EUROPEAN SOCIETY OF RADIOLOGY

eBook for Undergraduate Education in Radiology

CHAPTER: Vascular Imaging

Preface

Undergraduate teaching of radiology in Europe is provided according to national schemes and may vary considerably from one academic institution to another. Sometimes, the field of radiology is considered as a "cross-cutting discipline" or taught within the context of other clinical disciplines, e.g., internal medicine or surgery.

This e-book has been created in order to serve medical students and academic teachers throughout Europe to understand and teach radiology as a whole coherent discipline, respectively. Its contents are based on the *Undergraduate Level of the ESR European Training Curriculum for Radiology* and summarize the so-called ***core elements*** that may be considered as the basics that every medical student should be familiar with. Although specific radiologic diagnostic skills for image interpretation cannot be acquired by all students and rather belong to the learning objectives of the *Postgraduate Levels of the ESR Training Curricula*, the present e-book also contains some ***further insights*** related to modern imaging in the form of examples of key pathologies, as seen by the different imaging modalities. These are intended to give the interested undergraduate student an understanding of modern radiology, reflecting its multidisciplinary character as an organ-based specialty.

We would like to extend our special thanks to the authors and members of the ESR Education Committee who have contributed to this eBook, to Carlo Catalano, Andrea Laghi and András Palkó who initiated this project, and to the ESR Office, in particular Bettina Leimberger and Danijel Lepir, for all their support in realising this project.

We hope that this e-book may fulfil its purpose as a useful tool for undergraduate academic radiology teaching.

Minerva Becker

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Based on the ESR Curriculum for Undergraduate Radiological Education

Chapter: **Vascular Imaging**

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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.

=> See respective e-book chapters.

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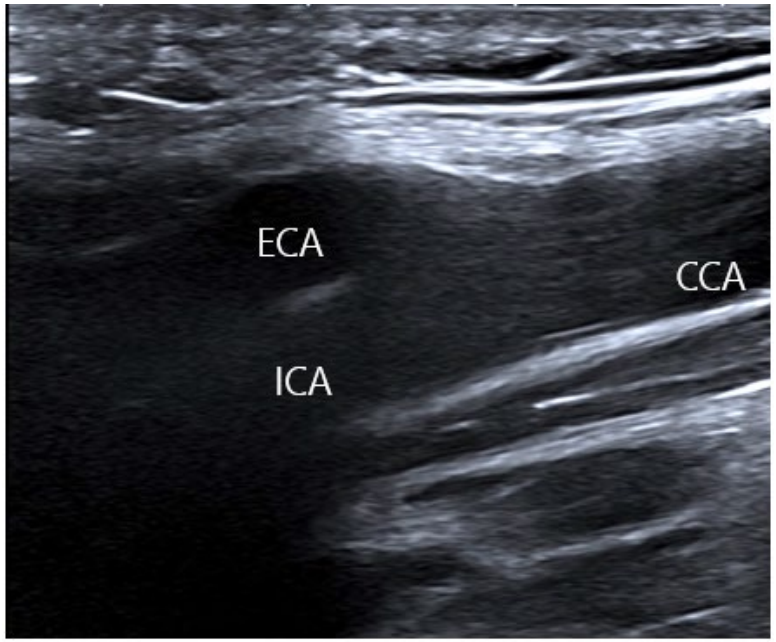
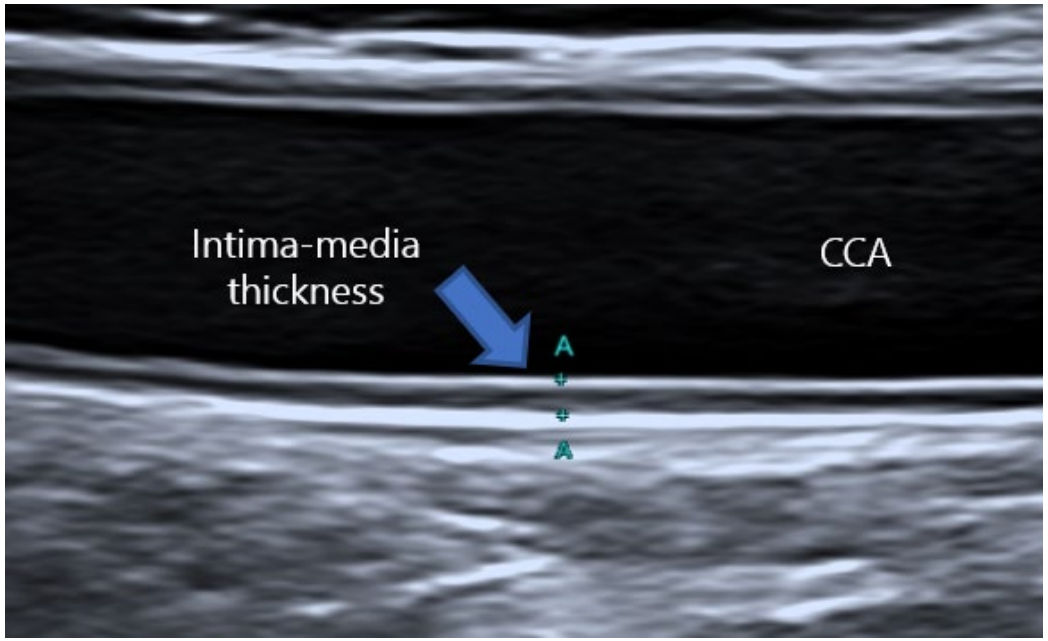
Ultrasound (US)

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).

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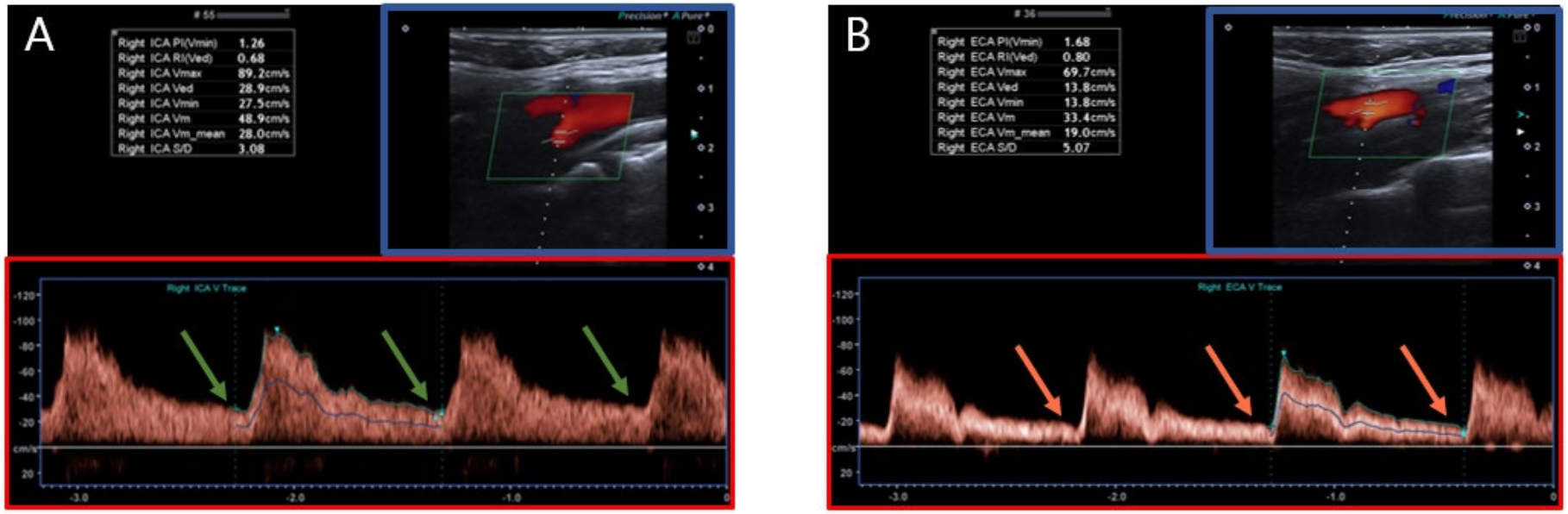
Doppler Ultrasound (Doppler-US)

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 (changes colour inside blood vessels depending on flow and speed of blood)
- Spectral Doppler (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*) (image A) and of the external carotid artery (*ECA*) (image 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).

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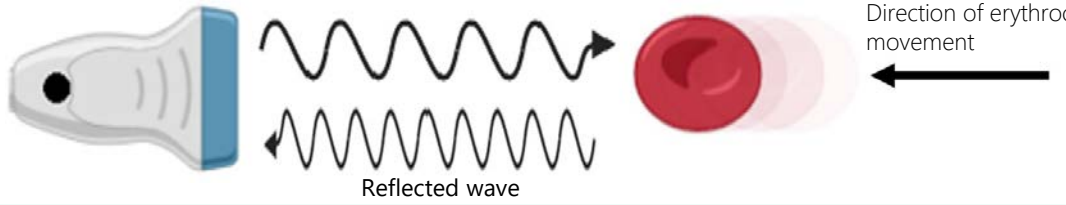
Doppler Ultrasound (Doppler-US)

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**.

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.

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

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

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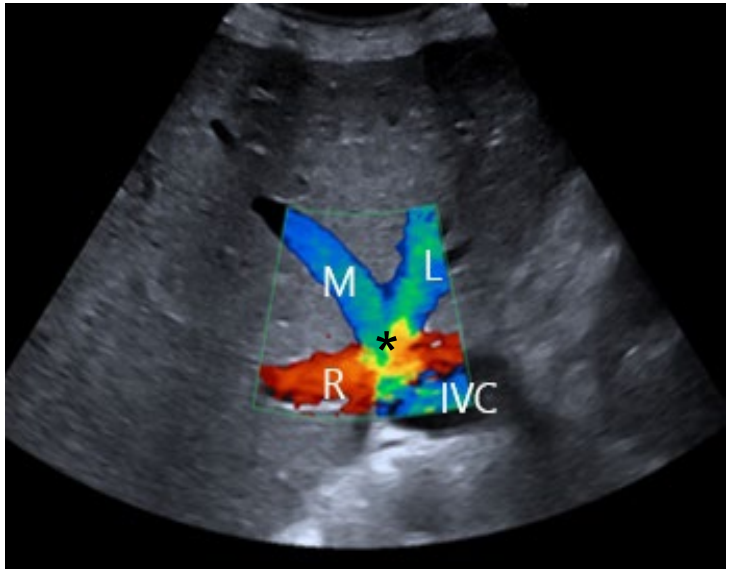
Doppler Ultrasound (Doppler-US)

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.



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.

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!

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Doppler Ultrasound (Doppler-US)

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
Femoral artery	80 -110
Popliteal artery	50 - 80
Internal carotid artery	80 -120
Vertebral artery	25 - 40
Vena cava	10 - 45

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!

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Doppler Ultrasound (Doppler-US)

Usually the PSV:

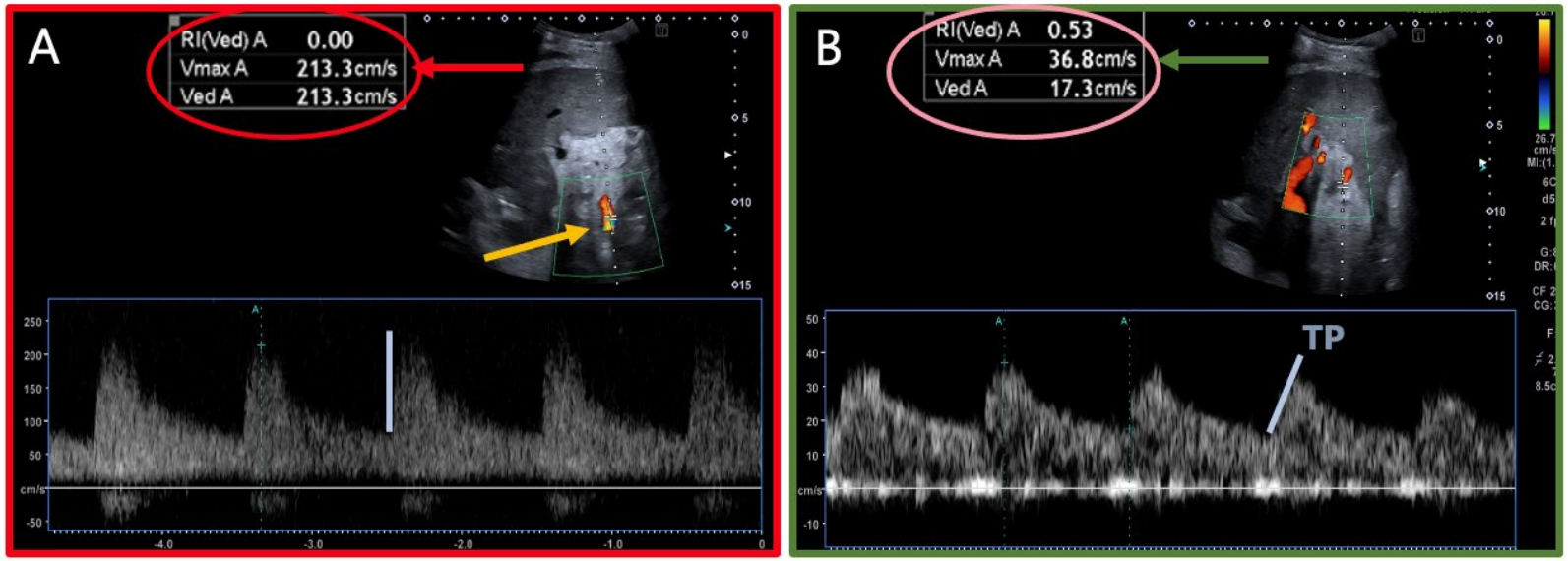
- Before the stenosis => is slightly higher
- At the site of stenosis => is significantly higher
- After stenosis => is lower

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.



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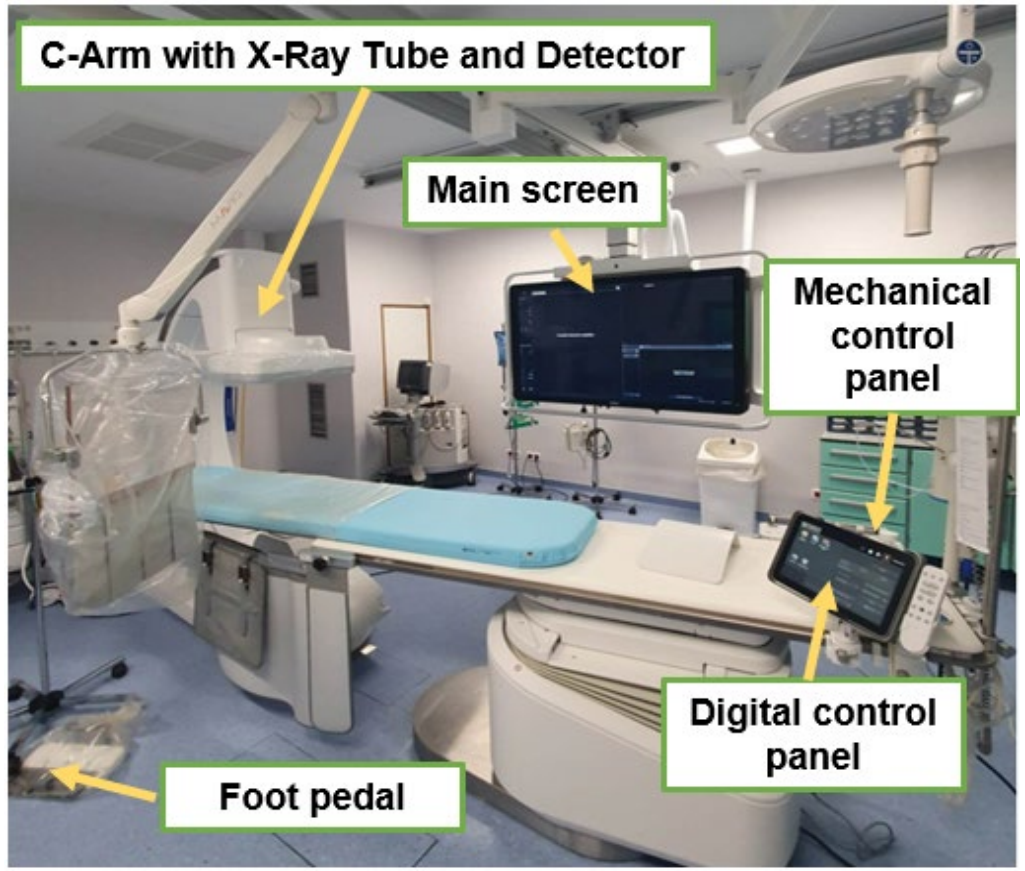
Digital Subtraction Angiography (DSA)

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 4-6h.



Angio-suite at *Coimbra Hospital* and *University* Centre

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Digital Subtraction Angiography (DSA)

Indications for DSA (diagnostic and/or treatment):

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

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)

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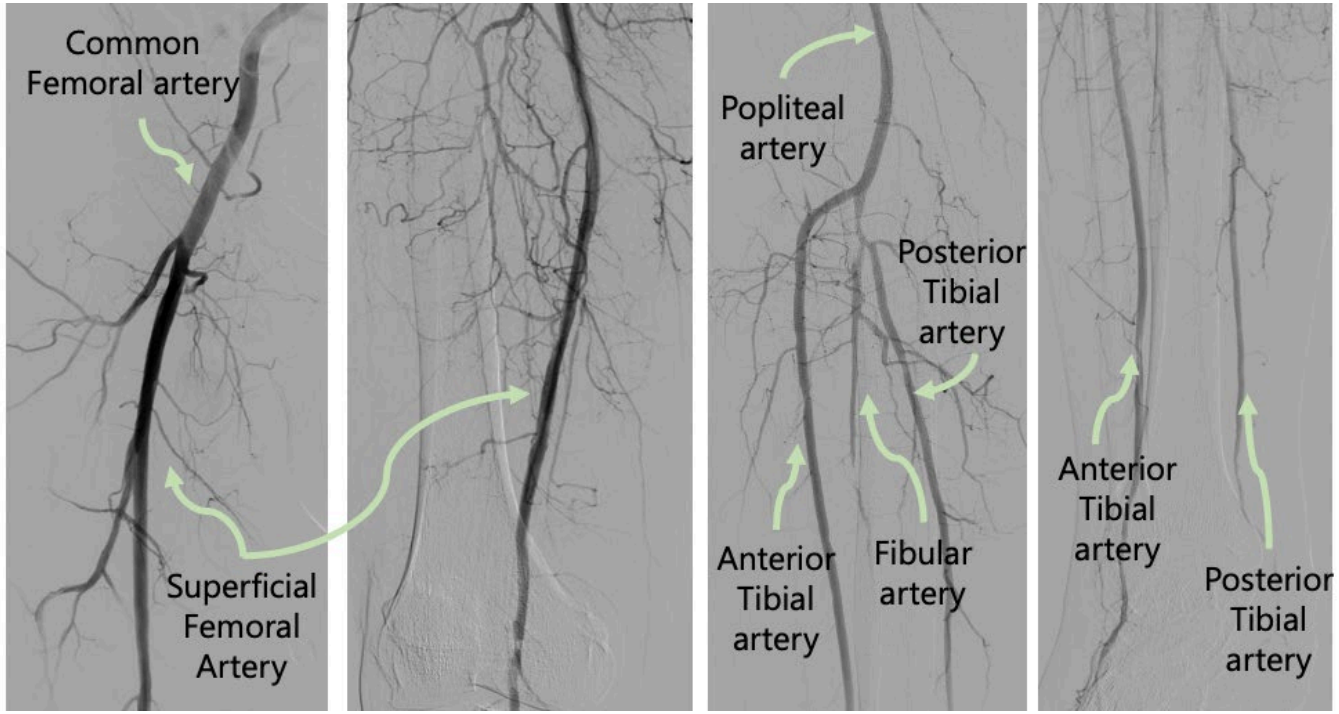
Digital Subtraction Angiography (DSA)

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

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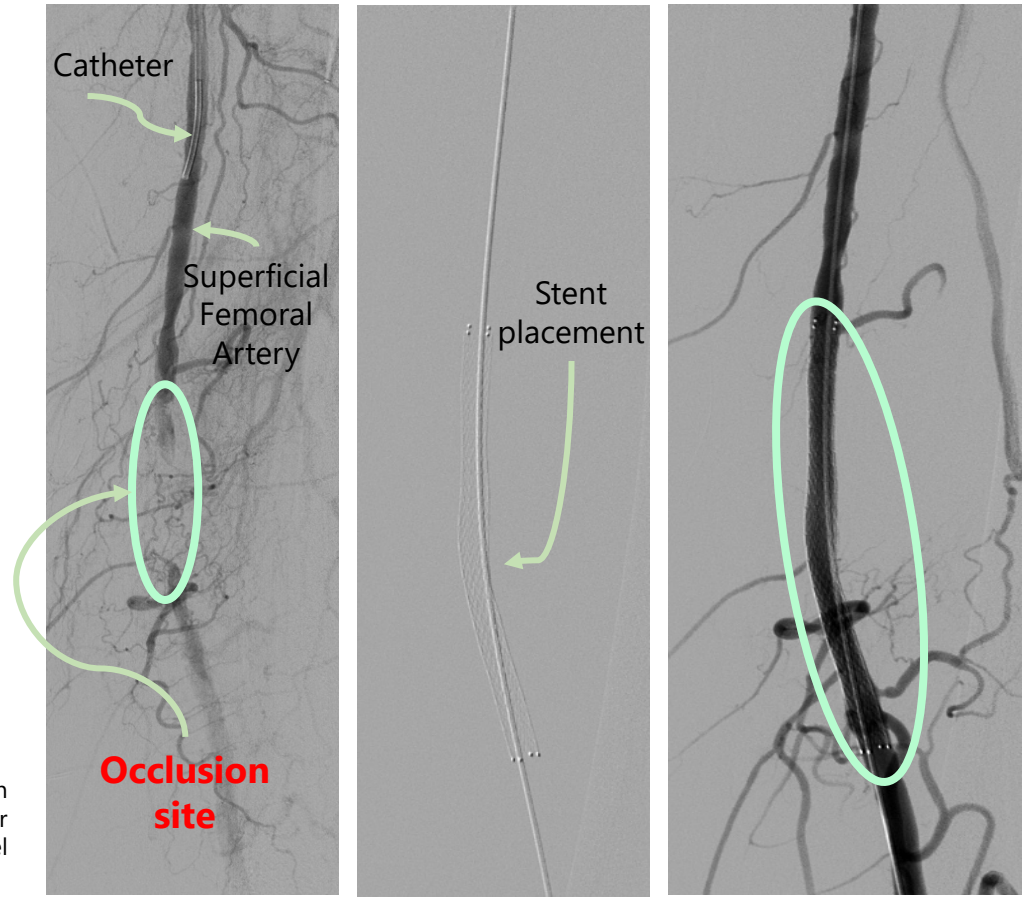
Digital Subtraction Angiography (DSA)

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

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

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

Sven Ivar Seldinger (1921-1998) - the Swedish interventional radiologist who pioneered the Seldinger Technique (= gold standard method for vessel catheterization)
https://www.sciencedirect.com/topics/medicine-and-dentistry/seldinger-technique
Image from: https://www.jvir.org/article/S1051-0443%2821%2901195-7/pdf



Superficial Femoral Artery Stenosis → Stenting→ Post-stenting Result

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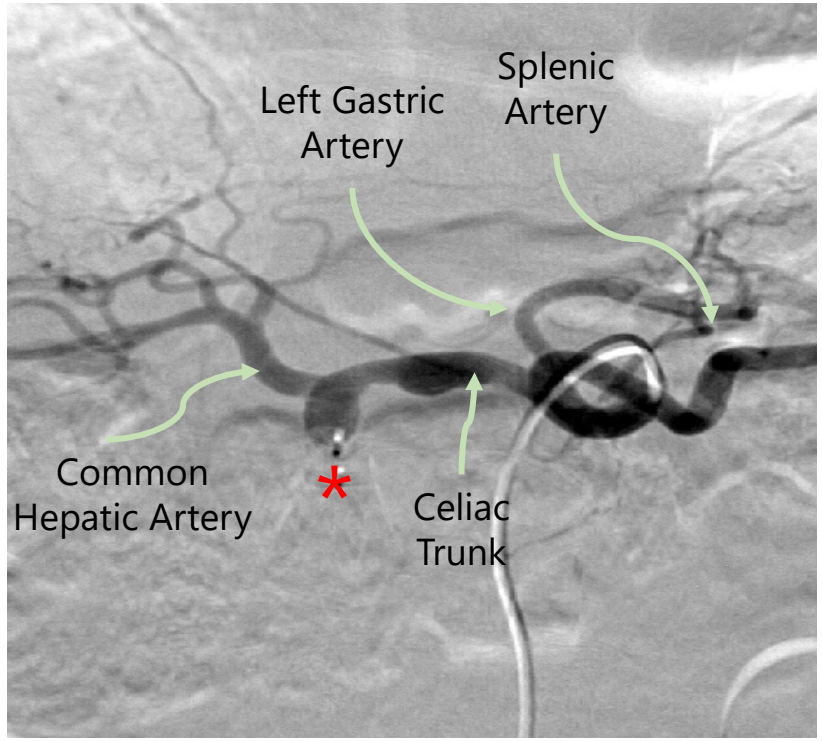
Digital Subtraction Angiography (DSA):
Visceral Angiography

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)

=> See chapter e-book chapter on interventional radiology



DSA of the celiac trunk and its branches:

- Common hepatic artery
- Left gastric artery
- Splenic artery

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 (*)

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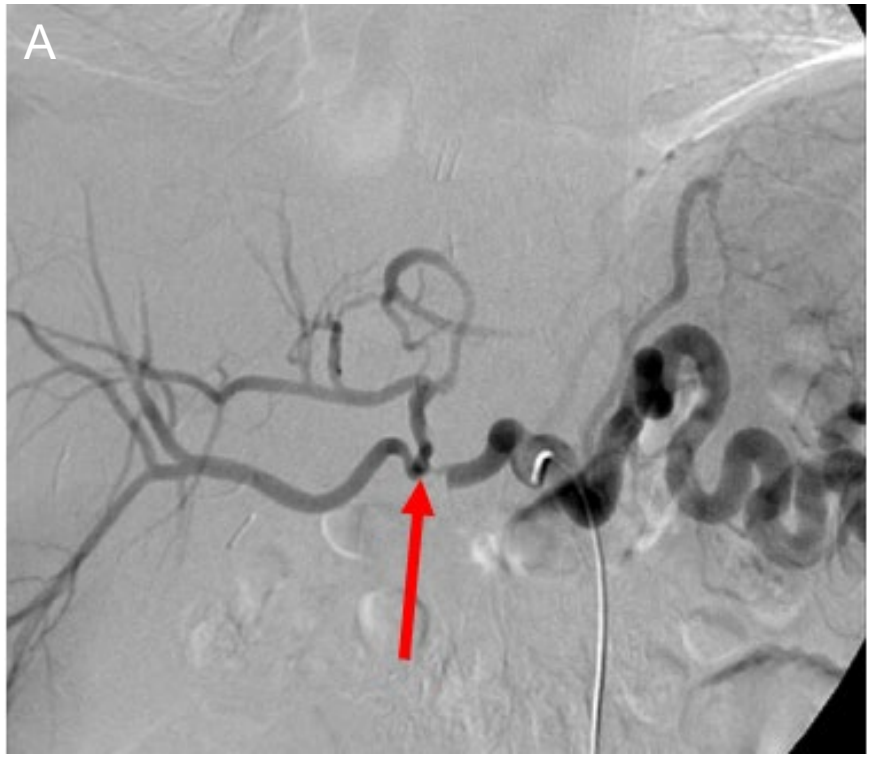

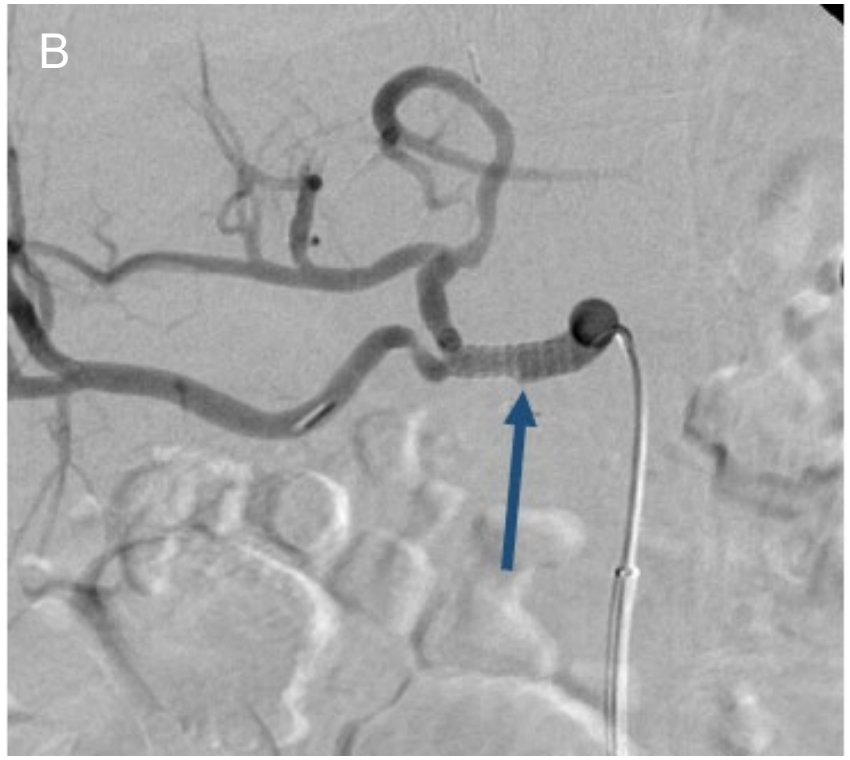

DSA of the celiac trunk showing a stenosis of the proper hepatic artery, right before it branches into the left and right hepatic arteries (A)

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

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Digital Subtraction Angiography (DSA):
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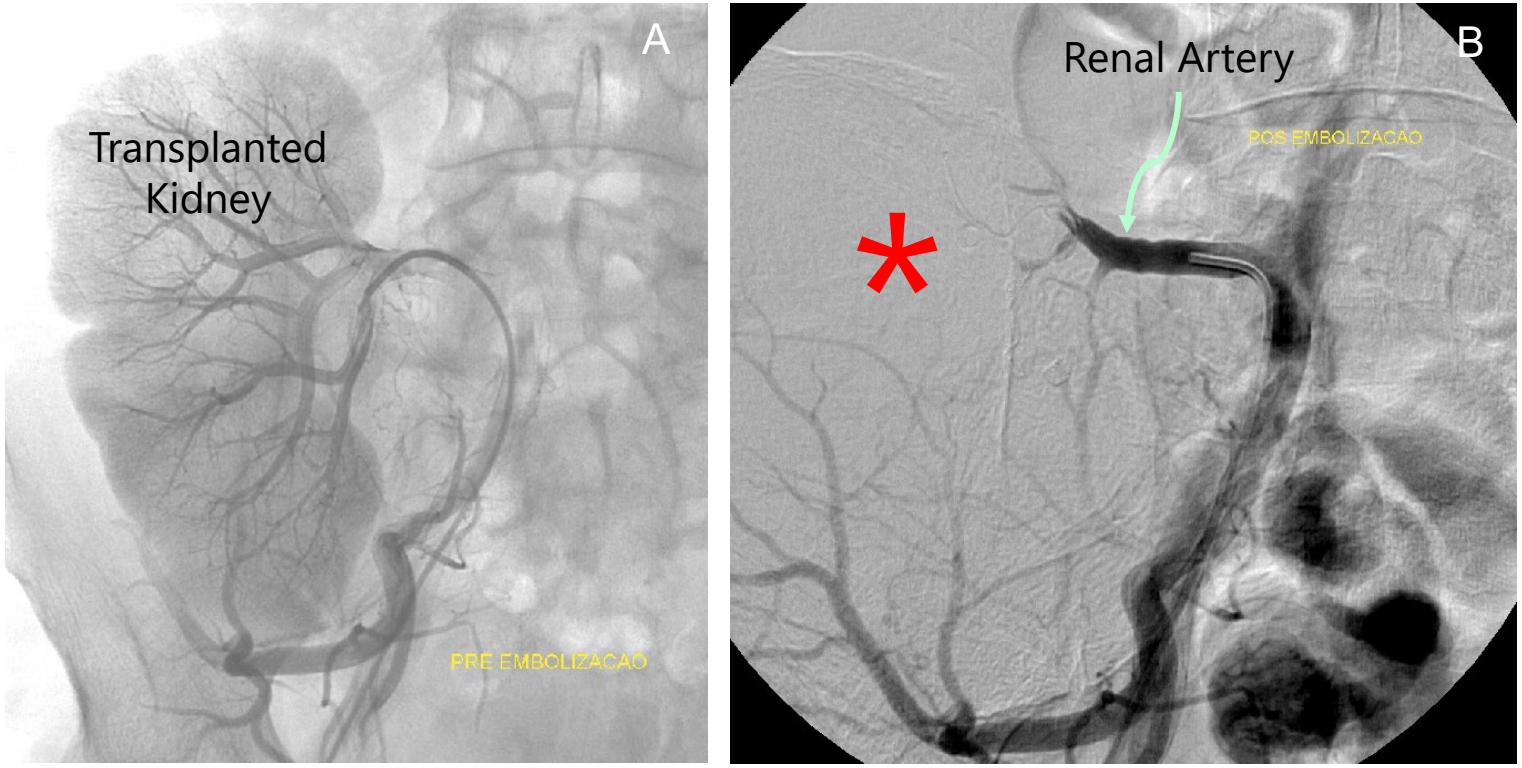

DSA of the renal artery of a non-functioning transplanted kidney (A). On B the kidney is no longer visible (*) as it was excluded by embolisation of the renal artery (arrow).

Right renal angiography catheter access route:

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

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Digital Subtraction Angiography (DSA):
Venous Angiography (Venography)

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

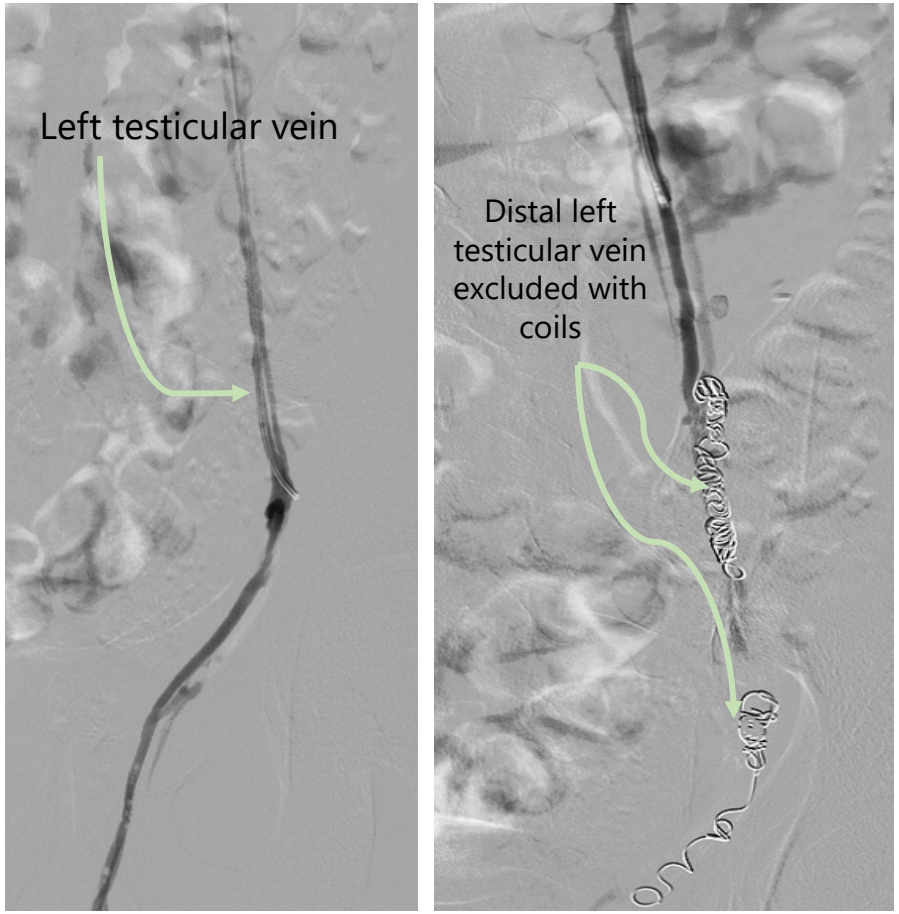
By performing venography keep in mind in which direction the blood flows.

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.

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 embolization (coiling)

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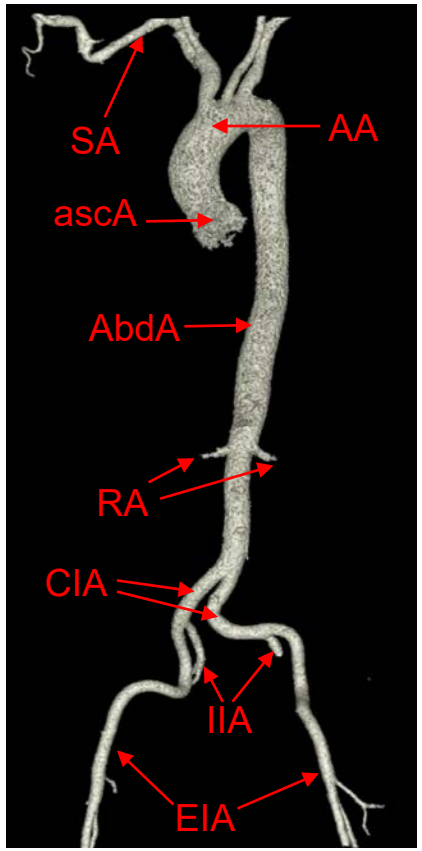
Computed Tomography Angiography (CTA)

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.

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).

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Computed Tomography Angiography (CTA)

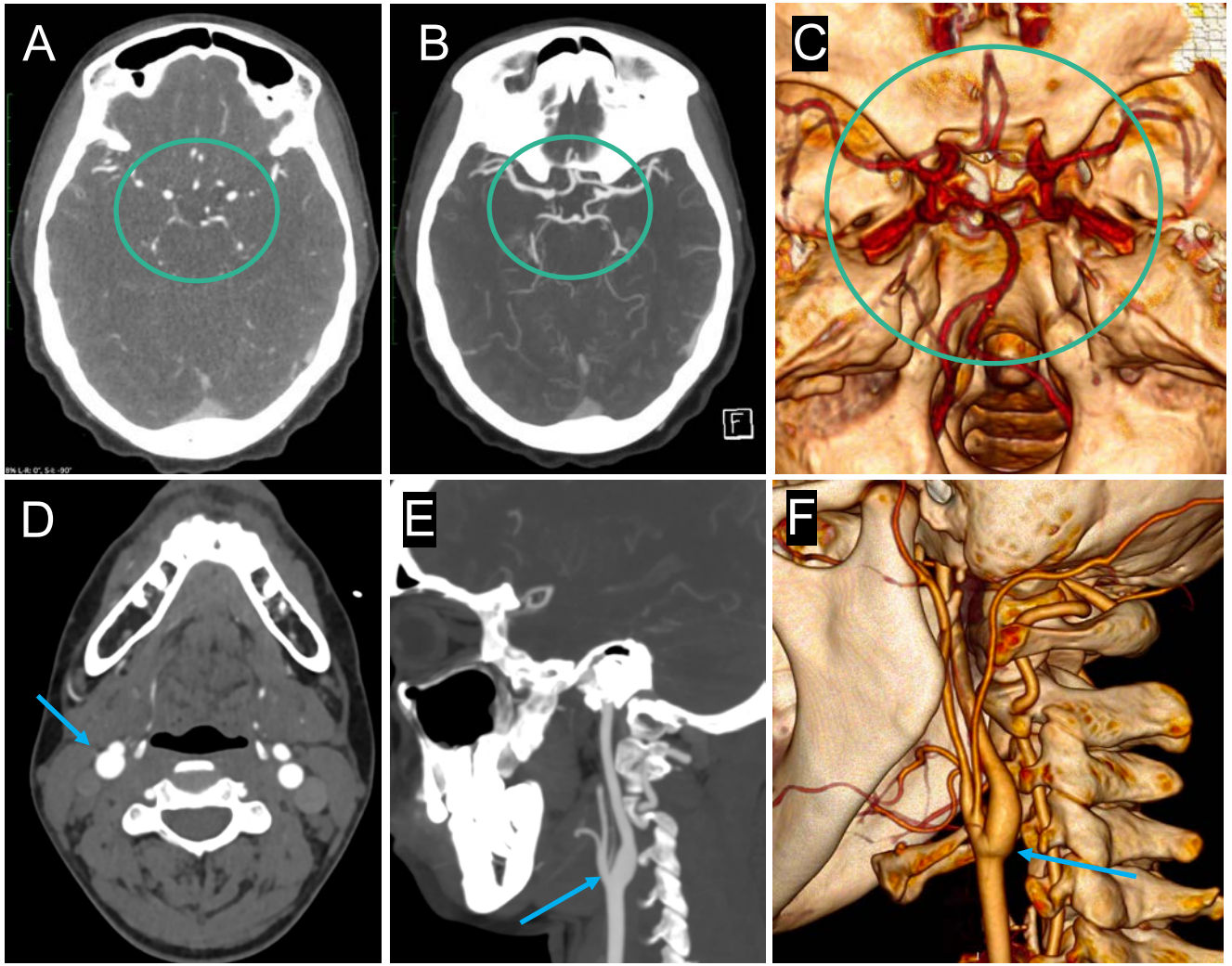
Different types of CTA reconstructions can be obtained from the same contrast-enhanced CT acquisition. In this figure, reconstructions of the polygon of Willis (A-C) and of the right carotid bifurcation (D-F) are shown. Both the polygon and the carotid arteries are normal.

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 (0.75mm slice thickness).

Axial (B) and sagittal (E) MIP

3D VR view of the polygon of Willis seen from above (C) and lateral view of the right carotid bifurcation (F).



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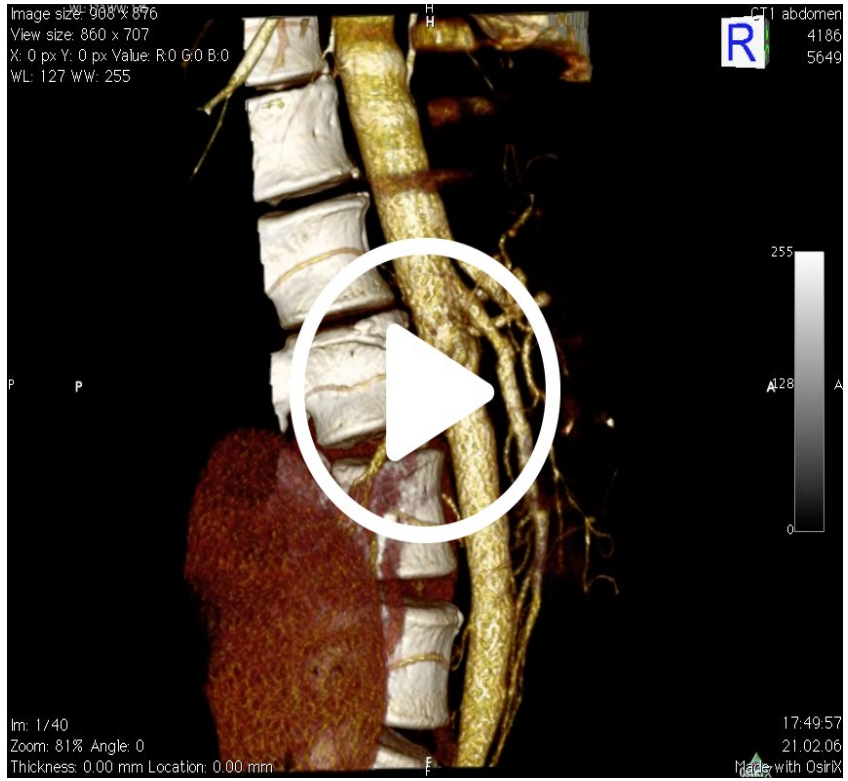
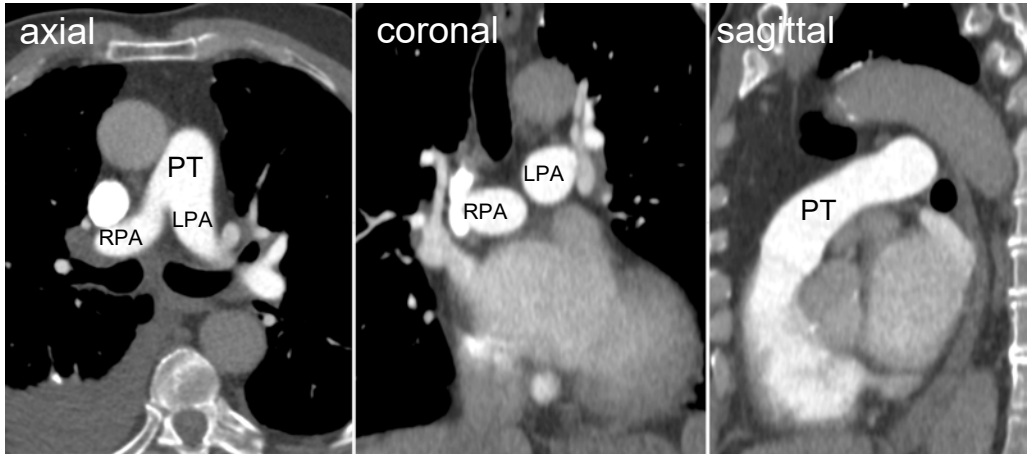
Computed Tomography Angiography (CTA)

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).

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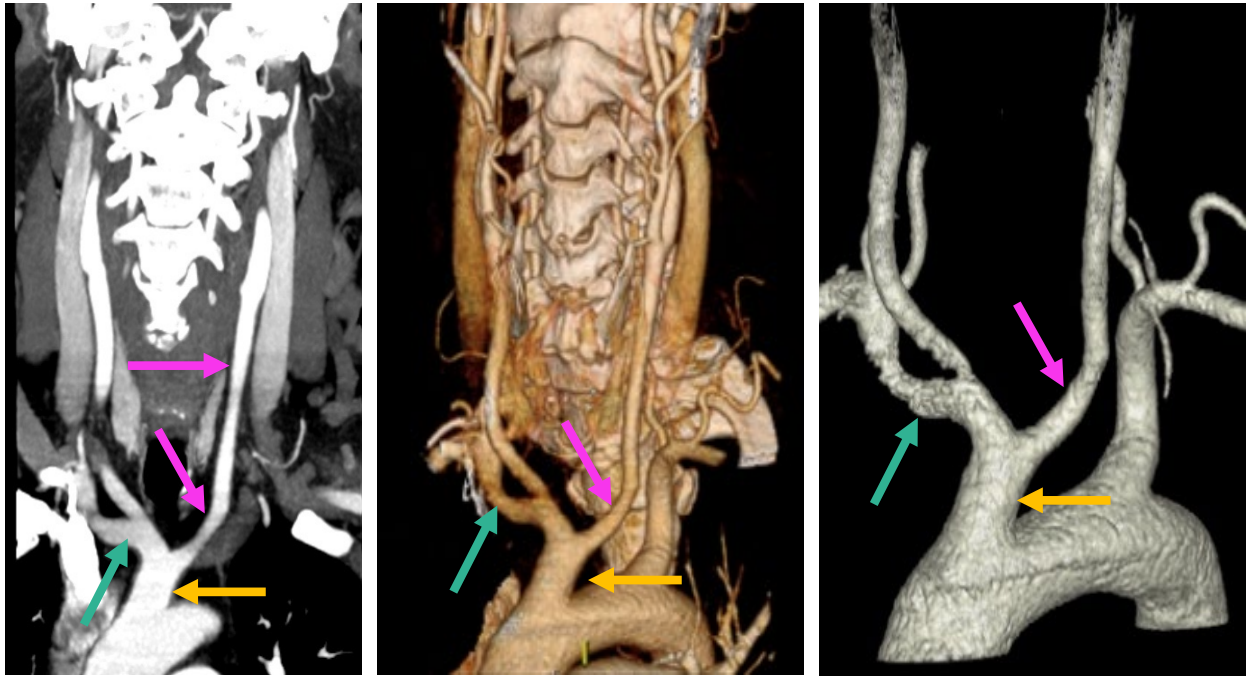
Computed Tomography Angiography (CTA)

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

Figure illustrating CTA reconstructions of vascular variants.



The bovine arch is the most common variant of the aortic arch in which the innominate artery has a common origin as the left common carotid artery. 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).

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

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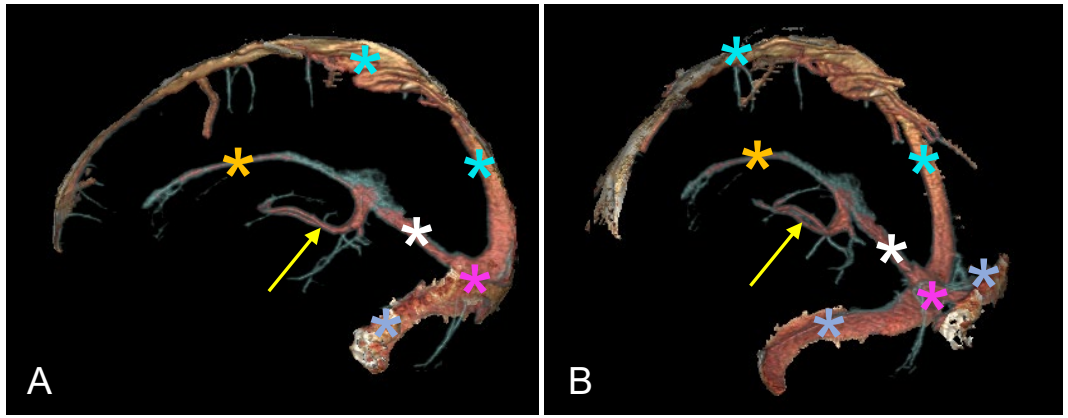
Computed Tomography Angiography:
CT Venography (CTV)

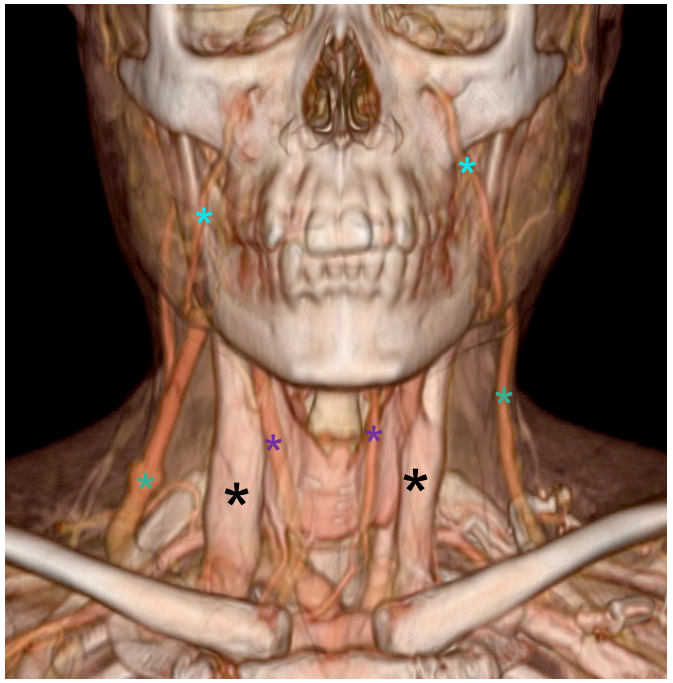
CT venography (CTV) of the head (also called cerebral CTV) is performed to visualise the cerebral veins and venous sinuses filled with contrast. The goal is to assess their patency in suspected cerebral venous thrombosis and to assess the venous anatomy prior to cranial surgery.

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

In CTV, the scan delay must be adapted (later acquisition compared to CTA). Usually, the scan delay is about ≥ 45s 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 Inferior sagittal sinus Straight sinus Confluence of sinuses Transverse sinus Great cerebral vein



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

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Computed Tomography Angiography (CTA):
Main Indications

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

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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.

=> See e-book chapter on MRI!

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

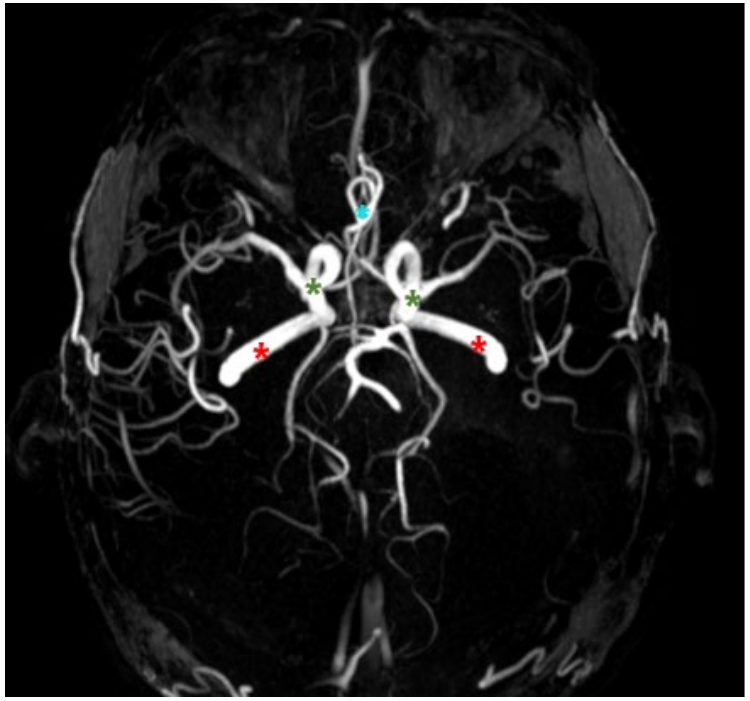
Also, it's possible to obtain MR angiography **with** or **without** contrast media, depending on indications.

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.

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

=> See e book chapter on MRI!



MRA using a 3D TOF sequence WITHOUT contrast media showing the intracranial blood vessels
Internal carotid arteries (*); Middle cerebral arteries (*); anterior cerebral arteries (*).

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

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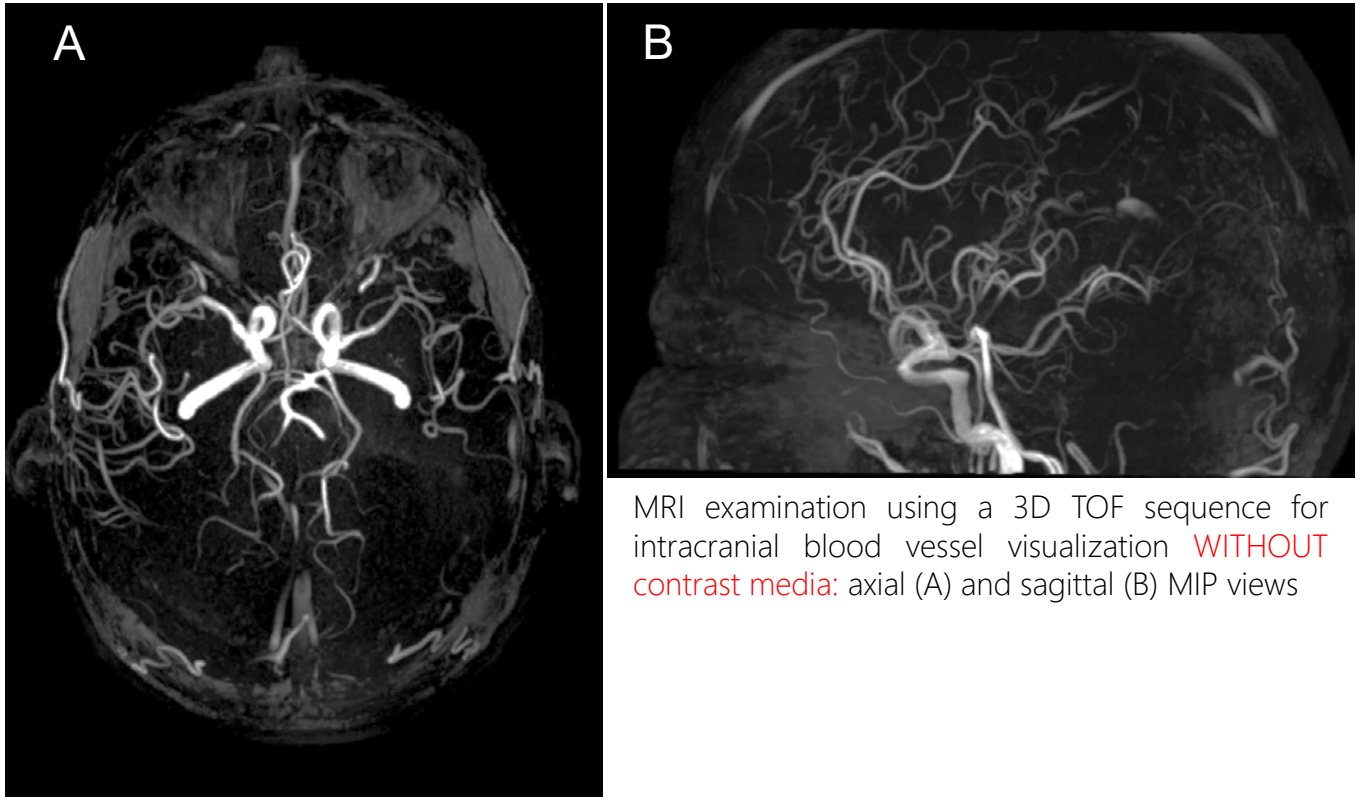
Magnetic Resonance Angiography (MRA): Without Contrast Media

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.**

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 visualization WITHOUT contrast media: axial (A) and sagittal (B) MIP views

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Magnetic Resonance Angiography (MRA):
With 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.

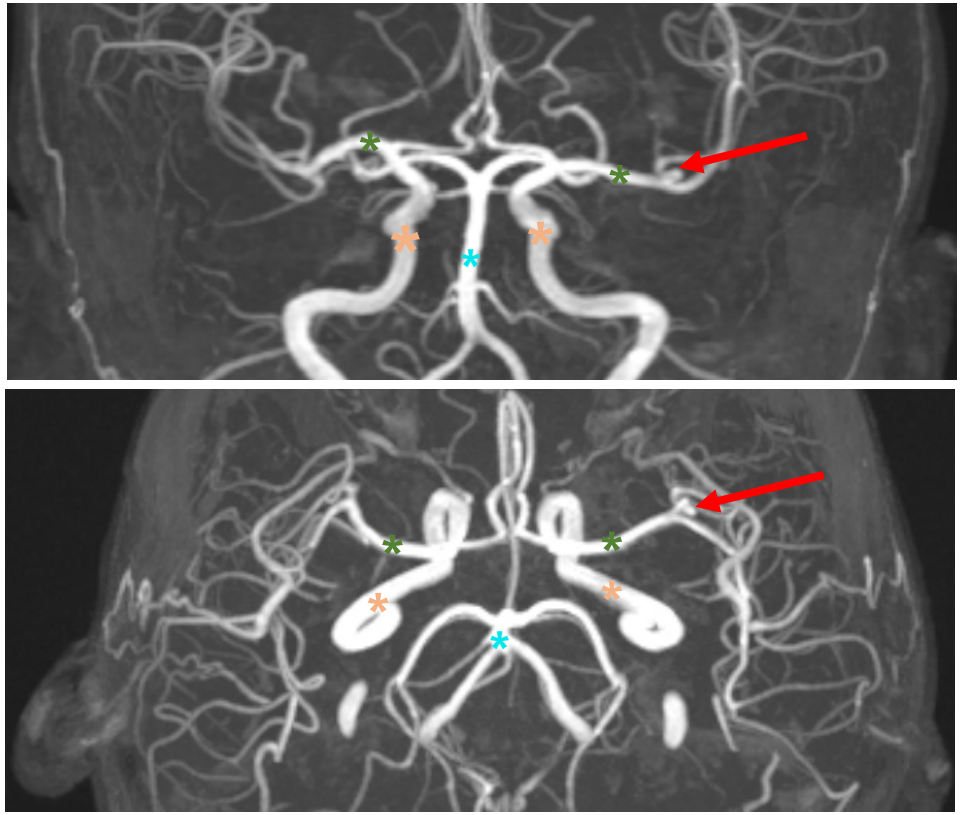
CE-MRA is an excellent alternative to CTA for vascular evaluation and follow-up without requiring iodinated contrast material and ionising radiation.

=> See e-book chapter on contrast media

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

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



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 (*) representing an aneurysm (arrow).

Internal carotid arteries (*); Middle cerebral arteries, MCAs (*); Basilar artery (*).

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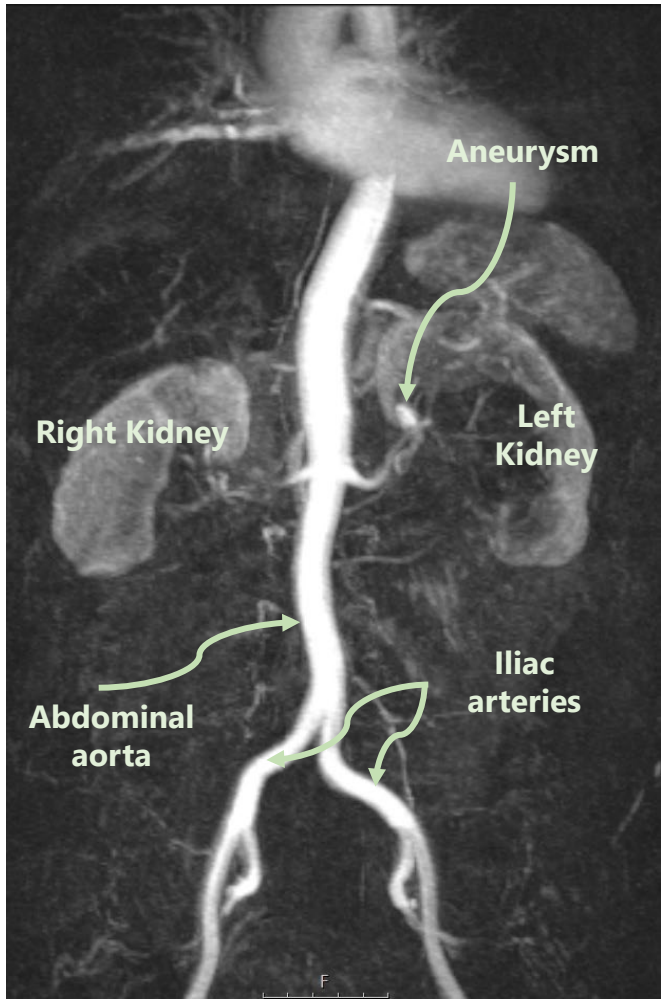
Magnetic Resonance Angiography (MRA):
With Contrast Media

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**

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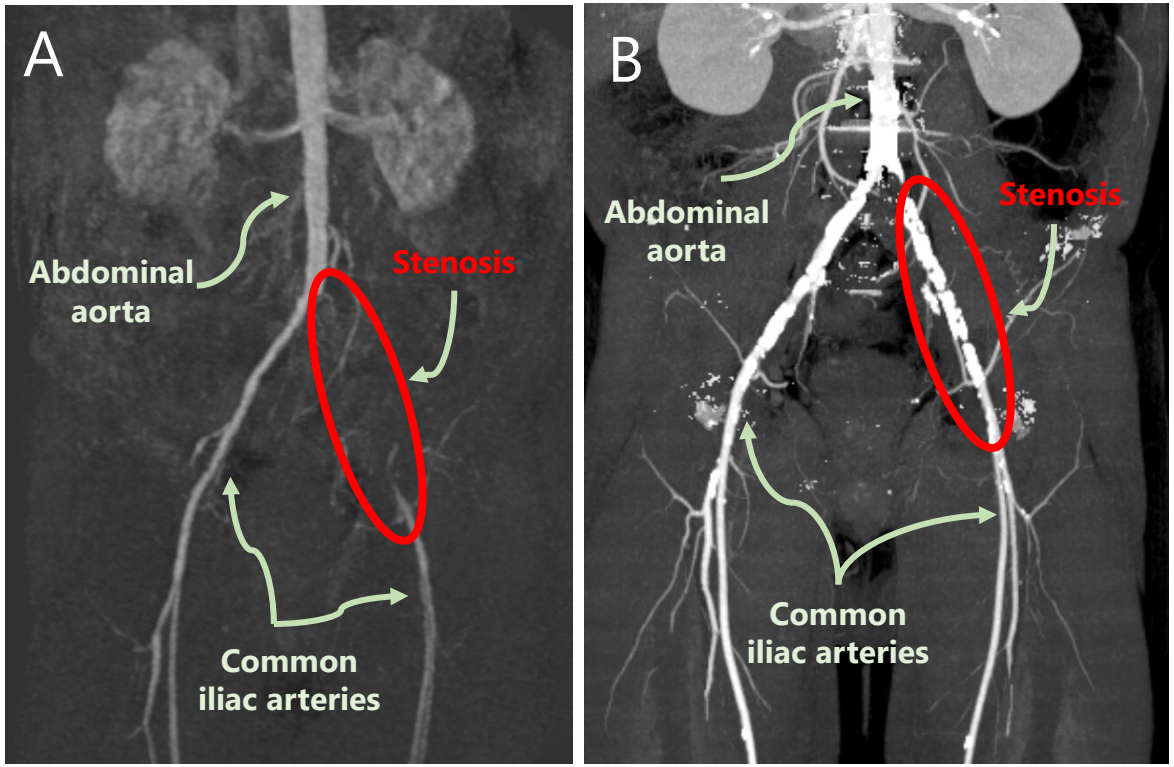
Magnetic Resonance Angiography (MRA):
With Contrast Media

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.

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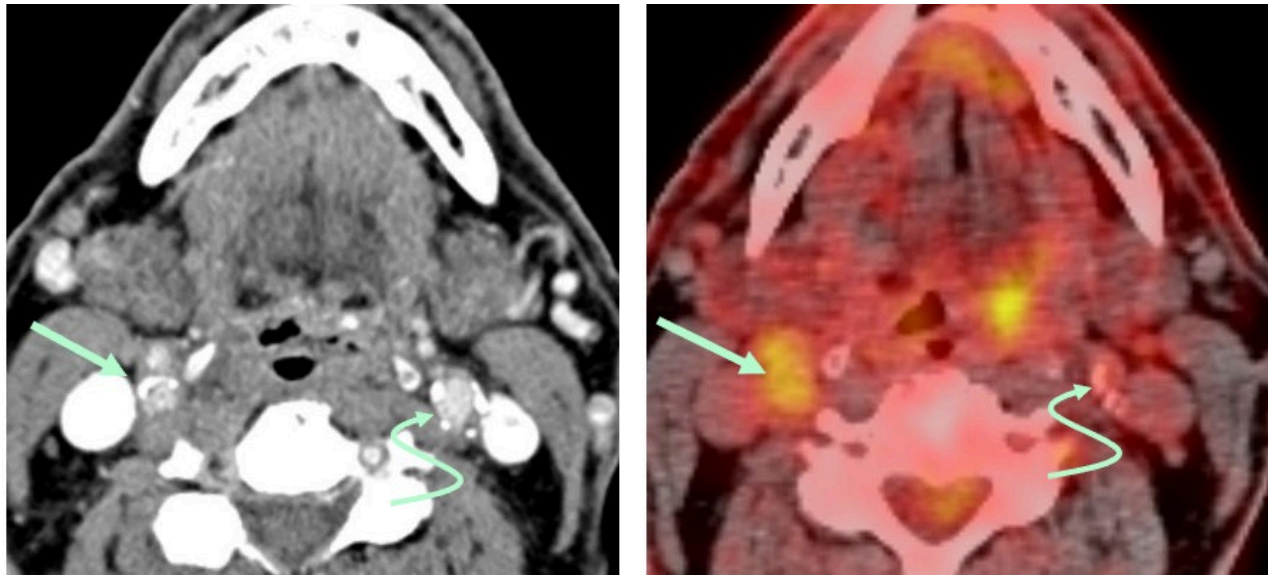
Positron Emission Tomography Computed Tomography (PET CT)

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.

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.

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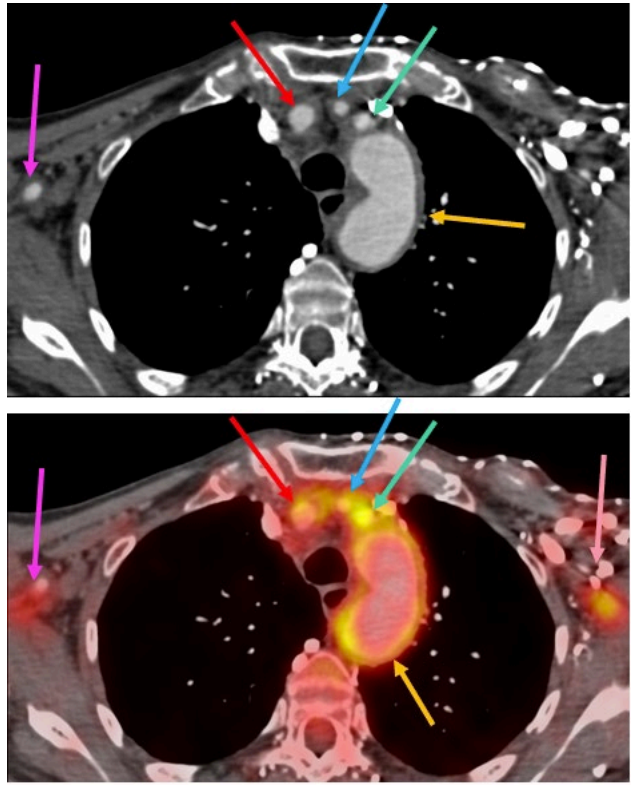
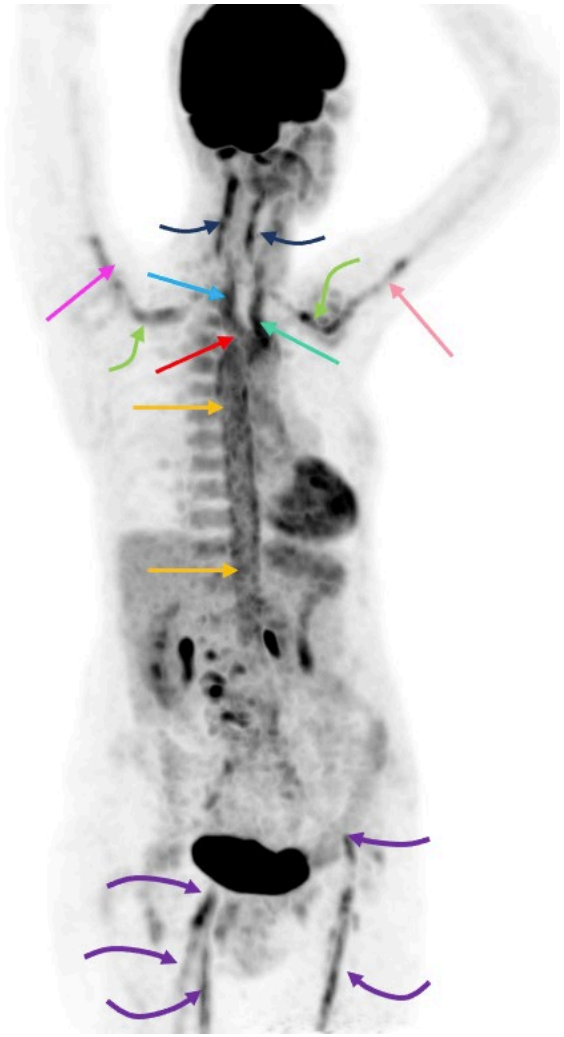
Positron Emission Tomography Computed Tomography (PET CT)

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 (3.9mm) and smooth and linear FDG uptake of the wall of the aorta and its branches (left common carotid artery, right common carotid artery, brachiocephalic trunk), right axillary and left axillary 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, vertebral and common, superficial and profound femoral arteries (curved arrows) on this MIP.

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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.

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.

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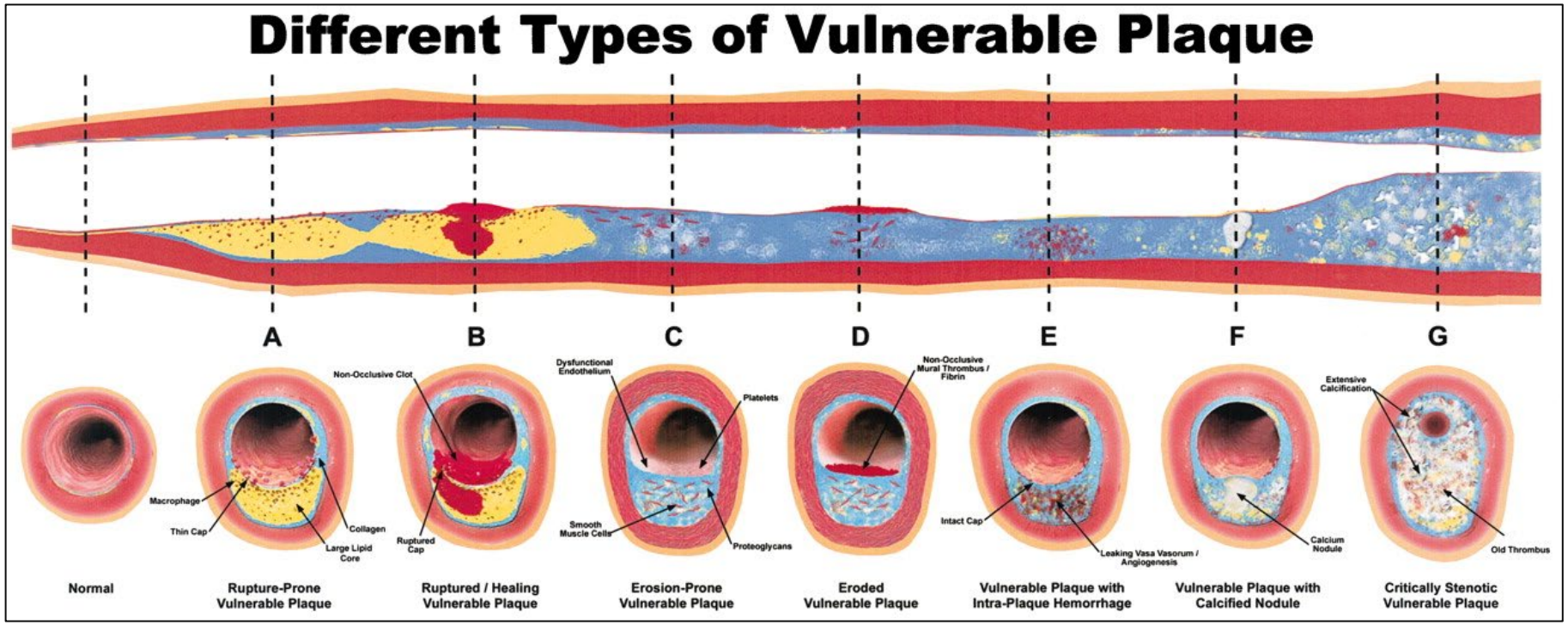
The **different types of vulnerable plaques** are shown in the figure below.



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.

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

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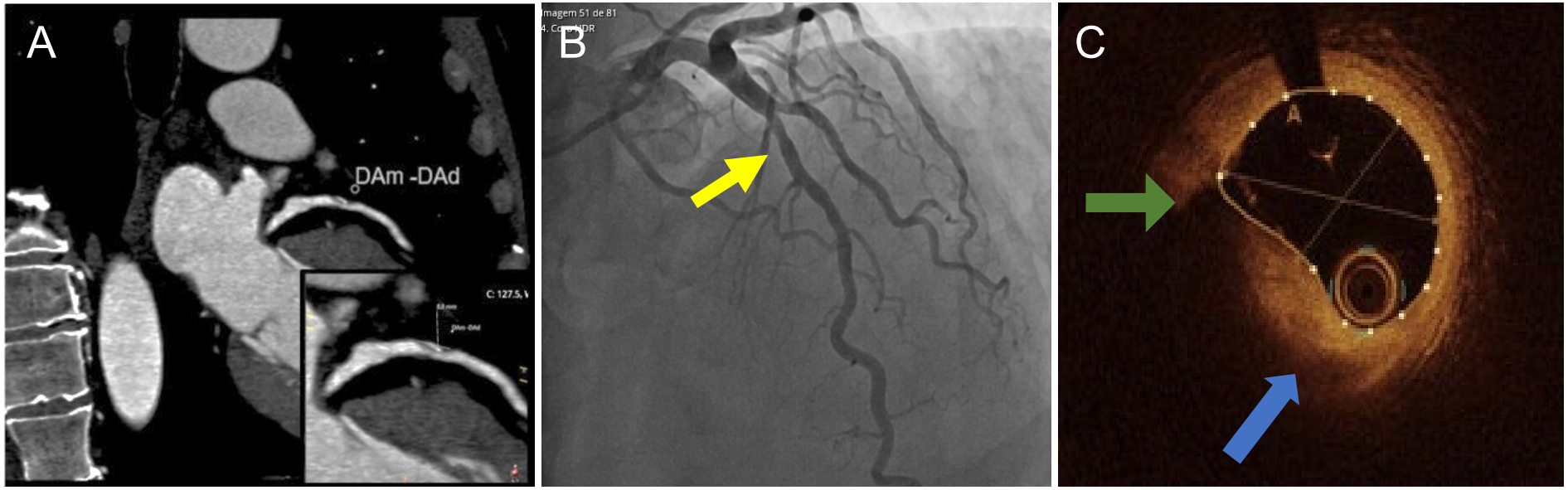
CT, MRI, ultrasound (including intravascular ultrasound) and - more recently - optical coherence tomography (OCT) are used to analyse the atherosclerotic plaque structure, thus playing an important role in therapeutic decisions.



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.

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.

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Atherosclerosis

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)
- Ischemia (= insufficient blood flow to provide adequate oxygenation); ischemia leads to tissue hypoxia or anoxia. Ischemia 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 ischemic attack, an ischemic stroke or cognitive impairment due to chronic white matter ischemia.

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).

=> See also e-book Chapter on CNS imaging!

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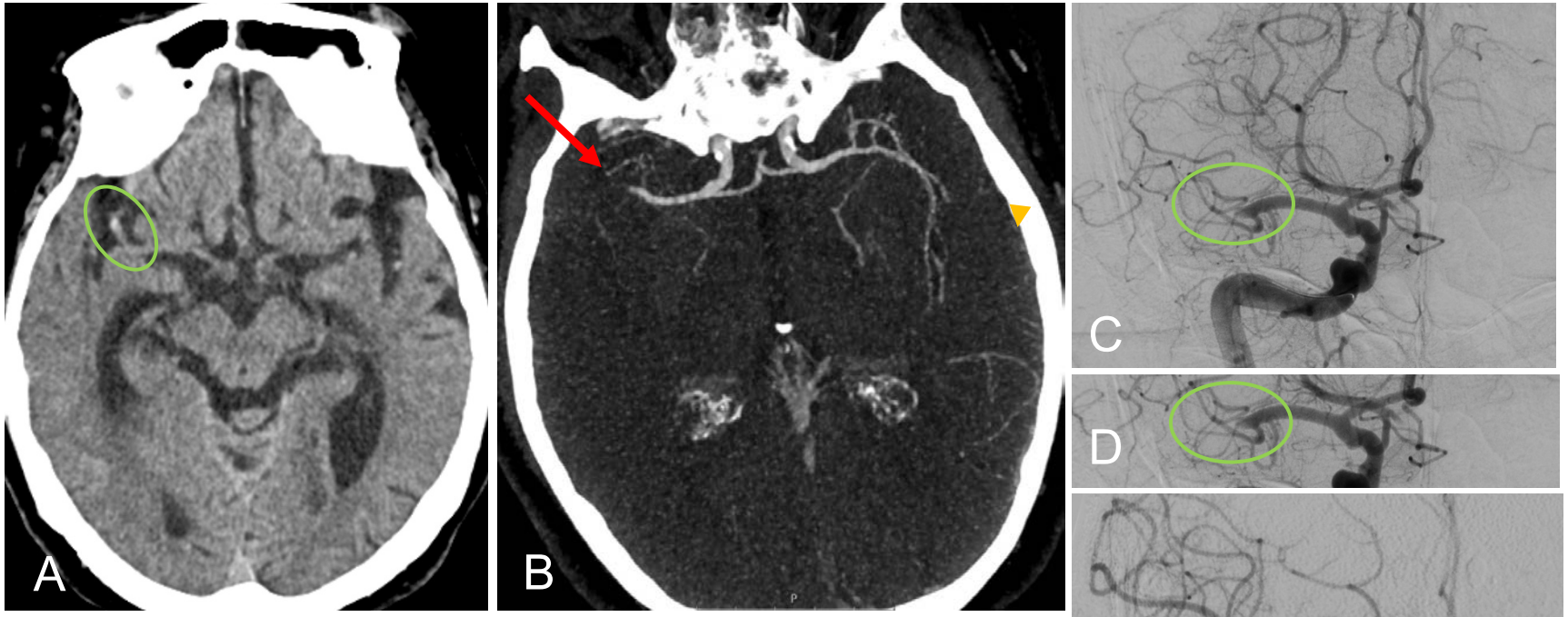
Atherosclerosis:
Middle Cerebral Artery (MCA) Occlusion



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).

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Atherosclerosis

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 ischemia, which can present clinically with intermittent claudication in mild to moderate PAD (ischemic pain during exercise due to tissue hypoxia caused by higher oxygen demand), with rest pain, ischemic 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.

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.

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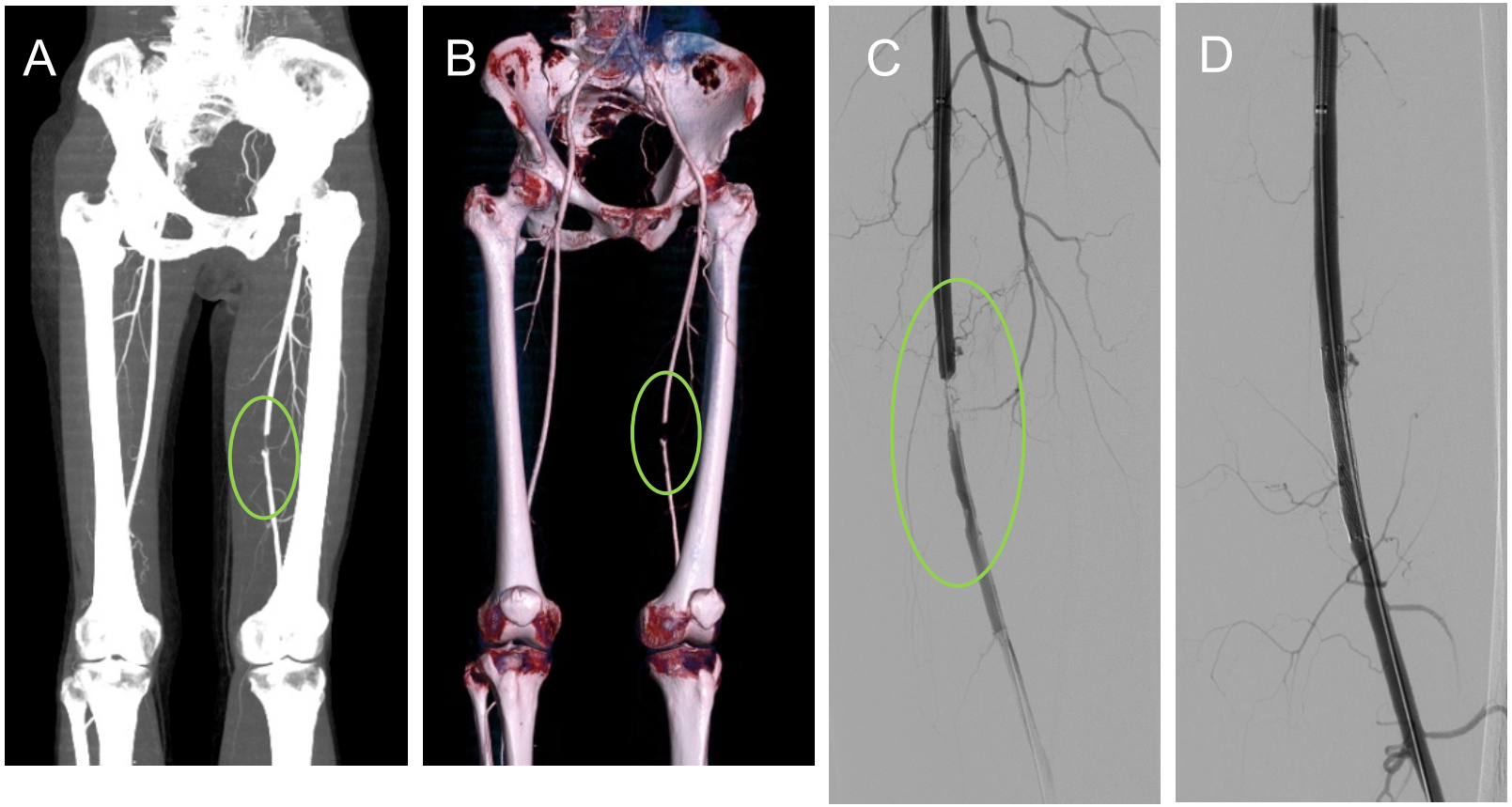
Atherosclerosis:

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).

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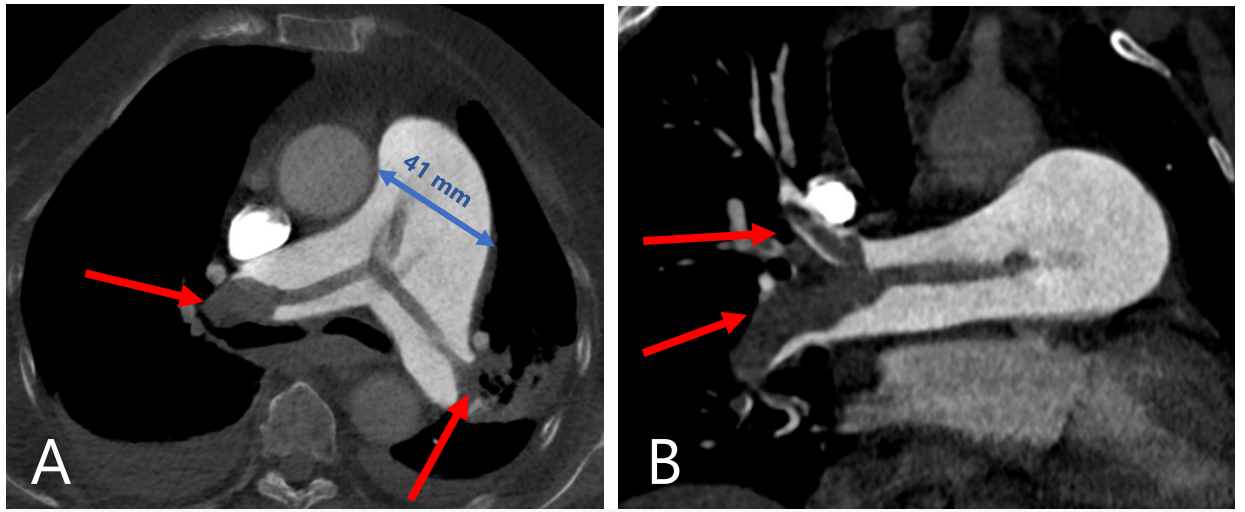
Pulmonary Embolism (PE)

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)



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).

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.

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

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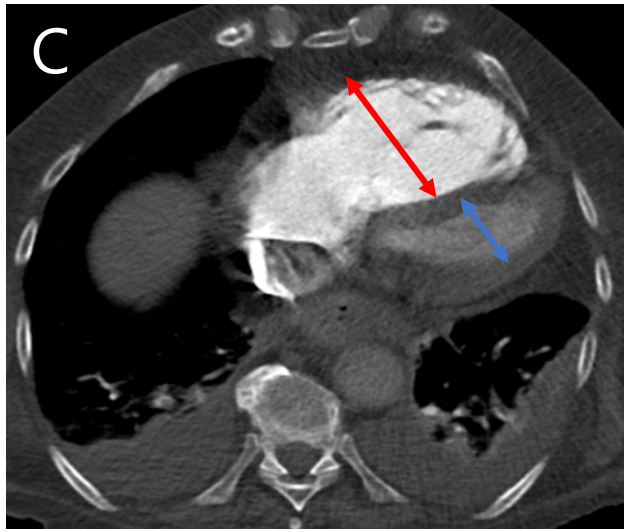
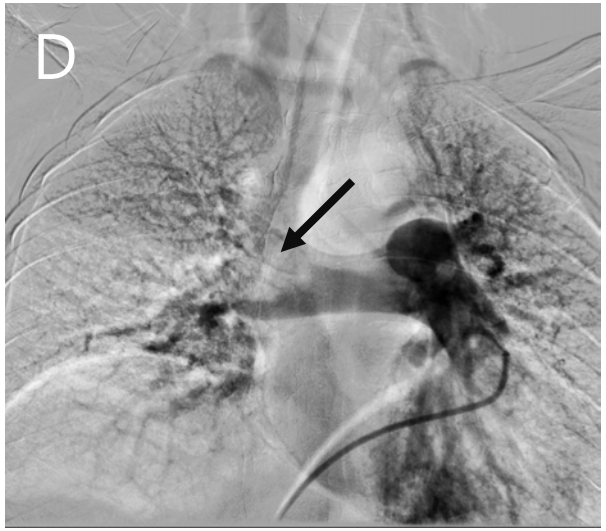
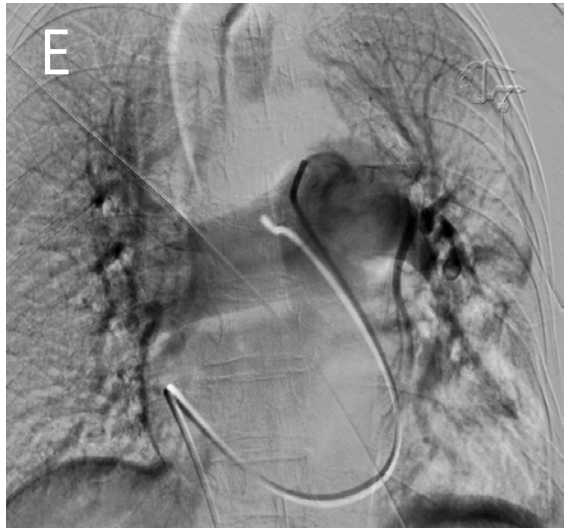
Pulmonary Embolism (PE)

PE treatment depends on risk stratification:

- Low 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
- High 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.

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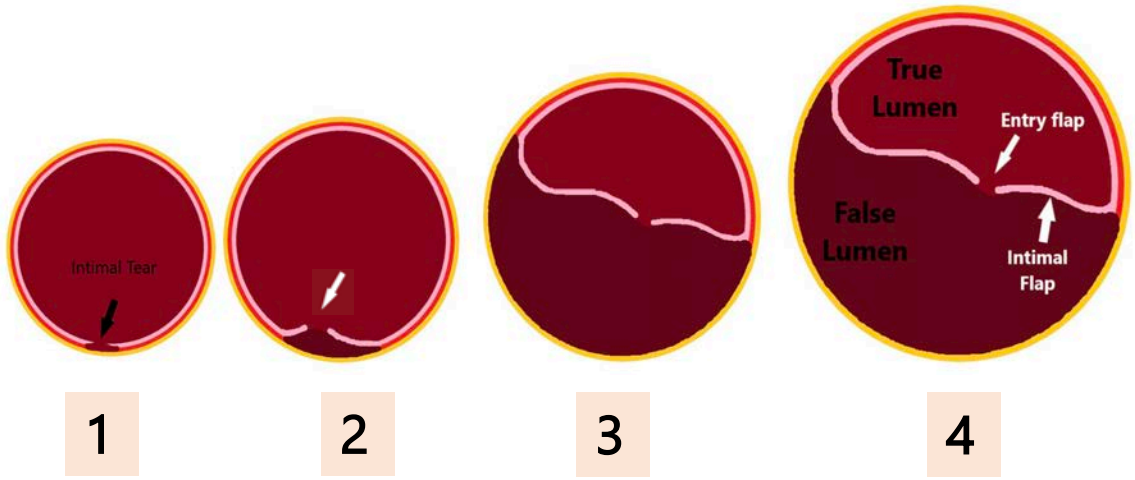
Dissection

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.

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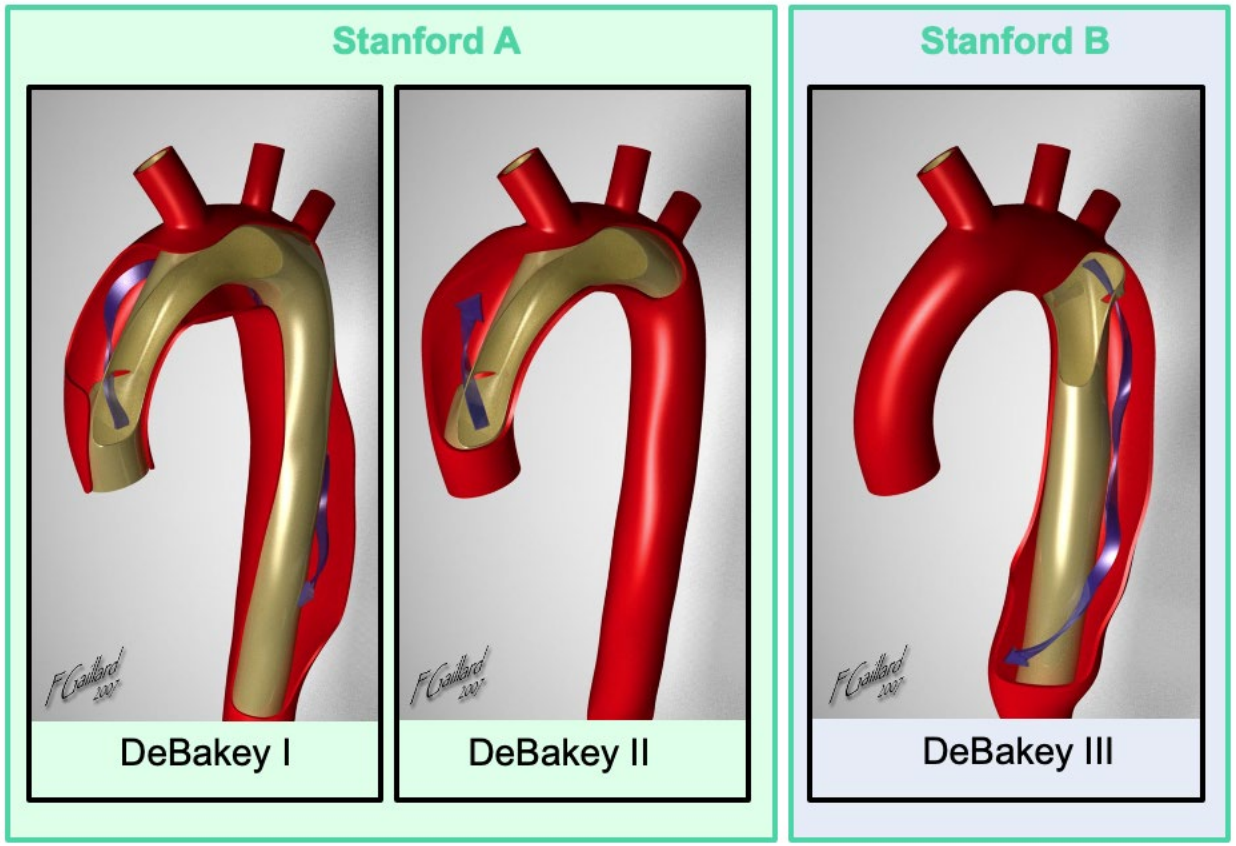
Dissection:
Aortic Arch Dissection Classification



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

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)

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

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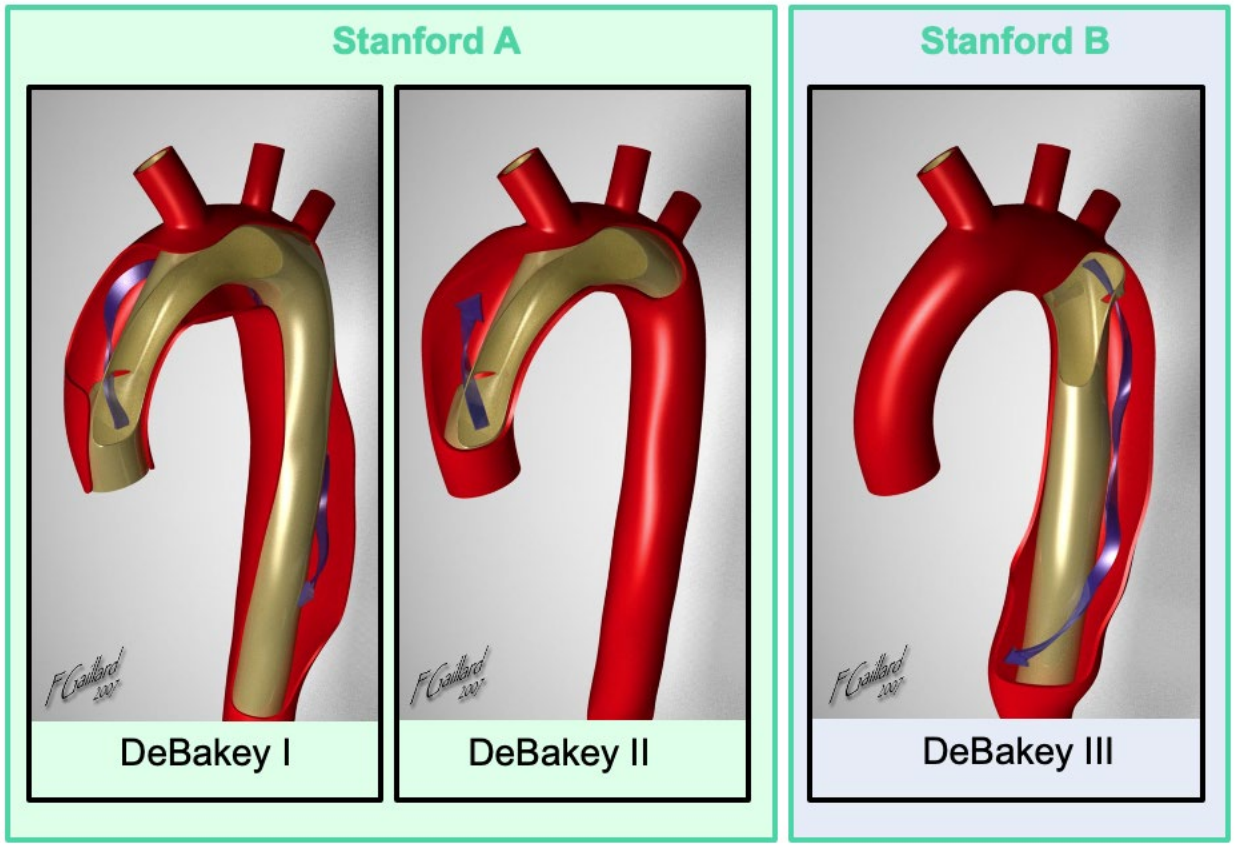

DeBakey classification (Type I-III):

- 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

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Dissection:

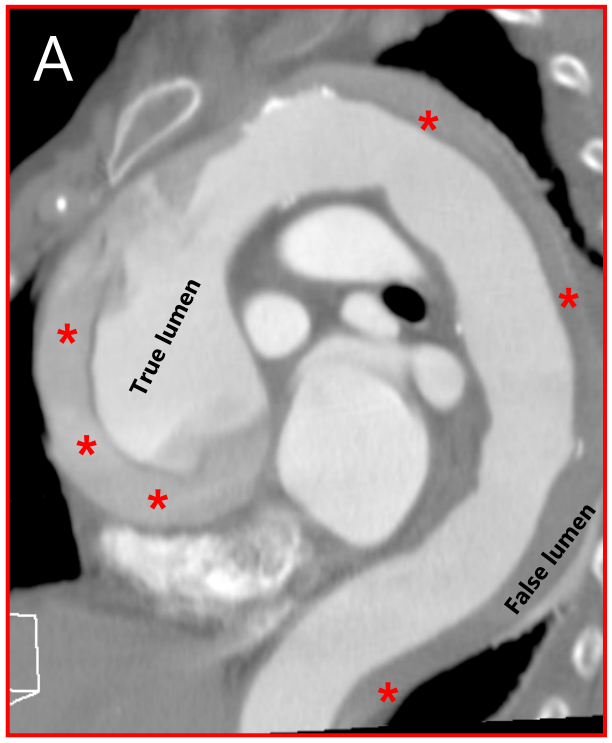
Aortic Arch Dissection Classification

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

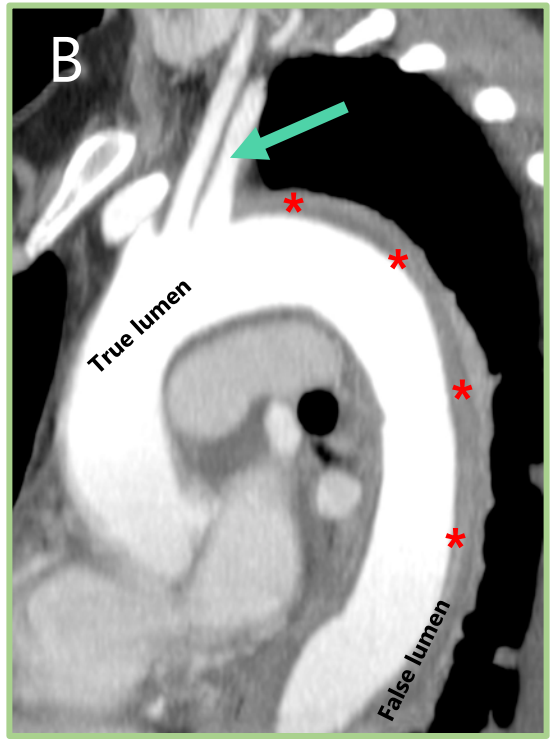
Type A dissections can lead to thrombosis of the coronary arteries and have an increased risk of rupture and cardiac tamponade = Urgent surgical intervention!

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)

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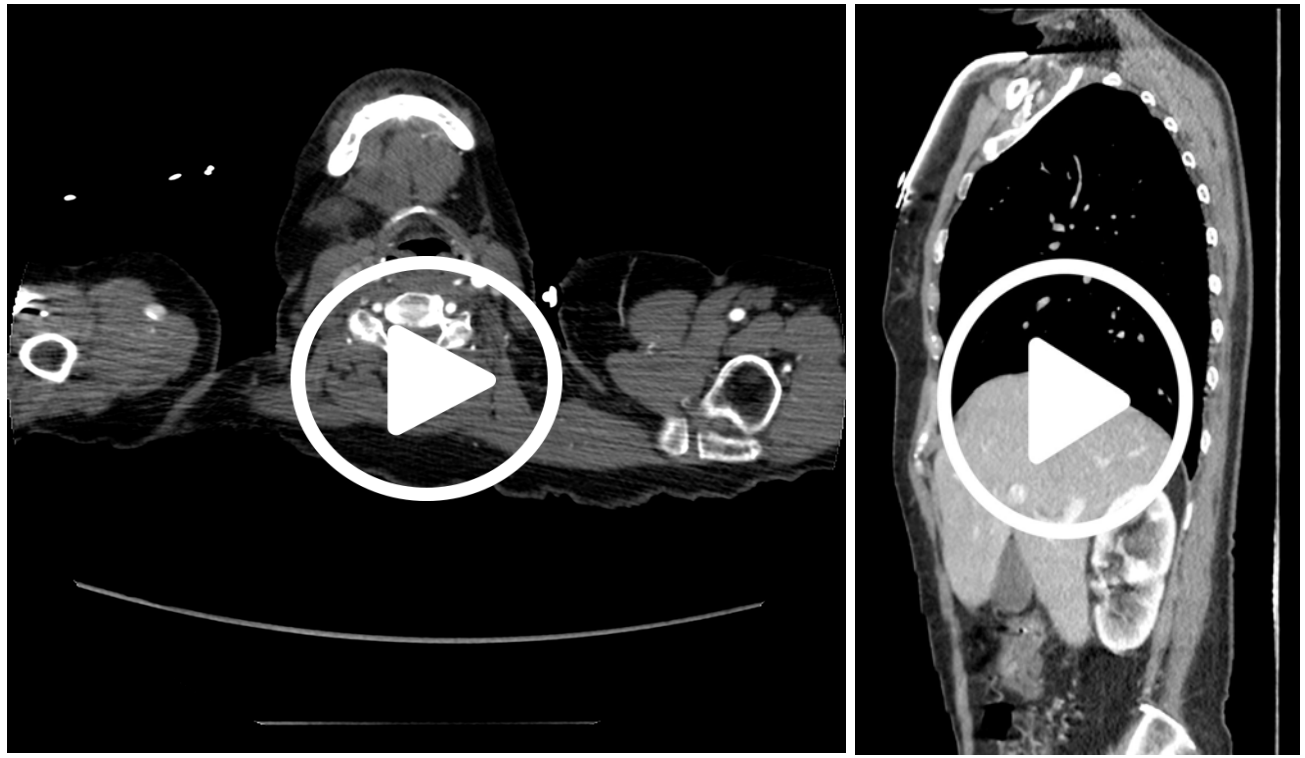
Dissection:

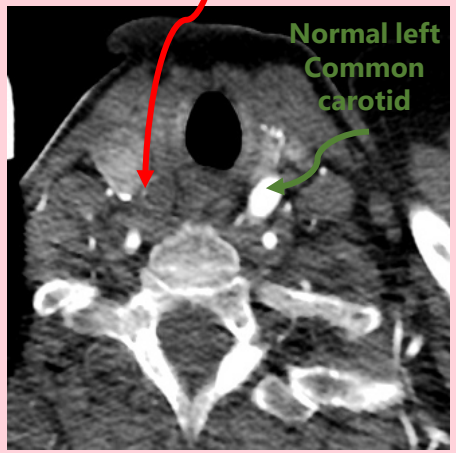
Stanford Type A Dissection



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)

Did you notice the right common carotid artery thrombosis?



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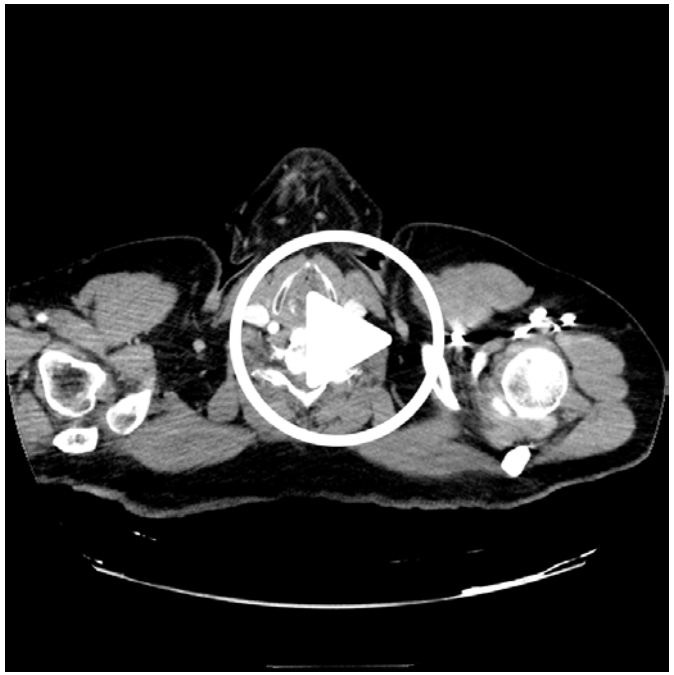
Dissection:
Stanford Type B Dissection

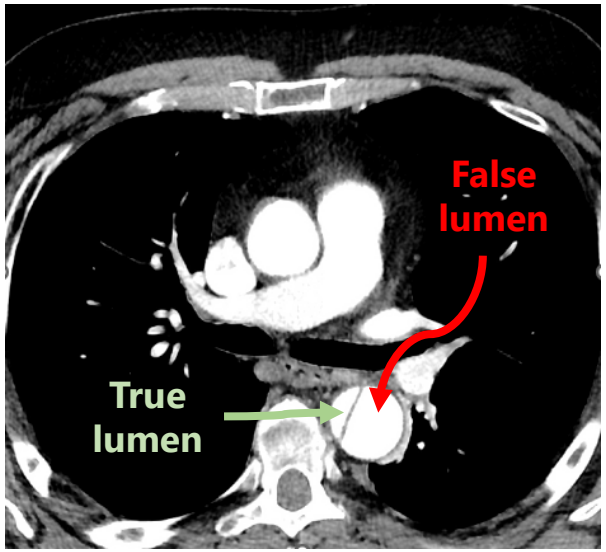


CTA of the aorta (videos): Axial and coronal reconstructions showing dissection starting bellow the left subclavian artery and extending to the infra-renal aorta – Stanford Type B dissection.

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



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Aneurysm

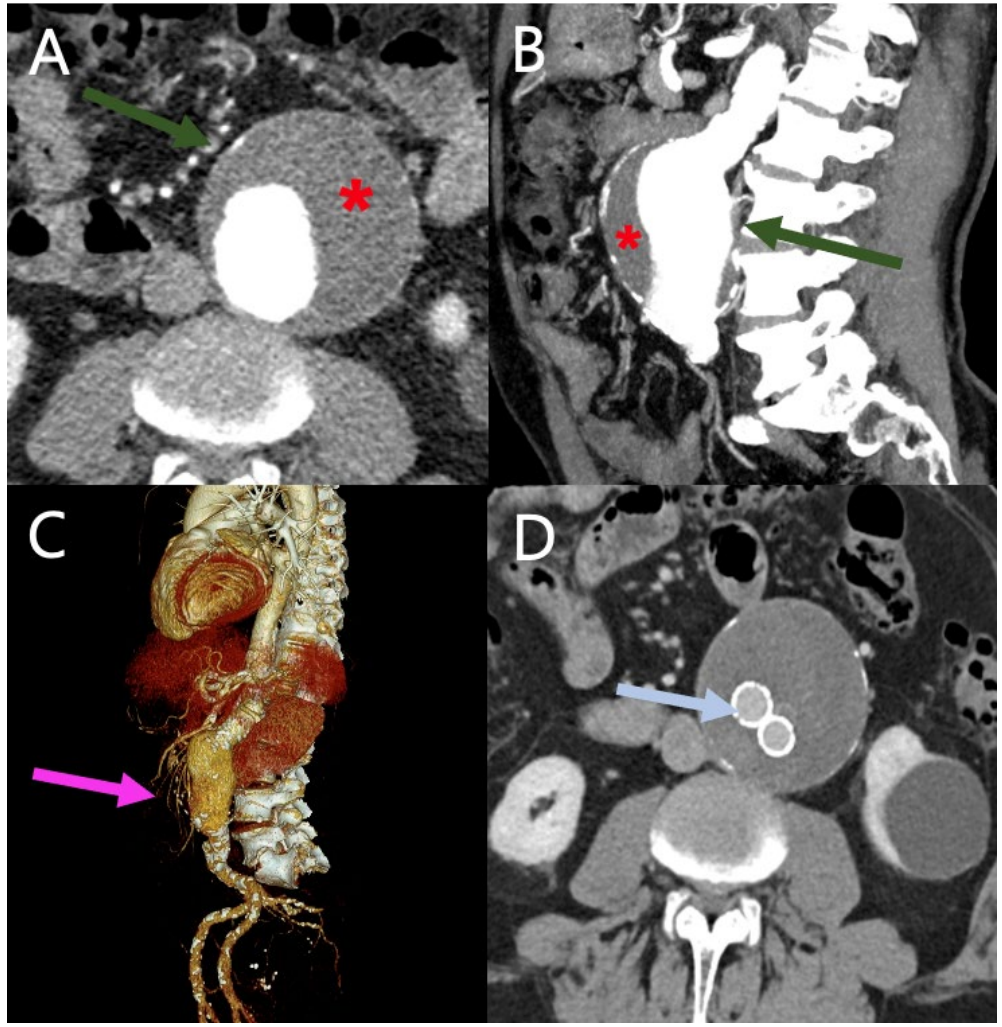
An aneurysm is an abnormal dilatation in a blood vessel due to the weakness of the vessel wall. While aneurysms can affect any blood vessel, they are most common in arteries rather than veins.

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** (arrows) measuring 7x7 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 (arrow). D: CTA axial slice showing abdominal aneurysm excluded by an Endovascular aortic repair (EVAR) graft (arrow).

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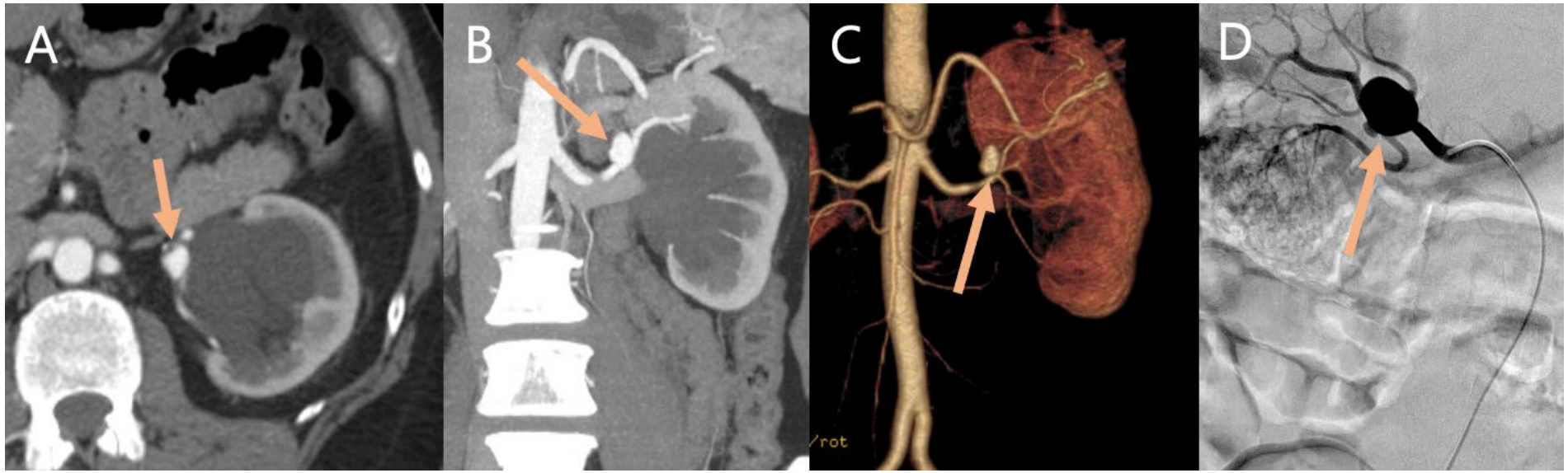
Aneurysm:
Renal Artery Aneurysm



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).

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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).

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

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Vasculitis:
Takayasu Arteritis

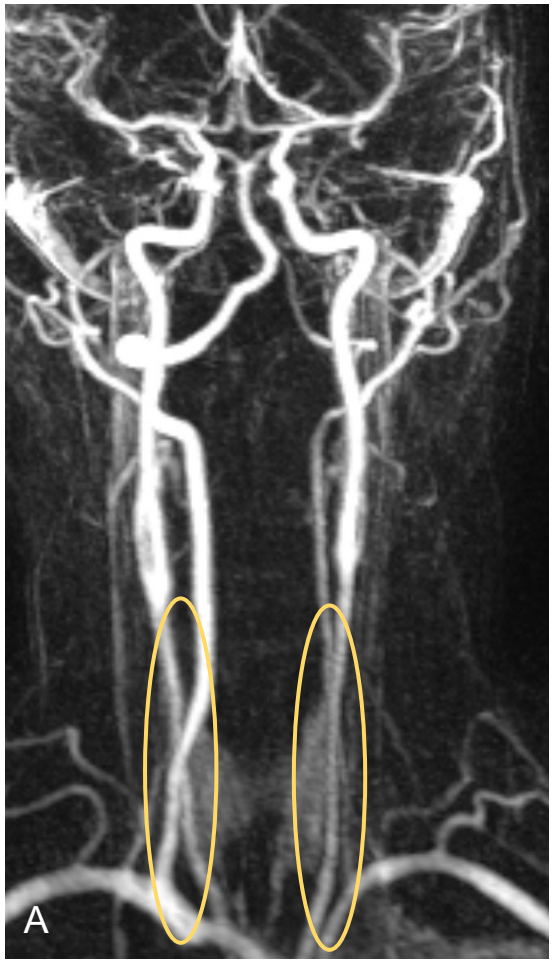
Takayasu Arteritis (Pulseless Disease)

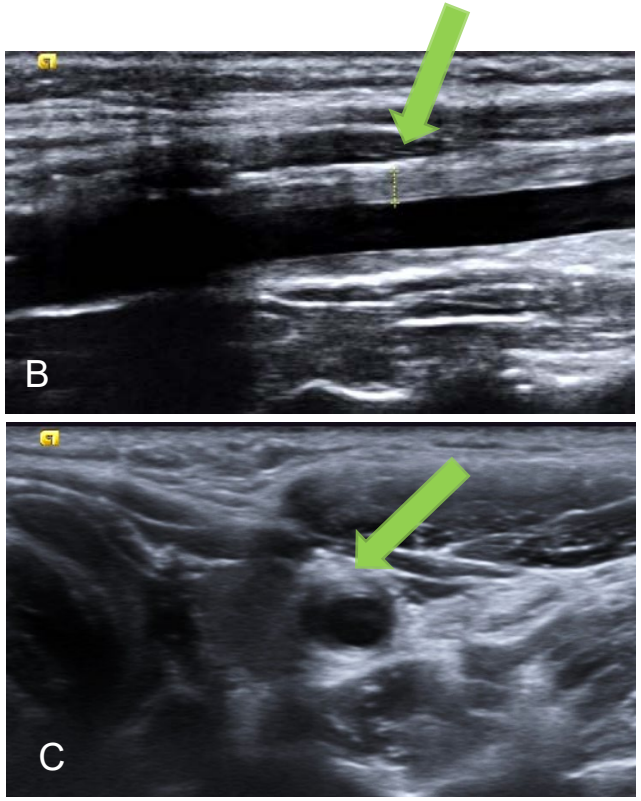
= Large vessel granulomatous vasculitis typically affecting the aorta and its main branches, e.g., the common carotid arteries, brachiocephalic trunk and subclavian arteries

After an initial systemic manifestation with fever, night sweats and arthralgia, follows a chronic phase with limb ischemia, 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

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Fibromuscular Dysplasia (FMD)

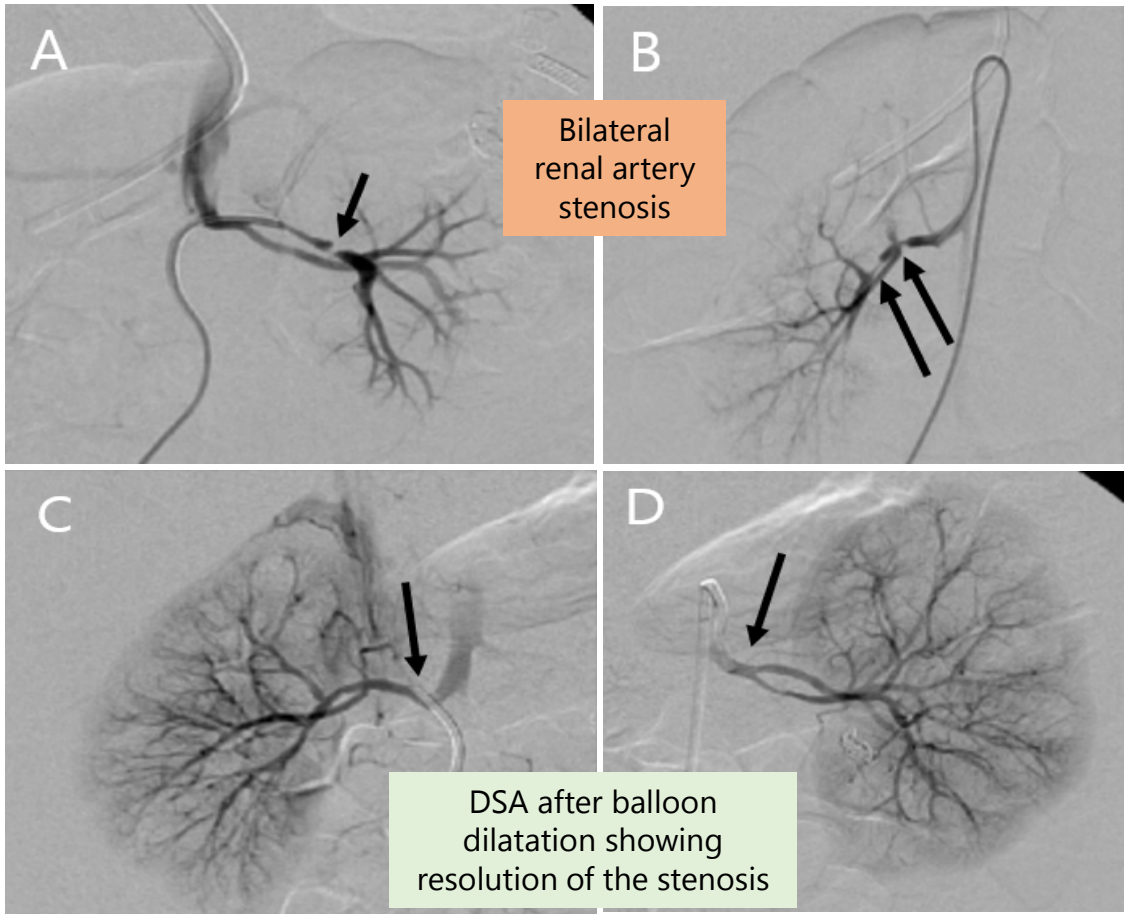
Fibromuscular dysplasia (FMD) is an idiopathic, focal, non-inflammatory and non-atherosclerotic disease that affects small and medium size arteries.

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.

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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:

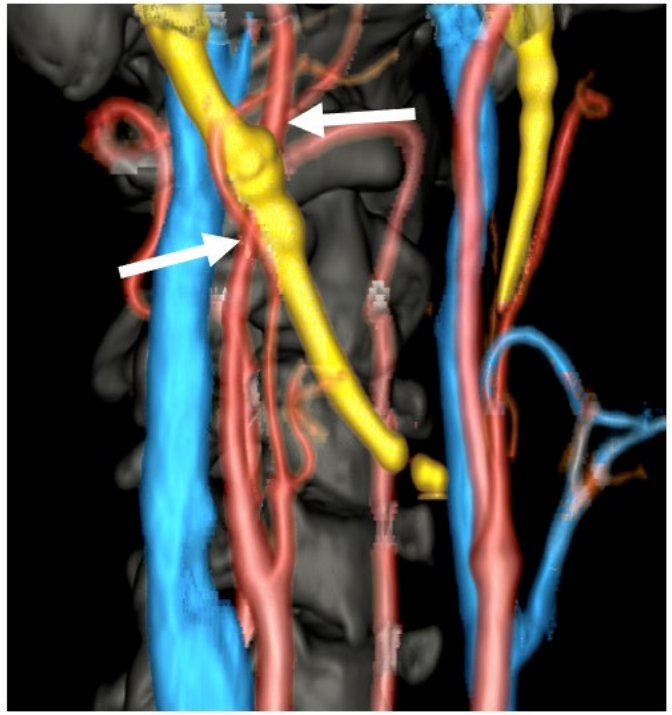
- Anomalous origin of the coronary artery (which courses between the ascending aorta and the pulmonary trunk)
- Hypothenar hammer syndrome (compression of the ulnar artery by the hypothenar muscles)
- Eagle syndrome (see below) and many more

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 > 3cm. 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.

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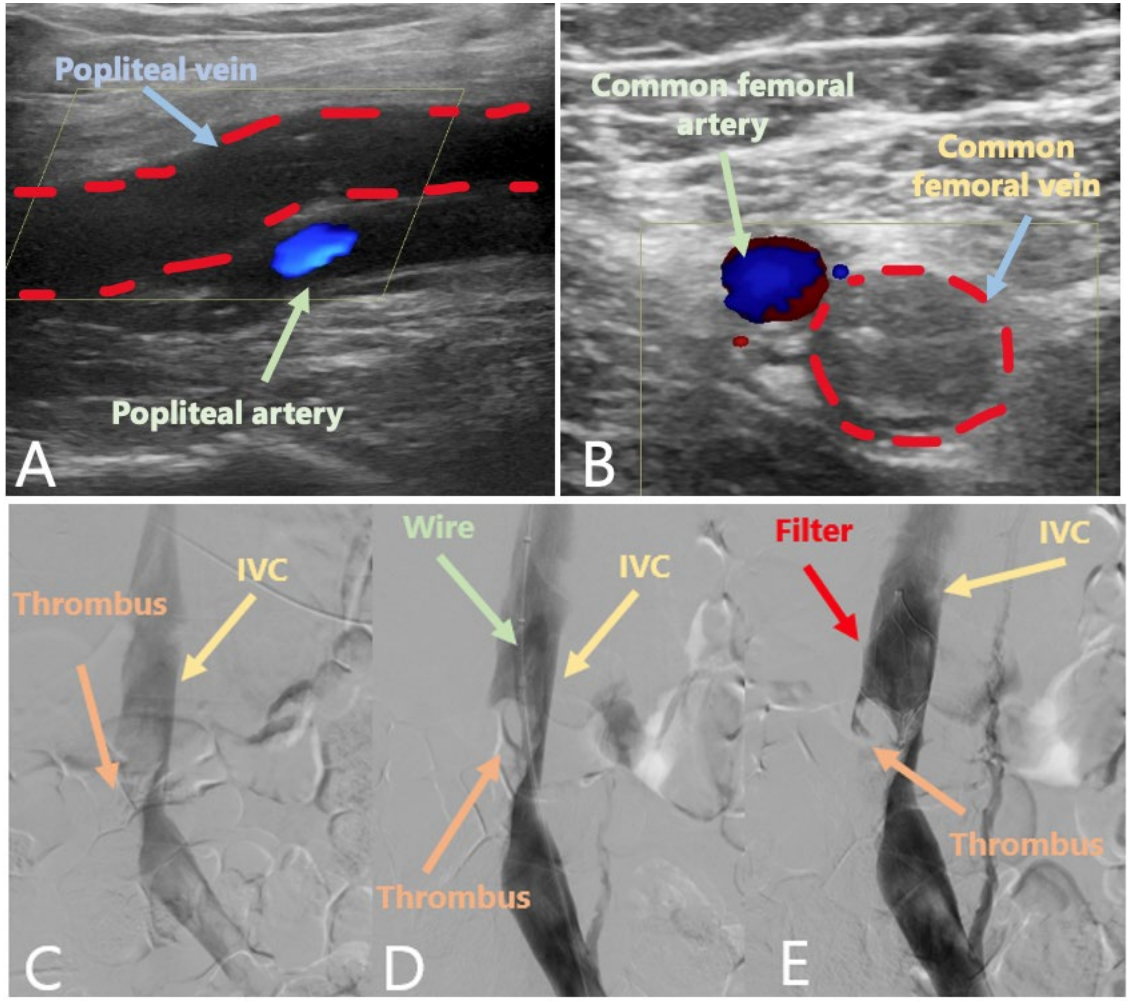
Deep Vein Thrombosis (DVT)

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).

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

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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.

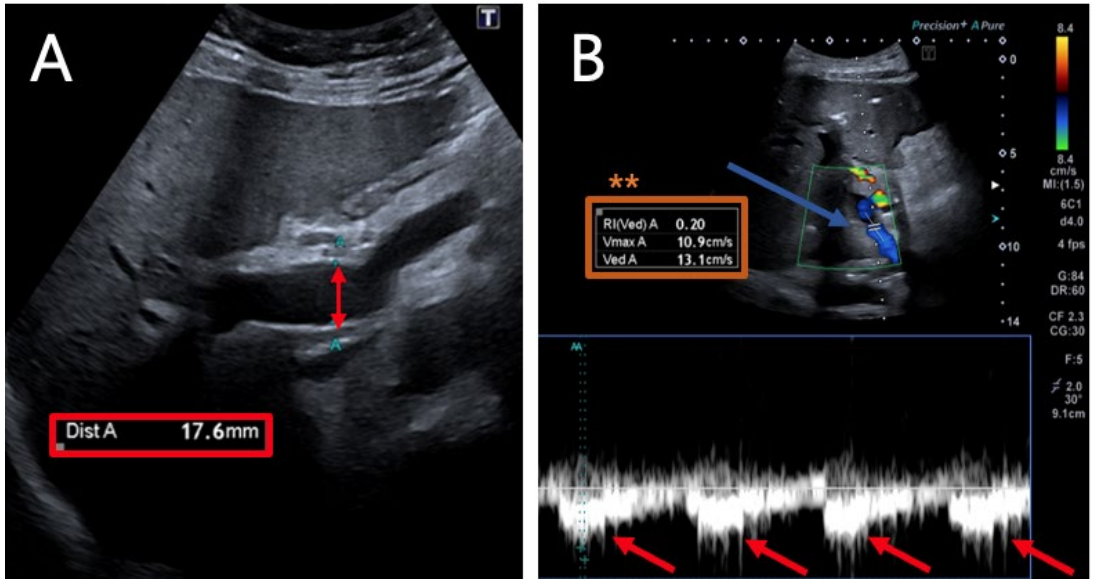
This becomes clinically important when the pressure gradient rises to over 10 mmHg, due to the increased risk of complications.

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.



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!

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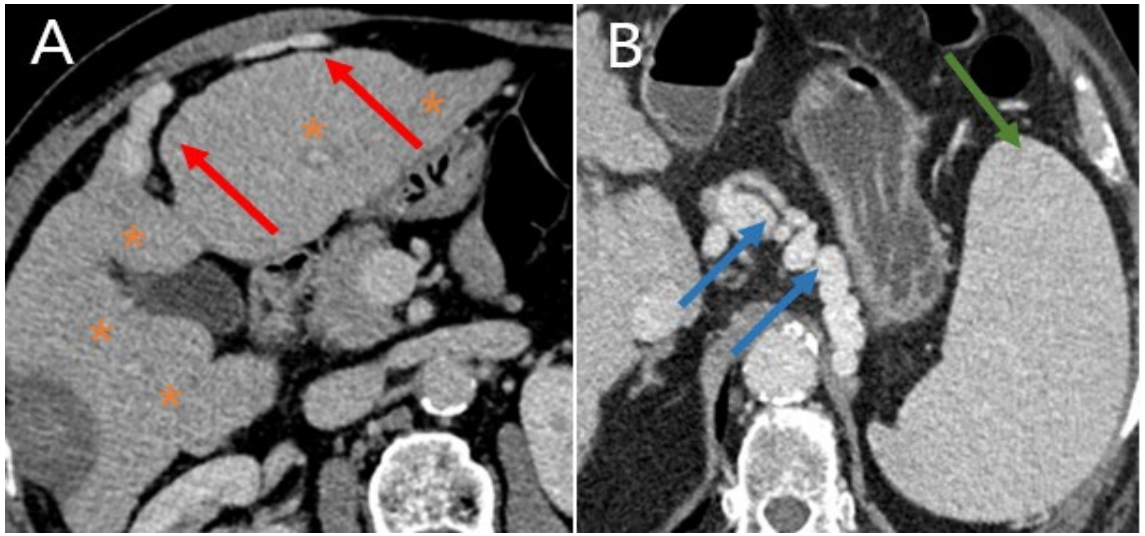
Portal Hypertension (PH)

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 in another patient showing an increased spleen size (splenomegaly, arrow) and an abnormally engorged and tortuous varicose left gastric vein (arrow).

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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.

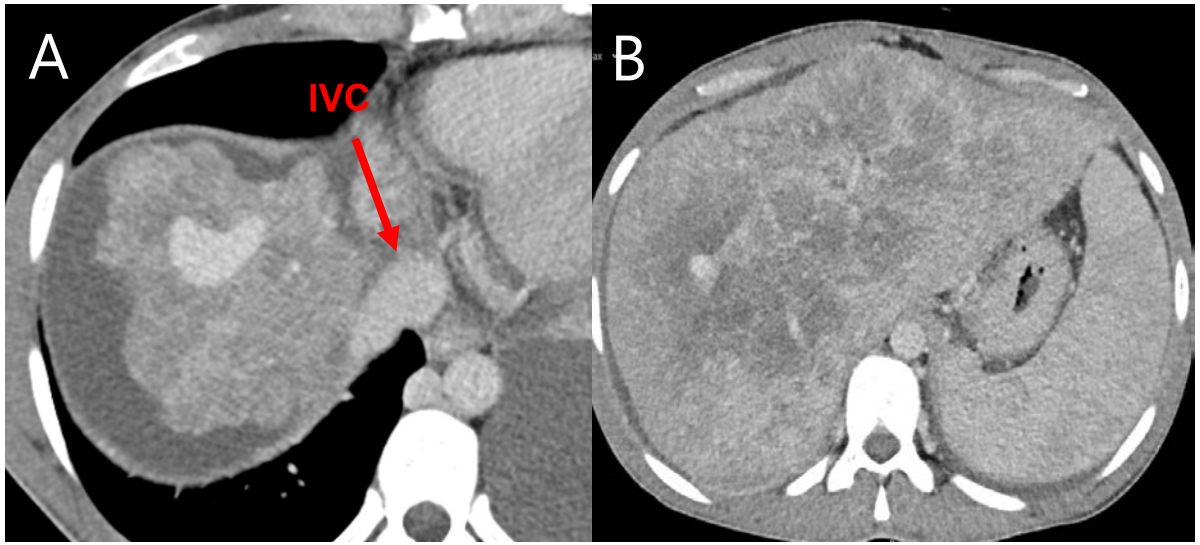
The classic acute presentation is the triad of ascites, abdominal pain and hepatomegaly.

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

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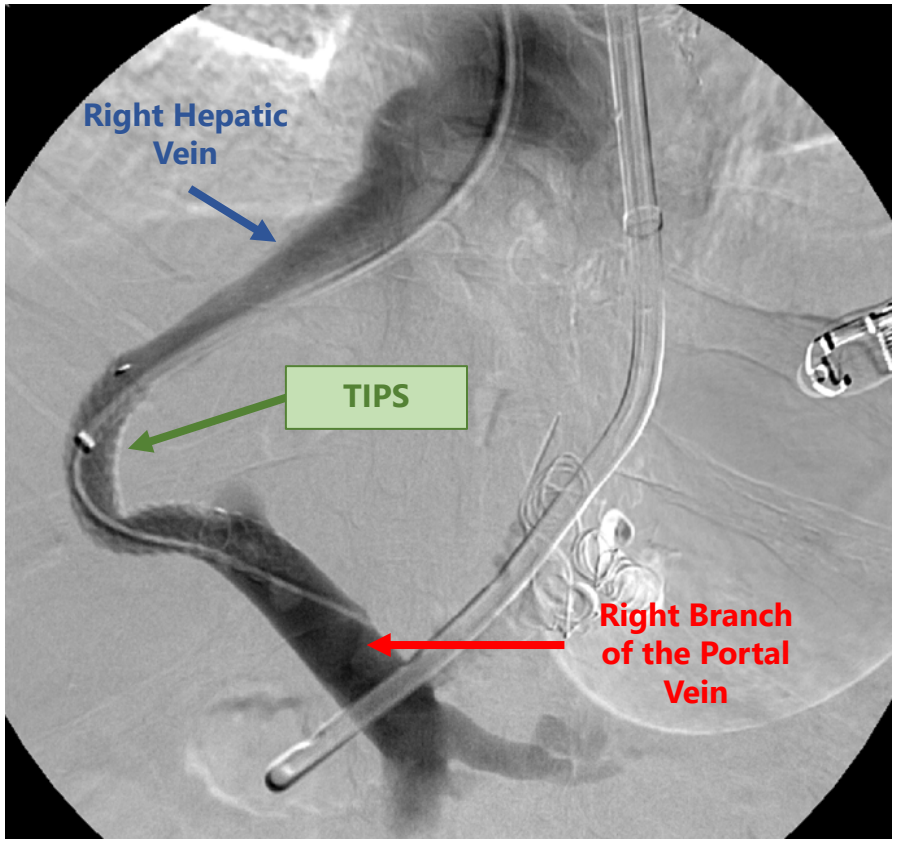
Budd-Chiari Syndrome (BCS)

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.

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 (arrow) and the right hepatic vein (arrow) – TIPS (arrow).

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Venous Compression Syndromes

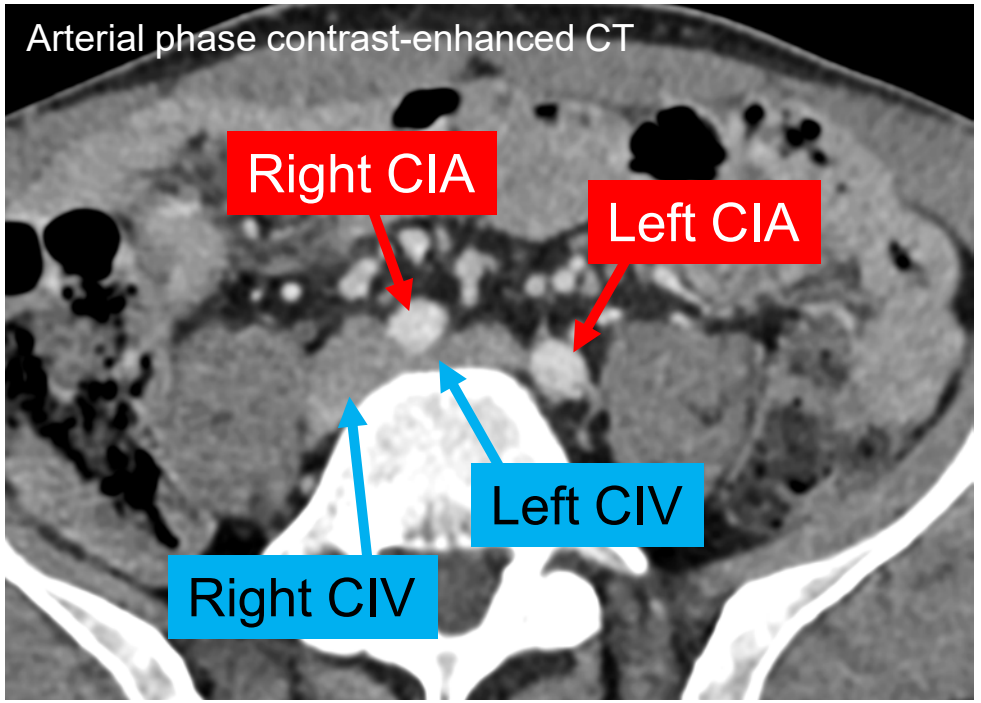
Venous compression syndromes include:

- 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)

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.



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Vascular Tumours and Malformations:
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.

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)

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.

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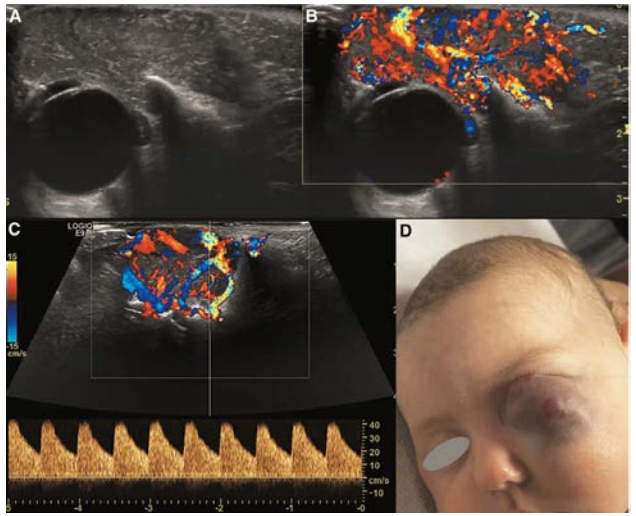
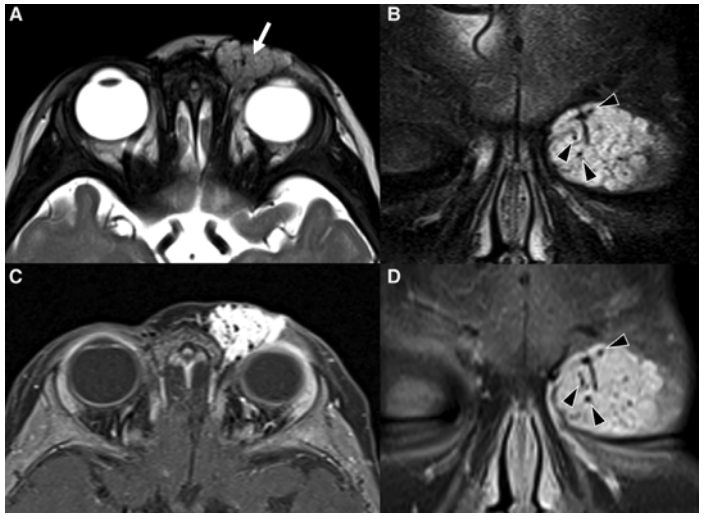
Benign Vascular Tumours

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.

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.

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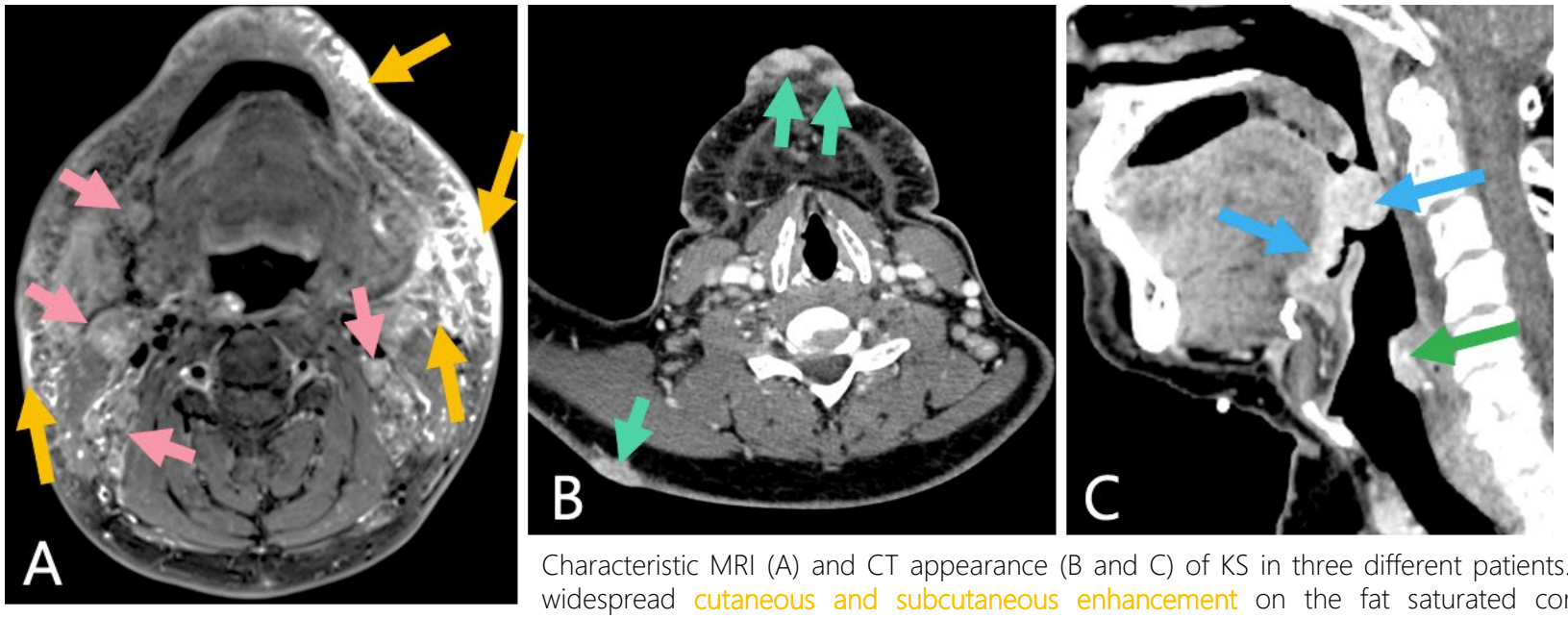
Locally Aggressive Vascular Tumours

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**.



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

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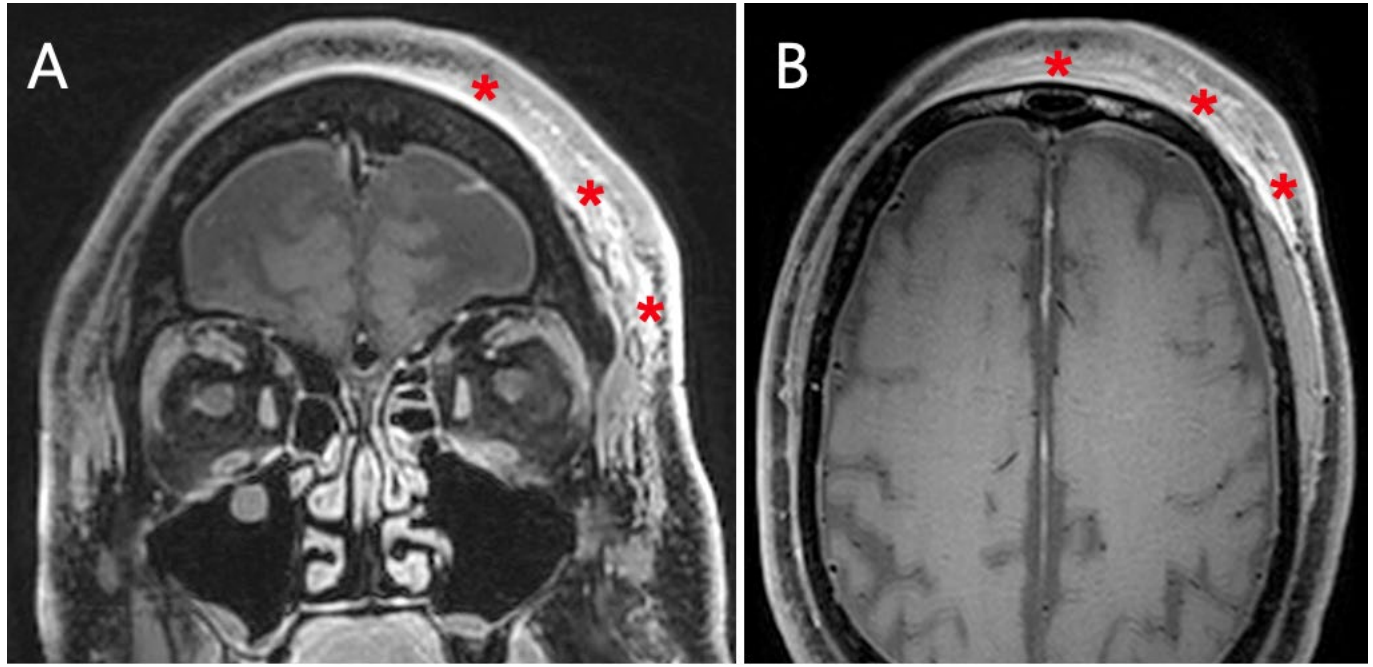
Malignant Vascular Tumours

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.



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.

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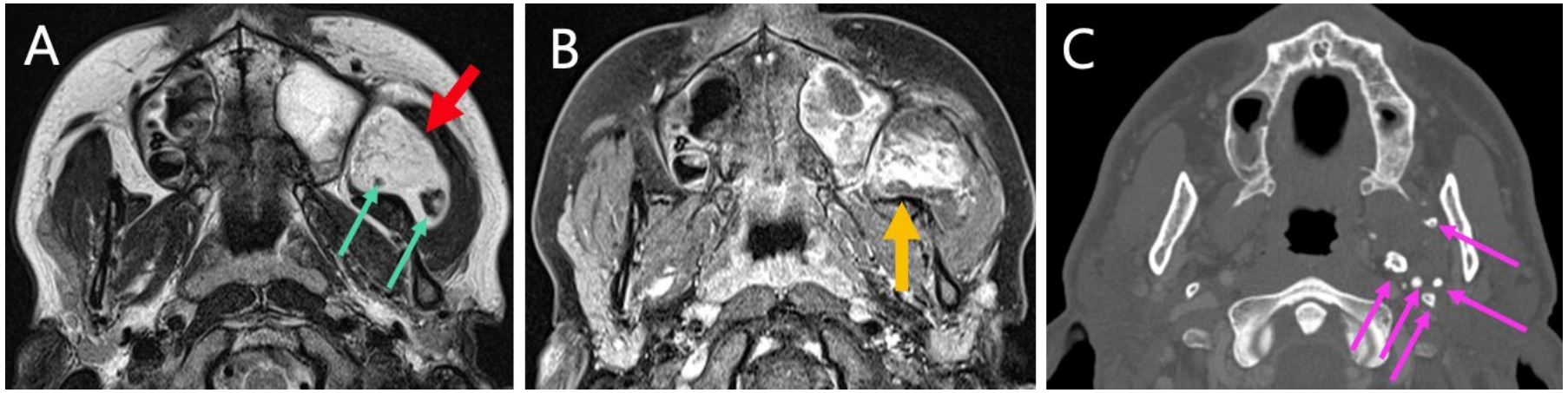
Vascular Malformations

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.

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).

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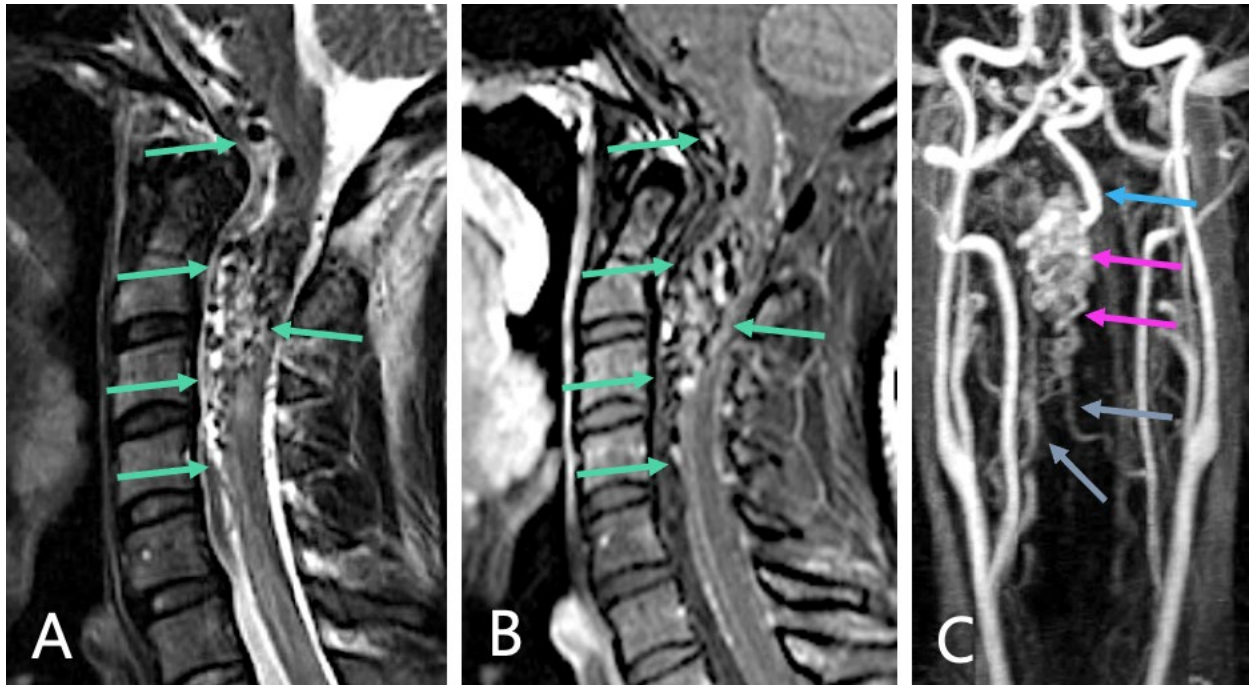
High-flow vascular malformations include AVMs and AVF, which **connect arteries and veins, bypassing the capillary bed**.

- **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.



Sagittal T2-weighted (A) and contrast enhanced fat saturated T1-weighted (B) images show the typical features of a spinal AVM. Note flow voids 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 and dilated draining veins and a nidus.

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

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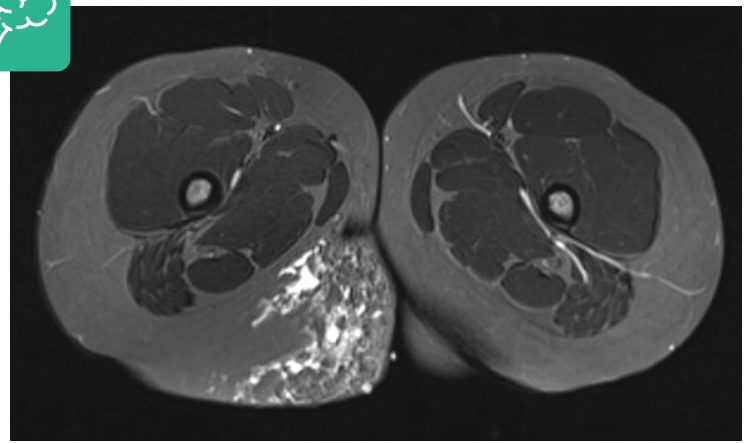
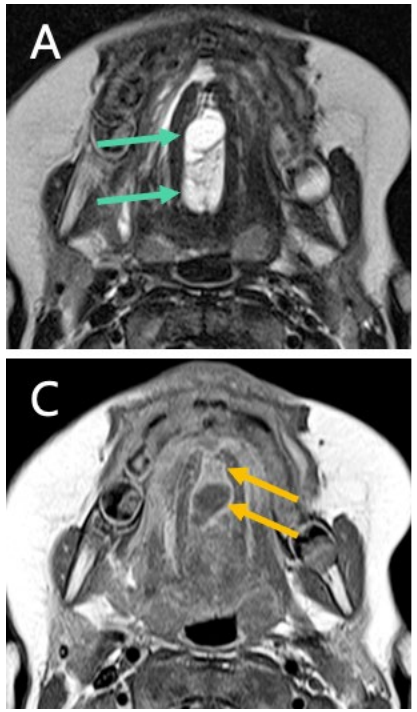
Lymphatic malformations are **low-flow** malformations that most often present in childhood; they tend to involve multiple anatomic spaces and structures. They do not communicate with normal lymph vessels and are most often seen in the head and neck subcutaneously. Lymphatic malformations can cause osseous 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.



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 on T2 (A), mixed low and high signal on T1 due to spontaneous bleeding. Minimal, smooth enhancement of the thin cyst walls (C).

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

a. Allergic reaction
b. Vessel damage from the catheter
c. Infection at the site of catheter insertion
d. Acute kidney injury

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

- ✓ a. Allergic reaction
- ✓ b. Vessel damage from the catheter
- ✓ c. Infection at the site of catheter insertion
- ✓ d. Acute kidney injury

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

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

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Test Your Knowledge

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

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- • d. The procedure is always performed under general anaesthesia

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

TRUE / FALSE

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Test Your Knowledge

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

TRUE / FALSE

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4) To perform CT angiography, it is always necessary to inject contrast media?

TRUE / FALSE

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4) To perform CT angiography, it is always necessary to inject contrast media?

TRUE / FALSE

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

a. Decreased
b. Increased
c. Not changed
d. Variable

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

- a. Decreased
- ✓ b. Increased
- c. Not changed
- d. Variable

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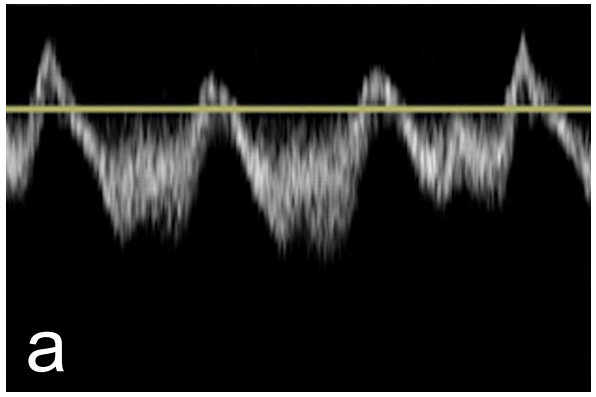
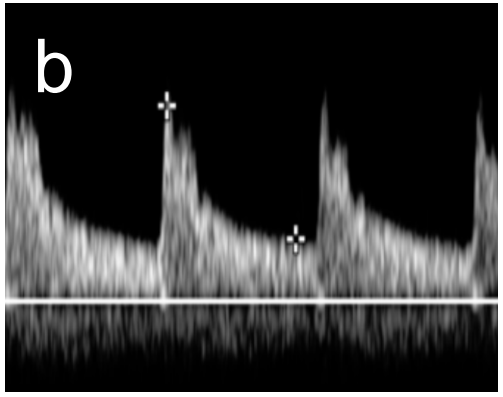
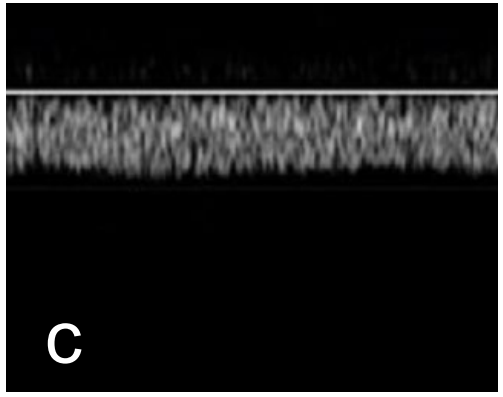
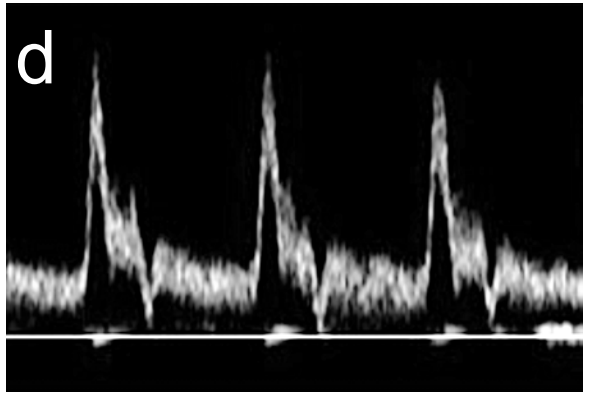
6) What type of waveform can you see at Doppler-US after a major arterial stenosis?

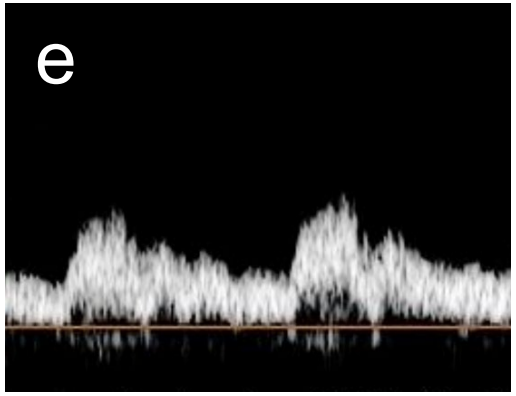
- a, b, c, d or e?











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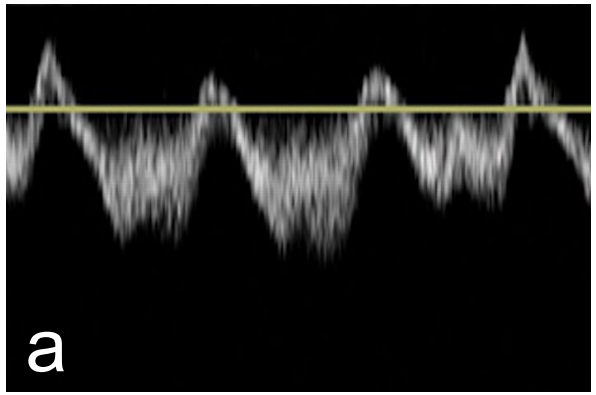
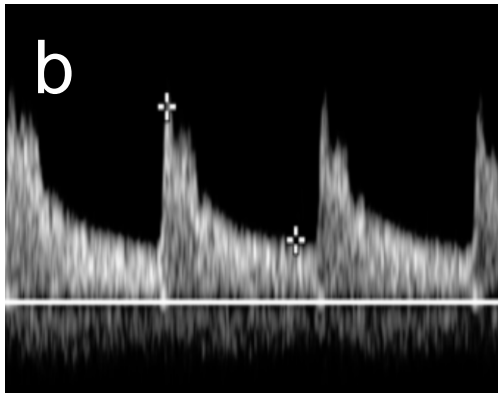
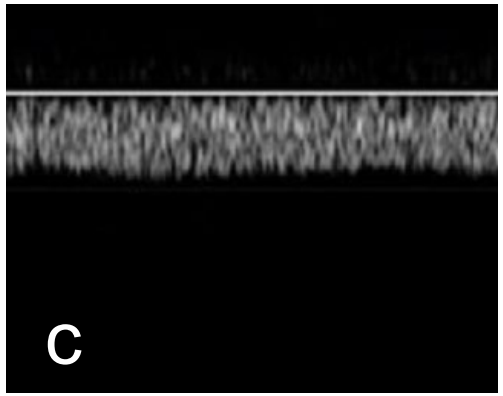
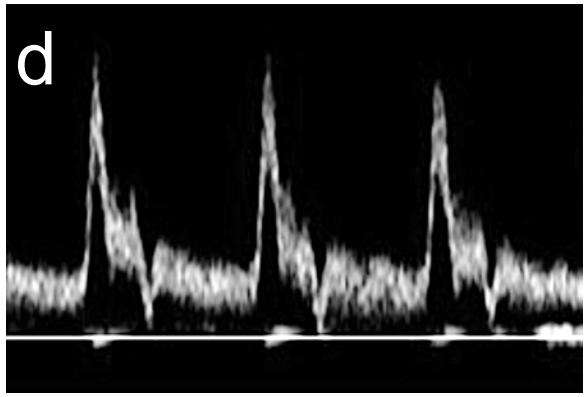
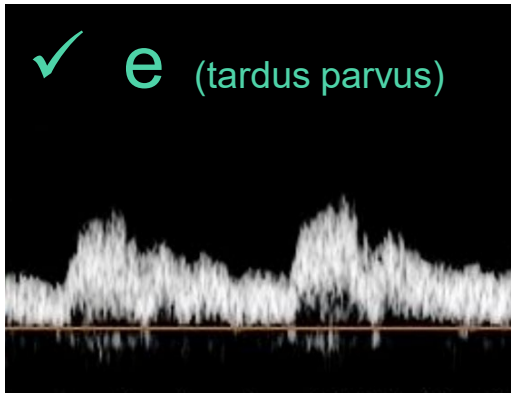
- a, b, c, d or e?











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

a. Larger than the true lumen
b. Less enhancement than the true lumen
c. Beak sign
d. Surrounded by calcifications (if present)

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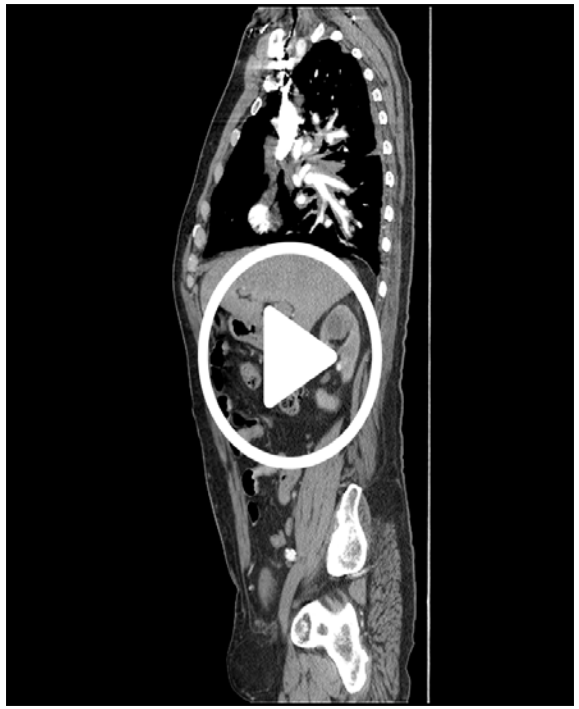
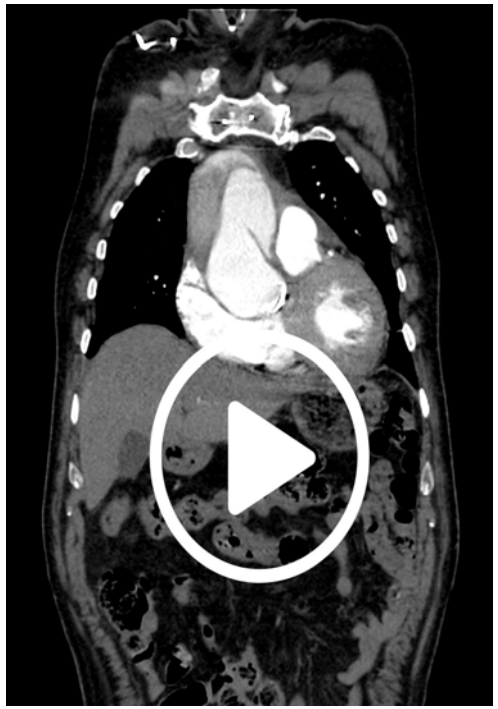
8) What type of aortic dissection is this (see videos)?

a. DeBakey I
b. Stanford B
c. DeBakey III
d. Stanford A or DeBakey II





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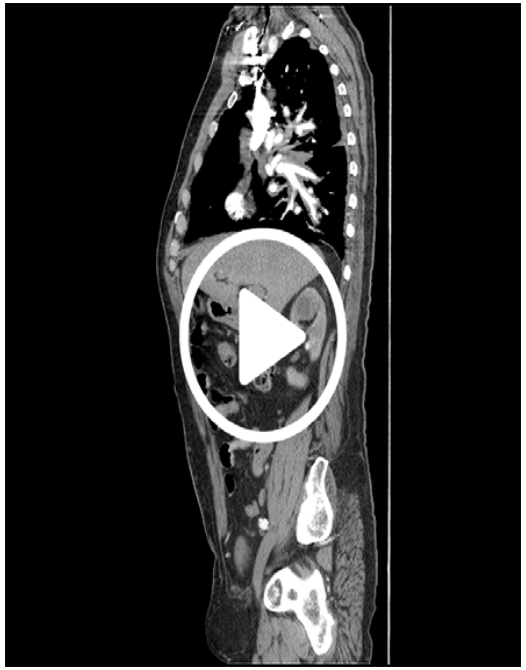

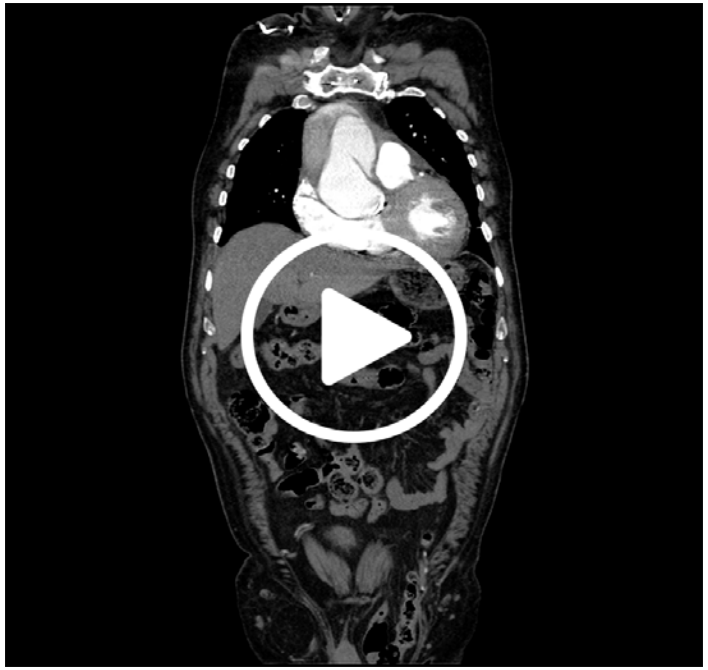

CTA of aorta (videos). Sagital and coronal reconstructions showing Stanford type A dissection (DeBakey II) affecting the ascending aorta.

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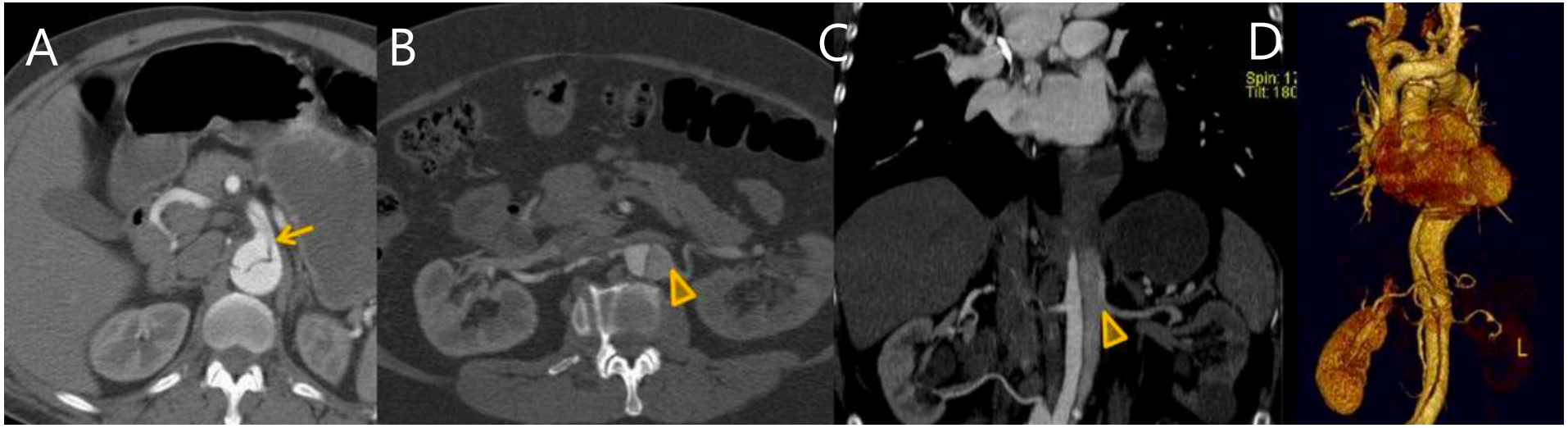
9) Which statements regarding the CTA images below obtained in the emergency setting are correct ?

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



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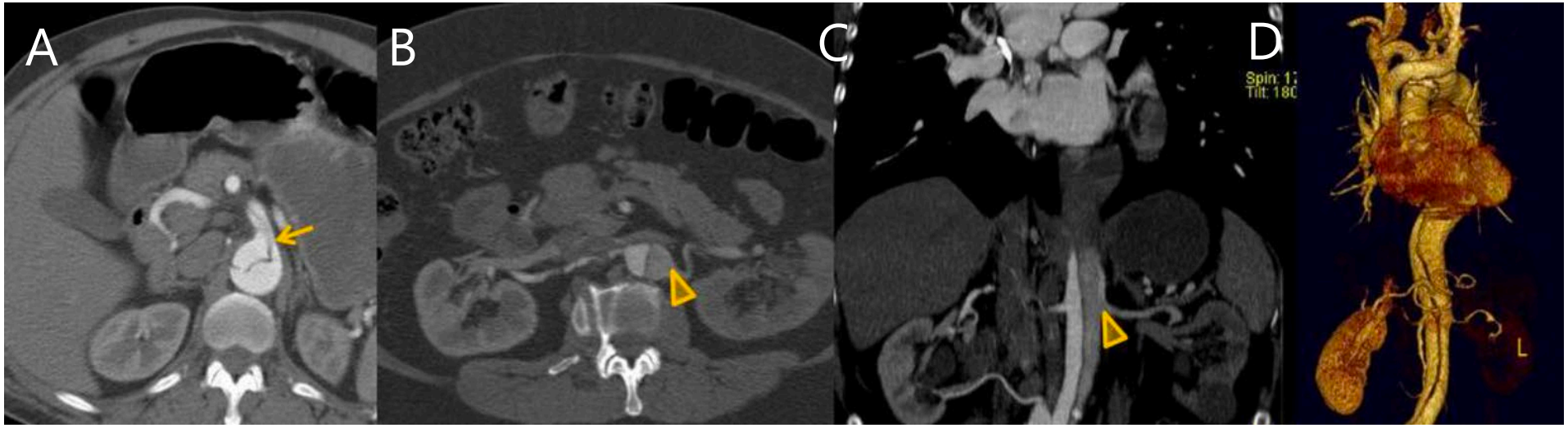

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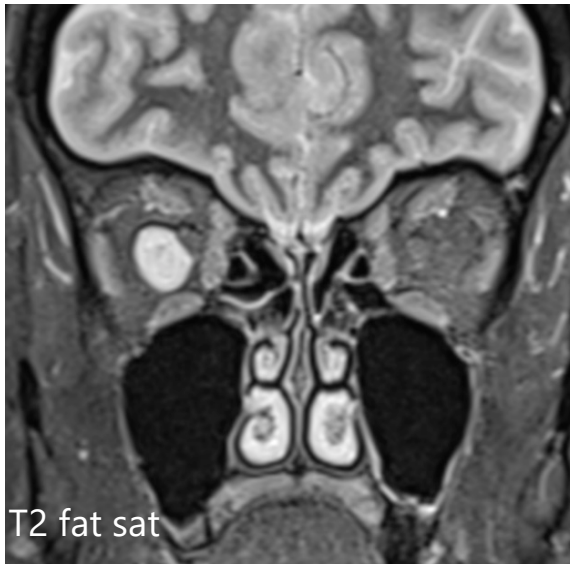
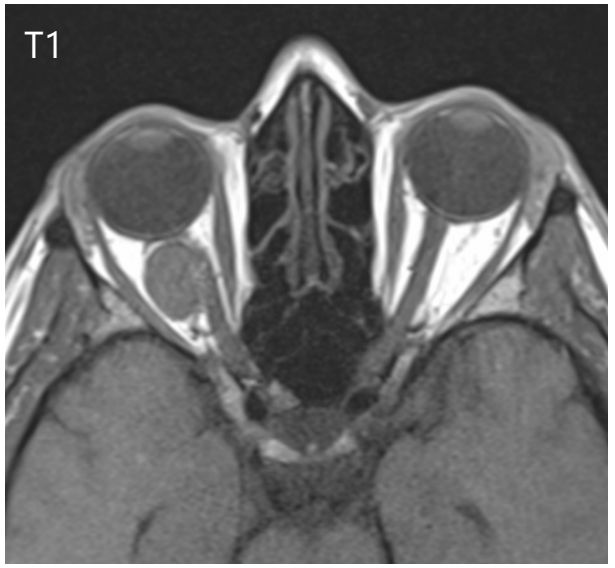
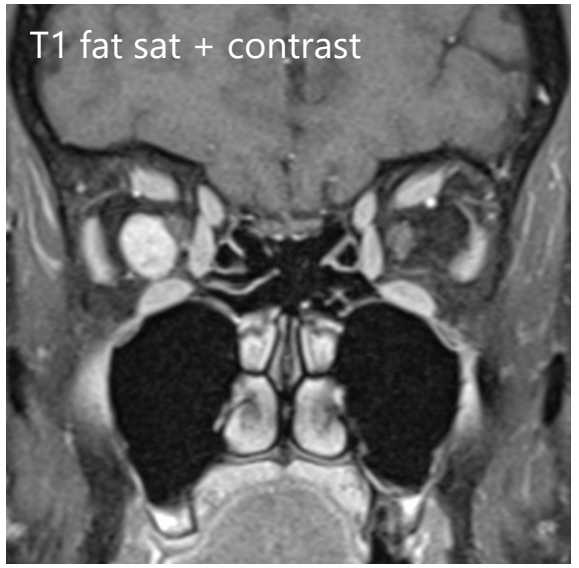
10) Which statements regarding the MR images below obtained in a 20-year-old patient are correct ?

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







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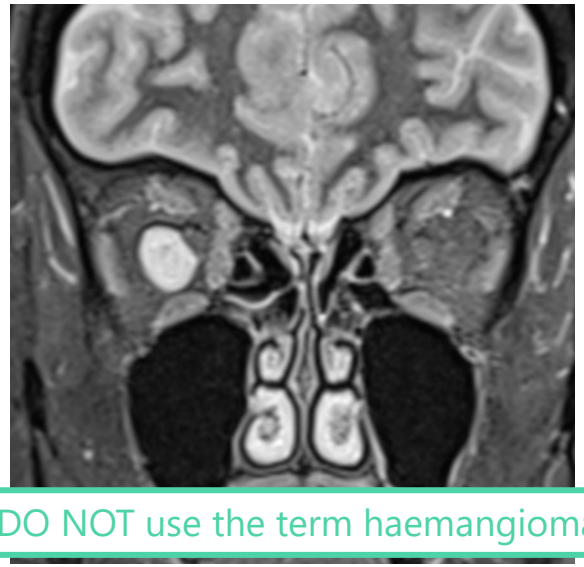

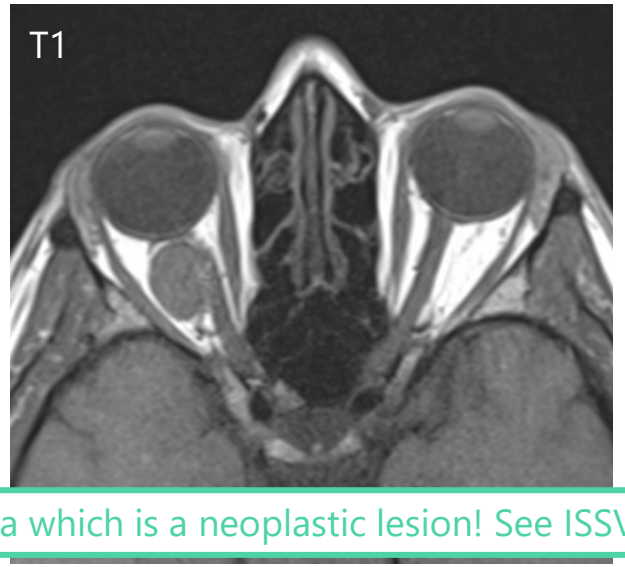

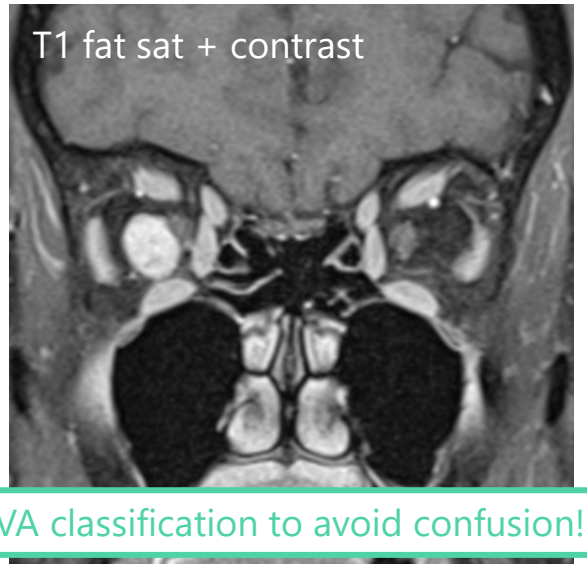

DO NOT use the term haemangioma which is a neoplastic lesion! See ISSVA classification to avoid confusion!

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