Cerebrovascular Diseases

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Key Learning Outcomes

1. Recognise risk factors for cerebrovascular diseases.
2. Understand pathophysiology of CVAs.
3. Recognise and describe gross morphology of CVAs.
4. Deduce and plan public health interventions to prevent cerebrovascular diseases.
5. Identify own learning goals for further reading.

Introduction

Cardiovascular diseases (CVDs) include:

- coronary heart disease: heart and its blood supply
- **cerebrovascular disease – disease of the blood vessels supplying the brain;**
- peripheral arterial disease – supply limbs affected.
- rheumatic heart disease – damage to the heart muscle and heart valves from rheumatic fever, caused by streptococcal bacteria;
- congenital heart disease – malformations of heart structure existing at birth;
- deep vein thrombosis and pulmonary embolism – blood clots in the leg veins, which can dislodge and move to the heart and lungs.

Epidemiology: Global

- CVDs are the number 1 cause of death globally: more people die annually from CVDs than from any other cause.

- An estimated 17.5 million people died from CVDs in 2012, representing 31% of all global deaths.
  - Of these deaths, an estimated 7.4 million were due to coronary heart disease and 6.7 million were due to stroke .

- **Over 3/4 of CVD deaths take place in low- and middle-income countries!**

WHO, 2016

Epidemiology: Global

- Out of the 16 million deaths under the age of 70 due to non-communicable diseases:
  - **82% are in low and middle income countries and 37% are caused by CVDs.**

- Most cardiovascular diseases can be prevented by addressing behavioural risk factors such as:
  - tobacco use, unhealthy diet and obesity, physical inactivity and harmful use of alcohol using population-wide strategies.

WHO, 2016

Epidemiology: Global

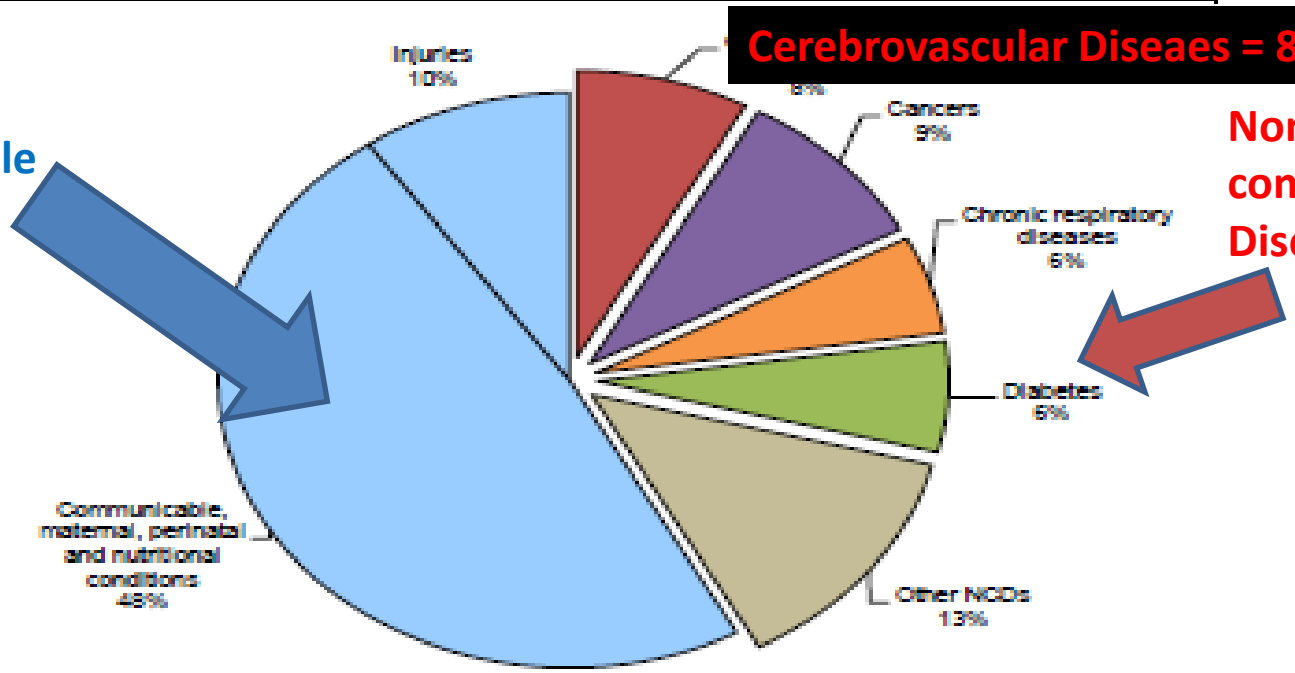
- People with CVDs or who are at high cardiovascular risk (due to the presence of one or more risk factors such as hypertension, diabetes, hyperlipidaemia or already established disease) need early detection and management using counselling and medicines, as appropriate

WHO, 2016

Epidemiology: PNG Perspective

Percentage of population living in urban areas: 12.5%
Population proportion between ages 30 and 70 years: 32.8%

Proportional mortality (% of total deaths, all ages, both sexes)*



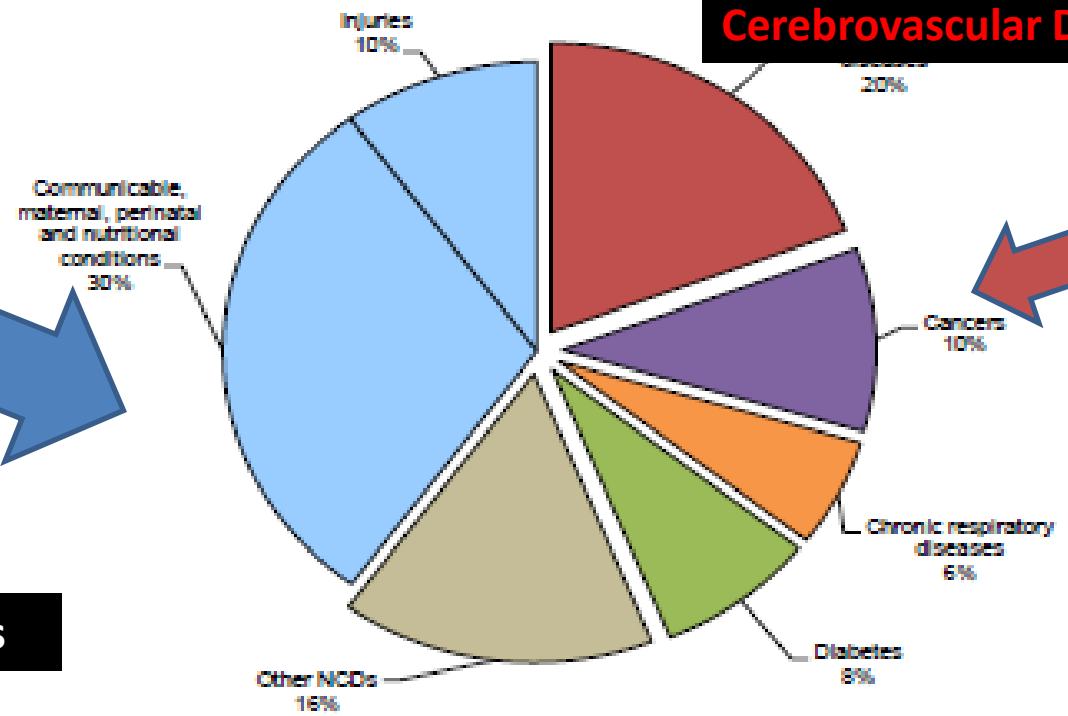
Total deaths: 56,000
NCDs are estimated to account for 42% of total deaths.

WHO, 2016

Epidemiology: Pacific

Percentage of population living in urban areas: 20.5%
Population proportion between ages 30 and 70 years: 30.8%

Proportional mortality (% of total deaths, all ages, both sexes)*



Total deaths: 3,000
NCDs are estimated to account for 60% of total deaths.

Cerebrovascular Diseases = 20%

Communicable Diseases

Non-communicable Diseases

Solomon Islands

WHO, 2016

Common Risk Factors for CVDs

TABLE 1: RISK FACTORS FOR CVD

Modifiable Risk Factors	Nonmodifiable Risk Factors
Overweight/obesity	Increasing age
Hypertension	Gender
Hyperlipidemia	Family history
Alcohol and tobacco use	Heredity and race
Stress	Menopause
High fat, high calorie diet	
Sedentary lifestyle	

CVD = cardiovascular disease.

Definitions

- Cerebrovascular diseases: abnormality of the brain caused by pathological process of blood vessels disrupting blood supply to the brain.
- Clinical term = stroke.

Pathophysiology

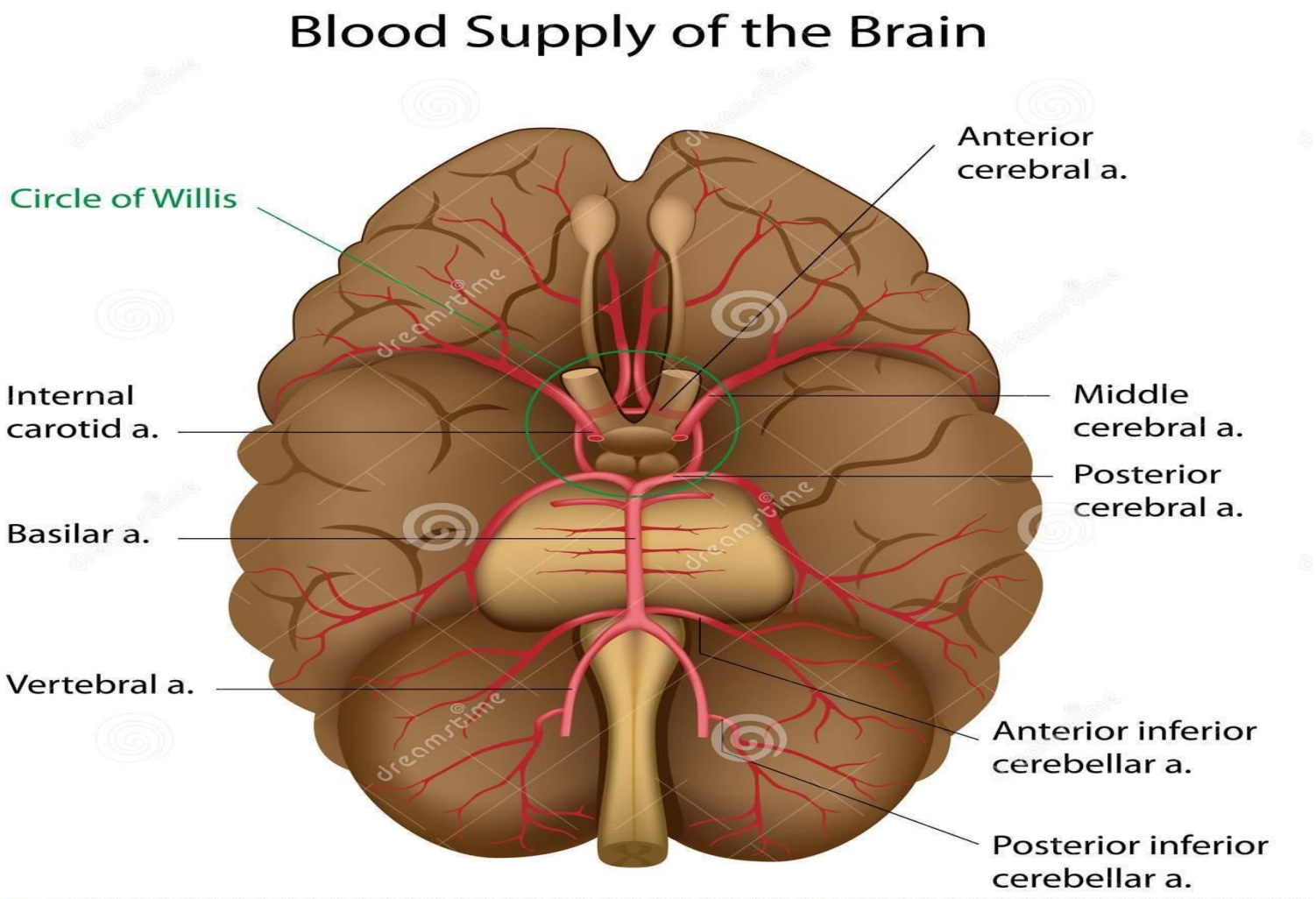
- 3 major categories:
- Thrombosis
- Embolism
- Haemorrhage

Pathophysiology

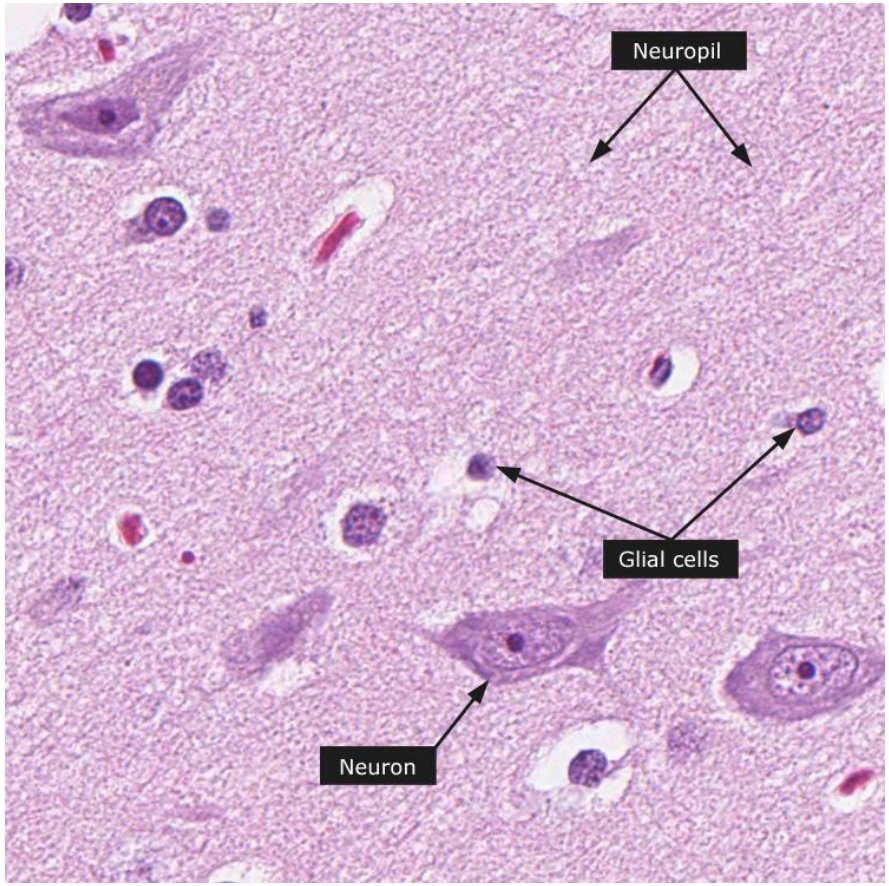
- Essentially 2 pathophysiolgical processes:

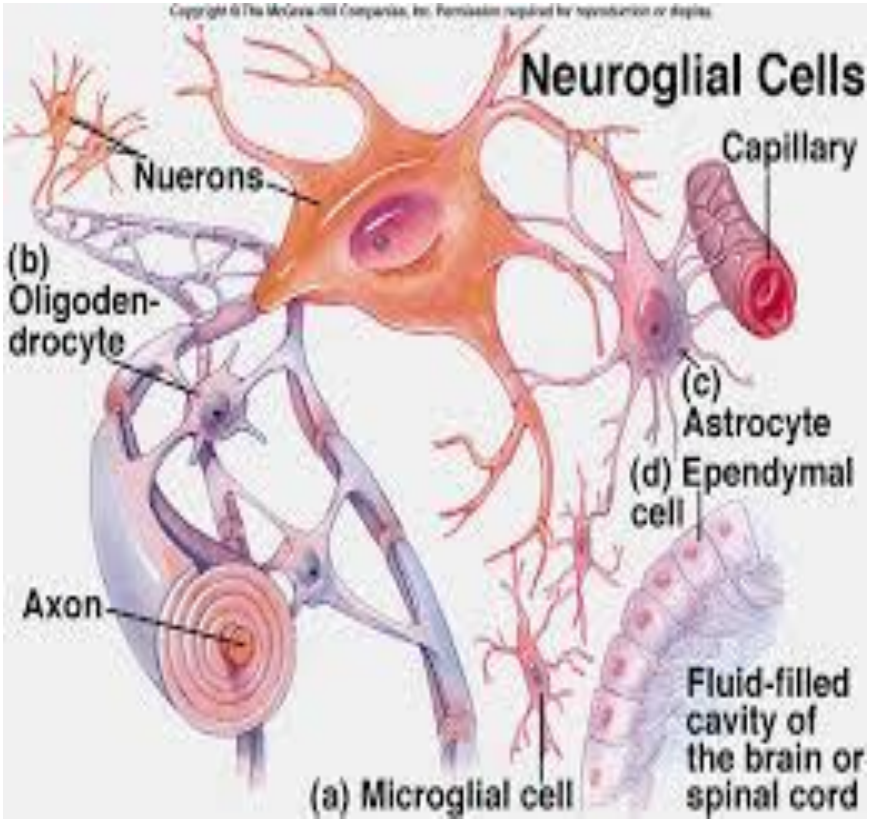
1. Hypoxia, ischaemia leading to infarction resulting from disrupted blood and oxygen supply to CNS tissue.
2. Haemorrhage resulting from rapture of CNS blood vessels (usually arteries).

Review



Normal cells





HYPOXIC INJURY:
1. GLOBAL CEREBRAL ISCHAEMIA
2. FOCAL CEREBRAL ISHAEMIA

Pathophysiology: Hypoxia, Ischaemia & Infarction

- 2 main types of acute ischaemic injury:

1. Global cerebral ischaemia: generalised reduction in cerebral perfusion. E.g. cardiac arrest, shock, severe hypotension, arrythmias.
2. Focal cerebral ischaemia: reduction in blood supply to localised areas of the brain. E.g. emboli, thrombotic occlusion, small vessel diseases (e.g. vasculities).

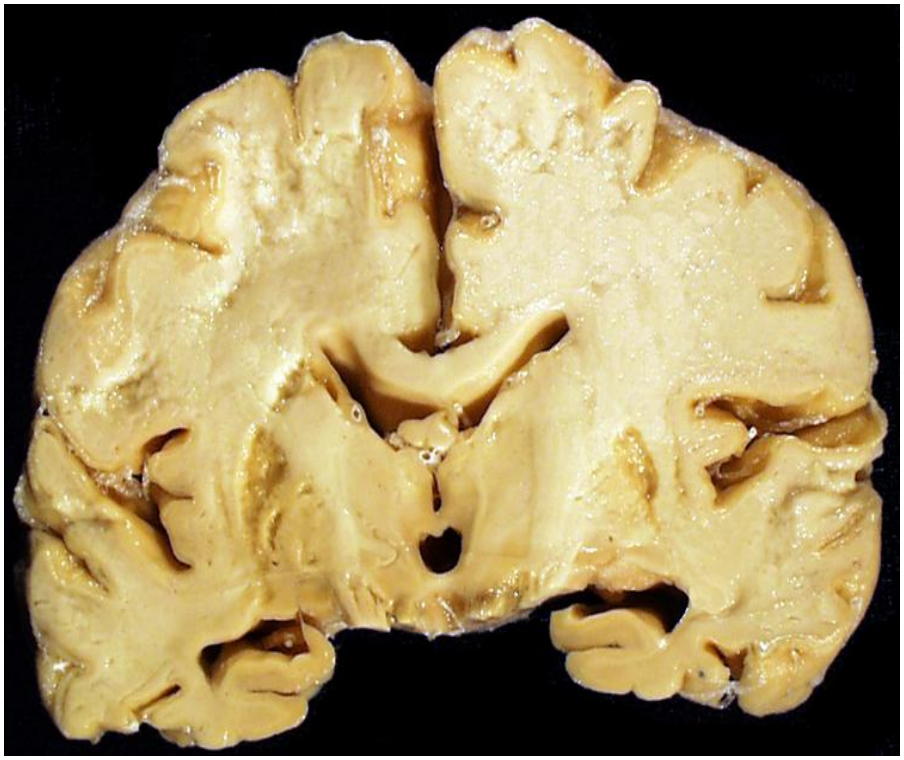
1. HYPOXIC INJURY: GLOBAL CEREBRAL ISHAEMIA

Global cerebral ischaemia: Morphology

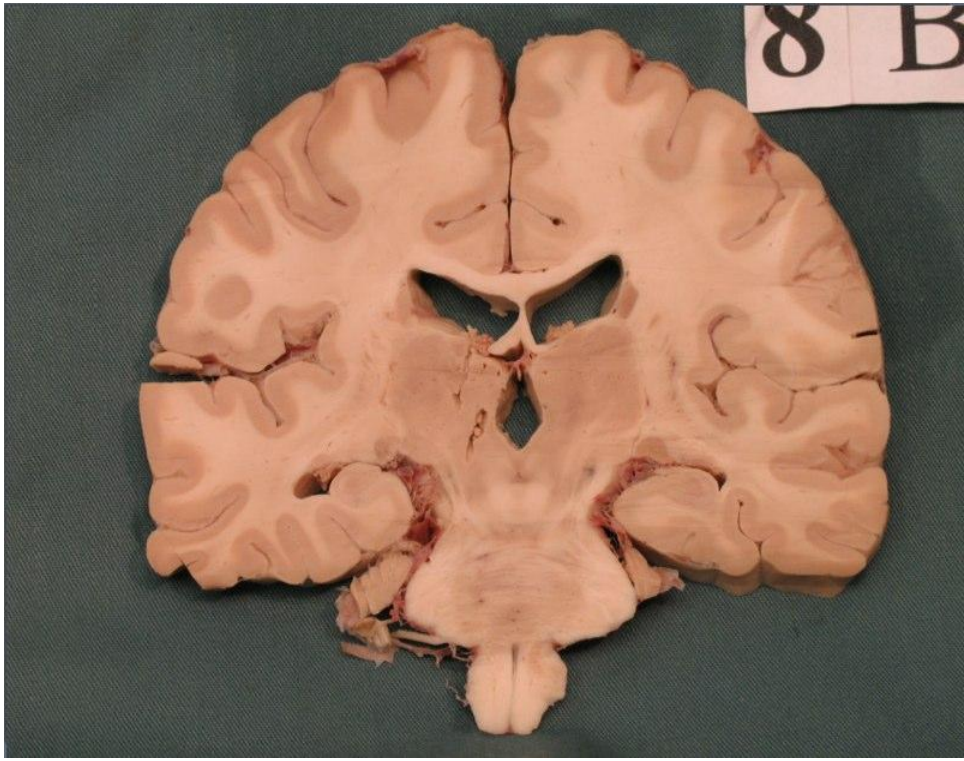
- Gross:
  - Brain is swollen
  - Gyri are widened and narrowed sulci.
  - Cut surface: poor demarcation between gray and white matter.
- Microscopic:
  - Early (12- 24 hours after injury)
  - Subacute (24- 2 weeks)
  - Repair (>2 weeks)

Gross

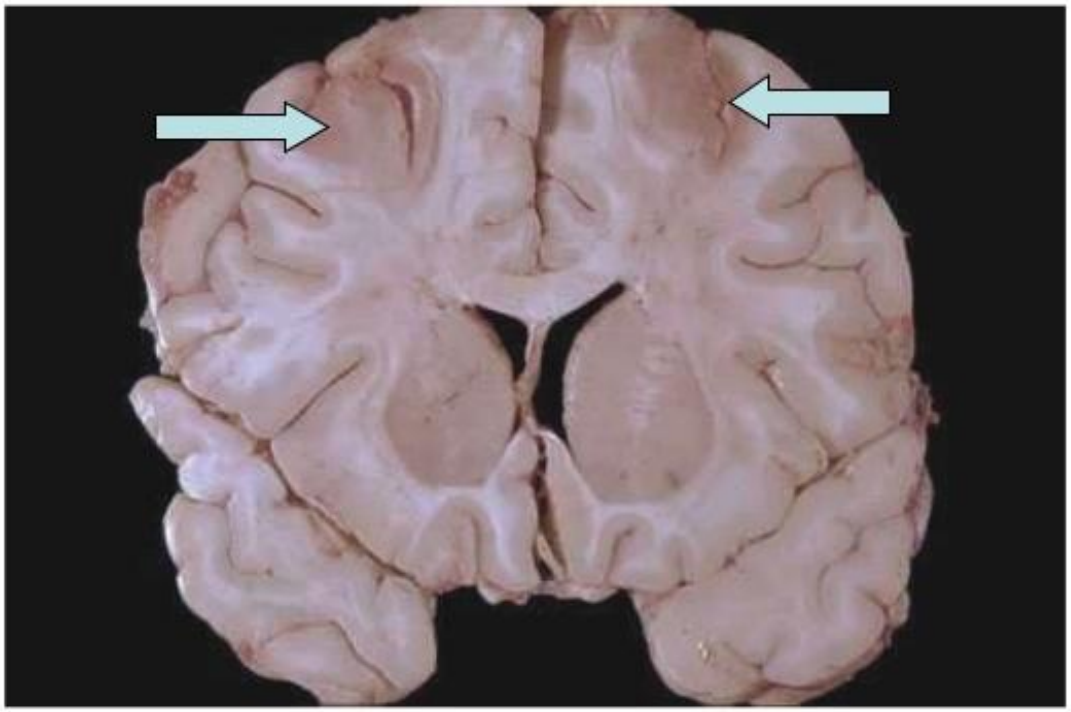
Hypoxic brain



Normal brain



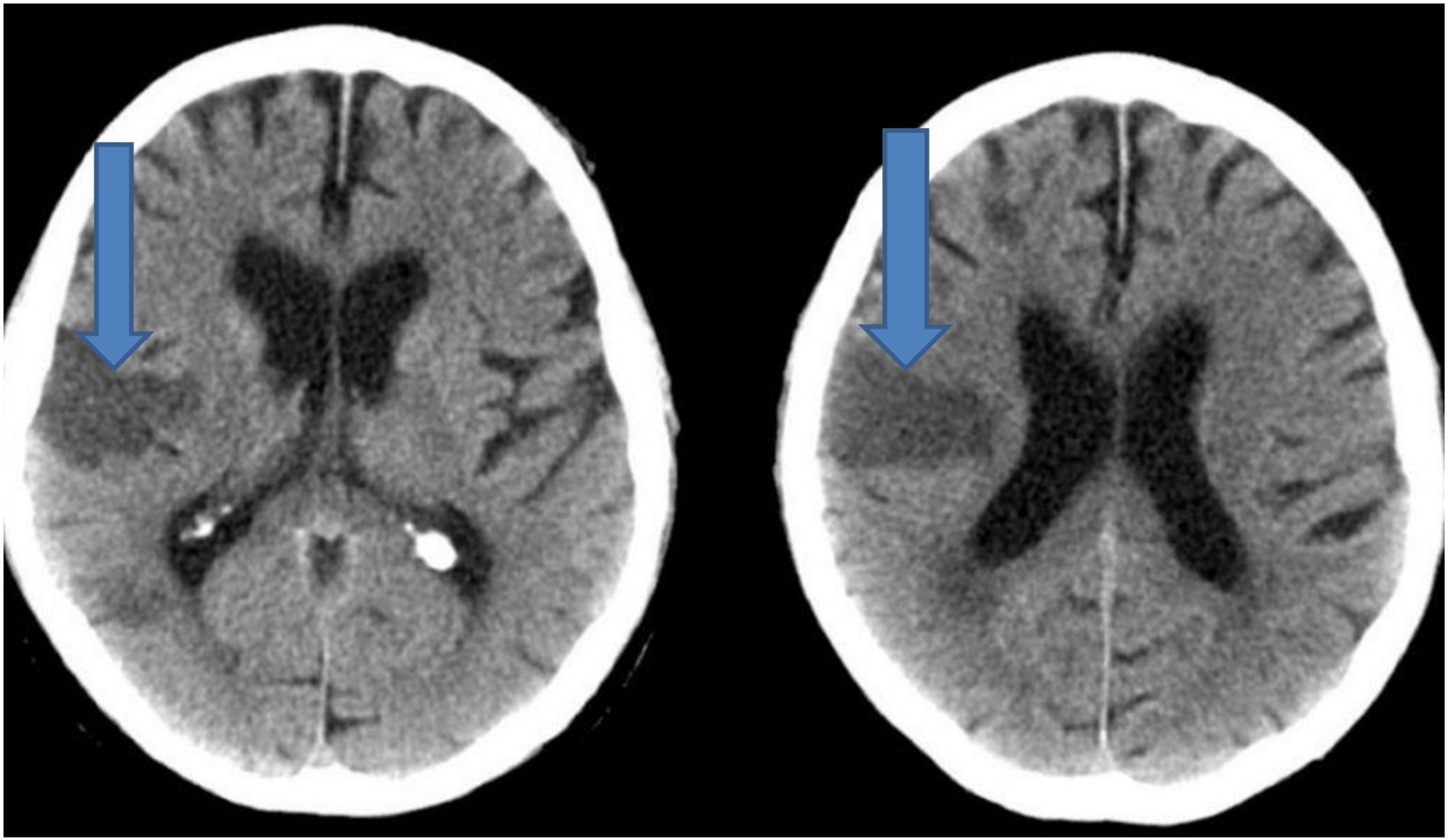
Watershed/Boundary zone infarcts:



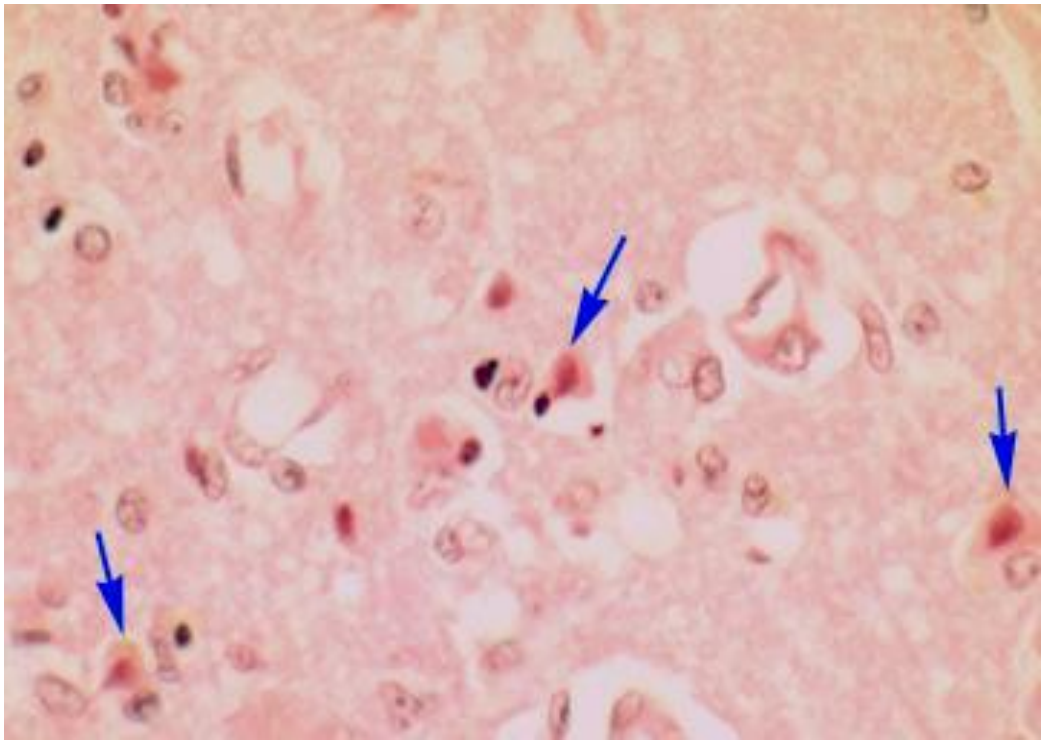
Areas farthest from the blood supply

Wedge shaped infarcted area

Gross: CT watershed area



Microscopy



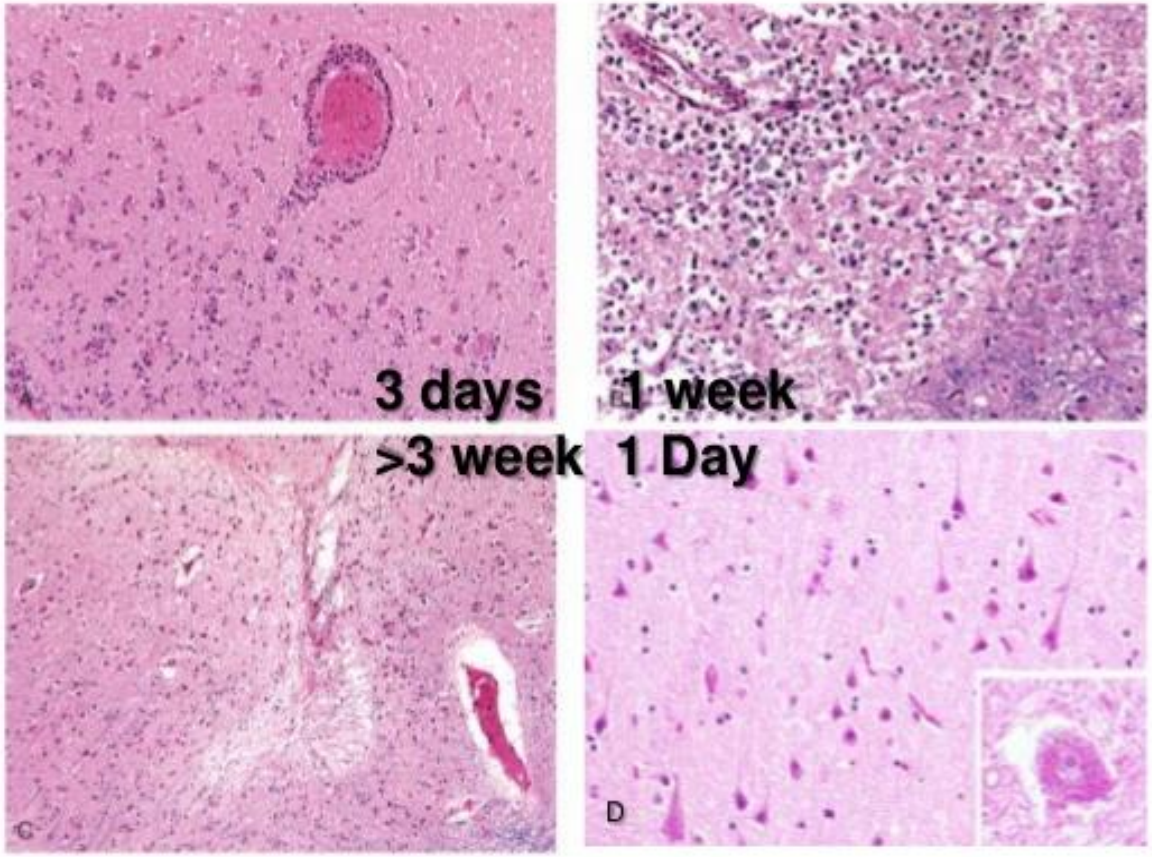
Acute neuronal cell change:

Red Neurons:
Microvacuolisation, eosinophila of cytoplasm, nuclear pyknosis and karyorrhexis

Similar changes affecting glial cells

Most susceptible: pyramidal cells of hippocampus, Purkinje cells of cerebellum, pyramidal neurons in the neocortex

Infarct : Microscopy



A- 3 days: neutrophils.
C-old: tissue loss + gliosis.

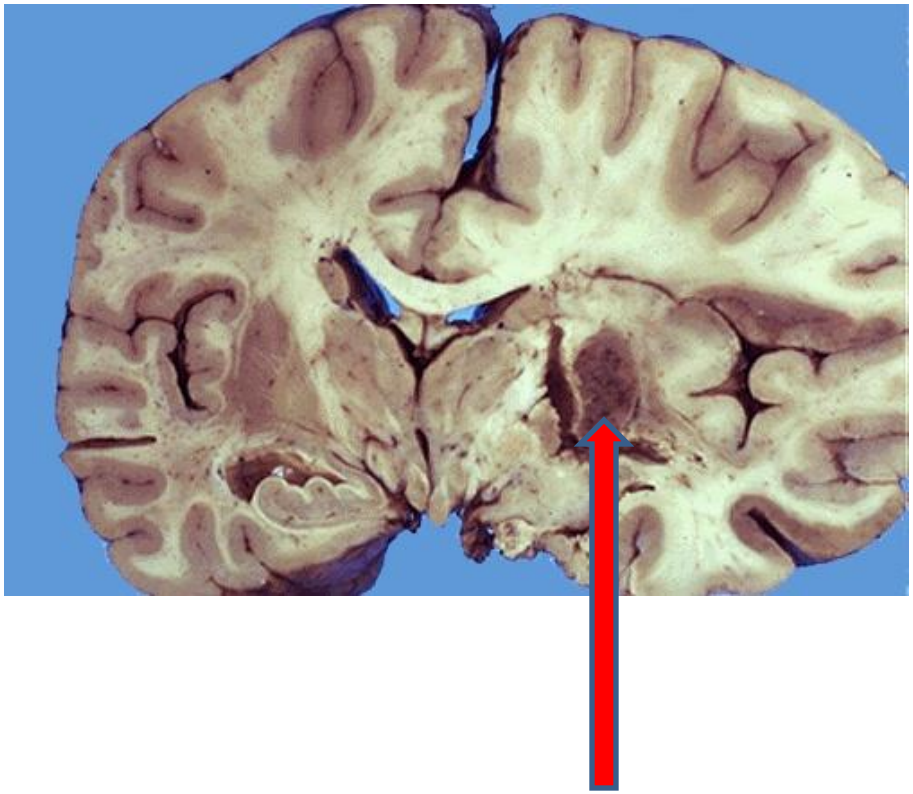
B-10 days: plenty of macrophages
D-1day: Red neurons & axon bulbs

2. HYOXIC INJURY: FOCAL CEREBRAL ISCHAEMIA

Focal cerebral ischaemia, infarction: Morphology

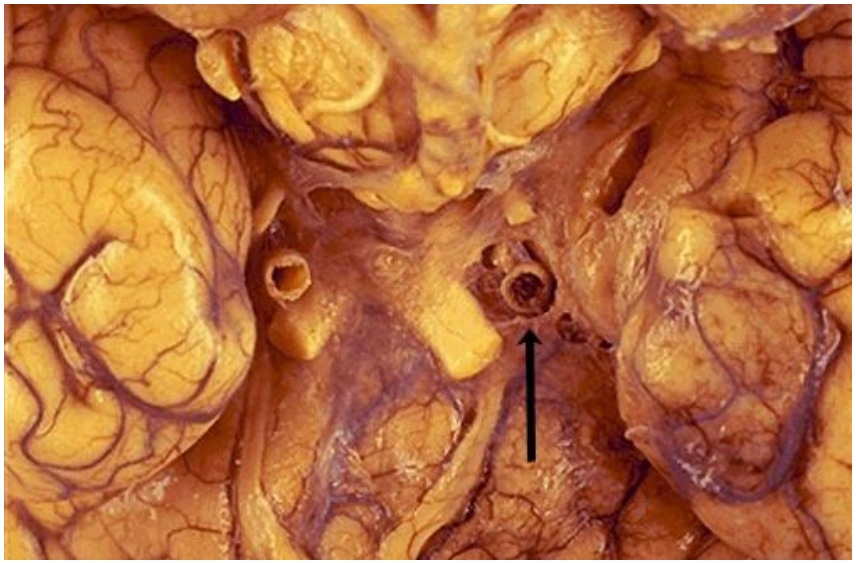
- Gross:
  - 48hrs: pale, soft, swollen, corticomedullary junction indistinct.
  - 2-10 days: gelatinous, friable, distinct boder between normal and abnormal tissue.
  - 10 days-3 weeks: tissue liquefication leaving fluid filled cystic spaces.
- Microscopy:
- 12-48 hours: Red neurons, edema, loss of white and gray matter disinction, glial cells swell, neutrophil infiltration.
- >48-<10 days: phagocytic cells predominate.
- 2-3 weeks: healing with astrocytes forming extensive network. New capillaries form with fibrous tissue.

Focal cerebral ischaemia, infarction: Morphology

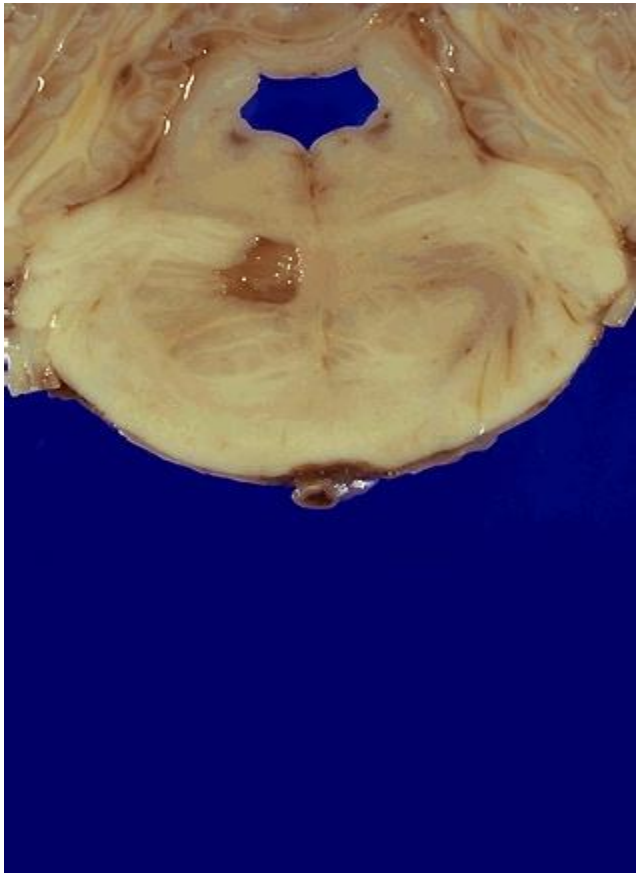


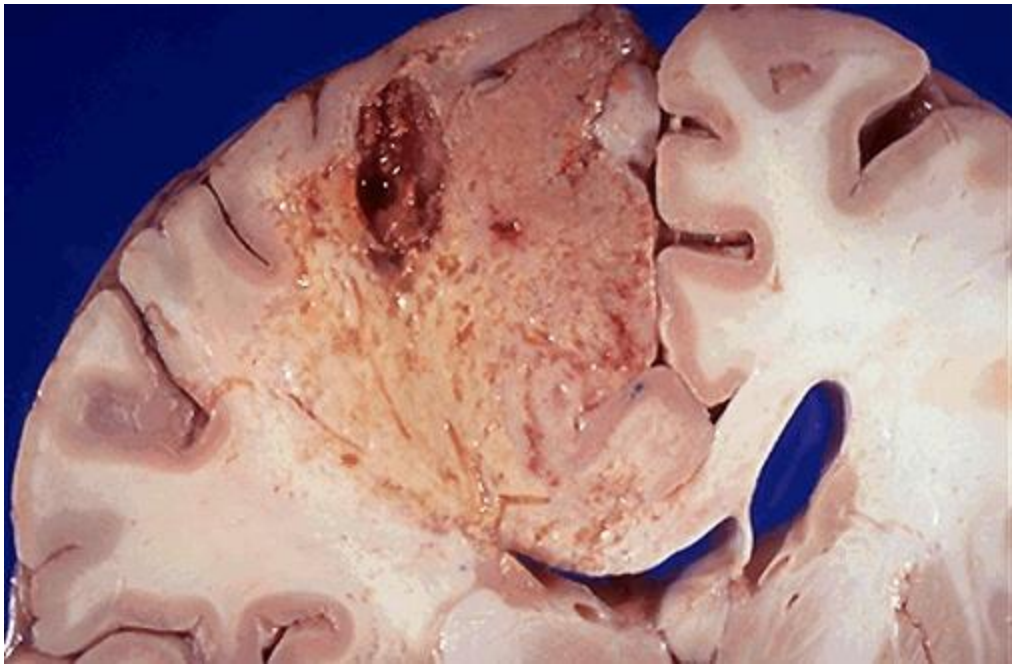
Infarcted area

Carotid artery thrombosis

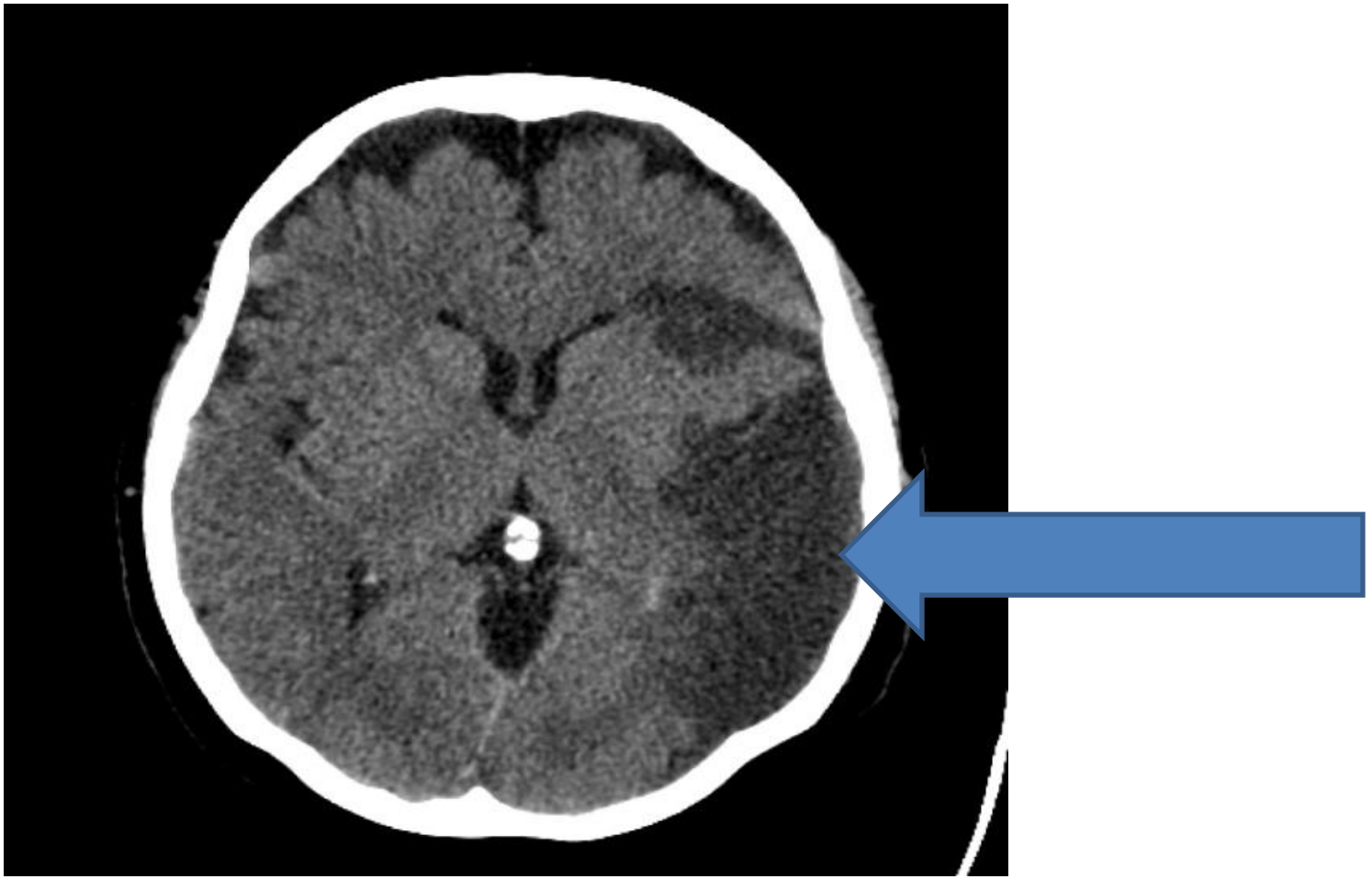


Focal cerebral ischaemia, infarction: Morphology

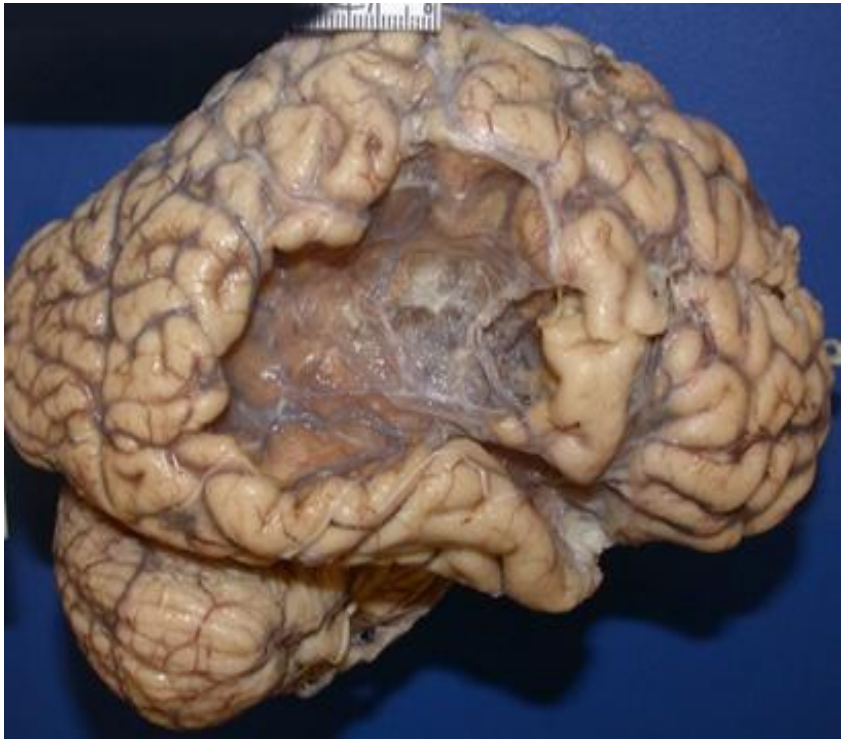


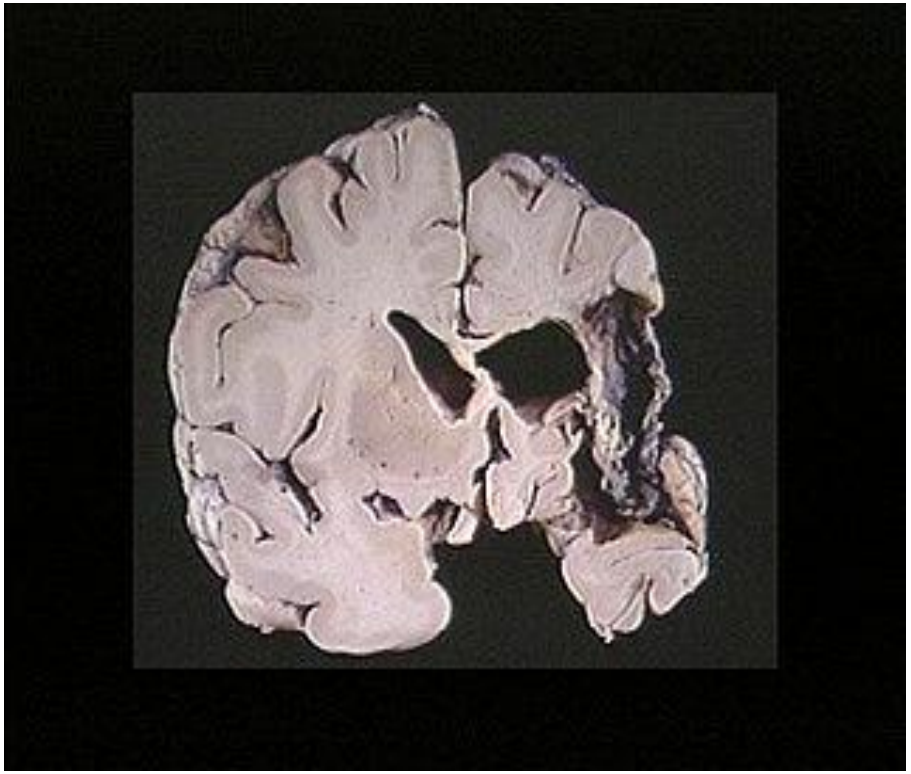


Focal cerebral ischaemia, infarction: Gross



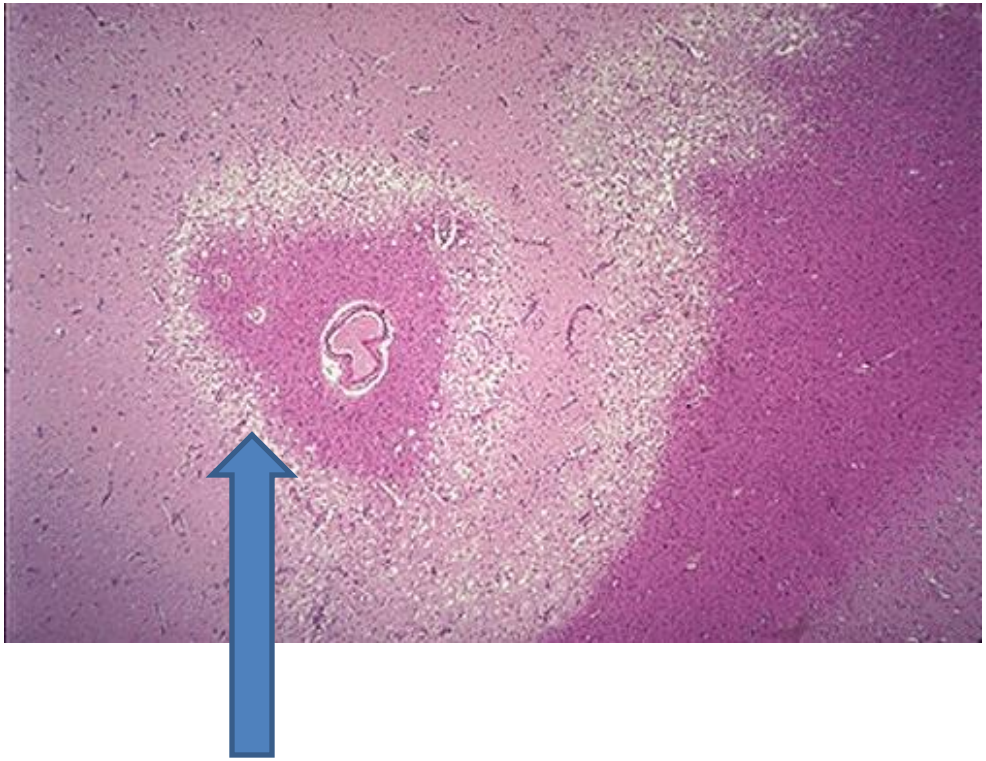
Focal cerebral ischaemia, infarction: Gross





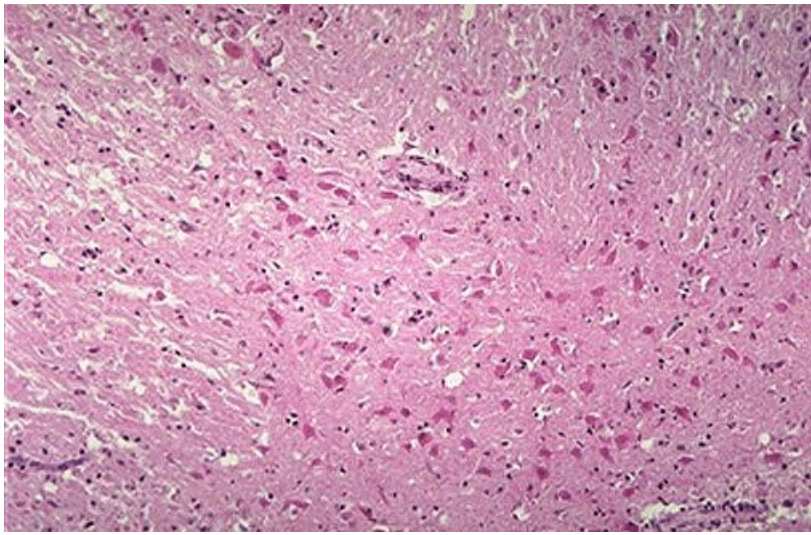
Old infarcted areas: fibrous tissue and cystic cavity. The cavity is separated from meninges and subarachnoid space by glial tissue

Focal cerebral ischaemia, infarction: Microscopy



Marked oedema (vasogenic oedema): pale white area created by loss of blood brain barrier.

Red neurons



Cerebrovascular diseases

1. Hypoxic, ischaemia and infarction:
    1. Global cerebral ischaemia
    2. Focal cerebral ischaemia

2. Haemorrhage

Cerebrovascular diseases: Haemorrhage

- Haemorrhage:

1. Intracerebral haemorrhage
2. Subarachnoid haemorrhage

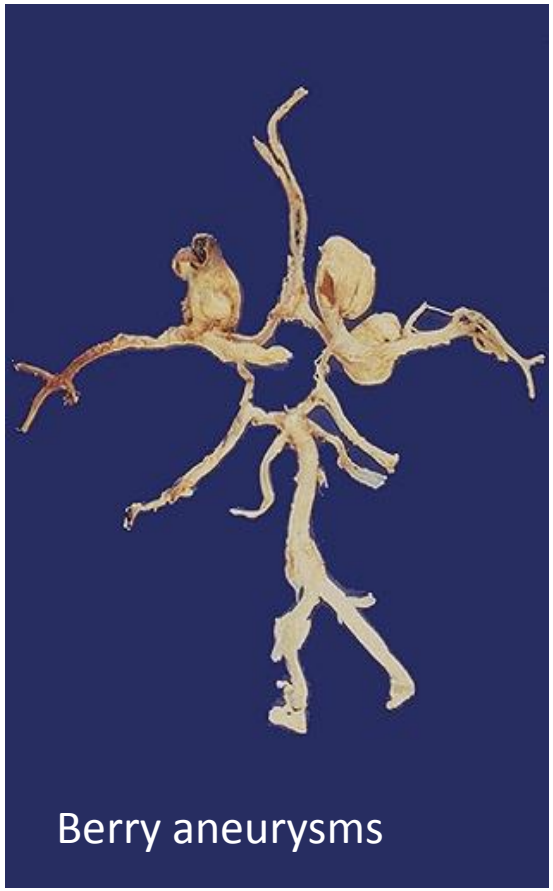
Intracerebral haemorrhage

- Spontaneous: non-traumatic.
  - hypertension accounts for >50% of causes.
  - Vascular malformations. E.g Charcot-Bouchard microaneurysms.
  - Coagulation disorders
  - Neoplasms
  - Vasculitis
  - Open heart surgery

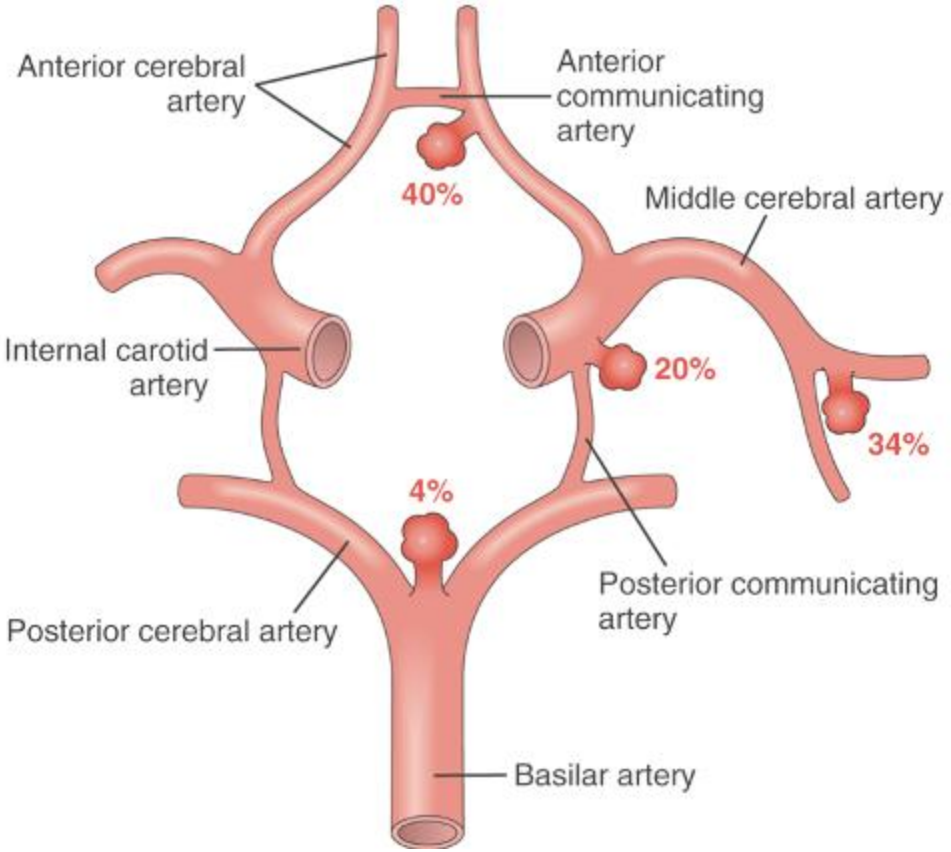
Chronic hypertension causing micro-aneurysms

Charcot-Bouchard aneurysms

Vascular malformations



Berry aneurysms

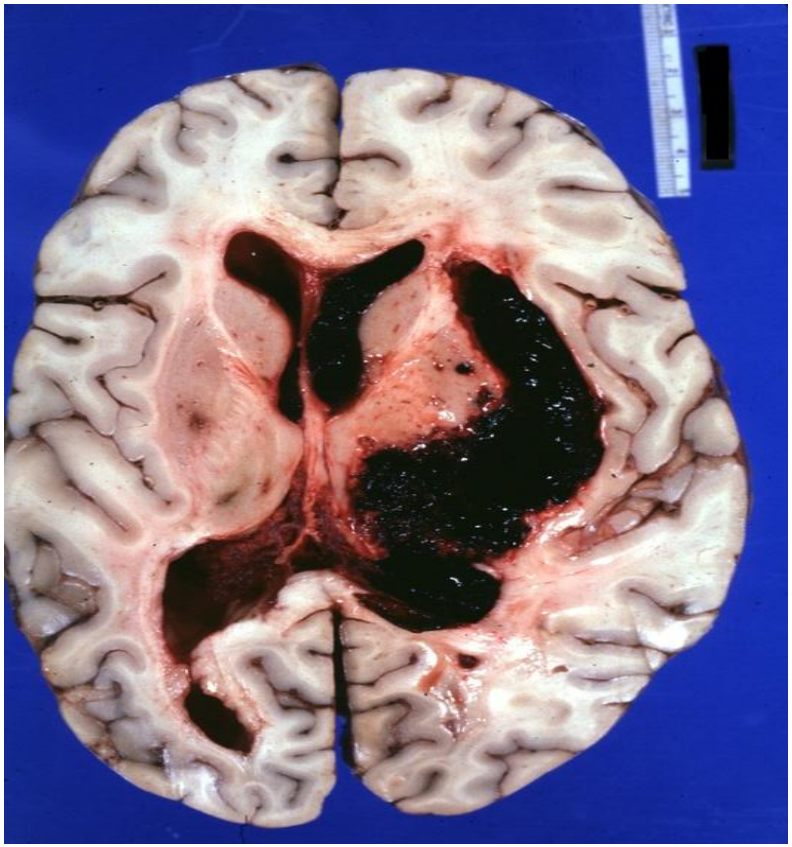


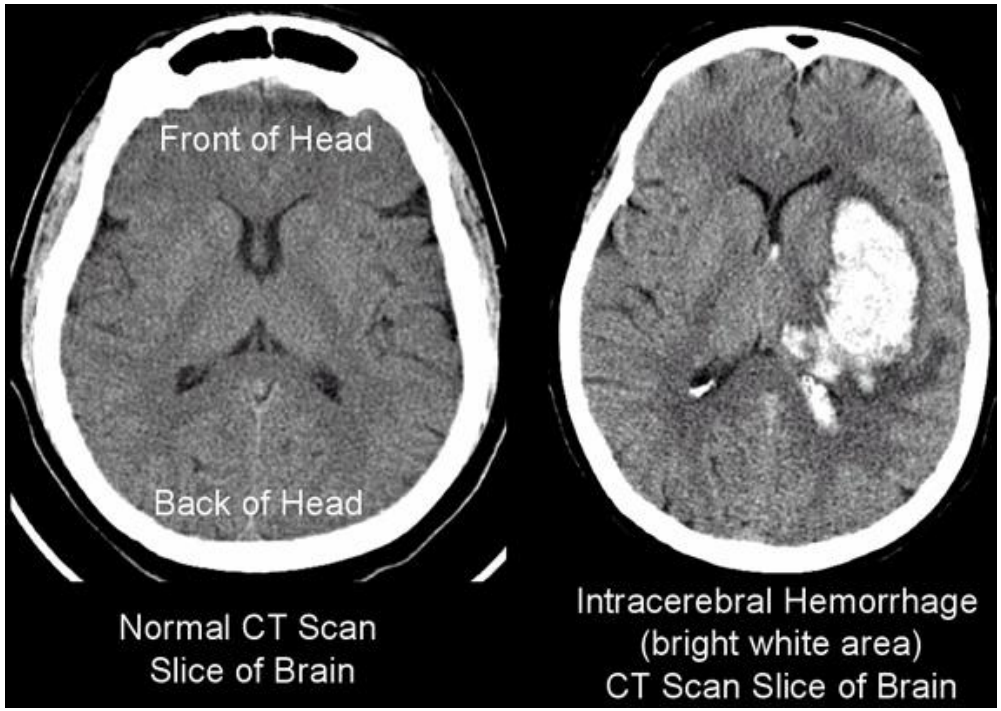
Sites of haemorrhage

- Putamen: 50-60%.
- Thalamus
- Pons
- Cerebral hemispheres.
- Other regions of the brain.

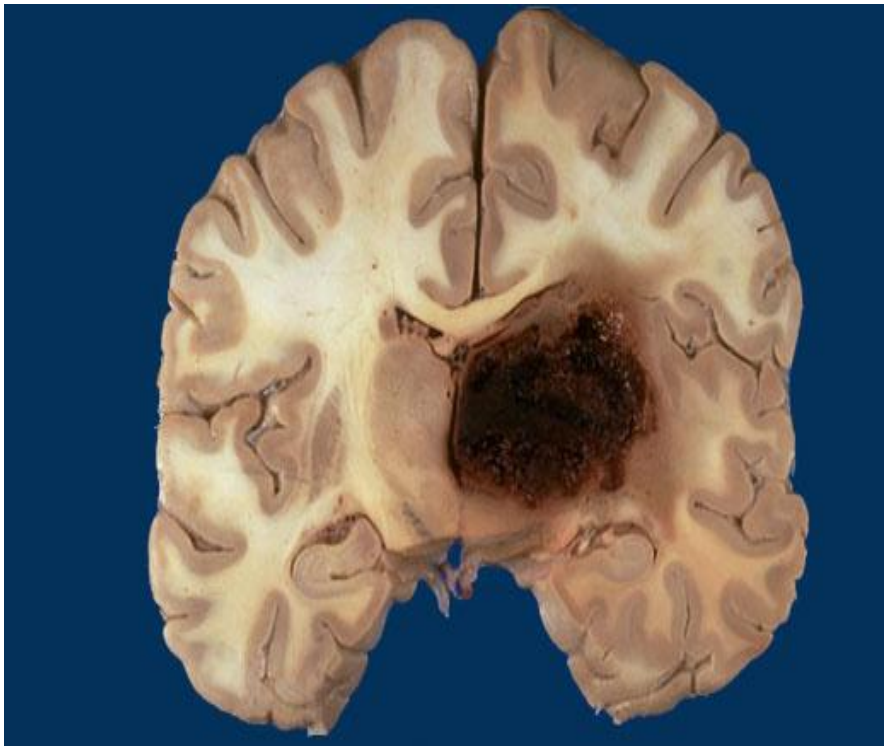
Characterised by bleeding into brain parenchyma with compression of adjacent tissues.

Haemorrhage: Gross

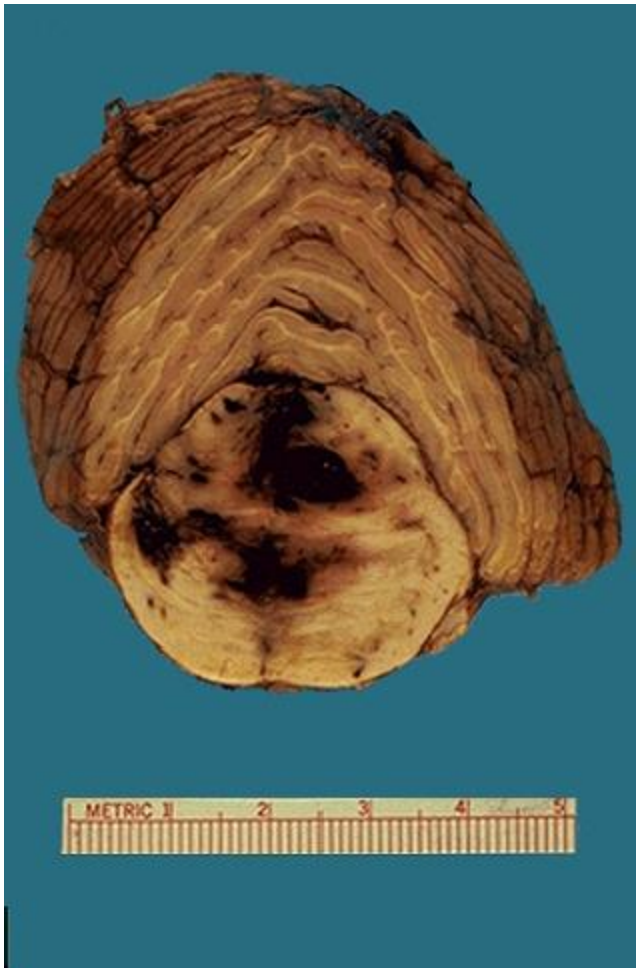




Haemorrhages: Gross



Basal ganglia involved

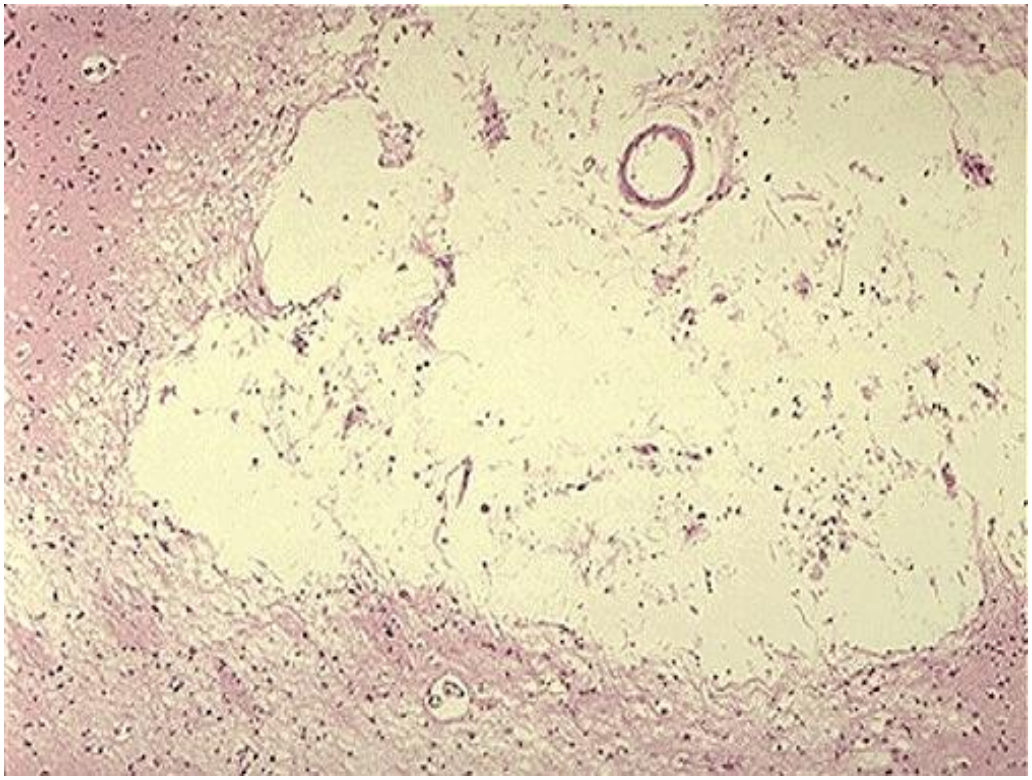


Pons involved (Duret haemorrhages

Lacunar Infarcts

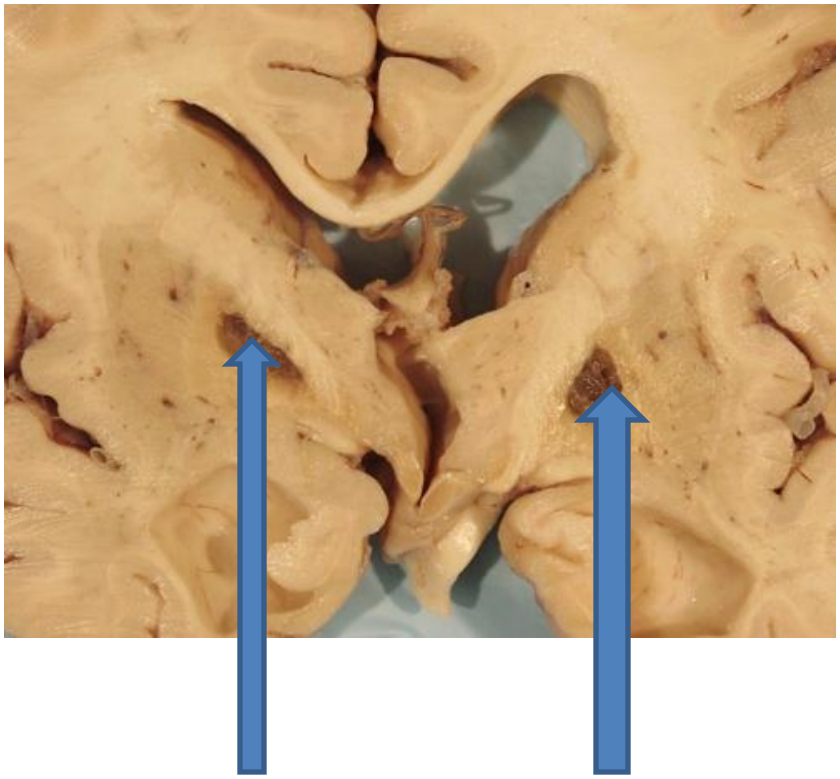
- Hypertension is the most common cause
- Small infarcts (micro-infarcts): <15mm
- Can be single or multiple forming cavitary infarcts (lacunar=cavity)
- Maybe clinically silent
- Can cause significant clinical impairment
- Sites: lenticular nucleus, thalamus, internal capsule, deep white matter, caudate &nucleus, pons.

Lacuna infarct: microscopy

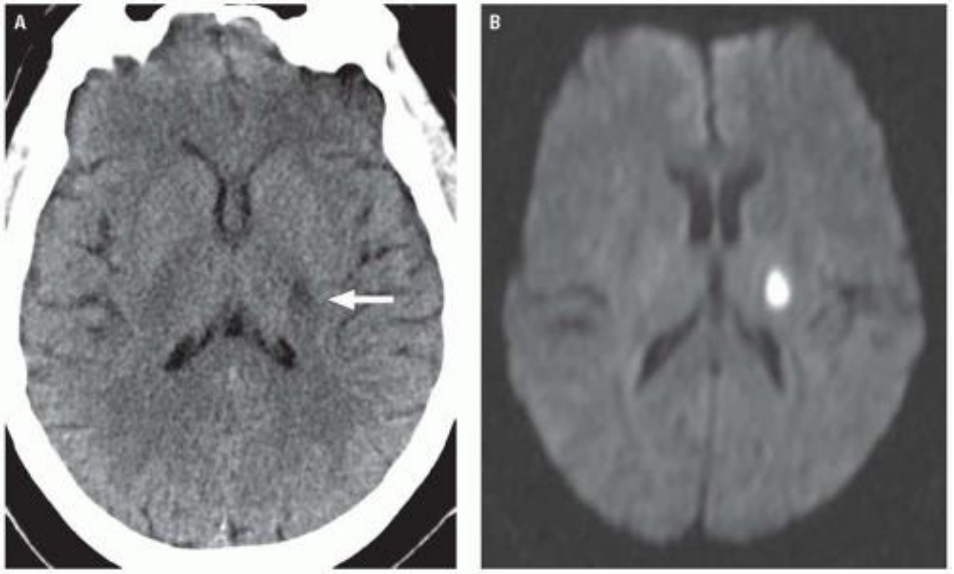


This is the microscopic appearance of a lacunar infarct. Note that it is a cystic space from the resolved liquefactive necrosis. There can be hemosiderin pigment from hemorrhage as well.

Lacunar infarct: Gross



Infarcted area



Acute lacunar infarct. A: Axial unenhanced CT image demonstrates an ovoid area of hypodensity centered in the posterior limb of the left internal capsule.

Diagnosis

- Clinical
- Medical Imaging: CT, MRI, Ultrasound (e.g. thrombus, blood flow analysis), angiography among others.

Management

- Specific: if presenting early in small number of cases. E.g removal of thrombotic emboli.
- Non-specific/supportive.
- Rehabilitation: physical (e.g. physiotherapy) & mental (e.g. depression).
- Preventive strategies:
  - Education/Awareness (life style changes)
  - Risk factor screening in high risk groups

Prognosis

- Variable depending on severity pathology and where in the world you live.

References for ongoing learning

- Robins Pathological Basis of Diseases.
- http://library.med.utah.edu/WebPath/