**Cerebrovascular Accident**

**History of Presenting Illness**

**BOX 5.3 Key points to elicit about focal neurological symptoms**
1. Nature - Was the deficit of the motor, somatosensory, visual and/or other system?
2. Quality - Was there a loss of function (e.g. weakness or numbness) or a gain of function (e.g. jerking, paresthesiae)?
3. Anatomical distribution - For example, did the deficit involve the face, arm or leg, or the face, arm and leg?
4. Onset - Was it sudden, stuttering or gradual?
5. Evolution - For example, did the deficit recover, stabilise or progress?

**ALSO ask about risk factors:**
- AGE?
- Smoking??

**BOX 5.1 Focal neurological and ocular symptoms**
- Motor symptoms
- Weakness or clumsiness of one side of the body, in whole or in part (hemiparesis)
- Simultaneous bilateral weakness (paraparesis, quadriparesis)*
- Difficulty swallowing (dysphagia)*
- Imbalance (ataxia)*

**BOX 5.2 Non-focal neurological symptoms**
- Generalised weakness and/or sensory disturbance
- Light-headedness
- Faintness
- "Blackouts" with altered or loss of consciousness or lancing, with or without impaired vision in both eyes
- Incon tinence of urine or faeces
- Confusion

**Symptoms are FOCAL, NEGATIVE and MAXIMAL AT ONSET? = STROKE!!**

**TIA and Stroke ONSET is ALWAYS SUDDEN**

**Strokes usually happen IN THE MORNING**

There is usually NO PRECIPITATING EVENT for stroke; haemodynamic TIA may result from a change in posture or strenuous activity

**Symptoms are FOCAL, NEGATIVE and MAXIMAL AT ONSET? = STROKE!!**

**When did it happen?? Crucial for thrombolysis**

**TIA differs only in duration:**
- resol ves in 24 hrs

**Risk of stroke doubles for every decade after 55 y.o.**

**TABLE 5.1 Neurological symptoms during transient ischaemic attacks**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral weakness, heaviness or clumsiness</td>
<td>50</td>
</tr>
<tr>
<td>Unilateral sensory symptoms</td>
<td>35</td>
</tr>
<tr>
<td>Slurred speech (dysarthria)</td>
<td>23</td>
</tr>
<tr>
<td>Transient monocular blindness</td>
<td>18</td>
</tr>
<tr>
<td>Difficulty speaking (dysphasia)</td>
<td>18</td>
</tr>
<tr>
<td>Unsteadiness (ataxia)</td>
<td>12</td>
</tr>
<tr>
<td>Dizziness (vertigo)</td>
<td>5</td>
</tr>
<tr>
<td>Homonymous hemianopia</td>
<td>5</td>
</tr>
<tr>
<td>Double vision (diplopia)</td>
<td>5</td>
</tr>
<tr>
<td>Bilateral limb weakness</td>
<td>4</td>
</tr>
<tr>
<td>Difficulty swallowing (dysphagia)</td>
<td>1</td>
</tr>
<tr>
<td>Crossed motor and sensory loss</td>
<td>1</td>
</tr>
</tbody>
</table>

* Percentage of 164 - the proportion of patients with TIA with various focal neurological symptoms from the Oxfordshire Community Stroke Project; many patients had more than one symptom (e.g. weakness as well as sensory loss) and no patient had isolated dysperta, ataxia, vertigo, diplopia or dysphagia.

**Symptoms indicating a TIA**

The diagnosis of TIA is clinical, and rests on the description by the patient or an eye-witness of symptoms:
- of loss of focal neurological or monocular function (see Table 5.1)
- of sudden onset
- that are maximal at onset, without spread or intensification
- that are thought to be due to inadequate blood supply to the brain or eye as a result of arterial thrombosis or embolism, associated with disease of the arteries, heart of blood

**Also consider stroke - For any acute focal neuro deficit or any acutely altered consciousness**
**MIDDLE CEREBRAL ARTERY:**
- Total occlusion
- CONTRALATERAL HEMI-EVERYTHING
  - Plegia, -anopia, -anaesthesia; DOWN TO THE THE HIP
  - Global aphasia: complete failure of comprehension and expression
- PLUS either DOMINANT or NON-DOMINANT symptoms:
  - Anosognosia, hemineglect, constructional apraxia

**MCA SUPERIOR DIVISION**
- WEAKNESS of UPPER LIMBS and FACE
- BROCA’S EXPRESSIVE APHASIA
- Wernicke’s Receptive Aphasia
- WEAKNESS of the CONTRALATERAL foot, leg, and genital failure

**MCA INFERIOR DIVISION**
- CONTRALATERAL HEMIPLEGIA
- SUPERIOR QUADRANTINOPIA
- Wernicke’s Receptive Aphasia

**ANTERIOR CHOROIDAL**
- Supplies Posterior Limb of Internal Capsule
- HEMIPLEGIA AND HEMIANOPIA

**POSTERIOR CEREBRAL ARTERY**
- Rarely will you infarct the whole thing. Usually only one of the branches:
  - VISUAL DISTURBANCE eg.
    - Homonymous hemianopia with macular sparing
    - HEMIBALLISMUS if subthalamic nuclei infarcted
    - IN THE DOMINANT HEMISPHERE:
      - ANOMIA, AGNOSIA, ALEXIA in occipitotemporal stroke

**BASILAR ARTERY OCCLUSION:**
- locked-in syndrome
- complete paralysis with preservation of consciousness
- almost uniformly fatal

**VERTEBRAL ARTERY OCCLUSION:**
- What happens depends on the quality of your other vertebral artery: if you don’t have one, this has the effect of a basilar artery stroke.

**ANTECESSOR CEREBRAL ARTERY:**
- the Seat of Manners
- BEHAVIOURAL ABNORMALITIES:
  - Paucity of insight, slowness of thought, apathy, distractability
  - usually happens when you congenitally have both ACAs arising from one stem; this way you infarct bilaterally
  + CONTRALATERAL FOOT, LEG and GENITAL FAILURE:
    - Hemiplegia and hemiaesthesia sparing the upper limbs and face
    - (therefore also INCONTINENCE)

**THE NON-DOMINANT MCA:**
- WEIRDNESS
  - In the Superior division:
    - Brachiofacial paralysis, as well as
    - Anosognosia: you stubbornly fail to notice that you have a serious neurological deficit
    - Hemineglect: everything on that side does not exist for you, you just ignore it
  - Constructional apraxia: inability to mentally rotate objects, follow maps, copy pictures
  - Dressing apraxia: unable to operate simple shirts
  - Constructional apraxia

**Non-Dominant PCA stroke:**
- Anomia for familiar faces (prosopagnosia)
- Spatial Disorientation
- BILATERAL TEMPORAL PCA STROKE:
  - Hippocampus damaged, thus PERMANENT AMNESIA

**SUPERIOR CEREBELLAR:**
- Ipsilateral limb and gait ataxia
- Horner’s syndrome
- tremor
- contralateral limb dysmetria
- contralateral loss of pain and temperature sensation over the body and face,
- loss of limb position sense
- DEAFNESS

**AICA: Pontine Infarct**
- DEAFNESS
- loss of pain and temperature sensation on the face AS WELL AS complete paralysis of the face;
- Ipsilateral limb and gait ataxia
- Dizziness
- Nystagmus

**PICA: Lateral Medullary Syndrome**
- Ipsilateral Horner’s Syndrome
- Loss of pain + temperature:
  - IPSI face- CONTRA body
- - Dysarthria
- - Dysphagia
- - Dysphonia
- - Vertigo
- - Nystagmus
- - Ataxia or Unsteady gait
- - Loss of taste and paralysis of the palate
Mimicking Stroke:
The most frequent stroke mimics include seizures (17%); systemic infections (17%); brain tumors (15%); toxic-metabolic causes, such as hyponatremia (13%); positional vertigo (6%).

REMEmber:
Hypoglycaemia
Head trauma
Brain Tumour
Epilepsy
Hemiplegic migraine
Syncope
TIA
Meningitis

Miscellaneous disorders
- syncope,
- trauma,
- subdural hematoma,
- herpes encephalitis,
- transient global amnesia,
- dementia,
- demyelinating disease,
- myasthenia gravis,
- parkinsonism,
- hypertensive encephalopathy,

Examination: Where did the clot come from?
ATRIAL FIBRILLATION? ENDOCARDITIS? Then, peripheral vascular system:
- carotid auscultation for bruits,
- blood pressure, and pressure comparison between arms
- examine extremities for ischaemia (peripheral emboli),
- retina [effects of hypertension and cholesterol emboli (Hollenhorst plaques)];
PLUS: look for signs of head trauma

Tests and Investigations
Investigate bruises with DOPPLER ultrasound

CT scan of the BRAIN:

CT excludes 90% of intracranial haemorrhages; the other 10% can be picked up with LUMBAR PUNCTURE
by12 hours after a subarachnoid bleed, blood breakdown products collect at the base of the spine, and can be drained for analysis – you are looking for XANTHOCHROMIA, or yellowness

CT will miss little infarcts, early infarcts, posterior fossa infarcts (obscured by artefacts)

NOTE: A CT scan done too early (before oedema begins) will yield nothing!!
thus, a NORMAL CT DOES NOT EXCLUDE A STROKE!!
But… even 5 hrs after the CVA the CT will give findings in 50% of cases
So scan anyway, just to have a baseline picture of their brain

WHAT ARE YOU LOOKING FOR?
The evolution of a haemorrhage:
- Immediately: a white, hyperdense area
- Within days: becomes less dense (turns to clot)
- after a few days to a few weeks (depending on its size) it becomes isodense with surrounding brain tissue (‘fogging’) and may be difficult to see. Smaller haemorrhages fog up faster
- Thereafter the haemorrhage becomes hypodense and may be mistaken for an old infarct

The evolution of an infarct:
hyperacute stage: the CT image often appears normal.
Sometimes might find a hyperdense artery (see the thrombus in it) within 3 hours there are usually subtle changes in the ischaemic brain
= loss of normal grey-white matter differentiation
= compression of the lateral ventricle due to focal brain edema
= and hypodensity!! Dark greyness…
during the first few days, becomes more clearly demarcated and well defined and more hypodense (black)
around days 3–5 the swelling of the infarct is usually maximal during the second and third week the swelling subsides Eventually a sharply demarcated, atrophic, hypodense (similar to CSF) defect remains: the INFARCT CAVITY

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MRI for stroke:
Diffusion Weighted Imaging is much more helpful than CT in the early phase
Infarcts of any size are more often and more quickly visible
However, even MRI can be normal in a clinically definite stroke!!

Minutes: loss of the normal flow void in the symptomatic artery within minutes of onset
(the MR equivalent of the hyperdense artery sign on CT)
3 hours: swelling of the ischaemic brain on T1-weighted images, but without signal change on T2-weighted images
8 hours: signal changes on T2-weighted images
16 hours: signal change on T1-weighted images.
Large infarcts are often visible on routine T1- and T2-weighted imaging within 6 hours,
... but small cortical and subcortical infarcts may never become visible.

Also may want to consider Magnetic Resonance Angiography

The advantages of MRI over CT are:
1. Better soft tissue contrast resolution;
2. Multiplanar capabilities;
3. No ionising radiation;
4. No bone artifacts which limit the sensitivity of lesion detection in the posterior fossa;
5. Very sensitive to changes in water concentration in tissues and therefore superior detection of lesions; and
6. Ability to perform both anatomic and physiological imaging.

The disadvantages of MRI are:
1. More expensive than CT
(between two and three times more);
2. Less accessible to patients because there are less units than CT in Australia;
3. Problems with cardiac pacemakers and other implants including aneurysmal clips; and
4. Claustrophobia

MRI has not been evaluated for (and is generally held to be useless at) picking up very acute haemorrhage

OTHER INVESTIGATIONS:

FBC, ESR: looking for infectious WBC changes
urea, creatine, electrolytes: looking for renal disease 2ndary to renal artery stenosis (comorbid with hypertension)
Liver Function Testing: looking to exclude hepatic encephalopathy
Random Glucose: looking to exclude ketoacidosis coma
Serum cholesterol & triglycerides looking for hyperlipidaemia (a risk factor)
Chest X-ray looking for signs of heart failure, in particular LV and LA enlargement
ECG: looking for signs of dysrhythmia, in particular atrial fibrillation
Echocardiogram: looking for atrial appendage clots and septal defects
Lumbar Puncture to support a diagnosis meningitis if that’s what it looks like
Echo doppler: looking for carotid stenosis and/or aneurism, even if there wasn’t a bruitt.

How is this diagnosis made?
By CT, MRI, and the exclusion of hypoglycaemia, infection, trauma and tumour
**MANAGEMENT**

### Immediately:

**CIRCULATORY AND RESPIRATORY SUPPORT:**
- Make sure they have an airway and are saturating
- Make sure they are not dehydrated

**REPLACE FLUIDS INTRAVENOUSLY:** *NIL BY MOUTH* until cleared as somebody who can swallow without choking.

**WATCH THEIR BLOOD PRESSURE:** *mainly out of morbid interest*
- Give them more fluids if they are hypotensive, BUT-
  - DO NOT attempt to lower even ridiculously high blood pressures:
    - High blood pressure helps perfuse the highly stenosed brain vessels
    - THUS wait 7 – 10 days before starting new antihypertensives
- HOWEVER if they are already on anti-HT meds, YOU CAN FEED THEM THEIR OWN PILLS …carefully watching their blood pressure and monitoring them for signs of deterioration.

**IF THE SYSTOLIC IS OVER 220,** lower their blood pressure: that way they will become eligible for thrombolysis.
- Use a short-acting drug which has a reduced effect on intracranial vessels, eg. **LABETALOL** (Alpha-Blocker and nonselective beta-blocker)

### ESTABLISH NORMOGLYCAEMIA

**ADMINISTER NEUROPROTECTIVE DRUGS**
- May be medicolegal suicide, as none have been proven to have a measurable effect on survival;

**ANTICOAGULATE with heparin**
- if particularly keen; but 1-4% of patients with stroke will upgrade to hemorrhagic stroke when anticoagulated (i.e. the clot you dissolved was holding the blood in a vessel somewhere)

**PLUS:** may consider anticoagulation if the patient is looking at a prolonged hospital stay

### Long-Term Rehabilitation:

**Administer therapy to address those functions lost as the result of CVA:**

- **SPEECH THERAPY**
- **OCCUPATIONAL THERAPY**
- **PHYSIOTHERAPY**
- **COUNSELLING**
- **PSYCHOTHERAPY**

**STROKE UNIT** is the best place to recover from a CVA because its swarming with the abovementioned specialist allied health staff

Most people stay for months…

Key words to spout in the barrier: “Therapeutic Alliance” and “Multidisciplinary Team Approach”
Prognosis

Stroke is the third leading cause of death and the leading cause of disability in the US. Cerebrovascular disease was the second leading cause of death worldwide in 1990, killing over 4.3 million people. Cerebrovascular disease was also the fifth leading cause of lost productivity.

Epidemiology

In the US: Incidence for first-time strokes is more than 400,000 per year. At current trends, this number is projected to jump to one million per year by the year 2050.

Risk factors:

Risk factors you cannot change

- **Age.** Nine out of ten strokes affect people over 55. The risk for stroke increases with age. The risk doubles every decade you are over 55.
- **Race.** African-Americans and Hispanics have 2 to 3 times the risk of ischemic stroke
- **Gender.** Stroke is more common in men than women. However, at older ages, more women than men have strokes. At all ages, more women than men die of stroke.\(^2\)
- **Family history.** The risk for stroke is greater if a parent, brother, or sister has had a stroke or TIA.
- **Prior history of stroke or TIA.** About 14% of people who have a stroke have another stroke within 1 year.\(^2\) Up to 25% have another stroke within 5 years.\(^3\)

Risk factors that you can change

- **Hypertension** = the second most important stroke risk factor after age.
- **Diabetes.** About one-quarter of people with diabetes die of stroke. Having diabetes doubles your risk for stroke
- **High cholesterol.**
- **Other heart conditions** such as atrial fibrillation, endocarditis, heart valve conditions, and cardiomypathy
- **Other diseases :** lupus, syphilis, hemophilia, pneumonia, high levels of homocysteine, and periodontal disease.

Pathophysiology

**CAUSES OF COLLAPSE**

= either with retention of consciousness or with loss of consciousness.

= Loss of consciousness =

**EITHER cerebral cortex** has been disturbed diffusely, **OR** that the **Brainstem Reticular Formation**

**Common causes =** **epilepsy**

- tonic-clonic,
- absence,
- akinetic

Recovery is usually rapid, though some patients may carry on having continuous seizure (status epilepticus).

**cerebrovascular disease**

especially if the reticular formation is involved in a **brain stem stroke** or in

**massive cerebral strokes such as subarachnoid hemorrhage.**

The collapse is usually of **longer duration** than in syncope or epilepsy

**syncope** - a sudden and brief loss of consciousness associated with a loss of postural tone, from which recovery is spontaneous.

ALL SYNCOPE = result of DECREASED CEREBRAL OXYGENATION

... Syncope can be

1. **neurally mediated (vasovagal syncope)**
   = reflex-mediated changes in vascular tone or heart rate. This is the commonest cause of syncope, and may be due to emotional factors or activation of receptors in organs such as the bladder (micturition syncope) or the carotid sinus.

2. **orthostatic hypotension**
   (volume depletion, medications, primary and secondary – e.g. diabetes - autonomic failure).

3. **psychiatric** (e.g. panic attacks).

4. Due to **primary cardiac** conditions such as structural **heart diseases (such as aortic stenosis) or arrhythmias.**

   *In about a third of cases a cause cannot be found.*
CEREBRAL ISCHAEMIA
- The blood flow to the brain is controlled relatively independently to that of the rest of the body.

VASCULAR CONTROL
- **CRITICAL NEED FOR STABLE BLOOD PRESSURE**
  - **REMAINS CONSTANT** for 50 – 150 mmHg
  - This is known as cerebral autoregulation.
  - (increased pressure = dilation of arterioles to drop the pressure)

METABOLIC CONTROL
- Increase in the partial pressures of CO₂ (pCO₂) = vasodilation,
- Decrease in pCO₂ = vasoconstrictor.
  - **THUS**: sudden and local changes to blood flow, matching neurone activity.

Increased local neural metabolic activity
- The release of CO₂ (which leads to a decrease in local pH)
- Local vasodilation.
- Increased blood flow
- Thus, need for more nutrition is met

**Cerebral blood flow** is also controlled by both sympathetic and parasympathetic autonomic nerves.

**Sympathetic** = from the **superior cervical ganglion** in the neck,
**Parasympathetic** = from seventh (facial) cranial nerve.

**IMPORTANT ROLE UNDER ABNORMAL CIRCUMSTANCES**
- **SYMPATHETIC**: extremes of pressure autoregulation (when b.p. approaches 50 or 150)
- **PARASYMPATHETIC**: dilating vessels under conditions of focal hypoxia or ischaemia
  - **THUS**: AUTONOMIC REGULATION = DAMAGE CONTROL

COMPROMISED BLOOD SUPPLY IS DUE TO:
- Blockage or rupture of an artery
- Blockage of a vein.
- A severe fall in arterial blood pressure

Less severe hypotension causes decreased perfusion at sites of potential limited cerebral circulation
- Leads to "boundary/watershed zone" infarcts between the territories of, for example, the anterior and middle cerebral arteries. If the reduction of blood supply is of brief duration (e.g., migraine or transient ischaemic attack) full recovery of neural function is the rule.
  - If reduction in blood supply is prolonged enough to cause ischaemic necrosis (i.e. infarction) very little recovery of neural tissue function can be expected.

Following a stroke, arterial pressure is often elevated.
However, there is usually severe disruption to normal autoregulatory mechanisms of cerebral blood flow.

Giving agents that lower arterial pressure, therefore, may seriously compromise cerebral perfusion.

**CAUSES OF STROKE**

<table>
<thead>
<tr>
<th>Stroke</th>
<th>Neurological deficit with sudden onset and a vascular basis.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transient ischaemic attacks</td>
<td>Short-term neurological deficit that resolves quickly.</td>
</tr>
</tbody>
</table>

Take history and examine to determine:
1. What is the cause of this patient's stroke?
2. Is there effective therapy for this stroke?
3. Can the chance of a further stroke be reduced?

Strokes can be due either to **OCCCLUSION** (ischaemic necrosis; infarction)
- Or **RUPTURE** (haemorrhage)
**INFARCT:**

**MOST OFTEN:**
cause of infarct is **atheroma in the internal carotid** artery near its origin from the common carotid artery.

**ALSO atheroma may form**
at the **termination of the internal carotid artery** and in the **basilar artery**.

**Small embolus** = only TIA, and more commonly to deep structures.

**Small penetrating arteries** to the deep parts of the brain can be occluded by atheroma, thrombus, or hypertensive thickening of the vessel walls.

**THUS, small infarcts will be found in the deep structures of the brain** such as the basal ganglia.

**HAEMORRHAGE:**
usually due either to
1. **rupture of a berry aneurysm** at the base of the brain,
   - giving rise to a **subarachnoid hemorrhage**,  
2. **rupture of a small penetrating artery**
   - gives rise to a **hematoma deep in the brain** (an intracerebral hemorrhage).
   - These have become less common with better control of arterial hypertension.

**NEUROPROTECTIVE AGENTS**

NORMALLY: treat stroke by addressing thrombogenesis or thrombolysis.

Eg: heparin, aspirin, tPA

NO LONGER!! Noveau therapies aim at cellular disturbances of ischaemia

**PATHOLOGY OF BRAIN INFARCTION:**

**ISCHAEMIA**

*Rise in intracellular Na+*  
**BECauses:**
- Na+ channels stay open for too long  
- No ATP means Na+/K+ATPase cannot pump Na+  
- **ThUS Na+ gradient collapses.**

**OVERSTIMULATION of NMDA glutamate receptor**
- Which has great permeability for Ca++

**ThUS:**
**CALCIUM IONS RUSH INTO THE CELL**
...and *cannot be removed* by the ATP-starved Ca++ ATPase

**IN ABSENCE OF REPERFUSION**

**MItochondrial injury**  
**ThUS: no more ATP, forever**

**ACTIVATION OF**
- Proteases
- Kinases
- Phospholipases
- Endonucleases

**Massive glutamate + aspartate release**
**Because:**
- Transporter for excitatory amino acids (which normally removes them from extracellular space)  
RUNS BACKWARDS and pumps shitloads of them out of the ischaemic neuron.

**WITH REPERFUSION**

**Free radical scavengers** (e.g.,

**REPERFUSION INJURY**
From production of oxygen free radicals
Neuroprotective agents act by targeting one or more of these abnormal cellular events. These drugs target the peri-infarct area which lies between the non-salvageable, ischaemic core and normally perfused brain.

The peri-infarct area is thought to be recruited into the infarcted core over several hours (evolving stroke), providing a therapeutic window for drug intervention.

Thus, it is hoped that neuroprotective agents will prevent "at risk" tissue progressing to infarction, thereby reducing brain damage caused by stroke.

They include:

- glutamate receptor antagonists (e.g., dizocilpine, dextorphan),
- voltage gated Na+ channel blockers (e.g., lubeluzole, riluzole),
- voltage gated Ca+ channel blockers (e.g., nimodipine, lifarizine)
- free radical scavengers (e.g., tirilazad).

PHARMACOLOGY OF ASPIRIN 6.02

Aspirin (acetylsalicylic acid) = COX enzyme system inhibitor.

This enzyme system, now called cyclo-oxygenase, converts arachidonic acid to products such as prostaglandins (PGs) and thromboxane (Tx). The major arachidonic acid metabolite in platelets is thromboxane A2, which is proaggregatory and the major metabolite in endothelial cells is prostacyclin(PGI₂) which inhibits platelet aggregation.

Aspirin acetylates many proteins, binding to them irreversibly. In the case of platelet cyclo-oxygenase, this irreversible binding inhibits thromboxane synthesis for the lifespan of the platelets.

Thus, prolonged bleeding tendency will last for several days after cessation of treatment with aspirin.

long-term treatment with aspirin reduces the incidence of cerebro-vascular events in patients with previous strokes or transient ischaemic attacks.

Clinical studies in normal volunteers suggested that low doses of aspirin (less than 100 mg) effectively inhibit platelet cyclooxygenase activity with only slight inhibition of vessel wall PGI₂ formation.

daily doses of 30 mg aspirin are no less effective than 283 mg and the lower dose has less side effects (Dutch TIA Trial, 1991).

Therefore:

use aspirin (in doses of 100-300mg/day) in patients needing secondary prevention of cerebrovascular disease provided that there are no contraindications (Orme, 1988).
Relevant anatomy

INTERNAL CAROTID, VERTEBROBASILAR SYSTEMS AND CIRCLE OF WILLIS

Orbital
Frontal
Parietal

Anterior cerebral

Heubner’s recurrent striate

Anterior communicating

Hypophyseal

Ophthalmic

Internal carotid in carotid canal

Anterior Choroidal

Anterior Circulation

POSTERIOR CIRCULATION

Middle cerebral (frontal, parietal, temporal)

Posterior communicating

Posterior cerebral (temporo-occipital parieto-occipital)

Basilar

Superior cerebellar

Pontine

Labyrinthine

Anterior inferior cerebellar

Medullary

Posterior inferior cerebellar

Dura

Vertebral

Labyrinthine usually arises from anterior inferior cerebellar. Posterior spinal may arise from vertebral

Anterior spinal

Posterior spinal (anterior/posterior branches)
In the medulla, as in the spinal cord, the different fibre tracts are segregated from each other. For example, the corticospinal tracts are found within the pyramids lying ventrally in the medulla; the dorsal spinocerebellar tracts are found within the inferior cerebellar peduncle located dorsolaterally; the medial lemnisci are found close to the midline, and the spinothalamic tracts are found laterally. Two major decussations occur in the medulla. First, the huge pyramidal (corticospinal) decussation which defines the caudal boundary of the closed medulla, and second, the sensory decussation of the axons from the gracile and cuneate nuclei, located more rostrally in the closed medulla.

The reticular formation of the medulla is made up of many distinct groups of cells concerned with specific functions, the majority of which, are crucial for the maintenance of life. For example, the reticular formation contains small cell groups regulating cardiovascular function, and other autonomic activities, as well as respiration. The reticular formation carries out these critical functions, in part, by controlling directly the activity of the spinal cord (e.g., via reticulospinal tracts). The reticular formation is, in turn, under the control of higher centres.

The brain receives its vascular supply from the internal carotid and vertebral-basilar arterial systems. The internal carotid arteries predominantly supply forebrain structures, while the vertebral-basilar system supplies the cervical spinal cord, brainstem, cerebellum, occipital and portions of temporal neocortex. The two systems meet and anastomose in the circle of Willis on the ventral surface of the brain. This has important clinical implications. For example, if a proximal artery (e.g., the internal carotid artery) is occluded, blood flow to a distal branch (e.g., the anterior cerebral) may be supplied from the other major arteries contributing to the circle.

The vertebral-basilar arterial system has many branches. The most important are the posterior inferior cerebellar artery (PICA), the anterior inferior cerebellar artery (AICA), the superior cerebellar artery (SCA) and the posterior cerebral artery (PCA). Many of the branches of the vertebral-basilar arteries that supply the brainstem also supply the cerebellum (e.g. PICA, AICA and SCA).

In the brainstem, the zone of supply of each arterial branch of the vertebral artery is characteristically "wedge-shaped". For example, in the caudal medulla, three wedges of supply are seen on each side. The most lateral wedge is supplied by the posterior inferior cerebellar artery, the middle wedge is supplied by the vertebral artery itself, and the most medial wedge is supplied by the anterior spinal artery. Thus a blockage of an individual artery will cause a distinctive wedge-like zone of infarction in the medulla, and this will be manifest as a distinct set of symptoms and signs which can be recognised on clinical examination.

**BRAINSTEM RETICULAR FORMATION**

* primitive core

* 2 general functions (related)
  (i) **Arousal-Mood Setter**
  - samples somatic/visceral worlds
  - sets forebrain activity (arousal/mood)
  (ii) **Autonomic Policeman**
  - monitors somatic/visceral world
  - influences crucial reflexes

* individual nuclei with distinct functions
* neurotransmitter systems within BRF
  - serotonin
  - acetylcholine
  - dopamine
  - noradrenaline
**RISK FACTORS:**
- Age.
- Hypertension
- Gender: women
- Family history
- Prior history of stroke or TIA
- Diabetes, doubles your risk for stroke
- High cholesterol
- Other heart conditions
- High levels of homocysteine

**BLOOD VESSEL OCCLUSION**

**RUPTURE of the blood vessel**

**HAEMORRHAGE**

**ISCHAEMIA**

NO ATP!! Thus:
- Na+/K+ ATPase cannot pump the Na+ out;
- PLUS Na+ channels stay open for far too long

Therefore

Influx of intracellular Na+
And hence

SWELLING OF NEURONS

Flipped by changing ionic gradients,
the excitatory amino acid transporter
runs BACKWARDS: pumps glutamate and aspartate OUT
of the cell: **THUS TO**

**CAVITATION**

A fluid-filled hole in the brain.

**Ca++ cannot be removed by the ATP-starved CA++ATPase**

** CA++ cannot be removed by the ATP-starved CA++ATPase**

**REPERFUSION**

**FREE RADICAL SCAVENGE**

**FREE RADICAL SCAVENGE**

**NEUTROPHIL INFLUX**

**NEUTROPHIL INFLUX**

**NO REPERFUSION**

**MITOCHONDRIAL INJURY**

Too much calcium causes mitochondria to lyse...

THUS: more free radicals, and what’s worse, **NO MORE ATP, EVER!**

Also...

**FREE RADICALS**generated by sudden influx of oxygen

EAT THE NEURAL TISSUE

but, the ischaemic penumbra is restored
(i.e. wherever damage was sublethal can be repaired)

**CAVITATION**

**CAVITATION**

**FREE RADICAL SCAVENGE**

**FREE RADICAL SCAVENGE**

**NEUTROPHIL INFLUX**

**NEUTROPHIL INFLUX**

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