Lecture 56, 57

Editing file

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# Ischemic and hemorrhagic strokes

## Objectives:

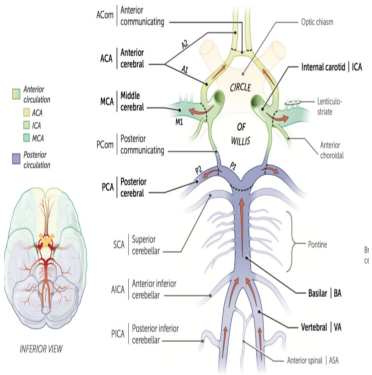
- ★ Know basic epidemiology and burden of stroke including risk factors
- ★ Understand pathophysiology of ischemic Stroke including subtypes/TIA
- ★ Know the clinical presentation of stroke/TIA
- ★ Know how to establish diagnosis and the essential work up including basic principle of Neuro imaging
- ★ Understand management principles of stroke /TIA.
- ★ Prevention/acute therapy/ Prevention of complications/Secondary prevention
- ★ Discuss the outcome of intracerebral hemorrhage
- ★ List the anatomical location of hypertensive hemorrhage
- ★ Summarize the current treatment modalities for intracerebral
- ★ hemorrhage

## Color index:

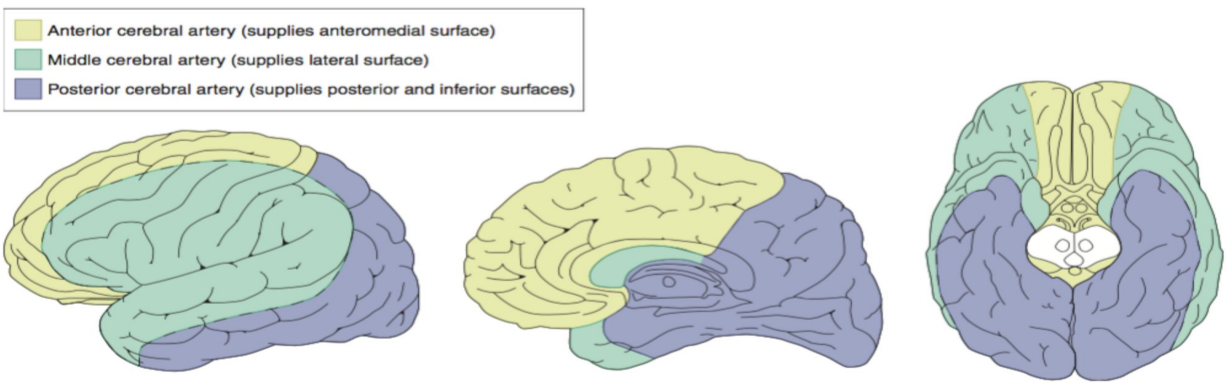
Original text Females slides Males slides
Doctor's notes Textbook Important Golden notes Extra

## ◀ Cerebral circulation

- The two vertebral arteries (arising from the subclavian arteries) join to form the basilar artery, the basilar artery ends in a bifurcation forming the **two posterior cerebral arteries**.
- The internal carotid arteries give rise to the ophthalmic artery before dividing into the **anterior and middle cerebral artery.**
- **Circle of Willis** is formed by: the two anterior cerebral arteries that are connected by the anterior communicating artery, the internal carotid arteries, the posterior cerebral, and the two posterior communicating arteries (connecting the internal carotids to the posterior cerebral arteries).




| Branch | Supplies |
|---|---|
| **Anterior cerebral artery** | • Medial surface of the frontal and parietal lobes.<br>• The anterior four-fifths of the corpus callosum.<br>• Approximately the superior 1 inch of the lateral surface of the frontal and parietal lobes. |
| **Middle cerebral artery** | • Most of the lateral surface of the frontal and parietal lobes.<br>• The posterior limb and genu of the internal capsule.<br>• Most of the basal ganglia. |
| **Posterior cerebral artery** | • The occipital and temporal cortex on the inferior and lateral surfaces of the hemisphere.<br>• The occipital lobe and posterior 2/3 of the temporal lobe on the medial surface of the hemisphere.<br>• Thalamus and subthalamic nucleus. |



# Introduction to Ischemic strokes

## Stroke

Abrupt onset of focal neurological deficits due to interruption of vascular supply.
Stoke can be either:

- Ischemic (blockage) → 80-85% of all strokes → has a better prognosis in comparing to Hemorrhagic stroke.
- Hemorrhagic (bleeding) → 15-20% of strokes

## ◀ Ischemic stroke

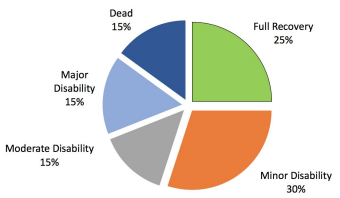
- Acute onset of neurologic deficits caused by impaired blood flow to CNS.
- ★ **Stroke:**
  - **Persisting neurologic deficit after 24 hours** and/or
  - **infarct on CT or MRI.**

## Transient ischemic attacks

- "mini strokes" or "warning strokes" stroke-like symptoms that last for a very short time( <1hr) with complete recovery (most are <5 min) with the **absence of infarct** in neuroimaging study.
- **Case:** A patient presented with stroke-like symptoms for <1hr. Upon doing CT, an infarct was found. What's the diagnosis? Ischemic stroke **NOT** TIA.
- A TIA indicates that conditions for an ischemic stroke are present.

## ◀ Worldwide Burden

Outcome of Ischemic Stroke




Adapted from Stegmayr B, et al. *Stroke* 1997;**28**:1367-1374

- **Leading cause of adult disability.**
- **Second cause of death in Saudi Arabia**[1]
- second leading cause of dementia after alzheimer disease
- 5 million deaths/year.
- 15-20 million people suffer a stroke each year.
- 1 in 4 people will have stroke in their lifetime
- Every six seconds someone is developing stroke somewhere in the world.
- Stroke kills more people than AIDS, malaria and TB combined
- Stroke kills more women than breast cancer does

➔ **In Saudi Arabia**

- New strokes: 20-25,000/year
- If you control all risk factors, 80% of strokes will be prevented
- Deaths (estimate): 4000-5000.
- Disabilities: 8000.
- Incidence:
  - New: 58/100,000
  - New & Recurrence: 70/100,000 total
- Cost to patient, burden to the family, and cost to the community.

1: Sometimes listed as the 3rd leading cause of death after cardiac diseases and cancer.

# Ischemic stroke

## ◀ Risk factors

### Modifiable

- ★ **Hypertension. (Most important one)**
- **Diabetes mellitus.**
- **Hyperlipidemia.**
- Cardiac diseases (particularly **Atrial fibrillation, CHF, IE).**
- Stroke, TIA, and carotid artery stenosis.
- Smoking.
- Sedentary lifestyle.
- Stress, type A personality.
- Air pollution
- Obstructive sleep apnea
- Polycythemia

VS

### Non-modifiable

- **Age**[2], risk after 60 double with each decade.
- **Sex,** generally men are more prone to stroke. However, young women are at higher risk than men due to pregnancy, hormonal changes.
- **Ethnicity** (African americans)
- **Genetic determinants** (e.g. sickle cell disease)
- **Previous vascular events** (MI, stroke, PVD)

## ◀ Mechanisms

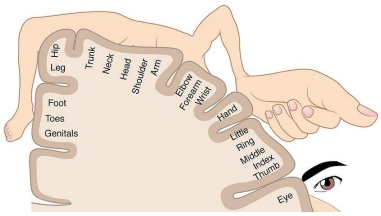
1. Due to **blockage** from:
   - **Cerebral thrombosis:** a thrombus (blood clot) that develops at the blocked part of the artery.
     - From an atherosclerotic plaque → **hypoperfusion**: only happen in narrow vessels, leads to reduced flow.
   - **Cerebral embolism:** typically caused by a blood clot that forms at another location and breaks loose and enters the bloodstream.

➔ Generally, strokes are divided into four subtypes: Large vessel stroke → atherosclerosis of major vessels (20%), small vessel stroke → lacunar strokes (20%), cardioembolic stroke (20-25%), blood disorders, or cryptogenic

| Blood vessels | Heart | Blood |
|---|---|---|
| • **Atheromatous (most common cause):**<br>  ○ Large vessels: internal carotid, proximal part of MCA, ACA<br>  ○ small vessels<br>• Non-atheromatous<br>  ○ Vasculitis (or any connective tissue disease)<br>  ★ Dissection of blood vessels (common in young patient "50 and less"). | **Cardioembolic,** due to:<br>• Atrial fibrillation<br>• Infective endocarditis<br>• Thrombus following an MI<br>• Valvular lesions | • haemoglobinopathies.<br>  ○ Sickle cell disease: Think of SCD when the pt is young with no conventional risk factors<br>• Coagulopathy<br>  ○ Thrombophilia[1]<br>  ○ Hypercoagulable state.<br>• Vasculitis |

1: Patient is usually a young female with recurrent history of arteriovenous infarctions eg: recurrent abortion, recurrent MI, history of DVT.
2: Its a disease of elderly, However 25% of patients are young in age (less than 55).

# Clinical presentation of Ischemic stroke

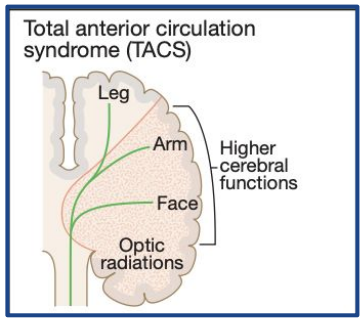



➔ **Depending on the site of occlusion:**

## Middle Cerebral Artery (MCA) occlusion
### (Total anterior circulation syndrome (TACS))

**Common cause:**

- Embolism from heart or major vessels




**Symptoms:**

Combination of:

- Hemiparesis: **Arm + face (UE) more than leg weakness (LE)**
- Hemisensory loss
- Higher cerebral dysfunction:
  - **Aphasia** if affecting the **dominant (left)** hemisphere.
  - Neglect if affecting the **non-dominant** hemisphere.
- homonymous hemianopia

**Types of aphasia**

1. **Broca's (expressive, anterior) aphasia: Damage in the left inferior frontal lobe** causes reduced speech fluency with relatively preserved comprehension. The patient makes great efforts to initiate language. Patients who recover say they knew what they wanted to say, but could not get the words out.
2. **Wernicke's (receptive, posterior) aphasia: Left temporo-parietal damage** leaves fluency of language but words are muddled. This varies from insertion of a few incorrect or non-existent words into speech to a profuse outpouring of jargon (i.e. rubbish with wholly non-existent words). Severe jargon aphasia is bizarre and often mistaken for psychotic behaviour. Patients could neither stop speaking nor understand speech.
3. **Nominal (anomic, amnestic) aphasia:** difficulty naming familiar objects (early feature in all types of aphasia)
4. **Global (central) aphasia:** combination of the expressive problems of Broca's aphasia and the loss of comprehension of Wernicke's with loss of both language production and understanding. Due to widespread damage to speech areas, the commonest aphasia after a severe left hemisphere infarct. Writing and reading are also affected.

## Anterior Cerebral Artery (ACA) occlusion

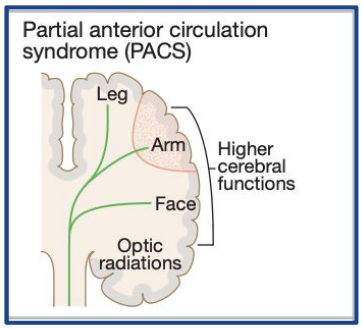
**Symptoms:**

- **Weakness LE more than UE** (Opposite to MCA)
- Emotional disturbance.

## Branch of MCA or ACA occlusion
## (Partial anterior circulation syndrome (PACS))

**Common cause:**

- Embolism from heart or major vessels




**Symptoms**

Could be:

- Isolated motor loss (e.g. leg only, arm only, face)
- Isolated higher cerebral dysfunction (e.g. aphasia, neglect)
- Mixture of higher cerebral dysfunction and motor loss (e.g. aphasia with right hemiparesis)

## Internal Carotid occlusion

**Symptoms**

- Above and ophthalmic.

# Clinical presentation of Ischemic stroke *(cont.)*

## Posterior Cerebral Artery (PCA) occlusion

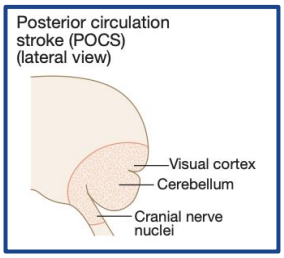
**Symptoms**

- Vision - visual field (homonymous hemianopia)
- memory

## Vertebrobasilar (posterior circulation stroke)

**Common cause:**

- Embolism from heart or thrombosis in situ leading to occlusion of vertebral, basilar, or PCA




**Symptoms**

- Cranial nerve syndrome with crossed motor
- cerebellum (cerebellar syndrome)
- altered LOC.
- homonymous hemianopia

## Midbrain

**Symptoms**

- CN III: signs of complete CN III palsy:
  - dilated pupil (if the left midbrain is affected → dilated and abducted left eye)
  - Unilateral complete ptosis (levator weakness)
  - Eye deviated **down and out** (unopposed lateral rectus and superior oblique
- **Weber's syndrome:** Ipsilateral IIIrd nerve palsy with contralateral hemiplegia is due to a unilateral infarct in the midbrain. Paralysis of upward gaze is usually present.

## Pons

**Symptoms**

- CN V → Sensory: facial numbness, anterior ⅔ of the tongue **sensory** loss. Motor: weakness of jaw movements.
- ★ **CN VI → lateral rectus palsy** (horizontal diplopia when looking into the distance, **maximal when looking to the side of the lesion**.)
- CN VII → facial weakness.

## Medulla

**Symptoms**

- CN VIII → vertigo, hearing loss.
- CN IX, X → dysphagia.
- CN XII → tongue weakness.

## Small penetrating arteries (Lacunar syndrome)

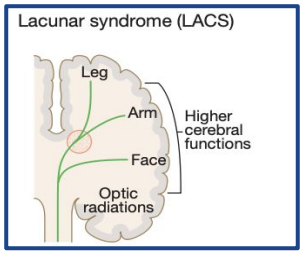
**Common cause:**

- thrombosis in situ of small penetrating arteries.

**Symptoms:**

unlike MCA and ACA ischemia, in lacunar syndrome legs, arms and face will be affected to the same degree, could be:

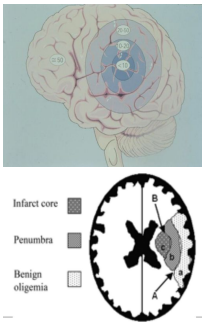
- pure motor stroke affecting two limbs
- Pure sensory stroke
- Sensory motor stroke




**Note:** no higher cerebral dysfunction (no cortical involvement, that leads to peripheral weakness when present) or hemianopia

# Ischemic stroke

## ◀ Pathophysiology




- Active and does not store energy.
- The brain is not adequately transfused, cells begin to die.
- **Core:** area of irreversible damage.
- **Penumbra:** tissue at risk (ischemic but still viable cerebral tissue)
- as seen in the picture, the frontal lobe has normal blood flow (~50cc/100g/min)
    - Reduced blood blow (20-30cc) ⟶ tissue is still viable but stops functioning (penumbra) ⟶ needs to be saved ASAP
    - A drop to 10 cc in blood flow ⟶ severe ischemia
      Note: each minute delay in restoring blood flow brain will lose 2 million neurons.

# History taking in Ischemic stroke

### Onset (Last time seen normal)

This is the single most important question to ask when taking history (when did it start?) it is taken from **the last time the patient has been seen normal,** for example, if the patient slept at 10pm and woke up at 8am with the symptoms, then we say the onset is 10pm. But if the patient woke up at 8am normal and the symptoms started 10 minutes later, then we say the time of onset is 8:10am.

Ask about:

- Symptoms (analysis of symptoms): **FAST:**
    - **F⟶ Face:** look for symmetry or sudden weakness of the face
    - **A ⟶ Arms** (or legs): weakness or numbness of one or both arms
    - **S⟶ Speech:** difficulty speaking, slurred speech
    - **T⟶ Time:** do not waste it, Go to ER ASAP, the sooner the treatment the better.
    - **Other symptoms:** Vision/balance disturbances
- **Headache (sudden and severe):** Headache and neck pain are more common with hemorrhagic stroke. However, ischemic strokes could cause headache in case of dissection (which causes ¼ of strokes in young patient) or posterior circulation stroke. A sudden severe headache may indicate subarachnoid hemorrhage.
- **Neck pain/ trauma** (In cases of dissection, usually in those with collagen disorders)
- Progression.
- Previous history of stroke or TIA.

### Past medical history

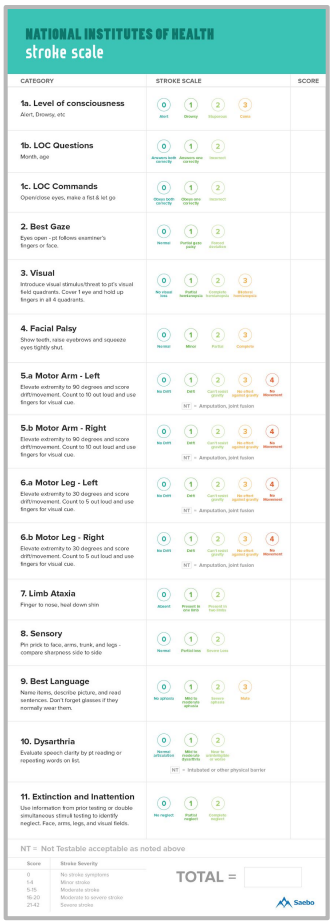
- Risk factors
- medication, particularly:
    - Oral contraceptives
    - HRT
    - Antiplatelets
    - Antithrombotics: antithrombotic/anticoagulants as they may lead to hemorrhagic shock if overused or ischemic stroke if underused (in those who suppose to take it routinely).

### HX from others

- when the patient cannot give proper history

# Physical examination in Ischemic stroke

→ keep it neurological (focused) and quick, use **National Institution of Health Stroke Scale (NIHSS)**, the higher the score, the more severe the stroke is.




## ABC (Sometimes they add D for dextrose)

## General examination

- e.g. in Pain, distressed, jaundiced, pale or febrile (suspect IE)

## Pulse

- (A.fib. → irregular)

## BP

- Will be high (in both ischemic and hemorrhagic strokes)

## Hand

## Listen for heart murmur, carotid bruits.

## Cortical infarcts

are suspect based on the presence of:

- language impairment (If dominant hemisphere is affected)
- neglect or anosognosia (If non-dominant hemisphere is affected)
- graphesthesia or stereognosis
- visual field impairment

## CN involvement and crossed motor

typical presentation of **brainstem strokes** (ipsilateral CN involvement & contralateral weakness)

## Tone

- **decreased** on side of weakness early on, **later on increased**

## Pyramidal pattern weakness (UMN)

- Upper Limbs: extensor > flexor
- Lower limbs: flexor > extensor

**Note:** Spasticity and hyperreflexia take time to develop

## Reflexes

hyperreflexia on side of weakness, with upgoing toe.

# Investigations of Ischemic Stroke

## 1 CBC

## 2 Coagulation profile

- PT.
- PTT.
- INR.

This is especially important in patients on anticoagulants

## 3 Chemistry

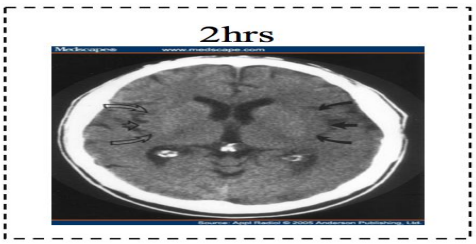
★ **Fasting glucose:** Glucose needs to be tested because patients with hypoglycemia (with history of a previous stroke) may present with similar symptoms of an actual stroke when in reality, it's the residual stroke exaggerated in the event of hypoglycemia (similar thing would happen with hyperglycemia)

- HbA1c.
- Lipids.

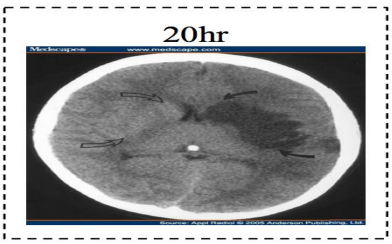
## 4 Imaging (immediate investigations)

➔ **CT scan:**

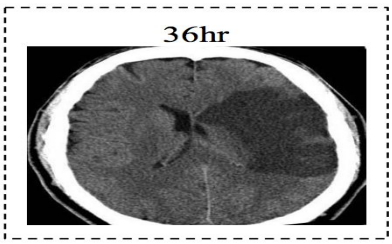
- CT scanning is the **mainstay of emergency stroke imaging**. It allows the rapid identification of intracerebral bleeding and stroke 'mimics' (i.e. pathologies other than stroke that have similar presentations), such as tumors. it is done to exclude hemorrhage and early infarct
- **non-contrast CT is the only way to differentiate between ischemic and hemorrhagic strokes**
- IF CT shows hemorrhage, give no drug that could interfere with clotting.




In the 2Hrs picture, early hypo-density can be seen on the left hemisphere (right side on the image) with fullness of the sulci, these are early signs of ischemia on CT




in 20hrs → infarct is seen




in 36hrs →the whole area is infarcted

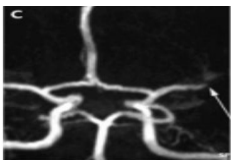
➔ **MRI:**

- MRI is **better overall, if immediately available**
- MRI is used when there is diagnostic uncertainty or delayed presentation, and when more information on brain structure and function is required.
- Contraindications to MRI include cardiac pacemakers, clips and claustrophobia.
- MRI is highly sensitive, it can detect ischemia within the first half hour, you can't do that on CT, However it takes so much time and **cannot be used in emergency**.
- diffusion-weighted MRI images shows ischemia within minutes, but is as accurate as CT for detection of haemorrhage.

# Investigations of Ischemic Stroke (cont.)

## 5 Vascular imaging (later investigations)

MRA



- **Carotid U/S:** the least invasive, shows extracranial lesions. If ultrasound suggests carotid stenosis, normotensive patients with TIA or stroke in the anterior circulation should have vascular imaging.
- **CTA:** shows everything, but is invasive
- **MRA: MRA and CTA are** valuable in anterior circulation TIAs to confirm surgically accessible arterial stenoses, mainly internal carotid stenosis.
- Cerebral Angio (conventional angio)
- Carotid doppler and duplex screening

## 6 Cardiac workup

- ECG: quick and convenient, it is especially important to be used in emergencies (to detect Afib)
- Echo (TTE or TEE): shows the structure, ejection fraction, valves, and cardiac muscle
- Holter, to record heart rhythm over 24-48hrs, used for impersistent rhythm issue.

## 7 In specific cases

- Hb
- electrophoresis.
- Hypercoagulable work up.
- CTD screen.
- HIV and syphilis

# Management of ischemic stroke

## ◀ Acute Stroke Management

Code stroke

### 1 ABC

| | |
|---|---|
| **Airway** | Perform bedside screen and keep patient nil by mouth if swallowing unsafe or aspiration occurs |
| **Breathing** | Check respiratory rate and give oxygen by mask if saturation **< 95%** |
| **Circulation** | Check peripheral perfusion, pulse and blood pressure, and treat abnormalities with fluid replacement, antiarrhythmics and inotropic drugs as appropriate |

# ◀ Acute Stroke Management (cont.)

## 2 Reperfusion

### 01 Intravenous thrombolysis (IV t-PA); *tissue plasminogen activator.*

- **Effective up to 4.5 hours from onset.**
- Sooner the better (time= brain).
- 30% chance of improvement 1/3, 1 out of 8 complete recovery.
- Risk of bleeding (ICH ) = 6%

➔ **Exclusion criteria (historical and laboratory)** Dr: no need to memorize them just know the concept

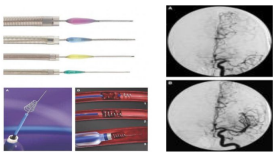
1. Intracerebral hemorrhage
2. platelet <100 000
3. recent MI
4. INR >1.7 if on warfarin
5. prior ICH
6. History suggestive of SAH
7. stroke in the past 3 months.
8. SBP >186 or DBP >110, Hg<100?
9. GI or GU hemorrhage in past 3 weeks
10. major surgery 14d
11. coming later than 4.5 hours of onset
12. Lumbar puncture or an arterial puncture at a non-compressible site, within 7 days
13. PT >15
14. Serum glucose <2.8 mmol/L or >21.2 mmol/L

➔ **Exclusion criteria (clinical)**

- Rapidly improving stroke syndrome
- Minor and isolated neurological signs
- Seizure at the onset of stroke if the residual impairments are due to postictal phenomena
- Symptoms suggestive of subarachnoid haemorrhage, even if the CT is normal
- post-MI pericarditis
- Pregnancy or lactation
- Active bleeding or acute trauma (fracture)

- Aspirin or other antiplatelets (started within 48 hours reduces the risk of early recurrent ischemic stroke without a major risk Hge and improves long-term outcome)
- In the absence of contraindications, aspirin (300 mg daily) should be started immediately after an ischaemic stroke **if the patient is not a candidate for thrombolysis.** If the patient has already received tPA, withhold aspirin for at least 24 hours.
  - In case of atrial fibrillation → add warfarin.
  - In case of significant carotid stenosis → surgery
  - In case of vasculitis → steroids

# Management of ischemic stroke *(cont.)*



## 02 Intra-arterial thrombolytic & Mechanical thrombectomy

- Invasive
- Only in case of blockage in **large vessels** e.g. MCA, ACA, Internal carotid or basillar
- you could do for **up to 6 hours.**
- very recently, patients have been treated beyond the therapeutic window using artificial intelligence, which help see core and penumbra → allowed for up to 24hrs intervention window.

## 03 Internal carotid endarterectomy

- Surgery is usually recommended in TIA or stroke patients with **internal carotid artery stenosis >70%.**
- Successful surgery reduces the risk of further TIA/stroke by around 75%.
- Endarterectomy has a mortality around 3%, and a similar risk of stroke.
- Percutaneous transluminal angioplasty (stenting) is an alternative.
- The value of surgery for asymptomatic carotid stenosis is debatable.

.

## 3 Prevent progression and complications

→ **Stroke unit:** Following acute intervention, or if patient is not candidate.

**BP and glycemic control.** → **NPO, avoid aspiration.** → **Dx and Rx temp** → **PT, OT and early rehab.** → **DVT prophylaxis**

**Blood pressure:**

- Unless there is heart or renal failure, evidence of hypertensive encephalopathy or aortic dissection, **do not lower blood pressure abruptly in first week as it may reduce cerebral perfusion.** Blood pressure often returns towards patient's normal level within days
- Control BP before thrombolysis bc of the risk of bleeding

**Blood glucose:**

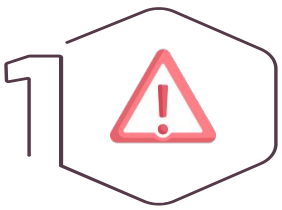
- Check blood glucose and treat when levels are ≥11.1 mmol/L (200 mg/dL) (by insulin infusion or glucose/potassium/insulin (GKI)
- **Monitor closely to avoid hypoglycemia**

# Management of ischemic stroke *(cont.)*

## ◀ Other components of Acute Stroke Management

| | |
|---|---|
| **Hydration** | If signs of dehydration, give fluids parenterally or by nasogastric tube |
| **Nutrition** | • Assess nutritional status and provide supplements if needed.<br>• If dysphagia persists for >48 hrs, start feeding via nasogastric |
| **Medication** | If dysphagic, consider other routes for essential medications |
| **Temperature** | • If pyrexic, investigate and treat underlying cause<br>• Control with antipyretics, **as raised brain temperature may increase infarct volume** |
| **Pressure areas** | • Reduce risk of skin breakdown:<br>  ○ Treat infection<br>  ○ Maintain nutrition<br>  ○ Provide pressure-relieving mattress<br>  ○ Turn immobile patients regularly |
| **Incontinence** | • Check for constipation and urinary retention; treat these appropriately<br>• Avoid urinary catheterisation unless patient is in acute urinary retention or incontinence is threatening pressure areas |
| **Mobilization** | Avoid bed rest |

## ◀ Long Term Stroke Management

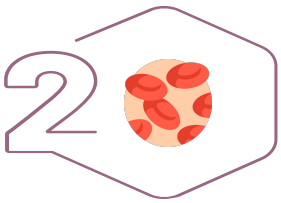


**Long term management of Risk factors (secondary prevention)**

- **HTN:** Recognition and good control of high blood pressure is the major factor in primary and secondary prevention of both ischemic and haemorrhagic strokes.
  - Transient hypertension, often seen in the first 24-48 hours following stroke, usually does not require treatment provided (let BP autoregulate) given diastolic pressure does not rise >100 mmHg, because high BP helps the cerebral circulation. unless
    - Patient is candidate for thrombolysis
    - patient has other risk factors that necessitate BP control
  - Sustained severe hypertension needs treatment
  - BP should be lowered slowly to avoid any sudden fall in perfusion.
- DM
- lipid
  - Patients with ischaemic events should be put on long-term statin therapy to lower cholesterol.
- smoking
- A-fib.
- Exercise

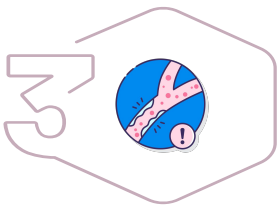
# Management of ischemic stroke *(cont.)*

## ◀ Long Term Stroke Management *(cont.)*



**Anti-platelet:** for atherosclerosis

- **Long-term soluble aspirin** (75 mg daily) reduces substantially the incidence of further infarction following thromboembolic TIA or stroke.
- Clopidogrel and dipyridamole are also used
- Combined aspirin 75 mg daily and clopidogrel 75 mg daily provide optimal prophylaxis against further thrombo embolic stroke or TIA.
  - Dipyridamole 200 mg twice daily is used if clopidogrel is contraindicated.

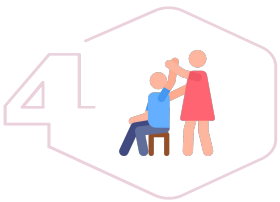


**Anticoagulant**

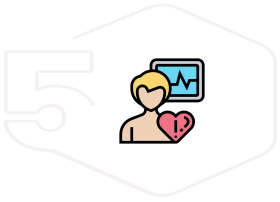
- Heparin and warfarin should be given when there is:
  - atrial fibrillation (give warfarin to achieve INR of 2-3)
  - other paroxysmal dysrhythmias
  - cardiac valve lesions (uninfected)
  - cardiomyopathies.
  - hypercoagulability
- Brain haemorrhage must be excluded by CT/MRI. Patients must be aware of the small risk of cerebral (and other) haemorrhage.
- Anticoagulants are potentially dangerous i**n the two weeks following infarction** because of the risk of provoking cerebral haemorrhage
- Antithrombins are now being used.

**Table 22.15 Anticoagulants and stroke prevention**

| Indication | Comment |
|---|---|
| Valvular heart disease | Heparin/warfarin of benefit in chronic rheumatic heart disease, particularly mitral stenosis |
| Recent MI | |
| Intracardiac thrombus | Heparin/warfarin if there is evidence of intracardiac thrombus |
| Atrial fibrillation | Anticoagulants long term reduce stroke incidence in atrial fibrillation |
| Acute internal carotid artery thrombus<br>Acute basilar artery thrombus<br>Internal carotid artery dissection<br>Extracranial vertebral artery dissection | Anticoagulants reserved for imaging-confirmed cases of arterial thrombosis or dissection. They have not been shown to be beneficial in stroke prevention after thromboembolism from carotid or vertebrobasilar sources |
| Prothrombic states, e.g. protein C deficiency | Anticoagulation, in consultation with haematologist |
| Recurrent TIAs or stroke on full antiplatelet therapy | If no remediable cause, a trial of anticoagulants may be justified |
| Cerebral venous thrombosis including sinus thrombosis | Benefits of anticoagulation outweigh risks of haemorrhage |



**Rehabilitation.**



**Treat the underlying cause**

- Carotid SX.
  - Surgery & stenting
- Cardio embolic.
  - Anticoagulants
- Hypercoagulable
  - rx with Coumadin.

# Transient Ischemic attack

## Definition

brief and **temporary reduction** in blood flow to a focal region within the brain **with no evidence of infarction on imaging.**

- TIA is a stroke that did not finish YET
- The term TIA traditionally also includes patients with **amaurosis fugax**[1], usually due to a vascular occlusion in the retina.

- **Duration:** most TIA's **last 5-20 mins**
  - if more than 1 hour usually infarction on MRI.
- Infarction is usually averted by autoregulation.
- Rarely, tumours and subdural haematomas cause episodes indistinguishable from thromboembolic TIAs.
- **DDX:** Seizure, migraine, syncope, labyrinthine LDH

## ◀ Pathophysiology

- TIAs are usually the result of **microemboli**, but different mechanisms produce similar clinical events. For example:
  - TIAs may be caused by a fall in cerebral perfusion (e.g. a cardiac dysrhythmia, postural hypotension or decreased flow through atheromatous arteries).
- Principal sources of emboli to the brain are:
  - cardiac thrombus → often results from atrial fibrillation or myocardial infarction.
  - atheromatous plaques/thrombus within the aortic arch
  - carotid and vertebral systems.
  - Cardiac valve disease, e.g. calcific material

## ◀ Features

| **Anterior circulation** ***carotid system*** | **Posterior circulation** ***Vertebrobasilar system*** |
|---|---|
| Amaurosis fugax | Diplopia, vertigo, vomiting |
| Aphasia | Choking and dysarthria |
| Hemiparesis | Ataxia |
| Hemisensory loss | Hemisensory loss |
| Hemianopic visual loss | Hemianopic visual loss or Bilateral visual loss |
| | Loss of consciousness (rare) |
| | Transient global amnesia (possibly) |

1: is a sudden transient loss of vision in one eye. When due to the passage of emboli through the retinal arteries, arterial obstruction is sometimes visible through an ophthalmoscope during an attack. A TIA causing an episode of amaurosis fugax is often the first clinical evidence of internal carotid artery stenosis – and forerunner of a hemiparesis.

# Transient Ischemic attack *(cont.)*

## ◀ Clinical findings

Diagnosis of TIA is often based solely upon its description. It is unusual to witness an attack as they are so brief. **Consciousness is usually preserved** in TIA.

**There may be clinical evidence of a source of embolus, e.g.:**

- Carotid arterial bruit (stenosis)
- Atrial fibrillation or other dysrhythmia
- Valvular heart disease/endocarditis
- Recent myocardial infarction.

**An underlying condition may be evident:**

- Atheroma.
- Hypertension.
- Postural hypotension.
- Bradycardia or low cardiac output.
- Diabetes mellitus.
- Rarely, arteritis, polycythaemia, neurosyphilis, HIV.
- Antiphospholipid syndrome.

## ◀ Approach to TIA

1. Needs **urgent assessment** (ER)
2. **Rule out other causes** of transient events (by HX and PE) TIA rarely march across body
3. **Work up:** (labs, CT scan or MRI)
   - Vascular image of carotid CTA, MRA, US.
   - Cardiac work up: (EKG، echo, +/- holter).
4. **Start stroke prevention measures** (like ischemic stroke) ASA, control HTN, DM and lipids, stop smoking and exercise.

## ◀ Prognosis of TIA

- Up to ⅓ will have a stroke (usually within 48 hours).
- The **ABCD2** score can help to stratify stroke risk in the first 2 days:
  - <4: minimal risk
  - >6: high risk for a stroke within 7 days of a TIA.
- If patients are considered to have had a high risk TIA, i.e.
- ABCD2 score >4, or have had two recent TIAs, especially within the same vascular territory, then the patient should ideally be admitted for *urgent* investigation and commencement of secondary prevention.

| | |
|---|---|
| **A**ge >60 years | 1 point |
| **B**P >140 mmHg systolic and/or diastolic >90 mmHg | 1 point |
| **C**linical features | |
| – unilateral weakness | 2 |
| – isolated speech disturbance | 1 |
| – other | 0 |
| **D**uration of symptoms (minutes) | |
| – >60 | 2 |
| – 10–59 | 1 |
| – <10 | 0 |
| **D**iabetes | |
| – present | 1 |
| – absent | 0 |

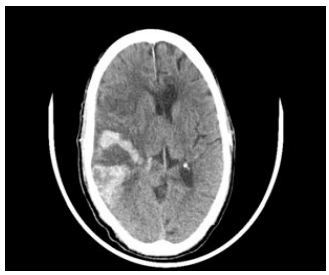
## ◀ Take home messages

- Stroke can be ischemic or hemorrhagic
- Every acute stroke patient should be viewed as an eminently treatable neuroemergency.
- Time window for effective therapy in stroke is brief ( Time is brain)
- TIA Is a stroke that did not finish YET
- Any one present with sudden severe Headache should be presumed to be SAH until proven otherwise .

# Transformation of ischemic stroke

## ◀ Hemorrhagic Transformation HI 1/2 – PH 1/2

- **> 50% of ischemic stroke have some hemorrhage**
- 0.6% - 3% → untreated patients
- 6% in treated patients
- **Risk Factors:**
  - **Older age**
  - **Larger stroke size**
  - **Anticoagulant use**
  - Cardioembolic stroke etiology
  - Fever
  - Hyperglycemia
  - Low serum cholesterol
  - Acutely elevated systolic blood pressure.
  - **Thrombolytic therapy/recanalization:** Intravenous thrombolysis with recombinant tissue plasminogen activator (rt-PA) **increases the risk of haemorrhagic transformation** of the cerebral infarct with potentially fatal results.



Ischemia (dark center) surrounded by haemorrhage

## ◀ Prognosis of Hemorrhagic stroke

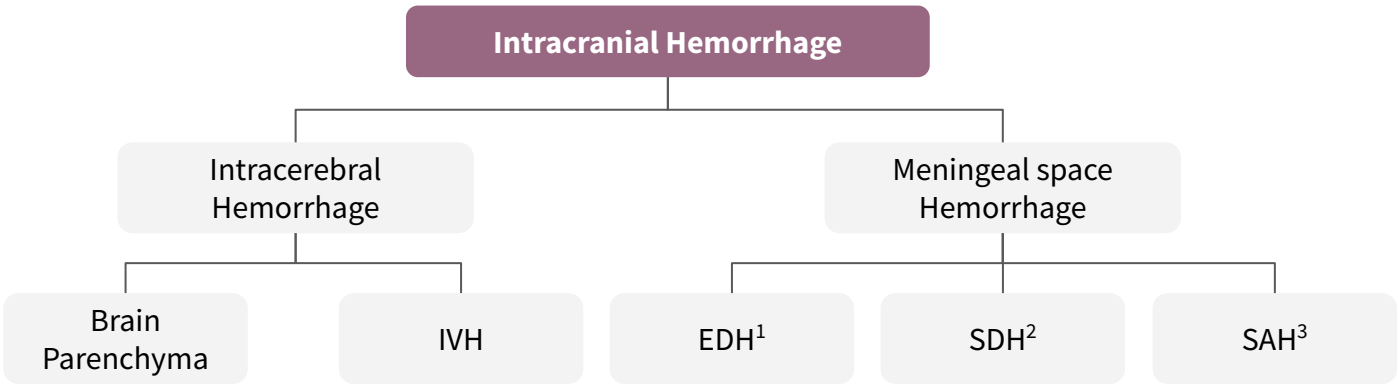
- **Why does hemorrhagic stroke carries worse prognosis?** Because the blood can compress the neurons, blood vessels. Also, it will cause edema and herniation which will compress the small blood vessels leading to ischemia

**Factors affecting prognosis:**

1. **Volume of haemorrhage**: The higher the volume/size the worse the prognosis is
2. **Patient's Age**
3. Extension of the hemorrhage into the ventricles
4. Low GCS score at presentation (e.g. Confusion, sleepiness)

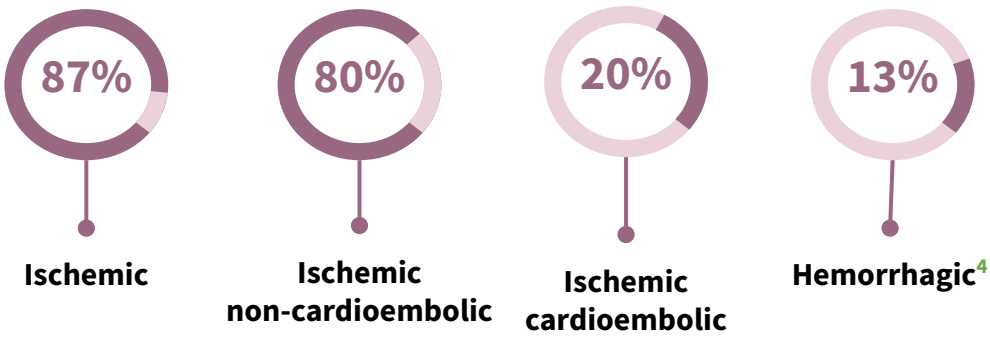
An area for your notes

# Introduction to haemorrhagic stroke



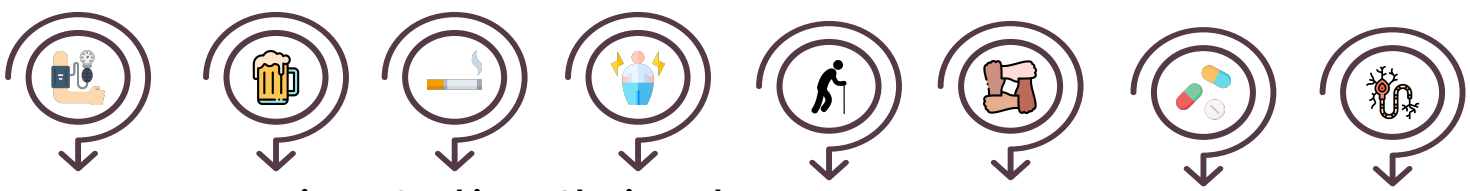

## ◀ Epidemiology

- **Asian countries** have a higher incidence of intracerebral hemorrhage:
  - Noted in China, Japan, and other asian population.
  - Possibly due to environmental factors (eg, a diet rich in fish oils) and/or genetic factors.
- **US distribution of types of strokes:** 800,000 total strokes annually.




- Incidence of intracerebral hemorrhage increases in individuals > 55 years and doubles with each decade until age 80.
  - Due to many risk factors such as uncontrolled or chronic HTN.

## ◀ Risk factors



**Hypertension**[5]

**Excessive alcohol use**[6]**.**

**Smoking** (Damage Blood Vessels)

**Obesity and physical inactivity.**

**Older age** ↑ incidence of aneurysm HTN.

**Ethnicity / race.**

**Medications** antiplatelets or anticoagulant

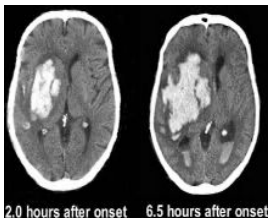
**Sympathomimetics**[7]

1: Epidural Hemorrhage → Hemorrhage from the **bridging arteries → usually following trauma and involving skull fracture.**
2:.Subdural hemorrhage → **Bridging veins** (it can happen from minor trauma)
3: Subarachnoid hemorrhage → rupture aneurysm.
4: Associated with worse prognosis.
5: Most important & Most common modifiable risk factor, controlling & preventing high BP is the key to reduce the incidence & prevalence of ICH
6: Can also lead to HTN and impaired coagulation/platelet function
7: Cocaine and amphetamines can cause intracerebral hemorrhage especially in the younger population

# Introduction to haemorrhagic stroke

## ◀ Mortality and disability

**Overall**, 40-44% mortality rate at 1 month and 54% at one year.




Only 12-40% are functionally independent long term.

In 2010, 62.8 million years lost DALYs[1] with ICH compared to 39.4 million in ischemic stroke.

Annually, more than 20,000 individuals in the United States die of intracerebral hemorrhage.

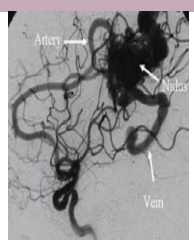
Pontine or other brainstem intracerebral hemorrhage has a mortality rate of 75% at 24 hours.

- In general, If you have a hemorrhage in the brain stem it's too bad with a high mortality rate.

## ◀ Etiology

| Hypertensive ICH | Other causes |
|---|---|
| - Essential<br>  - Rupture of microaneurysms[2] (Charcot–Bouchard aneurysms → 0.8-1 mm in diameter).<br>  - degeneration of small deep penetrating arteries[2]<br>- Eclampsia | - Cerebral venous thrombosis (CVT):in young females due to **OCP** use<br>- Intracranial neoplasm<br>- Moya Moya<br>- Vasculitis → ischemia or bleeding |

### Non-hypertensive ICH




- **Vascular Malformation:**
  - Arteriovenous malformation (AVM): common in young patients (pic), may often cause epilepsy.
  - Aneurysm
  - Cavernous hemangioma (cavernomas)
  - Bleeding disorders
  - venous and cavernous angiomas
- anticoagulant: more risky than antiplatelets
- Amyloid angiopathy: Happens in elderly, especially Alzheimer patients, Amyloid deposition in blood vessel → very fragile vessels. Patient will bleed every now and then and there is no Rx. it's usually cortical & subcortical areas ( lobar area) unlike Hypertensive ICH that is in deep structures (discussed in page 26)
- Trauma: could cause intraparenchymal hemorrhage, but most commonly causes subdural hemorrhage or epidural hemorrhage
- Tumor.
- **Drug abuse:** not uncommon, When young pts come with stroke you do drag screen
  - amphetamine: It cause blood vessel spasm, damage the vessels, can lead to both ischemic and hemorrhagic stroke
  - cocaine
  - PPA.

1:Disability Adjusted Life Years: The sum of years of potential life lost due to premature. mortality and the years of productive life lost due to disability.
2: these are the principal pathology. Such haemorrhage is usually massive, often fatal, and occurs in chronic hypertension and at well-defined sites – basal ganglia, pons, cerebellum and subcortical white matter.

# Intracerebral Hemorrhage (ICH)

## ◀ Pathophysiology

**Primary immediate effect[1]**

- Hemorrhage growth.
- Increase ICP

**Secondary effect**

- Downstream effect.
- Edema.
- Ischemia.

- **Site:**

1. **Basal ganglia (40-50%)**
2. **Lobar regions (20-50%)**
3. **Thalamus (10-15%)**
4. **Pons (5-12%)**
5. **Cerebellum (5-10%)**
6. **Other brainstem sites**

## ◀ Clinical presentation

- **Alteration in level of consciousness** (approximately 50%).
- **Nausea** and **vomiting[2]** (approximately 40-50%).
- **Headache** (approximately 40%)
  - At the bedside, there is no entirely reliable way of distinguishing between haemorrhage and thromboembolic infarction. Both produce stroke. Intracerebral haemorrhage tends to be dramatic with **severe headache**. It is more likely to lead to **coma** than thromboembolism.
- **Seizures** (approximately 6-7%).

### Focal neurological deficits:

➔ **depending on the location:**

| | |
|---|---|
| **Putamen** | - Contralateral hemiparesis, contralateral sensory loss, contralateral conjugate gaze paresis, homonymous hemianopia, aphasia, neglect, or apraxia. |
| **Thalamus** | - Contralateral sensory loss, contralateral hemiparesis, gaze paresis, homonymous hemianopia, miosis, aphasia, or confusion.<br>Any focal sensory loss, think of the thalamus |
| **Lobar** | - Contralateral hemiparesis or sensory loss, contralateral conjugate gaze paresis, homonymous hemianopia, abulia, aphasia (if affecting the left side), neglect (if affecting the right side), or apraxia. |
| **Caudate nucleus** | - Contralateral hemiparesis, contralateral conjugate gaze paresis, or confusion. |
| **Cerebellum** | - Ataxia on the same side |

1: ICH have bad prognosis because of hemorrhagic growth causes increase volume inside skull thus increasing ICP, damaging the brain and surrounding neurons, and compressing the surrounding vessels leading to downstream effect and ischemia.
2: due to increase ICP

# Intracerebral Hemorrhage (ICH)

## ◀ Investigations

### Laboratory studies

- CBC: look for thrombocytopenia
- Coagulogram: look for Increase PTT, INR, prothrombin time, any bleeding tendency or any disorder in factor VII or VIII

### Imaging

| CT brain (without contrast) (Essential to differentiate ischemic from hemorrhagic[1]) | CT Vessel |
|---|---|
| • Demonstrates acute hemorrhage as **hyperdense**[2] signal intensity.<br>• **Multifocal hemorrhages** at the frontal, temporal, or occipital poles suggest a **traumatic etiology**.<br>• Hematoma volume can be approximated by (AxBxC)/2: choose the CT cut that is showing the largest size of Hematoma, Calculate diameter (A,B), and for the depth (C). multiply the number of cut showing hematoma by 5mm (the depth of one CT cut image)<br>• Iodinated contrast may be injected to increase screening yield for underlying tumor[3] or vascular malformation.<br>• Brain haemorrhage is seen on CT imaging immediately (cf. infarction as intraparenchymal, intraventricular or subarachnoid blood | • CT angiography permits screening of large and medium-sized vessels for AVMs, vasculitis (String of beads appearance), and other arteriopathies. |

### The workup

- **CT head** → no contrast.
- **CTA head/neck** → suspect vascular etiology.
  - Careful interpreting noncon CT head after CTA or other dye study.
- **MRI brain** (shows the cause of the hemorrhage): with gado if looking for neoplasm, Routine MRI may not identify an acute small haemorrhage correctly in the first few hours but **MRI diffusion-weighted** (MRI-DW) is as good as CT.
- **MRA/MRV** → if allergic to CT dye or if you're looking at venous outflow.
  - MRA for AVM and aneurysms
  - MRV for cerebral venous thrombosis
- Cerebral angiography (avoid it as it carries risk of stroke).

1: ischemic stroke take hours to days to show in CT but hemorrhagic stroke will immediately show.
2: because blood contain metal
3: you could do MRI

# management ICH

## ◀ Medical

### Control Blood Pressure.

➔ **Guidelines:**

1. **Reduction of SBP to 140 is safe:** 120-140: why not less? to preserve blood perfusion to small vessel and preventing ischemia resulted from small blood vessel compression. in the area around the hemorrhage. The risk of recurrence after both ischaemic and haemorrhagic strokes can be reduced by blood pressure reduction, even for those with relatively normal blood pressures.
2. Anderson/Qureshi studies → Interact 2 and ATACH 2
   - **120-140 mmHg**
3. Use **labetalol** and/or **nicardipine** drip to titrate blood pressure.

➔ Between 15-23% of patients → hematoma expansion in first few hours.

◆ watch them very closely, if there is any deterioration you repeat CT, if there is an increase in the size of hemorrhage you should call the neurosurgeon to consider surgery.

➔ **Evidence-based practice nursing care:**

01 **Watch for neuro decline** → do immediate CT scan

02 **Type and cross with your labs** for transfusion

03 **head of bed > 30° elevation,** to decrease perfusion → less haemorrhage.

04 **Head midline**

05 **Prevent vagal maneuvers** (increases BP → more hemorrhage)

06 **Control SBP** (120-140)

07 **Treat hyperglycemia**[1]

08 **Treat hyperthermia**[1]

09 **Seizure prophylaxis**

10 **DVT prophylaxis** only after 48hrs & no haemorrhage[2]

➔ **Typically, do not make patients DNR (do-not-resuscitate) within the first 48 hours**

1: Associated with worse outcome

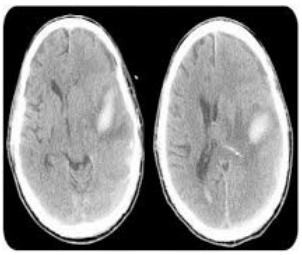
2: Because these Pts not moving a lot. In the first 48h you repeat CT scan, if there is no hemorrhage → start pharmacological DVT prophylaxis, but remember that Antiplatelet drugs and, of course, anticoagulants are contraindicated in the initial management of haemorrhagic stroke.

# management ICH *(cont.)*

## ◀ Medical *(Cont.)*

**Cerebral edema: sodium (hypoosmolar hyponatremia) and CO2**

- Use the ventilator to manage CO2: High CO2 levels → increased perfusion → hemorrhage, Put the patient on hyperventilation to lower it.
- Get the Sodium levels up to 145- 155.
- Mannitol 3% or even 23.4% (requires central line), Given to prevent brain herniation that's caused by the cerebral edema.
  - Give them hypertonic saline e.g. 3% sodium or mannitol to lower edema



edema is seen as hypodense area around the haemorrhage. midline shift is also noticed

➔ **Other medical measures:**
Anticoagulation should be rapidly reversed where possible (for patients on warfarin give intravenous vitamin K and clotting factor concentrates).

## ◀ Surgical

➔ Majority of ICH in deep location making the surgery not easy and associated with worse outcome. if Hemorrhage is lobular and superficial and the pt's condition worsening surgery should be considered

➔ Surgery **never works** except in only two scenarios:

- **Cerebellar hemorrhage** (it's close to brain stem and any edema or expansion of brain stem could cause death), if the hemorrhage is small we will observe the patient for any deterioration which we will do evacuation of cerebellar hematoma (has a good outcome as it is easy, accessible and superficial)
- **Lobar superficial hemorrhage** (if it's big then evacuate, if small observe the patient for deterioration to take him to the operating room)

**EVD (external ventricular drains).**

- CLEAR III trial → **no outcome benefit** with vent use of tPA
- Urgent neurosurgical clot evacuation is occasionally necessary when there is deepening coma and coning (particularly in cerebellar haemorrhage).

**Craniotomy depends on:**

- Etiology
- AC/APT status
- Timing
- Location:
  - **STICH II** → no overall favorable outcome.
  - **MISTIE II** → MIS techniques.
  - **MISTIE III** → underway.
- Cerebellar ICH:
  - Features:
    - Headache, often followed by stupor/coma and signs of cerebellar/brainstem origin (e.g. nystagmus, ocular palsies).
    - Gaze deviates towards the haemorrhage.
    - Skew deviation may develop.
    - Sometimes causes acute hydrocephalus, a potential surgical emergency.
  - In cerebellar ICH, it's superficial, easy surgery and associate with very good outcome.

# Subarachnoid haemorrhage (SAH)

## ◀ Etiology

1. **aneurysm rupture** 70%
2. **Arteriovenous malformation (AVM)** 10%
3. **No arterial lesion found** 15%
4. **Other rare causes** <5%

- SAH are usually due to **berry aneurysm rupture** → saccular or 'berry' aneurysms arising from the bifurcation of cerebral arteries, particularly in the region of the circle of Willis. The most common sites are:
  - **anterior communicating artery** (30%)
  - **posterior communicating artery** (25%)
  - **middle cerebral artery (20%).**
- Can be perimesencephalic SAH.
- Early treatment is crucial.

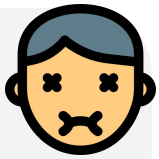
## ◀ Differential diagnosis of SAH

- **migraine**: sometimes difficult to differentiate from SAH – a short time to maximal headache intensity and the presence of neck stiffness usually indicate SAH.
- The syndrome of reversible cerebral vasoconstriction (**Call–Fleming syndrome**).
- **Acute bacterial meningitis** occasionally causes a very abrupt headache
- **Cervical arterial dissection** can present with a sudden headache.

## ◀ Clinical features

### Sudden acute severe Headache

- SAH typically presents with a sudden, severe, '**thunderclap**' headache (often occipital), which lasts for hours or even days.
- SAH is a ddx of any sudden headache
- could be followed by:
  - Vomiting
  - death



### Other features

- raised blood pressure
- neck stiffness or pain
- straining
- sexual excitement.
- Papilloedema
- There may be loss of consciousness at the onset (SAH should be considered if a patient is found comatose)

# Subarachnoid haemorrhage *(Cont.)*

## ◀ Clinical features *(cont.)*

**Physical examination**

- distress & Irritability
- Photophobia.
- **Positive kernig's sign** (neck stiffness due to subarachnoid blood), but this may take some hours to develop.
- Focal hemisphere signs, such as hemiparesis or aphasia, may be present at onset if there is an associated intracerebral haematoma.

## ◀ Investigations

**CT Brain scan**

- The diagnosis of SAH can be made by **CT** → a negative result does not completely exclude SAH, since small amounts of blood in the subarachnoid space cannot be detected by CT, CT can miss 10% of cases, **if CT is negative and the presentation is suggestive of subarachnoid hemorrhage then you do spinal tap** to look for blood in the CSF.

**Lumbar puncture**

- should be performed 12 hours after symptom onset if possible, to allow detection of xanthochromia → yellow CSF.

**CT Angiogram**

- If either CT or LP is positive, cerebral angiography is required to determine the optimal approach to prevent recurrent bleeding.

## ◀ Treatment

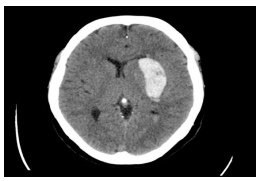
- **Surgery: Coil / Clip.**
  - Insertion of platinum coils into an aneurysm (via an endovascular procedure) or surgical clipping of the aneurysm neck reduces the risk of both early and late recurrence. Coiling is associated with fewer perioperative complications and better outcomes than surgery; where feasible, it is now the **procedure of first choice.**
- **Medication:**
  - NIMOTOP/ NIMODIPINE: all the patient should be given, because subarachnoid hemorrhage is associated with vasospasm that usually happen between day 3 to day 14 → multiple strokes and death. CCB prevent and treat the vasospasm, given for 21 days.
  - If patient develops vasospasm while on Nimodipine you should do angioplasty of the vasospasm
- Strict BP control.
- Check Sodium Levels → **Treat the central Hyponatremia:**
  - best managed by fluid restriction & 3% NaCl
- Check Urine output.
- Treat the obstructive hydrocephalus (a complication of SAH) → may require drainage via a shunt

# ◀ Hypertenstive haemorrhage

➔ **In order from most to least common site:**

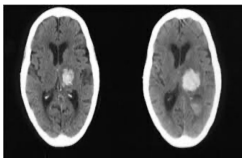
## 1 Putamen haemorrhage (Most common)

- hemorrhage is hyperdense in CT scan.
- **A typical location for hypertensive hemorrhage**.
- Hemorrhage is compressing the ventricles (Mass effect)
- Symptoms will be weakness in the contralateral side



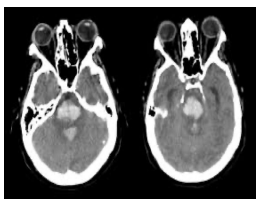
## 2 Thalamic haemorrhage

- picture shows a left thalamic haemorrhage.
- Patient presents with numbness and decreased sensation on the right side



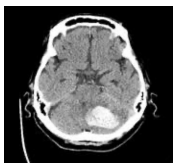
## 3 Pontine

- hemorrhage in the **bilateral pontine area.**
- very poor prognosis (brainstem hemorrhage) → to the grave (really bad).



## 4 Cerebellar haemorrhage

- Easy surgery, good prognosis
- left cerebellar hemorrhage, patient will present with ataxia on the left side.

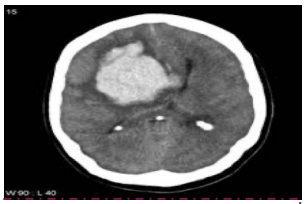


## 5 Lobar haemorrhage

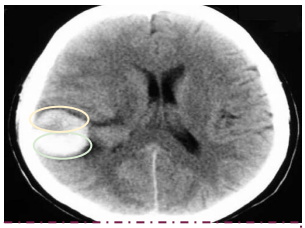
It is the 5th most common cause of hypertinstive haemorrhage, but it could also be caused by:

- **Tumor with ICH:**
  - lobar hemorrhage.
  - Hemorrhage in a tumor:
  - Very hyperdense area (haemorrhage) next to little hyper-dense area (tumor).
  - We have to do an MRI to pick up tumors and know which type it is, whether a metastasis or primary.

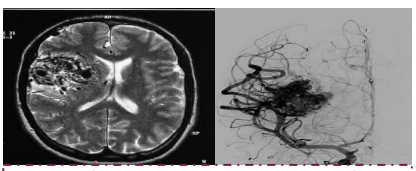
- **AVM:**
  - The artery will drain directly to the vein without passing into venuoles and capillaries which will cause dilatation and hemorrhage, Treatment is by embolization of artery
  - MRI showing **collection of worms** → typical appearance of AVM



lobar hemorrhage



Tumor with ICH

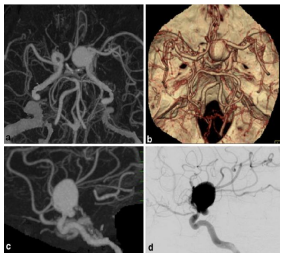


Arteriovenous malformation (AVM)

# Imaging interpretation *(cont.)*

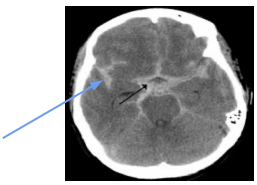
## ◀ Other

### 1 Aneurysm



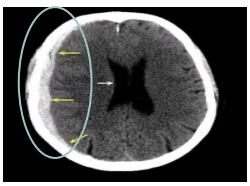
- It is going to rupture and patient is going to die, when patient comes with SAH it is only leaking. how do we treat it?
- They should undergo CT angiogram of the brain to coil it. it is done through a catheter through the groin and then they inject coil to close the aneurysm. Coiling is now the first line treatment.
- The picture shows the MCA with large aneurysm. To treat, insert a catheter in the groin that goes all the way to MCA → inject mesh to close the aneurysm

### 2 Subarachnoid haemorrhage (SAH)



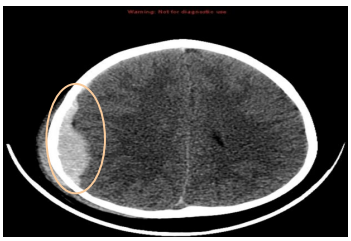
- Hemorrhage in sylvian fissure.
- If you miss it, it will rupture and it has a high mortality and morbidity.

### 3 Subdural haemorrhage (SDH)



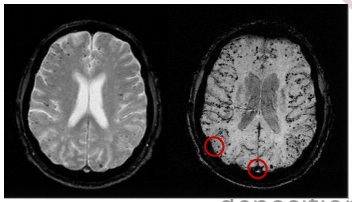
- Look like **a crescent**.
- It can happen in minor trauma, Treatment is by evacuation
- It is bleeding in meningeal veins or bridging veins,

### 4 Epidural hemorrhage (EDH)



- look like **lens**, swelling in subcutaneous. usually due to skull fracture. it's arterial hemorrhage.
- It is usually associated with trauma or fracture of the skull. It is very serious you need to watch the patient very closely, surgeons have to interfere very quickly because it's an arterial hemorrhage and they can compress and expand very quickly.

### 5 Amyloid Angiopathy



- Left (T2w): Show acute and subacute hemorrhage.
- Right (Swan or hemosiderin sequence): very sensitive to hemorrhage, will show old and new hemorrhage (Black dots) in lobar area → All these small dots are hemorrhage.
- Hypertensive hemorrhage and Amyloid Angiopathy: which is of amyloid protein on the vessel wall making it very fragile.
- To differentiate between hypertensive and amyloid, we do a sequence MRI:
  - In **amyloid** you will see **multiple old and new hemorrhages** this is indicative of amyloid (It means that the patient has been bleeding every now and then).
- High recurrence rate. no definitive treatment, may be associated with Alzheimer.

deposition

# Summary

Ischemic stroke is an acute onset of neurological deficit caused by impaired blood flow to the CNS.
Stroke is identified as ether neurological deficit that persist after 24hrs or infract on CT or MRI .

| | |
|---|---|
| **Mechanism of ischemic stroke** | • Due to blockage from :<br>○ Cerebral thrombosis: a thrombus that develops at the site on blockage.<br>○ Cerebral embolism: a blood clot that forms at another location and breaks loose.<br>• Hypoperfusion: Narrow vessels=reduced flow. |
| **Risk factors** | 1. Non modifiable: Age, Sex, Ethnicity, Genetic determinants.<br>2. Modifiable: HTN, DM, Smoking, Hyperlipidemia, Cardiac disease [AF], Stroke, TIA, carotid artery stenosis, Sedentary lifestyle. |
| **Investigation** | 1. CBC<br>2. Coagulation profile (Pt, PTT, INR)<br>3. Chemistry (Fasting glucose, Hba1c, Lipids)<br>4. Imaging (CT scan, MRI)<br>5. Vascular imaging (Carotid U/S, CTA, MRA, Cerebral Angio)<br>6. Cardiac work up (ECG, Echo, Holter)<br>• **Head CT without contrast** → can differentiate between hemorrhagic and ischemic, but ischemic changes may take 24 hours to appear.<br>• **MRI of brain** → more sensitive (changes may appear as early as 30 minutes). |
| **Management** | **TIME IS BRAIN: SAVE THE PENUMBRA!**<br>• Acute Stroke Management<br>○ ABC<br>○ Reperfusion<br>○ Prevent progression and complication<br>**At ER** (ABC's + history + blood work up + CT), then<br>- IV tpA (if pt >/= 18 years and before 270 minutes "4.5 hours" of stroke onset)<br>- Intra-arterial tpA<br>**DON'T GIVE TPA TO: pt with active or suspected bleeding (Ex: SAH)**<br>- Aspirin<br>- Mechanical → Endovascular thrombolysis<br>• Long Term Management<br>○ Risk Factor: HTN, DM, lipid, smoking, A-fib<br>○ Anti-platelet (atherosclerosis) or Anticoagulant ( afib or hypercoagulability)<br>○ Rehabilitation |

# Summary

## Hemorrhagic Stroke

high mortality rate (50% at 30 days).

**Risk Factors :**
HTN, Alcohol binge, Smoke, Obesity, Age, Race (Asians), Medications, Sympathomimetics (cocaine)

**Causes :**
1. **HTN** is the most common cause
2. **Ischemic stroke**
3. **amyloid angiopathy** (associated with Alzheimer)
4. **brain tumors**
5. **Vascular malformation**: .AVM (young and drug free), Aneurysm, Cavernous hemangioma.
6. **Bleeding disorders**/anticoagulant.
7. **Trauma.**
8. **Drug abuse:** amphetamine, cocaine, PPA (in young always drug screen)
9. **CVT** (young female using OCP)
10. **Moyamoya** (CT-angio:"puff of smoke")

**Locations**:
Basal ganglia → lobar regions → thalamus → Pons → cerebellum

**Investigations :**

1. **CBC, Coagulogram, Electrolyte**
2. **CT w/o contrast (will show Hyper density)**
3. **CT-angio (to look for the cause: aneurysm, AVM)**
4. **MRV (CVT)**

**Management:**

1. **Admit to ICU, Control BP, Watch for neuro decline, DVT prophylaxis**
2. **Surgery : only for cerebellar hemorrhage**
3. **Cerebral Edema Management: Ventilator, Sodium, Mannitol**

## Subarachnoid Hemorrhage

Berry aneurysm (associated with PCKD)

**Complications:**

1. **Vasospasm (prevent by nimodipine for 21 days)**
2. **-Hydrocephalus**
3. **Hyponatremia (put on hypertonic Saline)**

**Treatment:**
Intervention and inject coil in the aneurysm

*Pontine hemorrhage lead to locked in syndrome (aware but can only blink)

# Lecture Quiz

**Q1: After a patient experienced a brief episode of tinnitus, diplopia, and dysarthria with no residual effects, the nurse anticipates teaching the patient about**

A. cerebral aneurysm clipping.
B. heparin intravenous infusion.
C. oral low-dose aspirin therapy.
D. tissue plasminogen activator (tPA).

**Q2: A 73-year-old patient with a stroke experiences facial drooping on the right side and right-sided arm and leg paralysis. When admitting the patient, which clinical manifestation will the nurse expect to find?**

A. Impulsive behavior
B. Right-sided neglect
C. Hyperactive left-sided tendon reflexes
D. Difficulty comprehending instructions

**Q3: On observation, a patient has a left facial droop. On closer examination his nasolabial fold is flattened. When asked to smile, the left corner of his mouth droops. He is unable to keep his cheeks puffed out. Eye closure is only slightly weaker compared to the right and his forehead wrinkles when he is asked to look up high. What is the diagnosis?**

A.Right middle cerebral artery stroke
B. Parotid gland tumour.
C. Left internal capsule stroke
D. Bell's palsy
E. Cerebellar pontine angle tumour

**Q4: A patient is admitted with a stroke. On examination of her visual fields, she is unable to see in the right lower quadrant of her field. Where is the lesion?**

**A.**Optic chiasm
B. Left parietal lobe
C. Right temporal lobe
D. Right optic radiation
E. Left optic nerve

**Q5: A 35-year-old previously healthy woman suddenly develops a severe headache while lifting weights. A minute later she has transient loss of consciousness. She awakes with vomiting and a continued headache. She describes the headache as "the worst headache of my life." She appears uncomfortable and vomits during the physical examination. Blood pressure is 140/85, pulse rate is 100/min, respirations are 18/min, and temperature is 36.8°C (98.2°F). There is neck stiffness. Physical examination, including careful cranial nerve and deep tendon reflex testing, is otherwise normal. Which of the following is the best next step in evaluation?**

a. CT scan without contrast
b. CT scan with contrast
c. Cerebral angiogram
d. Holter monitor
e. Lumbar puncture

Answers: Q1:C | Q2:D | Q3:A | Q4:B | Q5:A

Answers Explanation File!

# THANKS!!

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Send us your feedback:
We are all ears!