The Vascular System

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Atherosclerosis

It occurs when atheromatous plaque are accumulated .
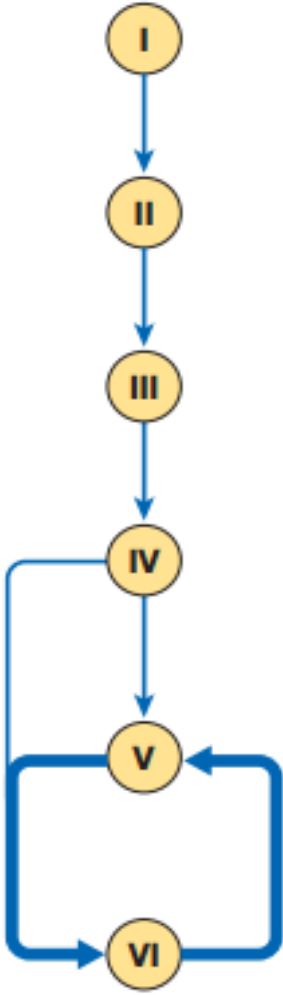
Initially, there are generally <u>no symptoms</u>.

<u>When severe</u>, it can result in coronary artery disease, stroke, peripheral artery disease, or kidney problems, depending on which arteries are affected.

Atheromatous plaque is accumulation of material in the inner layer of tunica intima.

The material consists from macrophage cells, cholesterol , calcium and a variable amount of fibrous tissue.

The accumulated material forms **a swelling** in the artery wall, which may intrude into the lumen of the artery, narrowing it and restricting blood flow.

Nomenclature and main histology		Sequences in progression	Main growth mechanism	Earliest onset	Clinical correlation
Type I (initial) lesion Isolated macrophage foam cells		I	Growth mainly by lipid accumulation	From first decade	Clinically silent
Type II (fatty streak) lesion Mainly intracellular lipid accumulation		II			
Type III (intermediate) lesion Type II changes and small extracellular lipid pools		III		From third decade	
Type IV (atheroma) lesion Type II changes and core of extracellular lipid		IV			Clinically silent or overt
Type V (fibroatheroma) lesion Lipid core and fibrotic layer, or multiple lipid cores and fibrotic layers, or mainly calcific, or mainly fibrotic		V	Accelerated smooth muscle and collagen increase	From fourth decade	
Type VI (complicated) lesion Surface defect, haematoma-haemorrhage, thrombus		VI	Thrombosis, haematoma		

Risk factors

1- increase age,

2- abnormal cholesterol levels,

3-high blood pressure,

4-diabetes,

5-smoking,

6- obesity,

7- family history,

8- unhealthy diet.

If this process grow gradually it will results in gradual symptoms as angina **but** if this plaque ruptured this will results in acute unstable condition due to formation of thrombous and results in sudden narrowing or closure of the lumen of the artery resulting in acute myocardial infarction.

Diagnosis

The heart medical imaging included

1. ECHO study

2. **CT coronary angiography**

3. PET STRESS TEST
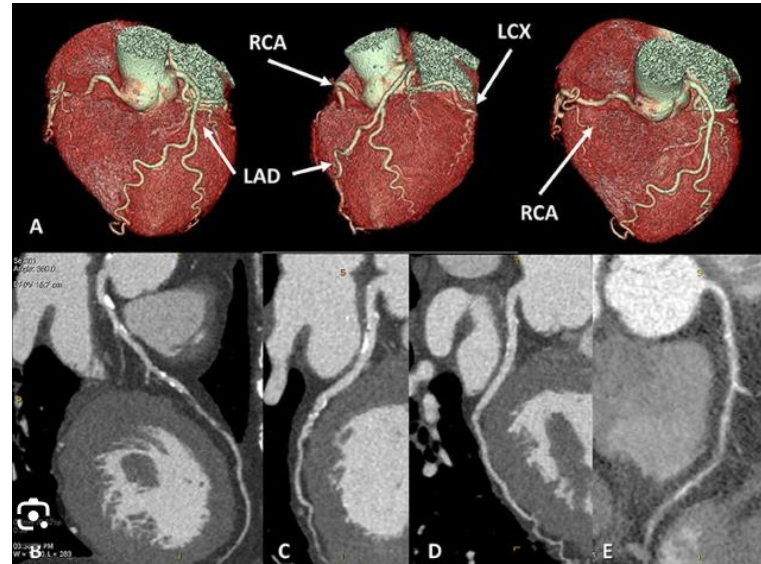
4. Percuataneous coronary angiography
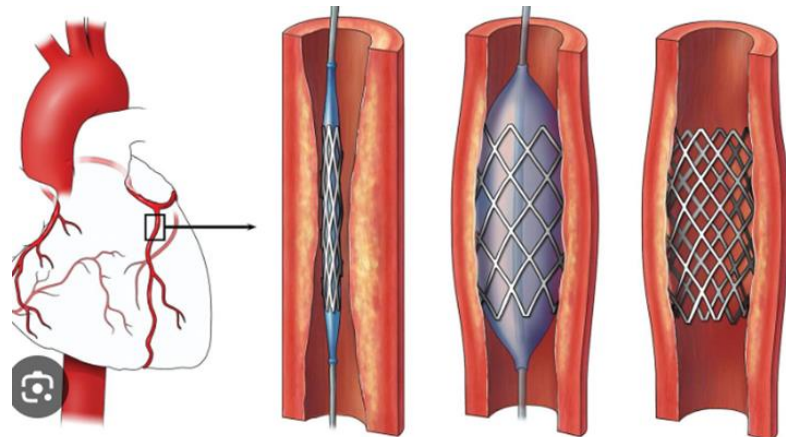
IF the cerebral vessels are included

CT scan

MRI, MRA, MRV.

Carotid Doppler study

If renal vessels included

Doppler of Renal artery

Angiography

Coronary artery disease

Coronary artery disease (CAD) is the most common cause of angina and acute coronary syndrome and the most common cause of death worldwide.

Occult CAD is common in those who present with other forms of atherosclerotic vascular disease, such as intermittent claudication or stroke, and is an important cause of morbidity and mortality in these patients.

i	16.36 Coronary artery disease: clinical manifestations and pathology	
Clinical problem	**Pathology**	
Stable angina	Ischaemia due to fixed atheromatous stenosis of one or more coronary arteries	
Unstable angina	Ischaemia caused by dynamic obstruction of a coronary artery due to plaque rupture or erosion with superimposed thrombosis	
Myocardial infarction	Myocardial necrosis caused by acute occlusion of a coronary artery due to plaque rupture or erosion with superimposed thrombosis	
Heart failure	Myocardial dysfunction due to infarction or ischaemia	
Arrhythmia	Altered conduction due to ischaemia or infarction	
Sudden death	Ventricular arrhythmia, asystole or massive myocardial infarction	

Angina pectoris

Angina pectoris is a symptom complex caused by **transient myocardial ischaemia**, which occurs whenever there is an imbalance between myocardial oxygen supply and demand

Clinical features

The history is the most important factor in making the diagnosis

Stable angina is <u>characterised by</u>: central chest pain, discomfort or breathlessness that is precipitated by exertion or other forms of stress, and is relieved by rest.

Acute coronary syndrome

Acute coronary syndrome is a term that encompasses both unstable angina and myocardial infarction.

Unstable angina is characterised by new-onset or rapidly worsening angina , angina on minimal exertion or angina at rest in the absence of myocardial damage.

Myocardial infarction <u>differs from </u>unstable angina, since there is evidence of myocardial necrosis.

The term acute myocardial infarction (MI) should be used when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischaemia.

16.49 Clinical features of acute coronary syndromes

Symptoms

- Prolonged cardiac pain: chest, throat, arms, epigastrium or back
- Anxiety and fear of impending death
- Nausea and vomiting
- Breathlessness
- Collapse/syncope

Physical signs

Signs of sympathetic activation
- Pallor
- Sweating
- Tachycardia
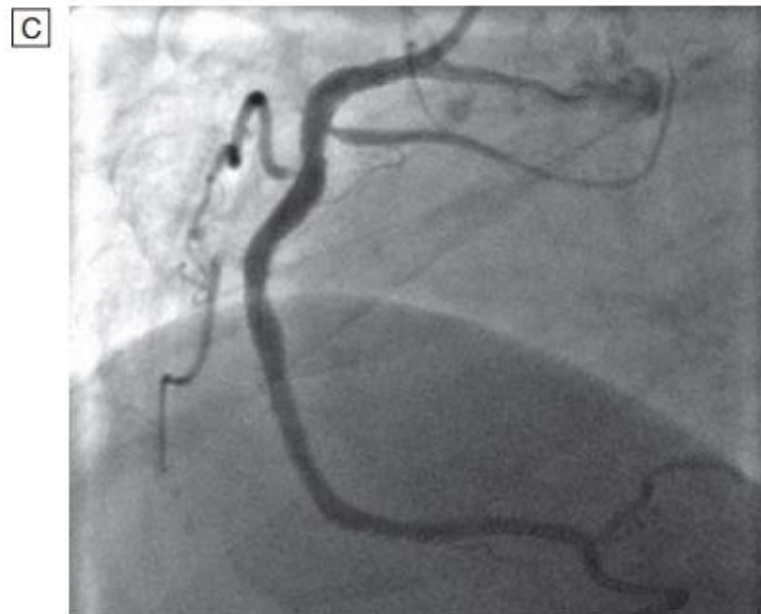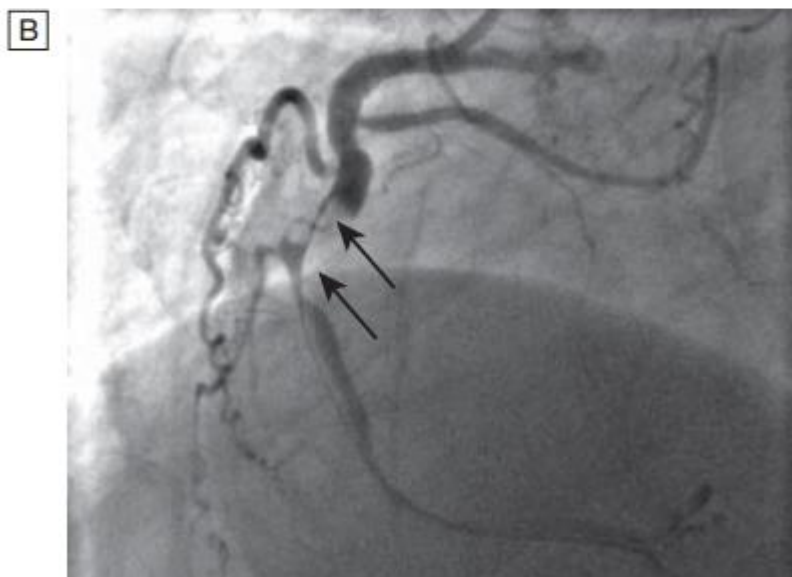
Signs of vagal activation
- Vomiting
- Bradycardia

Signs of impaired myocardial function
- Hypotension, oliguria, cold peripheries
- Narrow pulse pressure
- Raised jugular venous pressure
- Third heart sound
- Quiet first heart sound
- Diffuse apical impulse
- Lung crepitations

Low-grade fever

Complications
- Mitral regurgitation
- Pericarditis
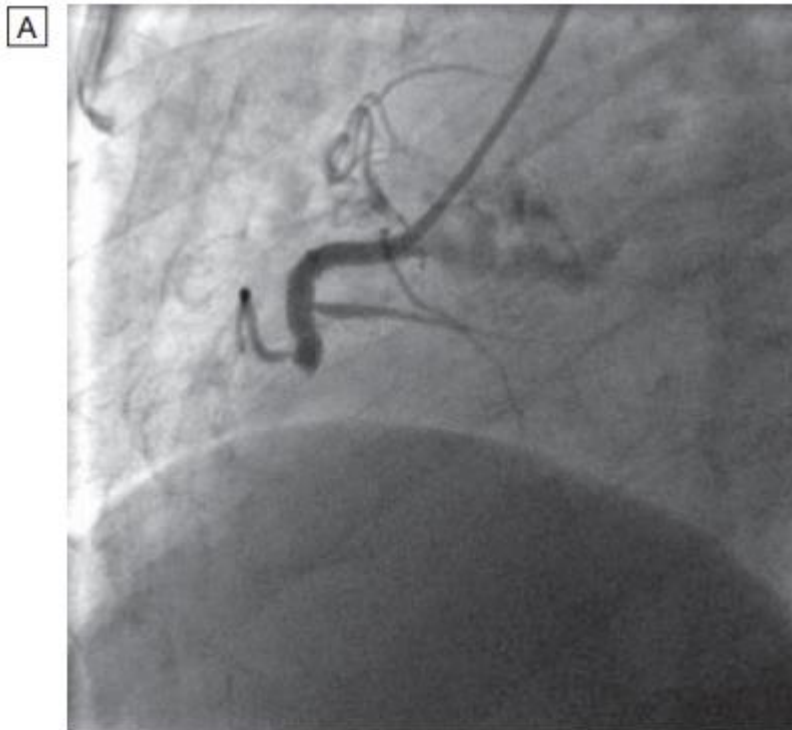
Fig. 16.60 Primary percutaneous coronary intervention. **A** Acute right coronary artery occlusion. **B** Initial angioplasty demonstrates a large thrombus filling defect (arrows). **C** Complete restoration of normal flow following intracoronary stenting.

Peripheral arterial disease

Peripheral arterial disease (PAD) has been estimated to affect about 20% of individuals aged 55–75 years in the UK.

Only 25% of patients present with symptoms, the most common of which is intermittent claudication (IC).

About 1–2% of patients with IC per year progress to a point where amputation and/or revascularisation are required.

The cause of death is typically an MI or stroke, reflecting the fact that IC nearly always occurs in association with widespread atherosclerosis.

Clinical features

Symptomatic PAD affects the legs **about eight times** more commonly than the arms.

Several locations may be affected, including the aortoiliac vessels, the femoropopliteal vessels and the infrapopliteal vessels. One or more of these segments may be affected in a variable and asymmetric manner.

In the arm, the subclavian artery is the most common site of disease.

Intermittent claudication

This is the most common presentation of PAD affecting the lower limbs. It is characterised by ischaemic pain affecting the muscles of the leg.

The pain is usually felt in the calf because the disease most commonly affects the superficial femoral artery, the pain comes on after walking, often once a specific distance has been covered, and rapidly subsides on resting

Critical limb ischaemia

is defined as rest pain requiring opiate analgesia

Acute limb ischaemia

This is most frequently caused by acute thrombotic occlusion of a pre-existing stenotic arterial segment, thromboembolism, and trauma that may be iatrogenic

16.57 Symptoms AND signs of acute limb ischaemia	
Symptoms/signs	**Comment**
Pain **P**allor **P**ulselessness	May be absent in complete acute ischaemia, and can be present in chronic ischaemia
Perishing cold	Unreliable, as the ischaemic limb takes on the ambient tempetature
Paraesthesia **P**aralysis	Important features of impending irreversible ischaemia

Aneurysm

Is an outward bulging caused by a localized, abnormal, weak spot on a blood vessel wall. Aneurysms may be a result of a hereditary condition or an acquired disease.

examples of congenital association of aneurysms is polycystic kidney disease which is associated with berry aneurysms.

Aneurysms can also be a nidus **(starting point) for clot formation (thrombosis)** and embolization.

As an aneurysm increases in size, the **risk of rupture**, which leads to uncontrolled bleeding, increases.

They may occur in any blood vessel, particularly lethal examples include aneurysms of the Circle of Willis in the brain, aortic aneurysms affecting the thoracic aorta, and abdominal aortic aneurysms.

Aneurysms can arise in the heart itself following a heart attack, including both ventricular and atrial septal aneurysms.

Aortic Aneurysm

Is an enlargement **(dilatation) of the aorta** to greater than 1.5 times normal size.

They usually cause no symptoms except when ruptured.

Occasionally, there may be abdominal, back, or leg pain.

The prevalence of AAA has been reported to range from 2 to 12% and is found in about 8% of men more than 65 years of age

Aortic aneurysms are classified by their location on the aorta.

**Aortic root aneurysm**, or aneurysm of the sinus of Valsalva.

Thoracic aortic aneurysms are found within the chest; these are further classified as ascending, aortic arch, or descending aneurysms.

Abdominal aortic aneurysms, "AAA" or "Triple A", the most common form of aortic aneurysm, involve that segment of the aorta within the abdominal cavity.

Thoracoabdominal aortic aneurysms involve both the thoracic and abdominal aorta.Thoracoabdominal aortic aneurysms comprise some or all of the aorta in both the chest and abdomen, and have components of both thoracic and abdominal aortic aneurysms

Aortic arch aneurysm

Aortic root aneurysm

Descending thoracic aortic aneurysm

Thoracoabdominal aortic aneurysm

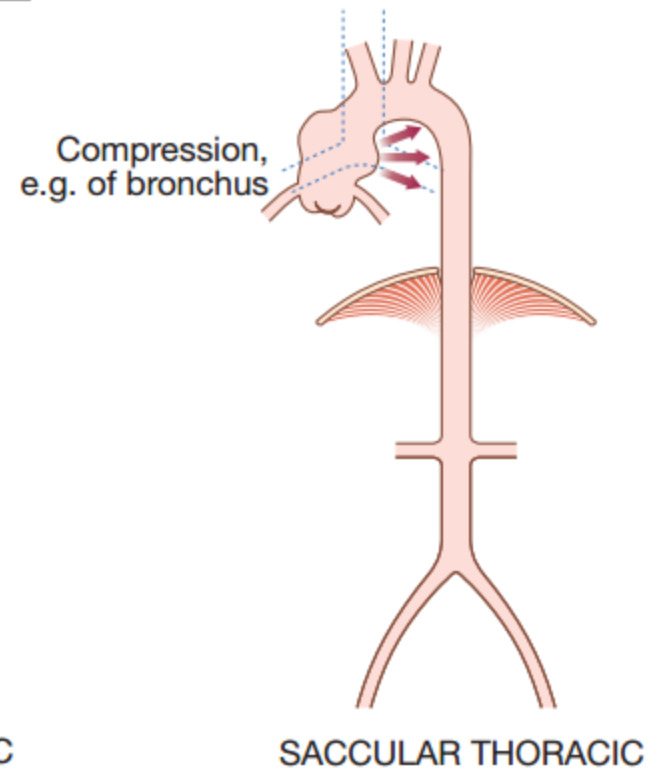**Ascending thoracic aortic aneurysm**
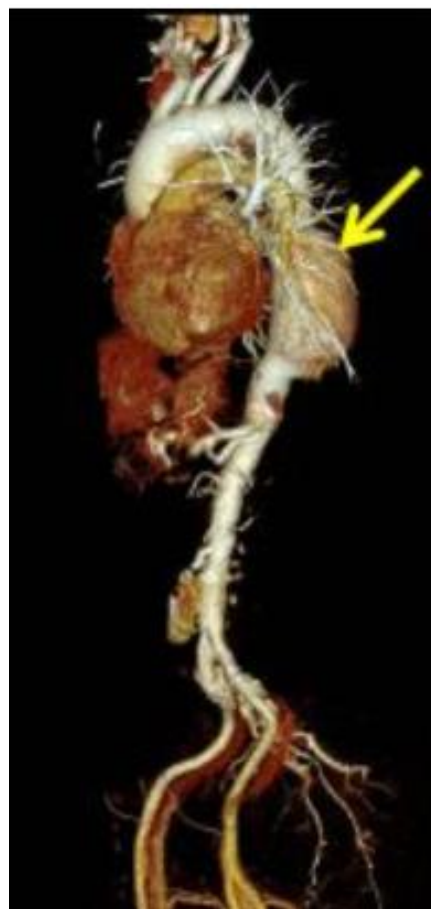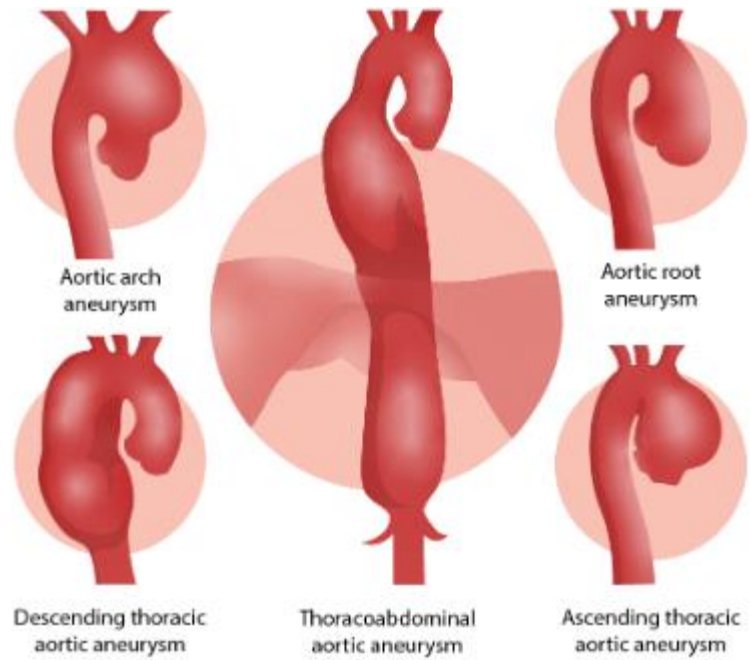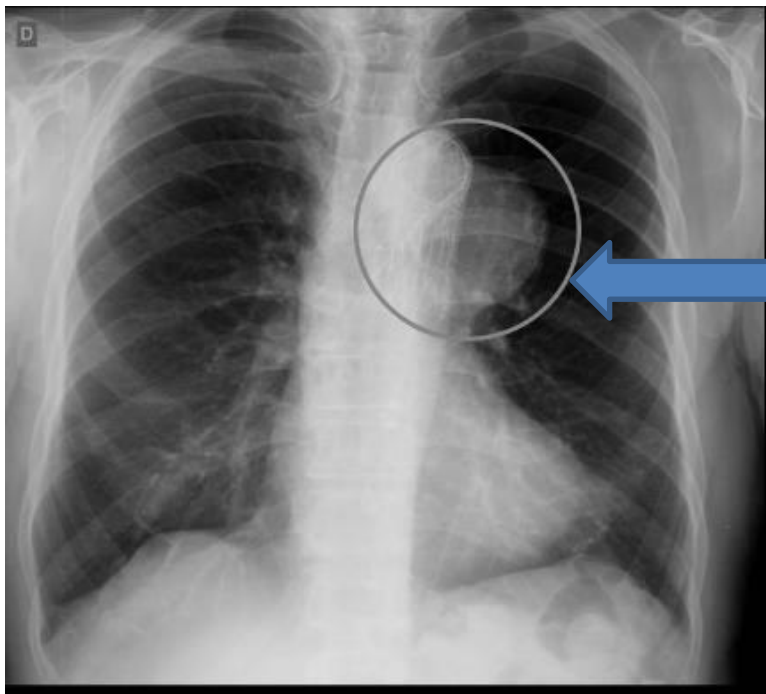
Aortic Aneurysm

Pathogenesis

The most common cause of aortic aneurysm is atherosclerosis.

Genetic factors that predispose to hypertension, hyperlipidaemia and diabetes all play a role in the pathogenesis of aortic aneurysm

This may explain in part why only some patients with risk factors for atheroma develop aneurysmal disease.

Aortic aneurysm

Diaphragm

Expansion

Thrombus

Embolism

ABDOMINAL

Aortic regurgitation

DILATED THORACIC

Compression, e.g. of bronchus

SACCULAR THORACIC

A
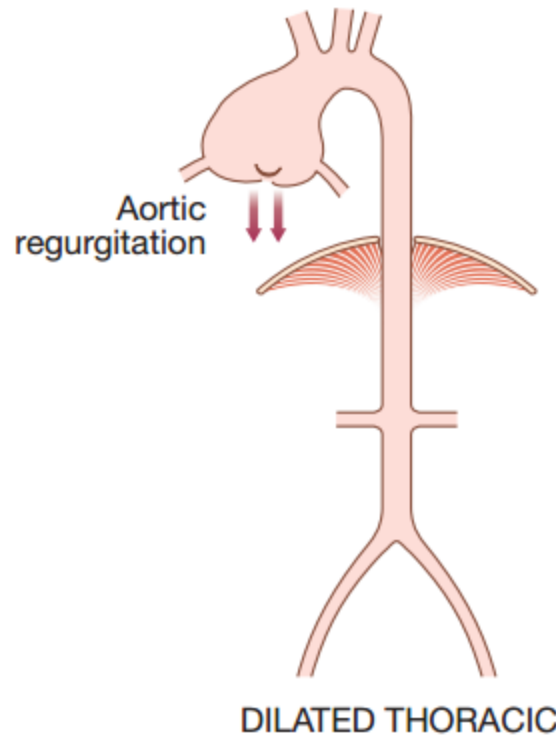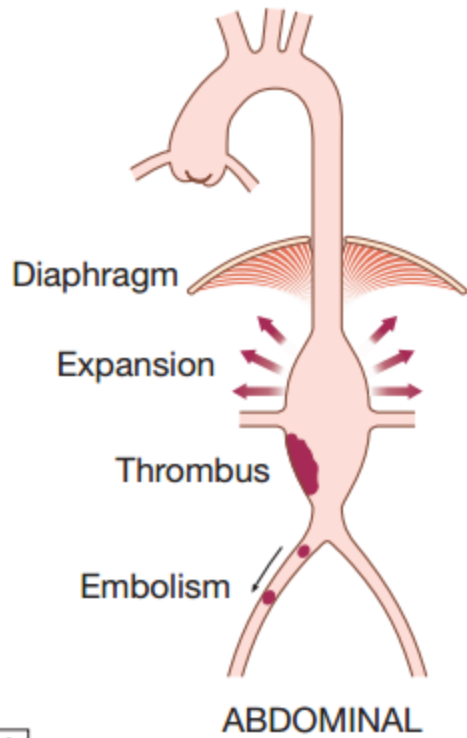
i 16.62 Abdominal aortic aneurysm (AAA): common presentations

Incidental

- On physical examination, plain X-ray or, most commonly, abdominal ultrasound
- Even large AAAs can be difficult to feel, so many remain undetected until they rupture
- Studies are currently under way to determine whether screening will reduce the number of deaths from rupture

Pain

- In the central abdomen, back, loin, iliac fossa or groin

Thromboembolic complications

- Thrombus within the aneurysm sac may be a source of emboli to the lower limbs
- Less commonly, the aorta may undergo thrombotic occlusion

Compression

- Surrounding structures such as the duodenum (obstruction and vomiting) and the inferior vena cava (oedema and deep vein thrombosis)

Rupture

- Into the retroperitoneum, the peritoneal cavity or surrounding structures (most commonly the inferior vena cava, leading to an aortocaval fistula)

Investigations

Ultrasound is the best way of establishing the diagnosis of an abdominal aneurysm and of following up patients with asymptomatic aneurysms that are not yet large enough to warrant surgical repair.

CT provides more accurate information about the size and extent of the aneurysm, the surrounding structures and the presence of any other intraabdominal pathology.

It is the standard pre-operative investigation but is not suitable for surveillance because of the high cost and radiation dose
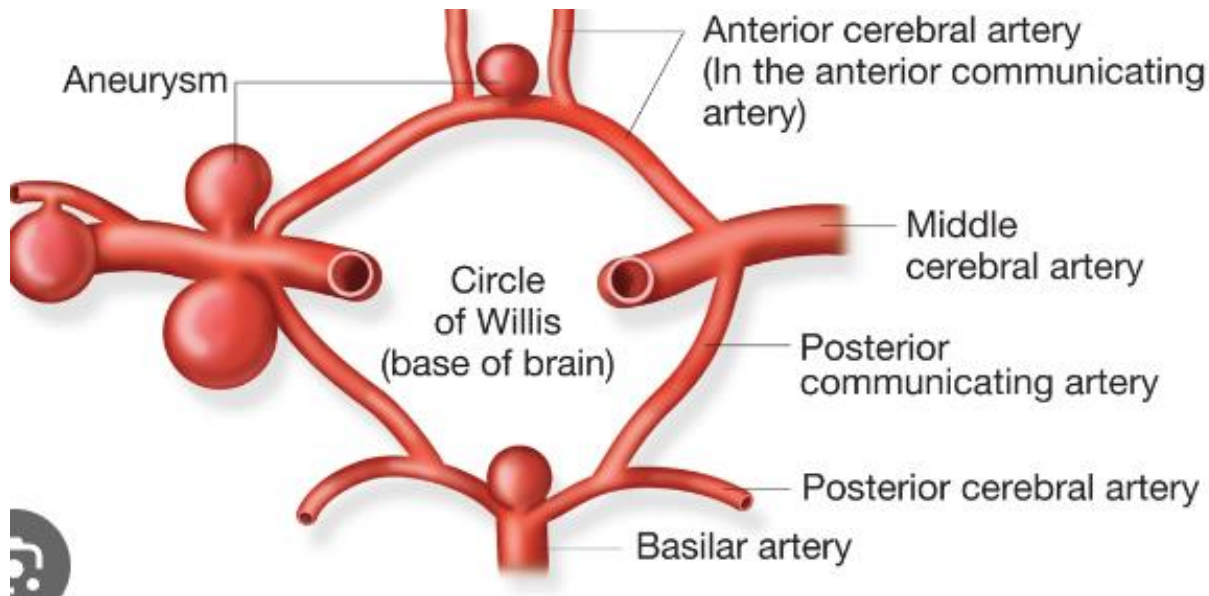
Management

The risks of surgery generally outweigh the risks of rupture until an asymptomatic AAA has reached a **maximum of 5.5 cm in diameter**.

All symptomatic AAAs should be considered for repair, not only to rid the patient of symptoms but also because pain often predates rupture.

Intracranial Aneurysms

Cerebral aneurysms are classified both by size and shape.

1- Small aneurysms have a diameter of less than 15 mm.

2- large (15 to 25 mm),

3- giant (25 to 50 mm), and

4- super-giant (over 50 mm).

Middle cerebral artery

Posterior communicating artery

Anterior cerebral artery

Ophthalmic artery

Internal carotid artery

Anterior choroidal artery

Posterior cerebral artery

Superior cerebellar artery

Pontine arteries

Basilar artery

Anterior inferior cerebellar artery

Vertebral artery

Anterior spinal artery

Posterior inferior cerebellar artery

Aneurysm

Anterior cerebral artery
(In the anterior communicating artery)

Middle cerebral artery

Circle of Willis (base of brain)

Posterior communicating artery

Posterior cerebral artery
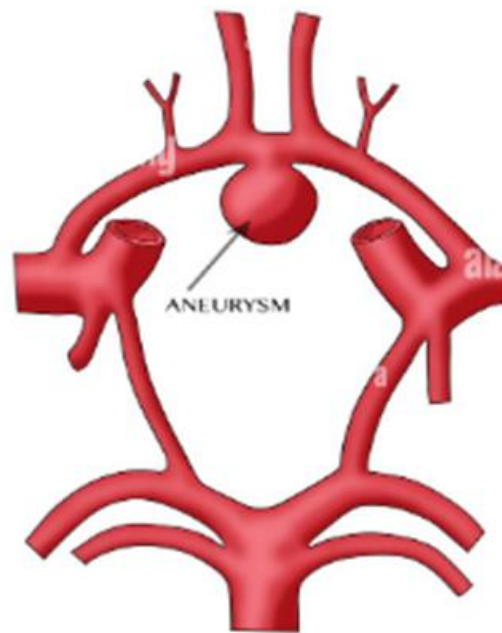
Basilar artery

Types

According to shape

Berry (saccular)aneurysms

Fusiform aneurysms

Microaneurysms, also known as Charcot–

Bouchard aneurysms

CIRCLE OF WILLIS and types of Aneurysms

ANEURYSM

Ruptured aneurysm

Saccular aneurysm

Fusiform aneurysm

Berry (saccular) aneurysms

Saccular aneurysms, also known as **berry aneurysms**, appear as a round out-pouching and are the most common form of cerebral aneurysm.

Causes: Connective tissue disorders, polycystic kidney disease, arteriovenous malformations, untreated hypertension, tobacco smoking, cocaine, and amphetamines, intravenous drug abuse (can cause infectious mycotic aneurysms), alcoholism, heavy caffeine intake, head trauma, and infection in the arterial wall from bacteremia (mycotic aneurysms).

in order of frequency affecting the following arteries:

Anterior communicating artery

Posterior communicating artery

Middle cerebral artery

Internal carotid artery

Tip of basilar artery

Fusiform aneurysms

Fusiform dolichoectatic aneurysms represent a widening of a segment of an artery around the entire blood vessel, rather than just arising from a side of an artery's wall.
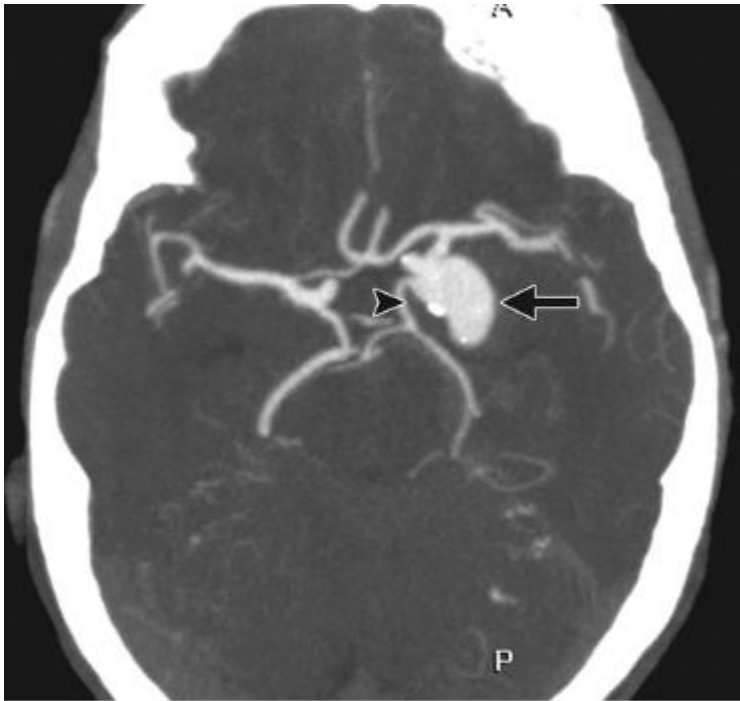
Microaneurysms, also known as Charcot–Bouchard aneurysms

Typically occur in small blood vessels (less than 300 micrometre diameter)

Are associated with chronic hypertension.

Charcot–Bouchard aneurysms are a common cause of intracranial hemorrhage.

Diagnosis is with CT angiography

Vasculitis

is a group of disorders that destroy blood vessels by inflammation.

Both arteries and veins are affected. Lymphangitis (inflammation of lymphatic vessels) is sometimes considered a type of vasculitis

Classification

According to the size of the vessel affected, vasculitis can be classified into:

Large vessel: Takayasu's arteritis, Temporal arteritis

Medium vessel: Buerger's disease, Kawasaki disease, Polyarteritis nodosa

Small vessel: Behçet's syndrome

Diagnosis with medical imaging

18F-fluorodeoxyglucose positron emission tomography/computed tomography (FDG-PET/CT)has become a widely used imaging tool in patients with suspected Large Vessel Vasculitis, due to the enhanced glucose metabolism of inflamed vessel walls.