenal abdominal aorta – Stanford Type A dissection.
Click to Play Video in Browser (External)

<!> ATTENTION

Did you notice the right common carotid artery thrombosis?



Stanford **Type B** dissection



CTA of the aorta (videos): Axial and coronal reconstructions showing dissection starting bellow the left subclavian artery and extending to the infra-renal aorta – Stanford Type B dissection.

<!> ATTENTION >=< FURTHER KNOWLEDGE

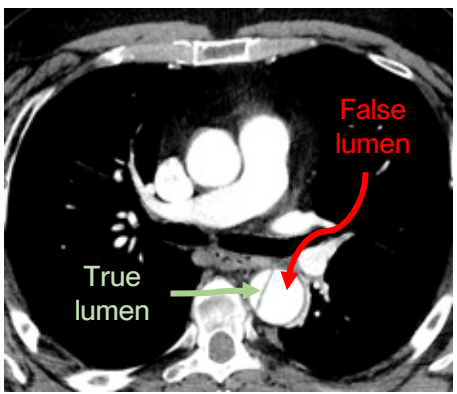
Some clues to distinguish the true lumen from the false lumen in a dissection:

False lumen:

- Larger than true lumen
- Delayed enhancement
- Outer curve of the arch
- Usually, origin of left renal artery
- Beak-sign (wedges around true lumen)

True lumen:

- Smaller than false lumen
- Surrounded by calcifications (when present)
- Usually origin of celiac trunk, superior mesenteric artery and right renal artery



CHAPTER OUTLINE:

/ Aneurysm

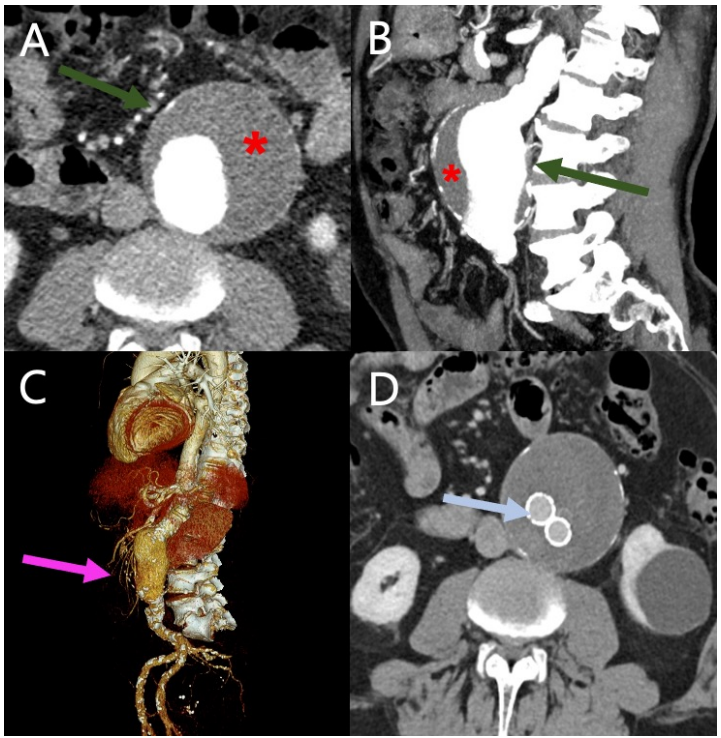
An aneurysm is an abnormal dilatation in a blood vessel due to the weakness of the vessel wall. While aneurysms can affect any blood vessel, they are most common in arteries rather than veins.

Aneurysms can be a **true** or **false**.

A **true** aneurysm contains all the three layers of the arterial wall (intima, media and adventitia)

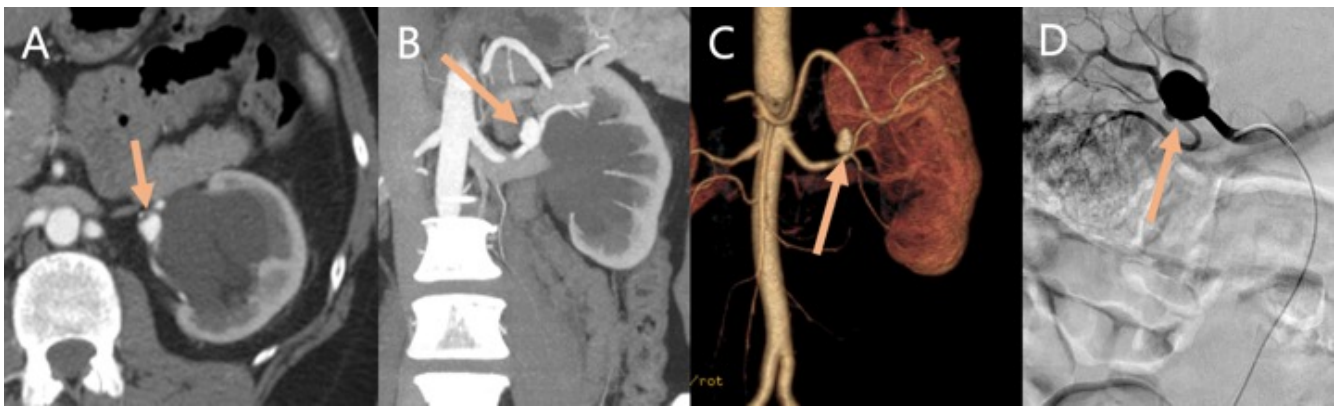
On the other hand, a **false** aneurysm (also known as pseudoaneurysm), involves only the adventitia.

According to their shape, they can be **saccular** or **fusiform**.



Images **A**, **B**: CTA of a fusiform infra-renal abdominal aortic aneurysm (greenarrows) measuring 7 x 7 cm (axial) and 11 cm (cranio-caudal extension). There is circumferential thrombosis (area with no enhancement surrounding the lumen with contrast) (*) measuring 13 mm. **C**: 3D reconstruction demonstrating the aneurysm (pink arrow). **D**: CTA axial slice showing abdominal aneurysm excluded by an Endovascular aortic repair (EVAR) graft (blue arrow).

/ Renal Artery Aneurysm



Images **A-D**: **Aneurysms of the renal arteries.**

A: Focal dilatation of the left renal artery at the renal hilum (arrow).

B: MIP coronal reconstruction showing enhancement of the blood vessels and of the left renal artery aneurysm (arrow).

C: 3D reconstruction of the abdominal aorta, left and right renal arteries with improved visualisation of the renal artery saccular aneurysm (arrow).

D is an example of a DSA of another patient with a right renal artery fusiform aneurysm involving the origin of the 3 renal branches (arrow).

/ Vasculitis

Vasculitis = generalised vessel inflammation. It can have many different clinical presentations and can involve any organ in the human body. Most vasculitis types are the consequence of immune related phenomena.

There are several classification systems, that partially overlap. The **Revised International Chapel Hill Consensus Conference Nomenclature of Vasculitis** distinguishes between primary and secondary vasculitis.

Primary vasculitis – The aetiology is unknown; these are heterogeneous, multi-system disorders characterised by inflammation and necrosis of large (e.g., Takayasu arteritis), medium (e.g., polyarteritis nodosa) and small blood vessels (e.g., granulomatosis with polyangiitis formerly called Wegener's granulomatosis). It also includes vasculitis affecting vessels only in a single organ (e.g., aortitis), as well as variable vessel vasculitis (e.g., Behçet syndrome).

Secondary vasculitis – The aetiology is known or is highly suggestive. It is subdivided into – **vasculitis associated with systemic disease** (lupus vasculitis, rheumatoid vasculitis, sarcoid vasculitis) and **vasculitis associated with a probable aetiology** (hepatitis C and B vasculitis, syphilis associated aortitis, vasculitis secondary to bacterial or viral infection).

>=< FURTHER KNOWLEDGE

2012 Revised International Chapel Hill Consensus Conference Nomenclature of Vasculitis

https://onlinelibrary.wiley.com/doi/10.1002/art.37715

Jennette, J.C., Falk, R.J., Bacon, P.A., Basu, N., Cid, M.C., Ferrario, F., Flores-Suarez, L.F., Gross, W.L., Guillevin, L., Hagen, E.C., Hoffman, G.S., Jayne, D.R., Kallenberg, C.G.M., Lamprecht, P., Langford, C.A., Luqmani, R.A., Mahr, A.D., Matteson, E.L., Merkel, P.A., Ozen, S., Pusey, C.D., Rasmussen, N., Rees, A.J., Scott, D.G.I., Specks, U., Stone, J.H., Takahashi, K. and Watts, R.A. (2013), 2012 Revised International Chapel Hill Consensus Conference Nomenclature of Vasculitides. Arthritis & Rheumatism, 65: 1-11. https://doi.org/10.1002/art.37715

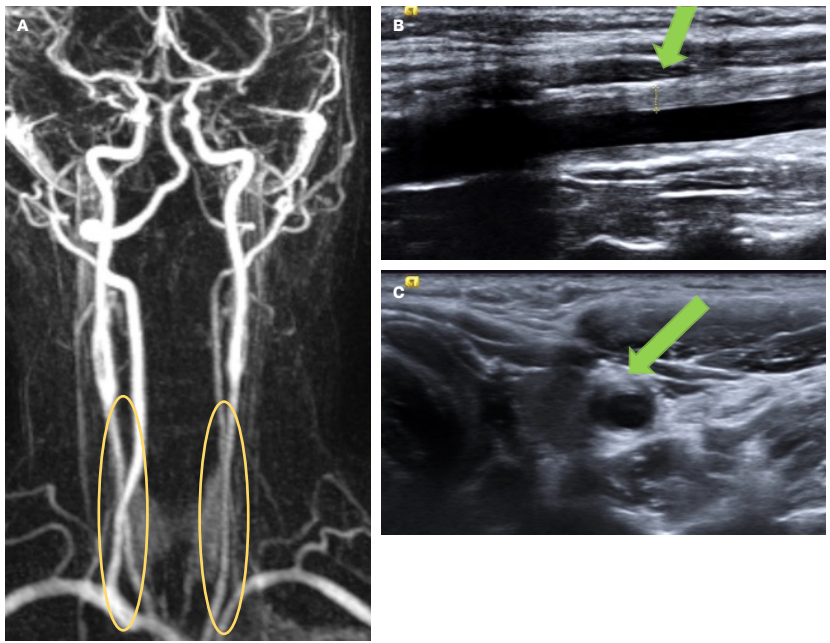
/ Takayasu Arteritis (Pulseless Disease)

= Large vessel granulomatous vasculitis typically affecting the aorta and its main branches, e.g., the common carotid arteries, brachiocephalic trunk and subclavian arteries.

After an initial systemic manifestation with fever, night sweats and arthralgia, follows a chronic phase with limb ischaemia, hypertension (renovascular), cardiac complications and pulmonary arterial involvement.

On US, there is thickening of the arterial wall ± secondary thrombus formation ± occlusion.

CT/MRI can additionally show vessel wall enhancement, aneurysm and pseudoaneurysm and diffuse narrowing of the distal aorta.



A. 3D Contrast-enhanced MRA (MIP) in a patient with Takayasu arteritis **shows stenoses of the common carotid arteries**, with normal sized internal and external carotids. Longitudinal (**B**) and axial US view (**C**) of neck vessels showing **thickening of left common carotid artery wall**.

/ Fibromuscular Dysplasia (FMD)

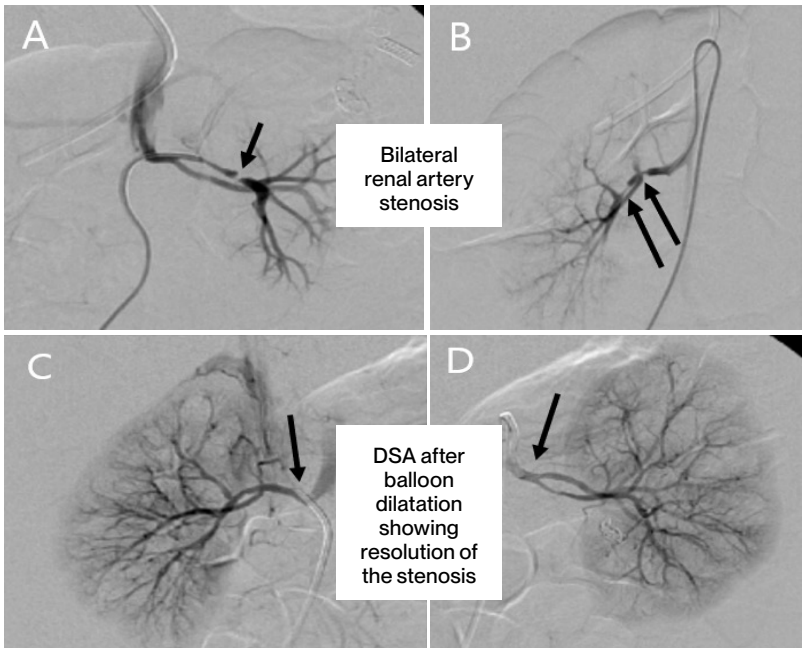
Fibromuscular dysplasia (FMD) is an idiopathic, focal, non-inflammatory and non-atherosclerotic disease that affects small and medium size arteries.

FMD most frequently affects the renal and neck arteries (carotid and vertebral). It's more prevalent in young women and is often asymptomatic.

Patients that are symptomatic usually present with:

- Hypertension (due to renal artery stenosis, which usually is bilateral)
- Headaches, TIA or even stroke (when the carotids and vertebral arteries are involved)
- Myocardial infarction or angina pectoris (due to coronary involvement)

Most common radiological findings include vessels with a “string of beads appearance” due to focal stenosis intercalating with small aneurysms typically affecting the mid segment of the vessel and sparing the origins.



Renal DSA of the left (**A**) and right (**B**) renal arteries in a patient with bilateral FMD of the renal arteries. For endovascular treatment, the interventional radiologist performs a DSA with insertion and subsequent inflation of a balloon dilation catheter at the site of stenosis, which usually results in stenosis resolution. Right renal artery (**C**) and left renal artery (**D**) after balloon dilatation. If dilatation failure, a stent can be placed.

/ Arterial Compression Syndromes

Vascular compression syndromes can be divided into several groups: (1) a vascular structure is the "compresser"; (2) the vascular structure is the "compressee"; (3) a vascular structure compresses another vascular structure.

Examples of vascular compression syndromes include:

- Anomalous origin of the coronary artery (which courses between the ascending aorta and the pulmonary trunk)
- Hypothenar hammer syndrome (compression of the ulnar artery by the hypothenar muscles)
- Eagle syndrome (see below) and many more

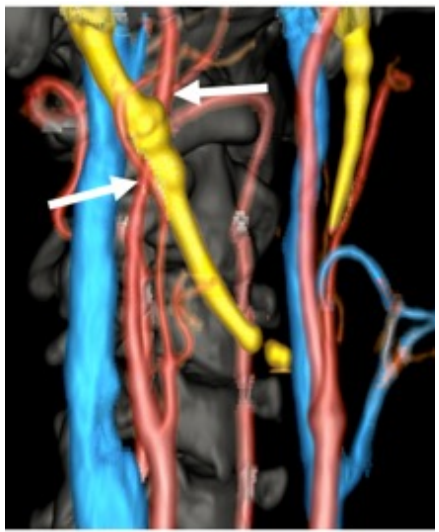
>=< FURTHER KNOWLEDGE

Eagle Syndrome

= elongation of the styloid process causing pain due to compression of the cranial nerves IX (glossopharyngeal nerve) or X (vagus nerve) or due to compression of the carotid artery, in which case the pain is mediated by the sympathetic plexus along the carotid artery. Compression of the carotid artery can also lead to stroke.

An elongated styloid process measures > 3 cm. It can be unilateral or bilateral.

Not all patients with an elongated styloid process are symptomatic. In fact, the vast majority are asymptomatic. In symptomatic patients, resection of the styloid process is carried out, which leads to immediate symptom relief.



Eagle syndrome with compression of the internal carotid artery
Compression of the internal carotid artery (arrows) by the calcified stylohyoid ligament (rendered in yellow). Arteries are rendered in red, veins in blue. This is a 3D reconstruction from a contrast-enhanced CT.

Venous Disease

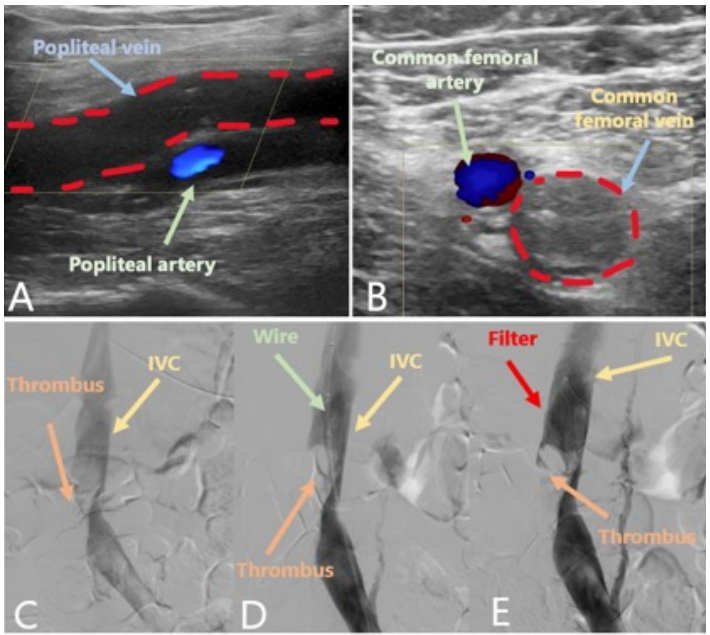
CHAPTER OUTLINE:

/ Deep Vein Thrombosis (DVT)

Clots within the deep veins are more likely to produce a clinically significant PE because these clots are usually larger than those in the superficial system. Also, because they are surrounded by muscle, the chance of the clot being dislodged during muscle contraction is higher than for a clot in the superficial veins. For these reasons, the focus in a venous duplex examination is on the deep system.

Don't forget that thrombosis can also happen on the upper limbs and neck vessels.

Doppler US is a perfect tool for identifying and evaluating blood clots, thus allowing physicians to take actions to minimise the risks of clot embolisation and pulmonary embolism.



A and **B**: Doppler US of the thigh demonstrating occlusive thrombosis (**highlighted in red**) of the deep venous system of the left lower limb extending from the popliteal vein (**A**) to the left common femoral vein (**B**) and to the infra-renal inferior vena cava (IVC). Note only partly occlusive thrombus seen as a filling defect in **C** - arrow. This patient could not have anti-coagulation therapy, therefore an **IVC filter** was placed (**D-E**).

>=< FURTHER KNOWLEDGE

Inferior Vena Cava Filter
https://www.ncbi.nlm.nih.gov/books/NBK549900/

/ Portal Hypertension (PH)

Portal Hypertension (PH) is defined as increased pressure in the portal venous system. A hepato-venous pressure gradient (HVPG) > 5 mmHg or "in simpler terms" - a pressure gradient between one hepatic vein and the portal vein > 5 mmHg makes the diagnosis.

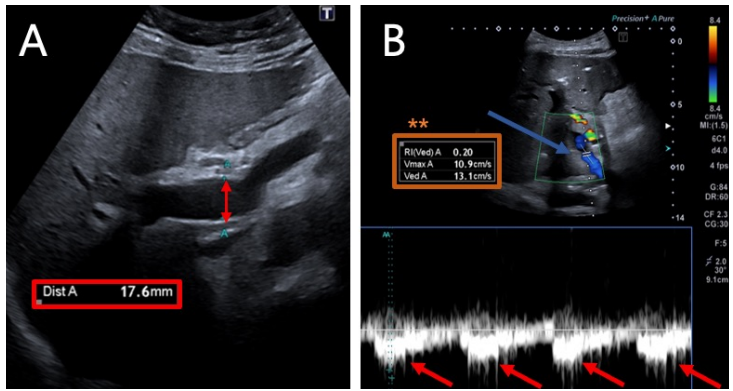
This becomes clinically important when the pressure gradient rises to over 10 mmHg, due to the increased risk of complications.

The causes of PH can be divided according to their relationship to the hepatic sinusoids into:

- **Pre-hepatic**: AV fistula, portal vein thrombosis
- **Hepatic**: cirrhosis (**most common**), hepatitis
- **Post-hepatic**: Budd-Chiari, congestive heart failure

Measuring the HVPG is an invasive task, and most of the time the diagnosis is made indirectly with surrogate markers of PH.

Duplex Doppler US, integrated with liver and spleen elastography represent the first line imaging method in suspected PH.



<!> ATTENTION

Most common findings of PH include:

- Portal vein dilatation (> 14 mm) – image **A**
- Splenomegaly (> 13 cm bipolar length)
- Ascites
- Low portal venous velocity (Doppler) < 16 cm/s (**)
- Porto-systemic shunts
- Reversal/hepatofugal flow in the portal vein (late finding)

Image **B** – colour mode shows **portal venous blood flow in blue**, and the **spectral waveform is displayed bellow the baseline** – meaning blood is moving away from the probe, i.e., portal blood is moving away from the liver when it should normally move towards the liver hilum!

/ **Vascular** Imaging

CHAPTER OUTLINE:

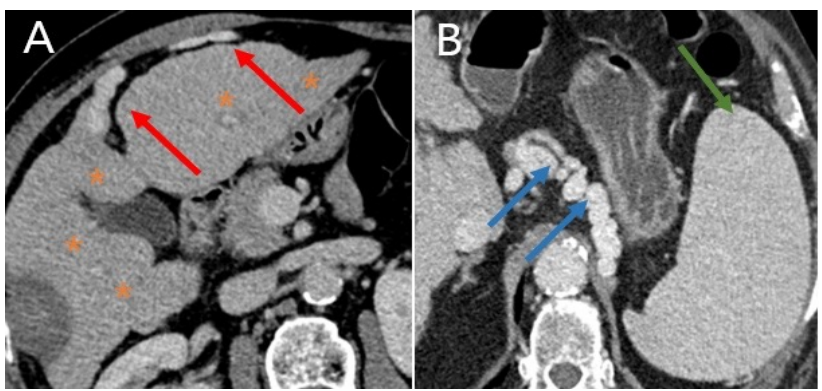
When the HVPG is above 10 mmHg, the pressure in the portal venous system (PVS) is so high that spontaneous porto-systemic collaterals begin to appear. These porto-systemic shunts or varices are connections between the portal venous system and the systemic circulation, allowing splanchnic blood to bypass the liver.

Most common varices include:

- Oesophageal and paraesophageal varices
- Left gastric varices
- Retro-gastric
- Paraumbilical vein recanalisation (so-called *Caput Medusae*)
- Superior rectal vein (haemorrhoids)
- Spleno-renal shunts

While these connections help alleviate the pressure in the PVS, they bring dire consequences to the patient:

- **Risk of rupture and massive bleeding**
- Hepatic encephalopathy
- Hepato-renal syndrome
- Hepatopulmonary syndrome



Axial contrast-enhanced CT slice of the portal phase (**A**) demonstrating an irregular and heterogenous liver (cirrhotic, *) with recanalisation of the paraumbilical vein (arrows). Axial contrast-enhanced CT slice of the portal phase (**B**) in another patient showing an increased spleen size (splenomegaly, arrow) and an abnormally engorged and tortuous varicose left gastric vein (arrow).

CHAPTER OUTLINE:

/ Budd-Chiari Syndrome (BCS)

Budd-Chiari syndrome (BCS) is a potentially life-threatening disorder characterised by occlusion of the hepatic outflow tract usually at the level of the hepatic veins or the inferior vena cava.

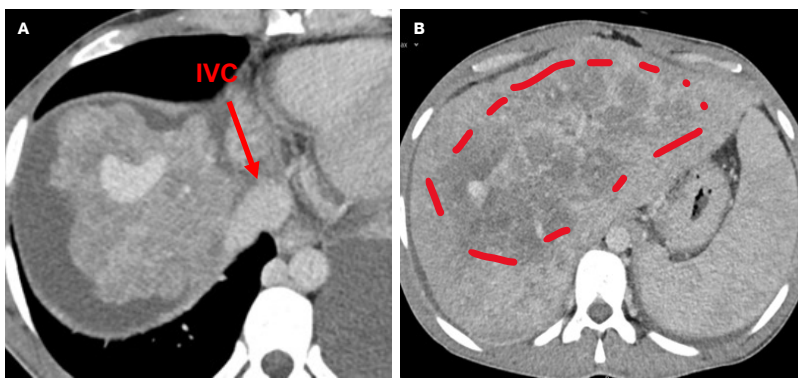
Most common causes include hypercoagulable conditions and myeloproliferative diseases resulting in thrombosis of at least two hepatic veins.

The classic acute presentation is the triad of ascites, abdominal pain and hepatomegaly.

While US is usually sufficient for confirming the diagnosis, CT and/or MRI is often necessary for planning further treatment. Imaging features depend on the extent and duration of the disease.

Most common findings include:

- No identifiable hepatic veins
- Hepatosplenomegaly
- Early enhancement of the caudate lobe
- "Flip-flop appearance"- i.e., delayed enhancement of the peripheral liver with a more hypodense central parenchyma



A and **B**: Axial contrast-enhanced CT (portal phase) demonstrating no apparent hepatic veins entering the IVC, associated with a very heterogenous liver showing different contrast enhancement between the peripheral and central liver parenchyma

Whilst anticoagulation is the cornerstone of treatment, most patients will need additional (more invasive) treatment like hepatic vein stenting, Transhepatic Intrajugular Portosystemic Shunt (TIPS) or liver transplantation due to PH.

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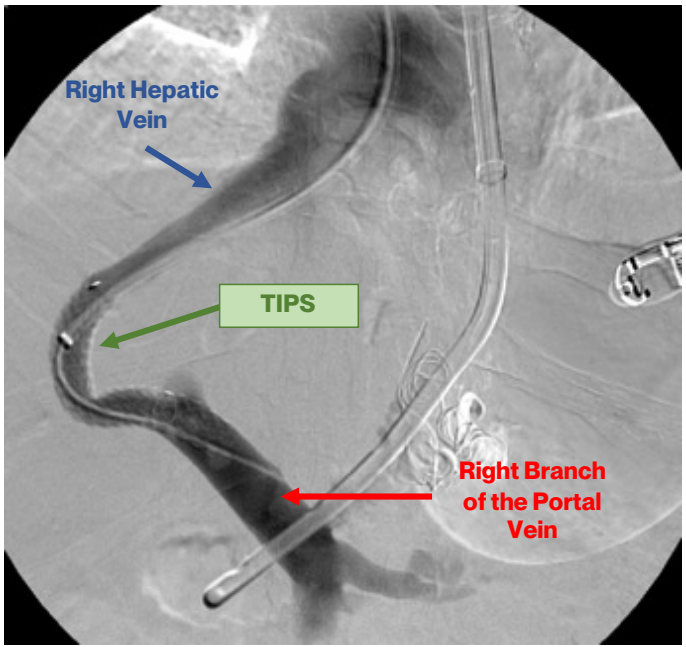
In cases of severe PH with refractory variceal bleeding, hepatorenal syndrome or hepatic vein occlusion, an interventional radiologist can perform a TIPS procedure. This involves the creation of a "bridge" between a branch of a portal vein and one the hepatic veins (usually the right one) allowing for the splanchnic blood to bypass the liver and therefore reduce PH to "safer" levels.



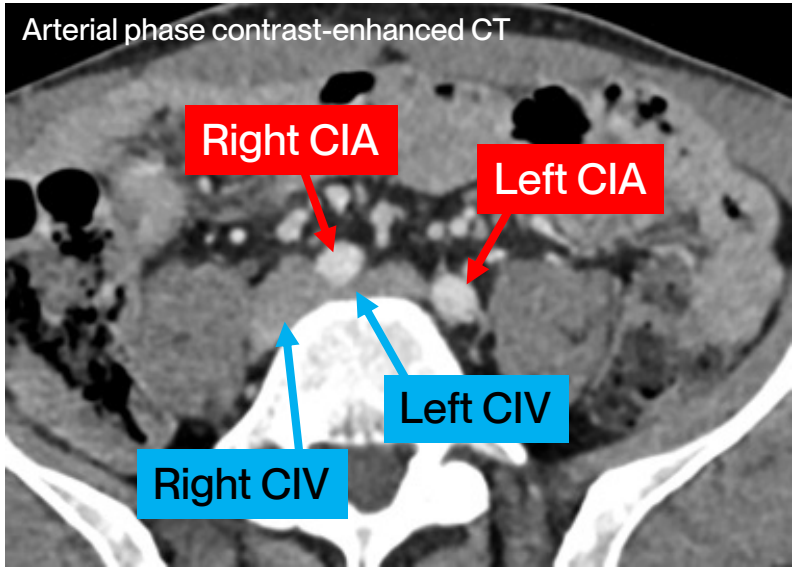
DSA demonstrating a stent creating a connection between the right branch of the portal Vein (red arrow) and the right hepatic vein (blue arrow) – TIPS (green arrow).

CHAPTER OUTLINE:

/ Venous Compression Syndromes

Venous compression syndromes include:

- / May-Thurner syndrome (compression of the left common iliac vein, see below)
- / Nutcracker syndrome (compression of the left renal vein by the superior mesenteric artery)
- / Posterior nutcracker syndrome (trapping of the retro-aortic left renal vein between the aorta and vertebral column)
- / Paget Schroetter syndrome («effort thrombosis» due to subclavian vein compression in the costoclavicular space)



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The May-Thurner syndrome is characterised by chronic compression of the left common iliac vein (CIV) against the lumbar vertebrae by the overlying right common iliac artery (CIA), with or without deep venous thrombosis.

Compression of the left CIV is more common than compression of the right CIV as the former has a more transverse course.

Pregnancy or long immobilisation are predisposing factors.

Vascular Tumours and Malformations

CHAPTER OUTLINE:

/ Definitions and Classification

Based on improved diagnostic and genetic features, according to the **International Society for the Study of Vascular Anomalies (ISSVA)**, vascular anomalies are divided into vascular tumours and vascular malformations.

Vascular tumours are neoplastic lesions, which can regress spontaneously (e.g., infantile haemangioma). They show increased proliferation of endothelial and vascular cells. Vascular tumours are classified into:

- Benign tumours
- Locally aggressive or low metastatic risk intermediate malignant tumours and
- Malignant tumours

<!> ATTENTION

Use of the contemporary nomenclature is important to ensure appropriate management of vascular anomalies.

Therefore, the term "lymphatic malformation" should be used instead of the older terms "lymphangioma" or "cystic hygroma". Likewise, the term "venous malformation" should be used instead of "cavernous haemangioma".

US, CT and MRI play an essential role for the diagnosis of vascular tumours and malformations and are, therefore, pivotal for patient management.

Vascular malformations are non-neoplastic structural anomalies. They can be subdivided into:

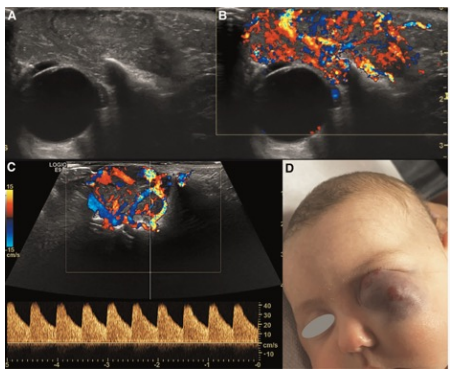
- Simple malformations > capillary, venous, lymphatic, arteriovenous malformations (AVMs) and arteriovenous fistulae (AVF) versus combined malformations > having > than one vascular component, e.g., lymphatic and venous
- High-flow malformations > with an arterial component, e.g., AVMs, AVF versus low-flow malformations > without an arterial component, e.g., capillary, venous or lymphatic
- Channel type malformations = malformations of major vessels
- Malformations associated with syndromes, e.g., syndromes with venous malformations (Sturge-Weber, Klippel-Trénaunay, Proteus, blue rubber bleb naevus, Maffucci and Gorham-Stout) versus syndromes with high-flow malformations (Rendu-Osler-Weber, Cobb, Wyburn-Mason, Parkes Weber)

/ Benign Vascular Tumours

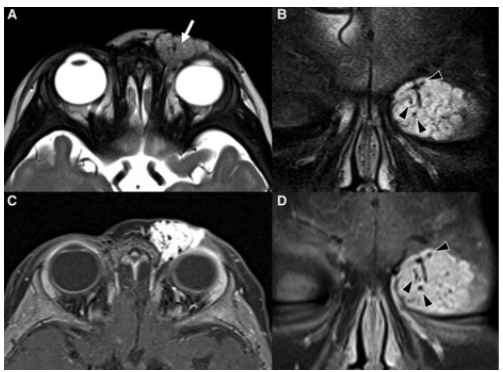
Benign vascular tumours include pyogenic granuloma, haemangioma and Masson tumours. **Haemangiomas** can be divided into infantile and congenital forms. Infantile haemangiomas (absent at birth) usually have a triphasic growth: rapid initial proliferation, stabilisation and then regression until total involution.

>=< FURTHER KNOWLEDGE

At Doppler-US, the tumours are hypervascular. At MRI, haemangiomas in the proliferation phase are strongly hyperintense on T2, they show flow-voids and display major enhancement after iv. contrast administration. Involuting haemangiomas have a fibro-fatty aspect (high T1 signal) and they display decreased enhancement.



US of an infantile haemangioma (IH) of the left orbit in a 5-month-old boy, during the proliferative phase. B-mode US shows a hyperechoic mass with well-defined margins overlying the globe (**A**) and a very high vascular density at Colour-Doppler US (**B**). Spectral analysis revealed a low resistance arterial flow (**C**). The clinical appearance of the lesion is depicted in (**D**).



MR images of a left periorbital IH in a 1-year-old girl. Axial (**A**) and coronal fat-saturated (**B**) T2 weighted images show a well-defined hyperintense mass (arrow in **A**) with multiple internal flow voids (black arrowheads in **B** and **D**), extending from the anterior periorbital soft tissues into the orbit. Axial (**C**) and coronal (**D**) contrast-enhanced fat-saturated T1 weighted images show vivid homogeneous contrast enhancement of the vascular lesion.

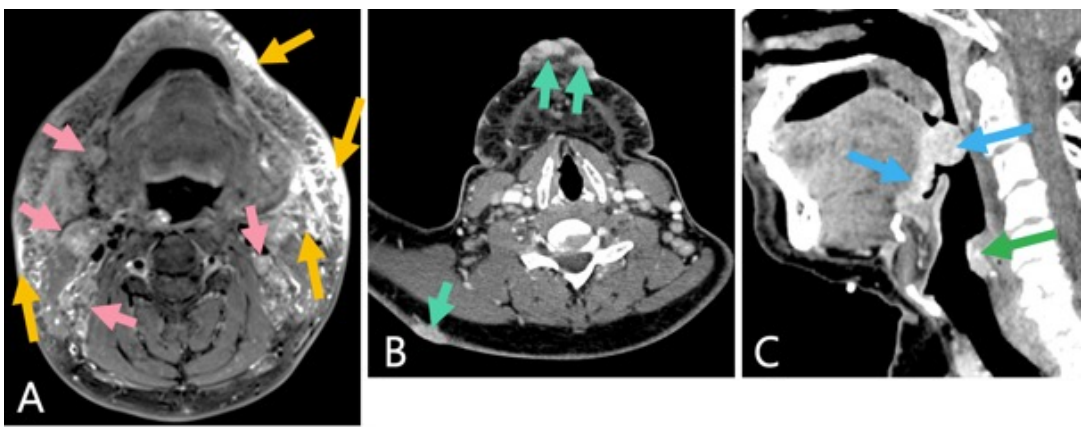
Both figures are reproduced from: Colafati GS, Piccirilli E, Marrazzo A, Carboni A, Diociaiuti A, El Hachem M, Esposito F, Zama M, Rollo M, Gandolfo C, Tomà P. Vascular lesions of the paediatric orbit: A radiological walkthrough. Front Pediatr. 2022 Nov 30;10:734286. doi: 10.3389/fped.2022.734286. PMID: 36533238; PMCID: PMC9748295.

/ Locally Aggressive Vascular Tumours

Locally aggressive vascular tumours include **kaposiform haemangioendothelioma** and **Kaposi sarcoma (KS)**.

KS arises from lymphatic endothelial cells. There are **four types of KS**: classic, post-organ transplant, AIDS-related and endemic (in Africa). Infection with the Human Herpes type 8 virus plays a major role in the aetiology of KS. The most common involvement in KS is subcutaneous and mucosal but deep organ involvement can also occur., e.g., the lungs. At CT and MR imaging, KS typically manifests with **strongly enhancing widespread cutaneous, subcutaneous and mucosal lesions**.

>=< FURTHER KNOWLEDGE



Characteristic MRI (**A**) and CT appearance (**B** and **C**) of KS in three different patients. Note widespread cutaneous and subcutaneous enhancement (yellow arrows) on the fat saturated contrast-enhanced T1, as well as enlarged enhancing lymph nodes (pink arrows),, enhancing cutaneous nodules (turquoise arrows) at contrast-enhanced CT and nodular strongly enhancing mucosal masses involving the base of the tongue (blue arrows) and the posterior larynx (green arrow)..

/ Malignant Vascular Tumours

Malignant vascular tumours include epitheloid haemangioendothelioma and angiosarcoma.

Angiosarcomas arise from vascular endothelial cells. Risk factors include lymphoedema, radiation therapy, exposure to toxic substances (e.g., vinyl chloride) and genetic predisposition (e.g., neurofibromatosis type I). Prognosis is poor. Angiosarcoma often involves the skin and subcutaneous vessels of the scalp. It can also involve the aorta and pulmonary arteries, the heart, chest wall and breast. Extravascular extension and metastases are common. Angiosarcomas are FDG avid.

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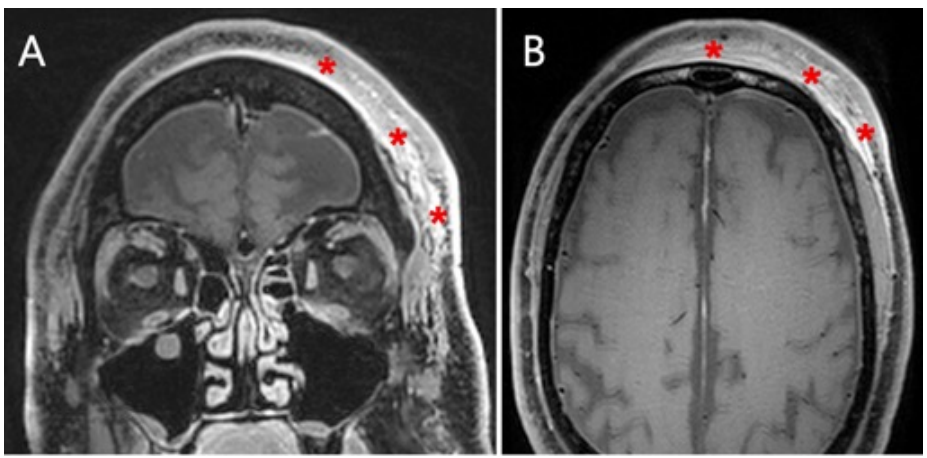


Characteristic MRI (A and B) appearance of an angiosarcoma of the scalp. Note infiltrative **cutaneous, subcutaneous and galea enhancement extending to the periosteum of the frontal bone** on the fat saturated contrast-enhanced coronal and axial T1-weighted images (A and B). At histology, the tumour invaded the galea aponeurotica and the periosteum of the frontal bone.

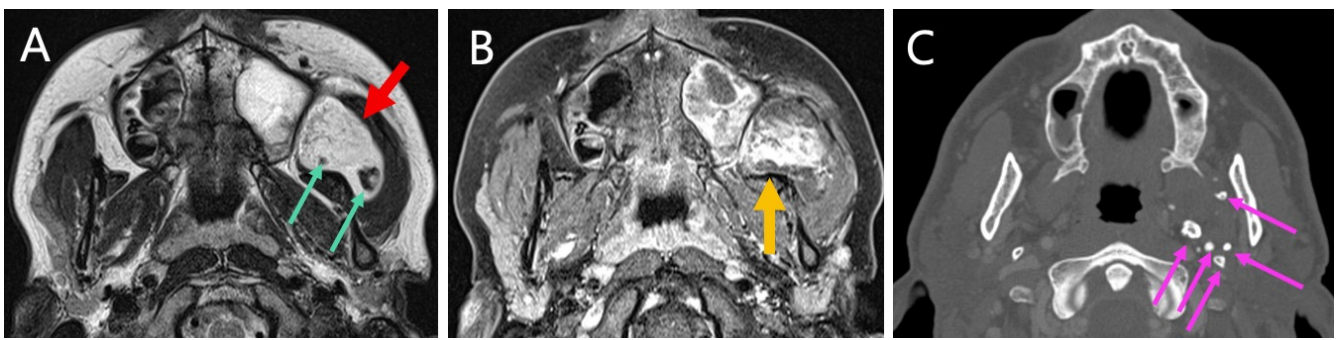
/ Vascular Malformations

Venous malformations are the **most common vascular** anomalies. They usually present early in life and do nor regress spontaneously. Most often, they involve multiple anatomic spaces and they are heterogeneous at imaging.

>=< FURTHER KNOWLEDGE

Characteristic imaging features include:

- US => venous flow or no flow
- Phleboliths (which can be seen at US, CT and MRI) in about 40% of cases
- MRI: high T2 signal and **absent** flow voids (as opposed to haemangiomas)
- Contrast enhancement: variable
- Contrast-enhanced **dynamic time-resolved** MRA: no arterial enhancement but gradual, persistent and late enhancement



Axial T2-weighted (**A**) and contrast enhanced fat saturated T1-weighted (**B**) images show the typical features of a venous vascular malformation of the masseter muscle. Note high signal on T2 (**A**) and patchy contrast enhancement (**B**). Thin arrows point at phleboliths. CT image in another patient (**C**) with a venous vascular malformation of the parapharyngeal space shows the typical aspect of phleboliths (arrows).

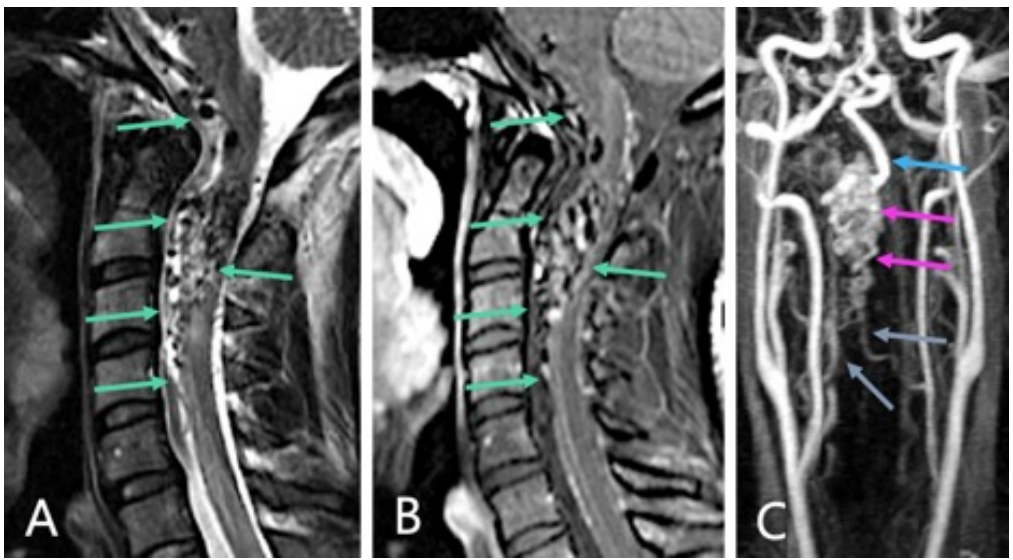
High-flow vascular malformations include AVMs and AVF, which **connect arteries and veins, bypassing the capillary bed**.

- **AVMs** - mostly congenital - have a dilated feeding artery, a nidus (tangle of vessels) and a dilated draining vein. They tend to increase in size over time.
- In contrast, **AVF** are most often acquired (post-traumatic) and they do not have a nidus => i.e., there is a direct communication between a dilated artery and a vein.

>=< FURTHER KNOWLEDGE

Sagittal T2-weighted (A) and contrast enhanced fat saturated T1-weighted (**B**) images show the typical features of a spinal AVM. Note flow voids (turquoise arrows) on T2 (**A**) and T1 (**B**) due to dilated feeding arteries and draining veins and no discernible soft-tissue component. MRA (coronal MIP image) in the same patient (**C**) shows the typical aspect of an AVM with a dilated vertebral artery (blue arrow) and dilated draining veins (gray arrows) and a nidus (pink arrows).

Images courtesy Maria Isabel Vargas, Geneva University Hospitals, Geneva University

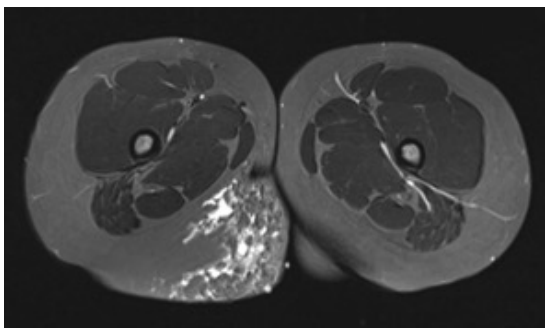


CHAPTER OUTLINE:

Lymphatic malformations are **low-flow** malformations that most often present in childhood; they tend to involve multiple anatomic spaces and structures. They do not communicate with normal lymph vessels and are most often seen in the head and neck subcutaneously. Lymphatic malformations can cause osseous hypertrophy and can, therefore, lead to discrepancy in limb length. Lymphatic malformations can be macrocystic (= multiple large cysts) or they can be microcystic (= multiple tiny cysts).

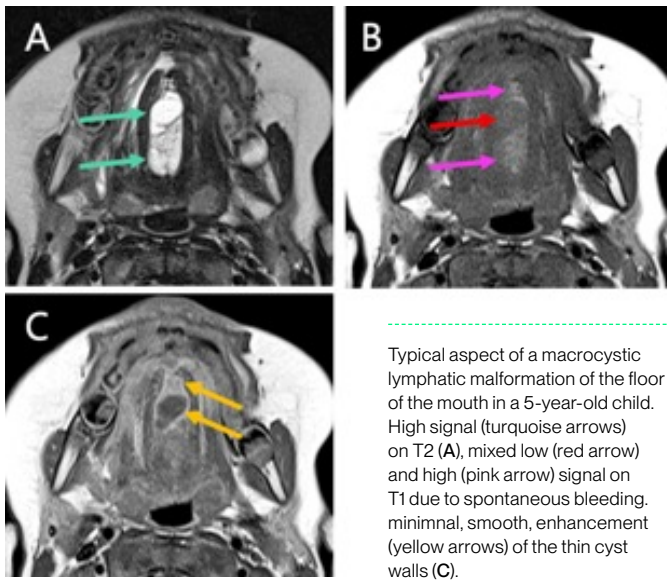
Superficial lesions are diagnosed clinically whereas deep lesions require imaging for diagnosis. The **main complications** of lymphatic malformations are bleeding and infection.

>=< FURTHER KNOWLEDGE



Lymphatic malformation of the right buttock. The subcutaneous lymphatic cysts, which are of high T2 signal, are of varying sizes and there is additional fat present between the cysts.

Image reproduced from: Gibson CR, Barnacle AM. Vascular anomalies: special considerations in children. CVIR Endovasc. 2020 Nov 22;3(1):60. doi: 10.1186/s42155-020-00153-y. PMID: 32886264; PMCID: PMC7474047.



Typical aspect of a macrocystic lymphatic malformation of the floor of the mouth in a 5-year-old child. High signal (turquoise arrows) on T2 (**A**), mixed low (red arrow) and high (pink arrow) signal on T1 due to spontaneous bleeding. minimnal, smooth, enhancement (yellow arrows) of the thin cyst walls (**C**).

/ Vascular Imaging

CHAPTER OUTLINE:

/ Take-Home Messages

- Vascular imaging includes intraluminal, vessel wall and extraluminal space evaluation.
- Blood vessels can be visualised with ultrasound, DSA, CTA and MRA, and each modality has advantages and disadvantages, including indications and contraindications to be taken in consideration.
- Ultrasound is a good initial diagnostic tool to assess blood vessels, being readily available and having low costs.
- CTA has a higher radiation dose than non-enhanced CT and uses iodinated contrast media. It has a high diagnostic accuracy in multiple vascular pathologies, e.g., atherosclerosis, arterial stenosis/occlusion, dissection, aneurysm, Budd-Chiari, PE, vascular compression, vascular tumours and many others.
- MRA can be performed in any body part and can be used with or without contrast media. MRA is a very versatile non-invasive imaging technique allowing screening, evaluation and follow-up of a multitude of vascular pathologies, e.g., arterial aneurysms, AV malformations, arterial dissection, arterial stenosis, neurovascular conflicts, PAOD and many more.
- During DSA not only diagnostic but also interventional radiologic therapeutic procedures can be performed.
- Sometimes it is necessary to combine information from different vascular imaging modalities.
- Distinction between vascular tumours and vascular malformations is essential and imaging plays an important role for diagnosis and treatment of these entities.

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Vascular Imaging

CHAPTER OUTLINE:

Test Your Knowledge

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<?> QUESTION

1 What are potential risks associated with DSA (several answers possible)?

- ☐ Allergic reaction
- ☐ Vessel damage from the catheter
- ☐ Infection at the site of catheter insertion
- ☐ Acute kidney injury

/ Test Your Knowledge

<?> ANSWER

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- Allergic reaction
- Vessel damage from the catheter
- Infection at the site of catheter insertion
- Acute kidney injury

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<?> QUESTION

2 How is DSA of the renal artery usually performed (several answers possible)?

- ☐ A small incision is made in the skin and a catheter is inserted into the femoral artery
- ☐ Contrast media is injected into the bloodstream via the catheter
- ☐ Sequential X-ray images are acquired to visualise and document the blood vessels
- ☐ The procedure is always performed under general anaesthesia

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/ Test Your Knowledge

<?> QUESTION

3 To perform MR angiography, is it always necessary to inject contrast media?

- ☐ True
- ☐ False

/ Test Your Knowledge

<?> ANSWER

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<?> QUESTION

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<?> QUESTION

5 How are blood velocity values at a site of a major arterial stenosis as compared to a normal artery?

- ☐ Decreased
- ☐ Increased
- ☐ Not changed
- ☐ Variable

/ Test Your Knowledge

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<?> QUESTION

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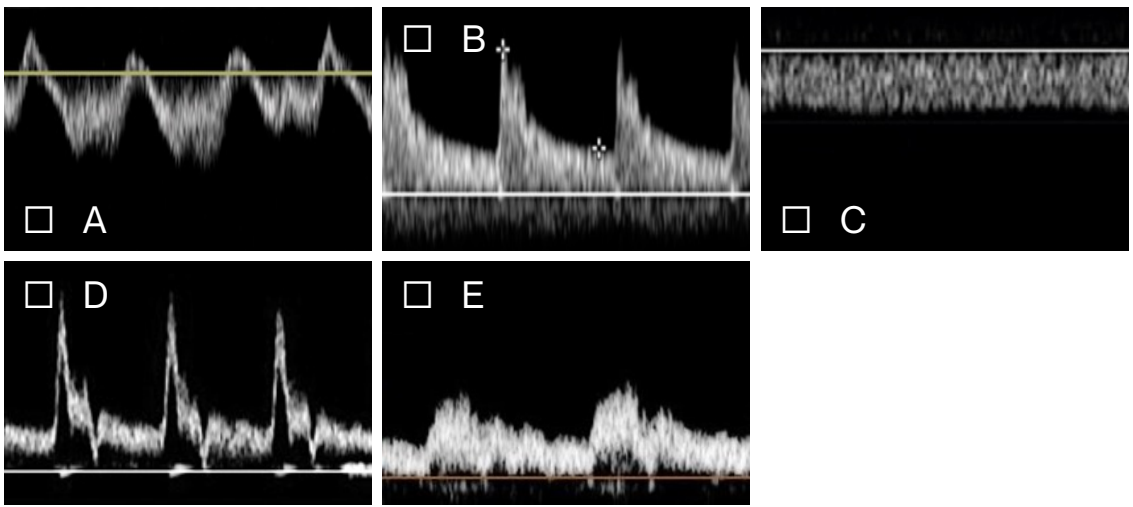

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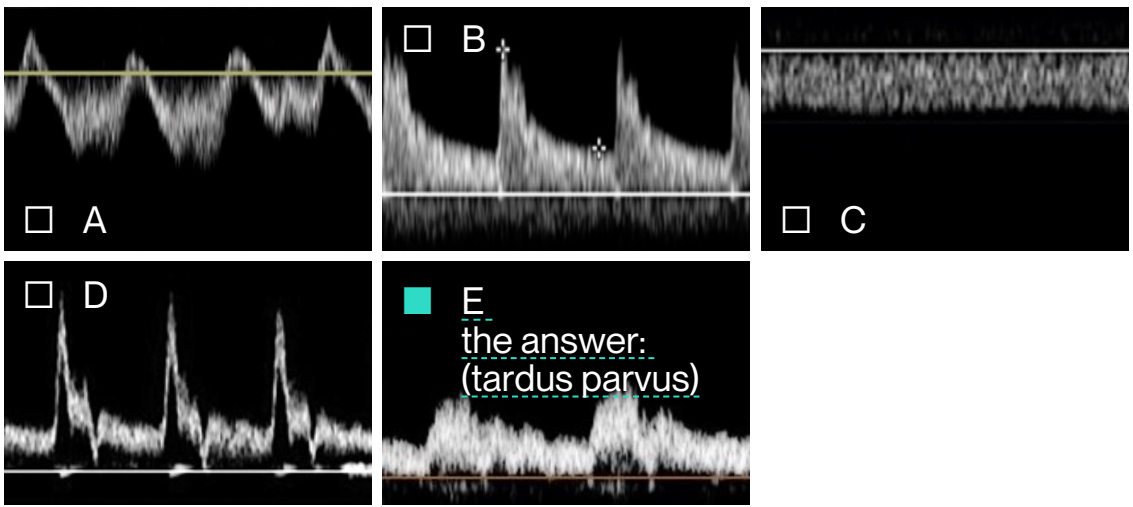

/ Test Your Knowledge

<?> QUESTION

7 What imaging features can you see in the false lumen of an aortic dissection compared to the true lumen (several answers are possible)?

- ☐ Larger than the true lumen
- ☐ Less enhancement than the true lumen
- ☐ Beak sign
- ☐ Surrounded by calcifications (if present)

/ Test Your Knowledge

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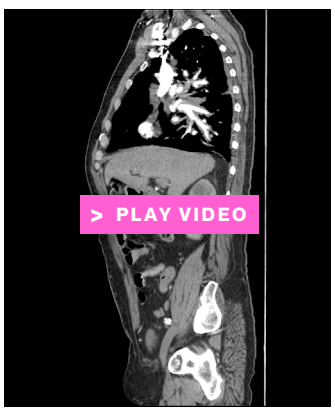
- ■ Beak sign
- ☐ Surrounded by calcifications (if present)

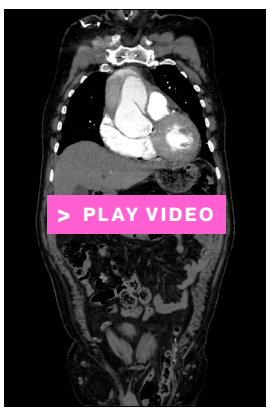
/ Test Your Knowledge

<?> QUESTION

8 What type of aortic dissection is this (see videos)?

- ☐ DeBakey I
- ☐ Stanford B
- ☐ DeBakey III
- ☐ Stanford A or DeBakey II





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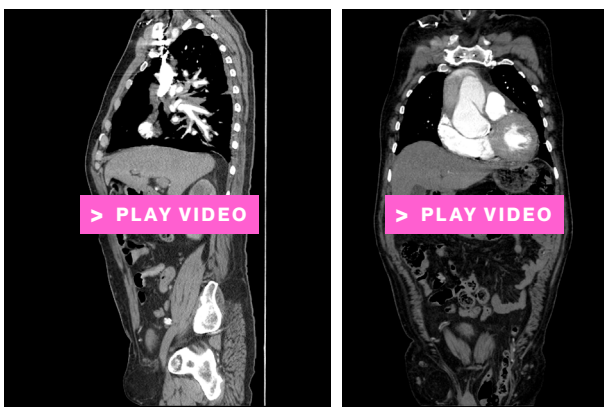


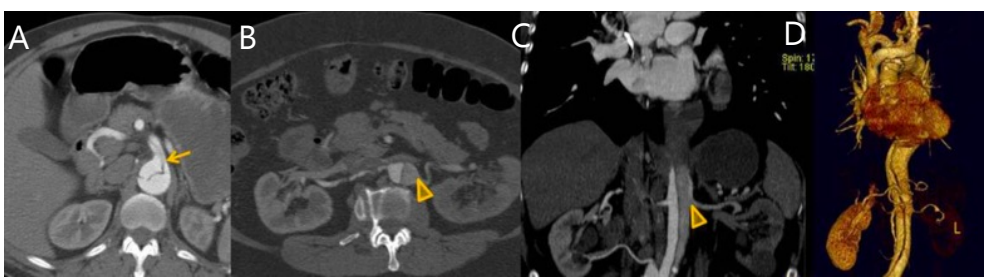
CTA of aorta (videos). Sagital and coronal reconstructions showing Stanford type A dissection (DeBakey II) affecting the ascending aorta.

/ Test Your Knowledge

<?> QUESTION

9 Which statements regarding the CTA images below obtained in the emergency setting are correct?

- ☐ The arrow in A shows that the dissection involves the celiac trunk
- ☐ The arrowhead in B points at the true lumen
- ☐ The arrowhead in C shows that the left renal artery arises from the true lumen
- ☐ Image D shows perfusion impairment of the left kidney



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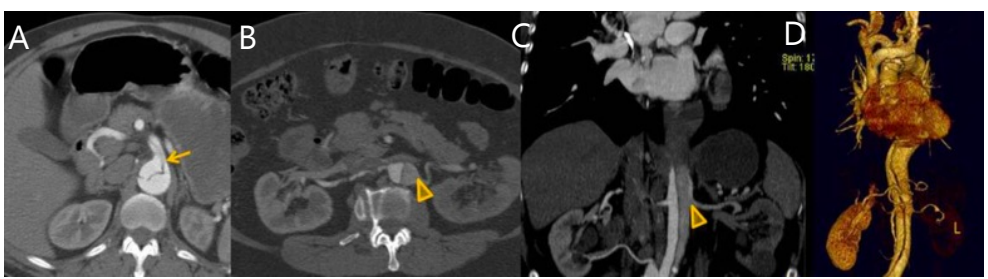
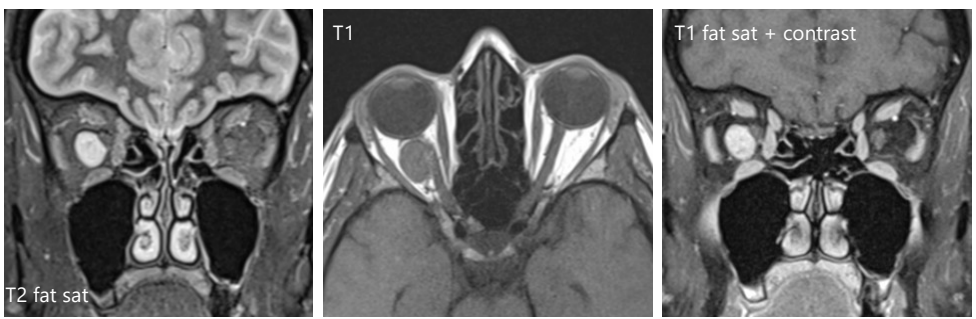


/ Test Your Knowledge

<?> QUESTION

10 Which statements regarding the MR images below obtained in a 20-year-old patient are correct?

- ☐ The orbital lesion is strongly hyperintense on T2 and enhances substantially
- ☐ There are flow voids suggesting an AVM
- ☐ This is most likely a haemangioma
- ☐ This is most likely a low-flow venous malformation



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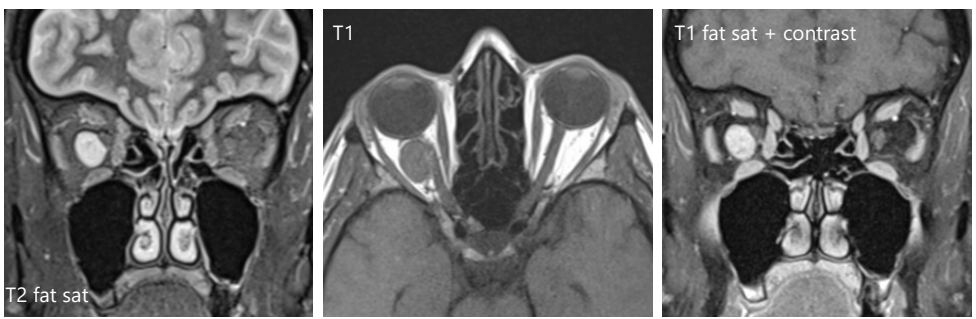

<!> ATTENTION

DO NOT use the term haemangioma which is a neoplastic lesion! See ISSVA classification to avoid confusion!