Stroke - Overview

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Acknowledgement

Thank you Dr Jason Gu
Overview

● Anatomy
  ○ The CNS: an overview
  ○ Cortical function
  ○ Cerebral vasculature
  ○ Motor and sensory pathways
  ○ Skull compartments
  ○ The ventricular system
  ○ Vision pathways

● Pathophysiology
  ○ Ischaemic stroke
  ○ Embolic stroke
  ○ Haemorrhagic Stroke
  ○ Young stroke

● Neuro-imaging
Anatomy

1) The CNS
Nervous System

- Central Nervous System (CNS): consists of brain and spinal cord

- Peripheral Nervous System (PNS): nerves joining CNS to peripheral structures
The Brain

Grey Matter - dense in nerve cell bodies eg central part of spinal cord and surface of cerebral hemispheres.

White Matter - contains nerve processes, often myelinated.
### Embryonic Development

<table>
<thead>
<tr>
<th>Primary Brain Vesicles</th>
<th>Secondary Brain vesicles (wk 7)</th>
<th>Mature Brain</th>
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</thead>
<tbody>
<tr>
<td>Prosencephalon (forebrain)</td>
<td>Telencephalon</td>
<td>Cerebral hemisphere</td>
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<tr>
<td></td>
<td>Diencephalon</td>
<td>Thalamus</td>
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<tr>
<td>Mesencephalon</td>
<td>Mesencephalon</td>
<td>Midbrain</td>
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<tr>
<td>Rhombencephalon (hindbrain)</td>
<td>Metencephalon</td>
<td>Pons, cerebellum</td>
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<tr>
<td></td>
<td>Myelencephalon</td>
<td>Medulla oblongata</td>
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Anatomy

2) *Cortical Function*
Frontal Lobe

- Lies anterior to central sulcus
- Precentral gyrus: **primary motor cortex**
- Inferior frontal gyrus of the dominant hemisphere is the motor speech area - **Broca’s area**.
- Middle frontal gyrus: **Frontal eye field**, controls voluntary conjugate deviation of the eyes when scanning, causes eye deviation towards the side of lesion.
- **Higher functions**: intellectual, judgemental, predictive faculties, planning of behaviour
The clinical picture
Parietal Lobe

- Postcentral gyrus: **primary somatosensory cortex**
- Inferior **visual field loss**
- Parietal association cortex
- Sensory information and **conscious awareness**
- **Interfaces** between visual and auditory association cortices.
- **Dominant lobe:** inability to name objects (anomia), loss of literacy, inability to read (alexia), to write (agraphia) and to calculate (acalculia)
- right parietal lobe: constructional apraxia
The clinical picture

Mrs Z, inability finding her jeans pocket, bumping into doors around corners and some right arm weakness. Difficulty with her speech, in particular with naming things.
Temporal Lobe

- **Primary auditory cortex:** conscious perception of sound. Bilaterally represented.
- Dominant hemisphere: **Wernicke’s area.**
- Inferior medial part is curled to form the **hippocampus:** memory and emotional aspects of behaviour.
- **Amygdala:** receive from olfactory tract.
The clinical picture

Aphasia. Word finding difficulty
Occipital Lobe

- Primary visual cortex
- Visual association cortex: interpretation of visual images.
The clinical picture

Aphasia, ataxia, arm and leg drift
Homonymous hemi
Cerebellum

- Midline: postural control
- **Ipsilateral fibres**
- Dysarthria
- Nystagmus
- Ataxia
The clinical picture
Basal ganglia

- Striatum, pallidum, substantia nigra, subthalamic nucleus, circuit connections
- Eye movements, in conjunction with the superior colliculus (midbrain)
- Motivation
- Movement disorders
- Ascending and descending tracts (internal capsule)
Brainstem

- **Midbrain**: nerve nuclei (3rd, 4th), substantia nigra, reticular formation (arousal and consciousness), central tegmental tract
- **Pons**: tracts between cerebrum, medulla, cerebellum. Sensory signals to the thalamus.
- **Medulla oblongata**: continuous with the pons. Contains cardiac, respiratory, vomiting and vasomotor centres (Heart rate, breathing and blood pressure)
The clinical picture
Anatomy

3) *Cerebral vasculature*
Blood supply to cerebral hemispheres

From stroke perspective, more important than “lobes”
Localise vessel
Clinical examples: symptomatic carotid stenosis
face + arm + leg
MCA
Lateral Medullary Syndrome (Aka Wallenberg Syndrome)

- PICA
- **vestibular nuclei** - vomiting, vertigo, nystagmus
- **inferior cerebellar peduncle**: ipsilateral cerebellar signs
- central tegmental tract: palatal myoclonus
- **lateral spinothalamic tract**: contralateral pain and temp (body)
- **Spinal trigeminal nucleus and tract**: ipsilateral pain and temp (face)
- **nucleus ambiguus**: ipsilateral laryngeal, pharyngeal and palatal hemiparalysis. dysphasia, hoarseness and diminished gag
- **sympathetic fibres**: ipsilateral horner’s syndrome
Watershed infarct

Hypoperfusion $\rightarrow$ Watershed Infarct
Venous infarction

Due to cerebral venous thrombosis:
- increased venous pressure, increased capillary pressure
- blood brain barrier disruption
- vasogenic oedema (leading to failure of energy metabolism), venous haemorrhage (capillary or venule rupture)

Presentation: headache, visual change (raised ICP), stroke symptoms, seizures.

Causes
- prothrombotic conditions
- prothrombotic states: pregnancy, OCP, malignancy
- Head injury and mechanical precipitants
Anatomy

4) Motor and Sensory Pathways
Motor and Sensory pathways
Anatomy

5) Skull compartments
Skull and compartments

Base of Skull, Internal Aspect

- Anterior cranial fossa
- Middle cranial fossa
- Sphenoid bone
- Petrous ridge of temporal bone
- Foramen magnum
- Internal occipital crest

Anatomy

6) The Ventricular System
Ventricular system
Circulation of Cerebrospinal Fluid (CSF)

1. CSF is produced by the choroid plexus of each ventricle.
2. CSF flows through the ventricles and into the subarachnoid space via the median and lateral apertures. Some CSF flows through the central canal of the spinal cord.
3. CSF flows through the subarachnoid space.
4. CSF is absorbed into the dural venous sinuses via the arachnoid villi.
Anatomy

7) Visual pathways
Optic radiation
Visual Field Testing
Pathophysiology

1) Ischaemic stroke
Ischaemic Stroke

80% of all strokes

- Ischaemia: an inadequate blood supply to an organ or part of the body
- Loss of oxygen (and other substrates) and removal of waste products
- Adenosine triphosphate (ATP) levels drop rapidly (4mins)
- Usually there is a switch to anaerobic metabolism
- Cells unable to maintain electro-chemical gradients
- Loss of cell functions, loss of cell integrity (cell death)
3 main types of ischaemia:

- **Thrombosis**: in situ (within vessel) obstruction of an artery

- **Embolism**: particle of debris originating elsewhere that blocks arterial access to a particular brain region

- **Systemic hypoperfusion**: more general circulatory problem
1. Cardiac source definite:
   a. LA thrombus, LV thrombus, AF and pAF, myocardial infarction, rheumatic MV or AV disease, bioprosthetic heart valve, dilated cardiomyopathy

2. Cardiac sources possible
   a. PFO, atrial septal aneurysm, LV aneurysm

3. Ascending aortic atheromatous disease

4. True unknown source
Thrombotic stroke

Large vessel (common and internal carotids, vertebral, circle of willis, proximal branches)
- Atherosclerosis
- Dissection
- Arteritis/vasculitis
- Fibromuscular dysplasia
- Vasoconstriction
- Aortic disease *

Small Vessel disease (intracerebral arterial system ie penetrating arteries from the distal vertebral, basilar, MCA, CoW):
- Lipohyalinosis (secondary to hypertension) and fibrinoid degeneration
- Atheroma formation
Pathophysiology

2) Haemorrhagic stroke
Haemorrhagic stroke

Intracerebral vs subarachnoid haemorrhage

Common causes for spontaneous intracerebral haemorrhage:
- Primary: Hypertension (lacunar), trauma, bleeding diatheses, amyloid angiopathy, illicit drug use (amphetamines and cocaine), vascular malformation.
- Secondary: bleeding into tumours, aneurysmal rupture, vasculitis
- consider age, size, location, past history, imaging
Pathophysiology

3) Young stroke
Young stroke

- Often still atherosclerosis or AF
- Dissection
- Cardiac defects
- Vasospasm
- Hypercoagulable state
- Vasculitis
- Migraine and other headache disorders
- Metabolic (CADASIL, Fabry disease, MELAS)
Blood disorders

Should especially be considered in young patients eg <45yrs of age, history of clotting dysfunction, history of cryptogenic stroke:

- sickle cell anaemia, polycythaemia rubra vera, essential thrombocytosis, HITS, Protein C or S deficiency, Prothrombin gene mutation, Factor V Leiden (APC resistance), AT III deficiency, Anti-phospholipid syndrome, hyperhomocysteinaemia

Infectious and inflammatory disorders

- cause a rise in acute phase reactants such as fibrinogen, CRP, coagulation factors VII and VIII
Neuro-imaging
CT

- Exclude haemorrhage
- Immediate
  - hyperdense vessel
- Early (1-3 hrs or hyperacute stage)
  - loss of grey-white differentiation and hypoattenuation of deep nuclei
  - cortical hypodensity with associated parenchymal swelling and gyral effacement
- first week:
  - more attenuation
  - More swelling
- Onwards:
  - swelling subsides, gliosis begins to occur
  - eventually results in low density lesion with negative mass effect
CT: Advanced techniques

1. CT Angiography
2. CT Perfusion
   a. Cerebral blood volume
   b. Cerebral blood flow
   c. Mean transit time
   d. Time to peak
   e. Penumbra: MTT - CBV
Perfusion MTT vs CBV
MRI

Greater sensitivity for acute ischaemic infarction in the first few hours

1. T1:
   a. Water/CSF is dark
   b. Sensitive for tissue
   c. Low density
2. T2/FLAIR:
   a. Water/CSF is bright
   b. Less immediately sensitive, but does show high signal by 6-12hrs
   c. Sulcal effacement, mass effect
3. DWI/ADC: correlates with infarct core
4. GE/SWI: sensitive for haemorrhage
Key points
1) Where is it?
2) Does it fit the vasculature?
3) What is the mechanism?

- **Ischemic Stroke**: Area deprived of blood
- **Hemorrhagic Stroke**: Area of bleeding

**Ischemic Stroke**
- Obstruction blocks blood flow to part of the brain

**Hemorrhagic Stroke**
- Weakened vessel wall ruptures, causing bleeding in the brain
4) Don’t forget the other parts of the brain
5) Don’t forget the rest of the patient
Thank you

Any questions?
Resources:

- Illustrated atlas neuroanatomy
- Nolte, neuroanatomy
- Uptodate
- Radiopedia