Medicine
TEAM 437

MED437
KING SAUD UNIVERSITY

Ischemic Stroke

Objectives :

1. What is the ischemic stroke and why ?
2. Pathophysiology and subtypes of ischemic stroke
3. Clinical presentation of ischemic stroke
4. Diagnosis of ischemic stroke
5. Management of ischemic stroke
6. What is TIA ?

Done by :

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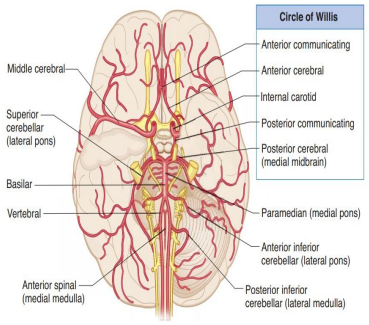
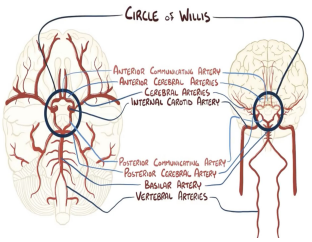
Resources :

Doctor's slides + Team 436
Lecturer: Dr. Adel Alhazzani
Same as 436 slides: Yes
Step up

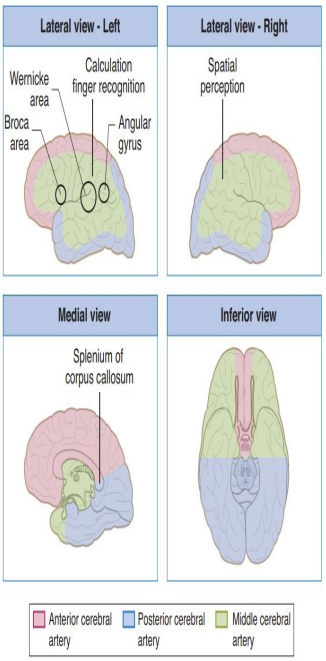
❖ Cerebral Circulation: imp

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



❖ BASIC INFO...

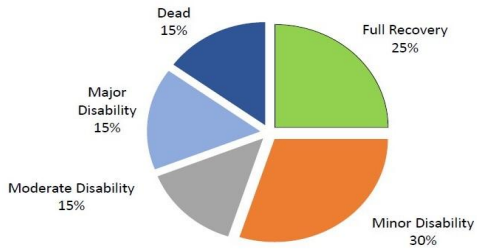
Worldwide Burden of Stroke

1. Leading cause of adult disability
2. 2nd cause of death
3. 20 million people worldwide suffer a stroke each year.
4. 1/4
5. 5 million deaths/year
6. Every 6 seconds

In Saudi Arabia

- 20 25,000 new strokes
- 4000 5000 deaths (estimate)
- 8000 disabilities
- Incidence 58 100,000 new
- Total 70 100,000 total recurrence
- Cost to patient, family , community

Outcome of Ischemic Stroke:



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

❖ Definition

- Abrupt onset
- Focal neurological deficits
- Due to interruption of
- vascular supply
- Can be ischemic (blockage) or hemorrhagic (bleeding).

❖ Ischemic Stroke

- 85% of all strokes.
- Acute onset of neurologic deficits caused by impaired blood flow to CNS.
- Stroke:
  - persisting neurologic deficit after 24hrs and/or
  - infarct on CT or MRI.

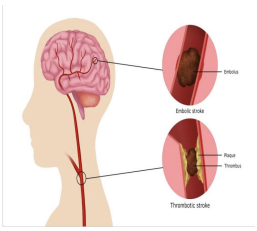
- Transient ischemic attacks (TIAs) AKA “mini strokes” or “warning strokes” stroke-like symptoms that last for a very short time(<1hr) with complete recovery (most are <5 min)
- A TIA indicates that conditions for an ischemic stroke are present.

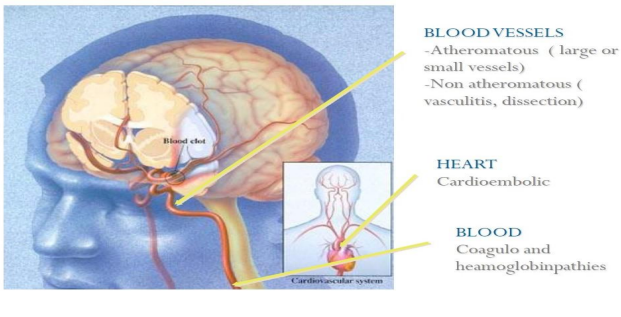
❖ Risk Factors

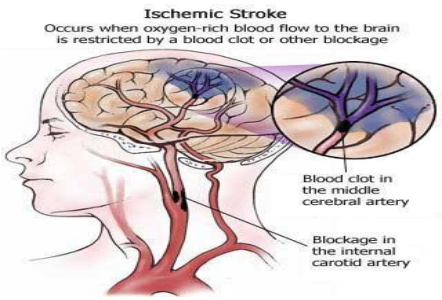
Non-modifiable:	Modifiable:
• Age, • Sex, • Ethnicity, • Genetic determinants.	• HTN, • DM, • Smoking, • Hyperlipidemia, • Cardiac disease (particularly atrial fibrillation [AF], • Stroke, TIA,carotid artery stenosis, • Sedentary lifestyle.

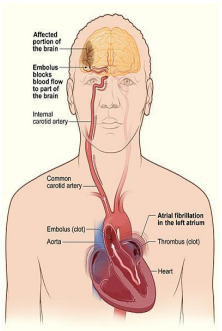
❖ Ischemic Stroke Mechanisms:

1. Due to <u>blockage</u> from :
   - Cerebral **thrombosis**: a thrombus (blood clot) that develops at the blocked part of the artery.
   - Cerebral **embolism**: typically caused by a blood clot that forms at <u>another location</u> and breaks loose and enters the bloodstream.
2. Hypoperfusion (Narrow vessels reduced flow)



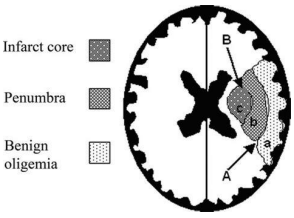


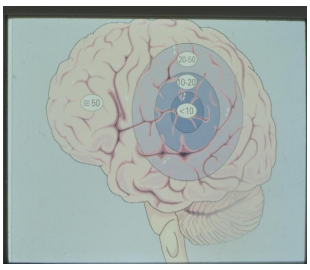




Pathophysiology

- Active and does not store energy.
- the brain is not adequately perfused , cells begins to die.
- Core (area of irreversible damage)
- Penumbra (tissue at risk can

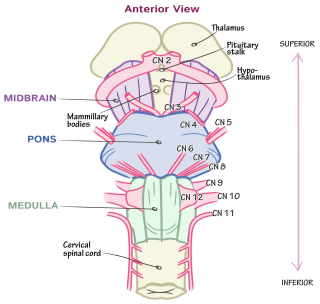


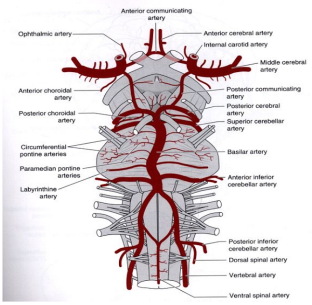


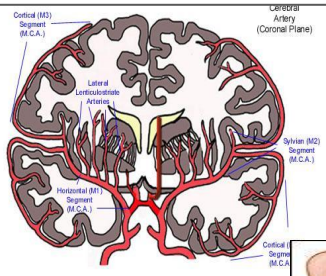
❖ Clinical presentation

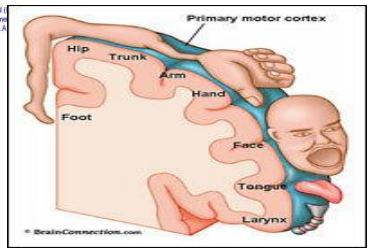
Location of occlusion:	Symptoms
Middle Cerebral Artery(MCA):	arm + face > leg weakness, sensory loss, aphasia, neglect, homonymous hemianopia.
Anterior Cerebral Artery(ACA):	weakness LE >UE, emotional disturbance.
Internal Carotid	above and ophthalmic.
Posterior cerebral artery (PCA)	vision-visual fields and memory.
Vertebrobasilar	CN with crossed motor, cerebellum, altered LOC.
Midbrain	CN III –, dilated pupil.
Pons	-CN V –facial numbness, weakness jaw movements. -CN VI –lateral rectus palsy. -CN VII –facial weakness.
Medulla	-CN VIII –vertigo, hearing loss. -CN IX, X –dysphagia. -CN XII –tongue weakness.

Lacunar syndrome (small penetrating arteries)









❖ Investigation

CBC

Coagulation profile
- PT,
- PTT,
- INR

Chemistry
- Fasting glucose,
- Hba1c,
- Lipids

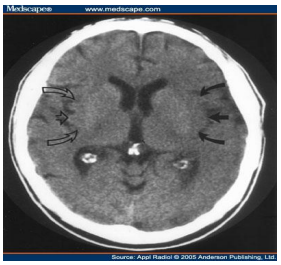
Specific cases
- Hb electrophoresis,
- hypercoagulable work up,
- CTD screen,
- HIV and syphilis

Vascular imaging
- Carotid U/S,
- CTA,
- MRA,
- Cerebral Angio

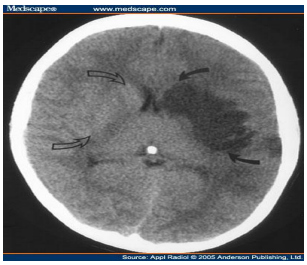
Cardiac work up
- ECG
- Echo (TTE or TEE)
- Holter

Imaging
- CT scan

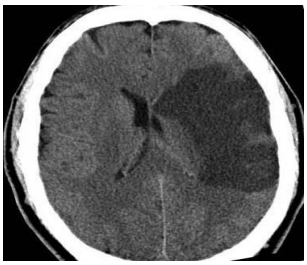
2hrs



20hr

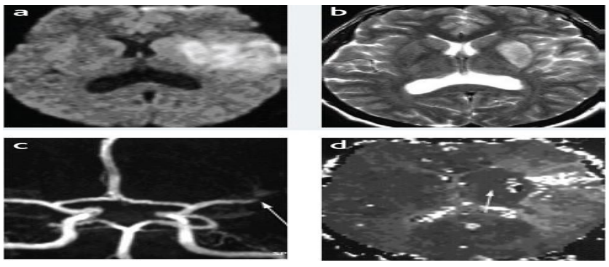


36hr



- MRI
  - More sensitive
  - C/I

<u>acute stroke</u>



❖ Management

- Acute Stroke Management
  - ABC
  - Reperfusion
  - Prevent progression and complication
- Long Term Management
  - Risk Factor: HTN, DM, lipid, smoking, A-fib
  - Anti-platelet (atherosclerosis) or Anticoagulant (afib or hypercoagulability)
  - Rehabilitation

Reperfusion

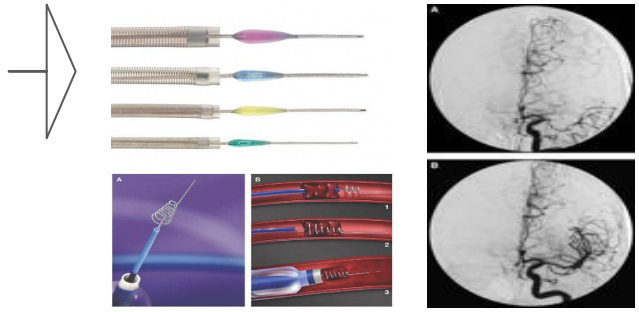
Intravenous thrombolysis (IV t-PA); Tissue plasminogen activator:

- Effective up to 4.5 hrs 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:

1. ICH
2. prior ICH, Hx suggests of SAH, stroke past 3mts
3. GI or GU hmg in past 3wks, recent MI, major surgery 14d
4. platelet <100 000,
5. INR >1.7, PT >15
6. SBP >186 or DBP >110, Hg<100?

Intra-arterial thrombolytic



Stroke unit

- BP and glycemic control
- NPO, Avoid aspiration
- Dx and Rx Temp .
- PT , OT and early rehab.
- DVT prophylaxis

Other medications

- 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)

Secondary Prevention

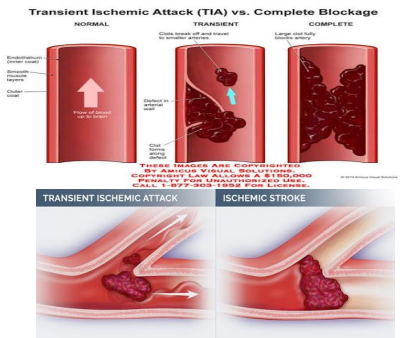
- Long Term Management
  - HTN
  - DM
  - Stop smoking
  - Lipid lowering agent
  - Exercise

Treat underlying condition

- Carotid SX,
- cardio-embolic
- hypercoagulable rx with Coumadin

❖ TRANSIENT ISCHEMIC ATTACKS

- Brief and temporary reduction in blood flow to a focal region within the brain with no evidence of infarction on imaging.
- Is a stroke that did not finish YET
- Up to 1/3 with have stroke (usually first 48 hrs)
- most TIA's last 5-20 minutes
- if >1hr usually small infarction on MRI
- DDX (Seizure, migraine , Syncope , Labyrinthine SDH,



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 ,
   a. vascular imaging of carotid CTA , MRA, US
   b. Cardiac work up (EKG, echo +/-Holter
4. Start stroke prevention measures (like ischemic stroke) ASA , control HTN ,DM and lipids, stop smoking and exercise .

❖ History

- ONSET (Last time seen normal)
  - Symptoms (analysis of symptoms)
    - Headache (sudden and severe)*
    - Neck pain/ trauma*
  - progression
  - Previous HX of stroke or TIA,
- PMHX : Risk factors/medication
- HX from others

❖ Physical examination

- ABC
- General examination
- Pulse (A.fib)
- BP
- Hand
- listen for heart murmur, carotid bruits
- Cortical infarcts are suspect based on the presence of:
  1. language impairment
  2. neglect or anosognosia
  3. graphesthesia or stereoagnosia
  4. visual field impairment
- CN involvement and crossed motor
- Tone –decreased on side of weakness early on, later on increased
- Pyramidal pattern weakness (UMN)
  1. UE extensor > flexor
  2. LE flexor > extensor
- Reflexes –hypereflexic on side of weakness, with upgoing toe

❖ Take Home Message

- 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 .

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 :
	  ○ Cerebral thrombosis: a thrombus that develops at the site on blockage.
	  ○ Cerebral embolism: a blood clot that forms at another location and breaks loose.
	• Hypoperfusion: Narrow vessels=reduced flow.

Risk factors	1. Non modifiable: Age, Sex, Ethnicity, Genetic determinants. 2. Modifiable: HTN, DM, Smoking, Hyperlipidemia, Cardiac disease [AF], Stroke, TIA, carotid artery stenosis, Sedentary lifestyle.

Clinical presentations	
Location of occlusion:	Symptoms:
Middle Cerebral Artery(MCA):	arm + face > leg weakness, sensory loss, aphasia, neglect, homonymous hemianopia.
Anterior Cerebral Artery(ACA):	weakness LE >UE, emotional disturbance.
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Lacunar syndrome (small penetrating arteries)

Summary

Investigation	1. CBC 2. Coagulation profile (Pt, PTT, INR) 3. Chemistry (Fasting glucose, Hba1c, Lipids) 4. Imaging (CT scan, MRI) 5. Vascular imaging (Carotid U/S, CTA, MRA, Cerebral Angio) 6. Cardiac work up (ECG, Echo, Holter)
Management	● Acute Stroke Management ○ ABC ○ Reperfusion ○ Prevent progression and complication ● Long Term Management ○ Risk Factor: HTN, DM, lipid, smoking, A-fib ○ Anti-platelet (atherosclerosis) or Anticoagulant (afib or hypercoagulability) ○ Rehabilitation

Questions

1-A 65-year-old male presents with dizziness and generalized weakness early this afternoon while shopping shortly after dinner. The patient recently underwent open heart surgery to have a prosthetic heart valve placed. His current medications include simvastatin, metoprolol and coumadin. He describes the dizziness as occurring gradually over the past few days and not succumbing to losing his balance and having a traumatic fall. He describes the dizziness as being bearable with a constant level of intensity, while also denying any nausea, vomiting or ringing in his ears. On physical exam, the patient exhibits vertical nystagmus but no horizontal or rotatory nystagmus. A Dix-Hallpike maneuver was performed, which proved to be negative. A pneumatic otoscope was also used to insufflate air into both his ear canals, which also failed to provoke an episode of vertigo with nystagmus. Which of the following is the correct diagnosis?

A- Physiological vertigo
B- Central vertigo due to stroke
C- Benign positional vertigo
D- Perilymph fistula
E- Meniere's disease

Answer is: B
Explanation: The patient's gradual onset of vertigo, low-intensity dizziness, presence of only a vertical nystagmus, and lack of nausea, vomiting and tinnitus are all more indicative of a pathology reflecting central vertigo, and a high index of suspicion should be maintained. On the other hand, peripheral vertigo is described as having sudden onset, intense dizziness, frequent nausea and vomiting, tinnitus and a rotatory-vertical or horizontal nystagmus, although all these symptoms may not be present at once. Nystagmus with only a vertical component generally represents a brainstem abnormality, helping to support pathology reflective of central vertigo here. The patient also has many risk factors for a stroke given his recent cardiac surgery and use of warfarin. In any patient taking warfarin, central vertigo should always be evaluated to rule out pathology. During the physical exam, the physician should look for other signs of a central pathology, such as neurological deficits like diplopia, dysarthria or visual abnormalities. This also includes loss of a corneal or gag reflex. All the other answer choices are incorrect since they represent pathologies that reflect a peripheral vertigo. The Dix-Hallpike test evaluates for BPV, which it is 50 to 80% sensitive for, while the insufflation of air with the pneumatic otoscope tests for a perilymph fistula, an opening in the oval or round window creating an abnormal communication between the middle ear and vestibular apparatus.

Questions

2- A 60-year-old male with a history of diabetes and hypertension presents with weakness, dizziness and abnormal speech for the past 24 hours. The patient's spouse states that he recently started acting "funny," and his symptoms have been worsening. Upon examination, the patient appears confused but is able to follow your commands. During the cardiac examination, a left carotid bruit is auscultated. During the neurological examination, a sensory and motor loss is noted in the right lower leg. The patient's speech sounds halting and effortful, but he is able to repeat words back to you. The patient does not have evidence of cranial nerve abnormalities on exam. What syndrome describes the ischemic stroke this patient suffers from?

A- ACA infraction
B- MCA infraction
C- PCA infraction
D- Vertebrobasilar infarction
E- Cerebellar infarction

Answer is: A
Explanation: Patients with an anterior cerebral artery infarction suffer from contralateral sensory and motor loss in the lower extremities sparing the face and upper extremities. A left-sided lesion is associated with mutism and transcortical motor aphasia - with repetition retained, while a right-sided lesion is associated with motor hemineglect. A middle cerebral artery infarction (choice B) is incorrect, because an infarction here affects the face and upper extremities. Choice C is incorrect, since the patient presents with no visual field deficits. Choice D is incorrect, since the patient does not present with simultaneous signs and symptoms of vertigo, headache, nausea, visual disturbances, oculomotor palsies, limb weakness or oropharyngeal dysfunction. Finally, choice E is incorrect since the patient does not have vertigo, ataxia, headache, dysarthria, nausea, vomiting, or cranial nerve abnormalities.

3- A 75-year-old female patient presents with headache, nausea and vomiting for the past 48 hours. Her past medical history is significant for an aortic valve replacement 15 years ago. The patient is alert, and she tells you she suffered from no recent trauma. Upon examining her, you note the patient's visual acuity is 20/20 in both eyes, but her speech is abnormal. During the neurological examination, the patient is able to understand your questions, respond appropriately, and repeat words back to you, but her words are poorly articulated. In addition, you ask the patient to walk across the room, and she nearly falls. As the physician, you suspect the patient may be suffering from a cerebellar infarction. What is your next step in the management of this patient?

A- Emergent surgical decompression
B- Administer unfractionated heparin
C- Administer recombinant tissue plasminogen inhibitor
D- Emergent MRI or MRA
E- Infuse hypertonic saline

Questions

Answer is: D

Explanation: The patient's signs: dysarthria from her poorly articulated words and gait ataxia from her inability to walk, and symptoms: headache, nausea and vomiting, are all consistent with a cerebellar infarction. Due to the obscuration by a posterior fossa bone artifact, an emergent MRI or MRA is preferred after a noncontrast brain CT may be used to rule out a hemorrhagic infarction. Choices A and E are incorrect, because they involve therapeutic interventions for the development of cerebellar edema. The development of an obstructing hydrocephalus is a possible complication of a cerebellar infarction that can result in poor outcomes. Choices B and C are therapeutic interventions that may be utilized in an ischemic stroke, but diagnosis should always precede treatment.

4- Which of the following is the most important modifiable risk factor for ischemic stroke?

A- Patent foramen ovale
B- Oral contraceptive
C- Sickle cell disease
D- Hypertension

5- Which of the following is not a common sign or symptom of ischemic stroke?

A- Hemisensory deficit
B- Quadriparesis
C- Hemiparesis
D- Visual field loss

6- Which of the following is the most commonly used form of neuroimaging in the acute evaluation of patients with apparent stroke?

A- Noncontrast CT scan
B- Transcranial doppler ultrasonography
C- Echocardiography
D- Single photon CT scan

7- Which of the following fibrinolytic agents can be used in patients with acute ischemic stroke?

A- Alteplase (rt-PA)
B- Urokinase
C- Streptokinase
D- Tenecteplase

4- D, 5- B, 6- A. 7- A

Doctor's notes:

Stroke is defined as an abrupt onset of neurological deficits caused by interruption of blood supply. It could either be ischemic or hemorrhagic.

-Stroke is common and serious, it is the leading cause of death worldwide but here it the second and comes after car accidents. It is the third cause of death worldwide, some say it's the second. It is one of the top 3 causes of dementia, it comes after alzheimer disease vascular dementia. 50% of patients that have ischemic stroke will either die or be disabled.

-TIA is basically transient blockage in which neurological symptoms will quickly go away (usually less than 5 minutes and maximum of one hour). A TIA will never produce an infarct in imaging, so if you see one it's NOT TIA. Usually they end up with complete recovery but ⅓ of them will have a stroke, especially in the 1st 48h so try to prevent that.

In ischemic stroke, blockage is either a thrombus or an embolus. An embolus could be from the heart (AFib, MI, …) or could be from the atherosclerotic aortic arch that dislodges emboli. Hypotension rarely leads to an ischemic stroke unless there was an artery in the brain that was already very narrow and stenotic due to atherosclerosis.

When someone gets a stroke, ask yourself: Where is the source of the problem? So that you can prevent another stroke from happening. "Think as a plumber"...blood vessels? Heart? Or blood?

Blood vessels:
1- So an old patient, heavy smoker and is diabetic, hypertensive and presented with stroke, you wouldn't be surprised and most likely he also has IHD or PVD. The cause here is atherosclerosis and it could be in the large vessels (ex: carotid) or small vessels (lacunar infarcts).

2- young female who has an autoimmune disease (ex: SLE), think of vasculitis.

3- One out of four strokes in the young population is due to carotid dissection. Classically, they present with **neck pain** and headache. It usually happens in someone who has some type of collagen disorder (ex: Marfan Syndrome) after some kind of neck injury as if he had a car accident which led to hyperextension injury, a barber hit his neck, following riding a roller coaster, I have even seen it بمطب سيارة.

Heart: AFib is the commonest, VHD, IE or even MI.

Blood: typically happens in a young patient (polycythemia, thrombocytosis, young female taking oral contraceptives…).
-DVT doesn't cause stroke unless very rarely in which the patient has a PFO + pressure in RA higher than LA (Straining, valsalva or like Eisenmenger syndrome).

When talking about risk factors, males are usually at more risk to get a stroke. However, younger women are at more risk than younger males since they have a hypercoagulable state and also considering their hormones (estrogen). Asians are at more risk to get a hemorrhagic stroke than other ethnicities, while african americans have a risk to get ischemic strokes. Nowadays, 80% of strokes are preventable if we control modifiable risk factors.

Once blood supply is interrupted to an area in the brain. At first, it stops functioning, but later on if this interruption persists, the area will infarct and die, especially if there are no collaterals developed to supply that are during the prolonged time of blockage. Usual blood flow to brain is 100 ml / 100 gm / minute, if blood flow to an area in the brain gets below 10 ml and persists, it will infarct. It depends on how low the supply is and how long (duration). You need to understand this pathophysiology because we have a large area in the brain with reduced flow but still alive and viable, it's called the **Penumbra**. This is the area of intervention!

Someone presenting with one sided weakness that started gradually and for 2 weeks, this is very unlikely to be stroke! Stroke is sudden and acute. Classical symptoms of stroke are:
-One sided weakness
-Speech problem, either expressive, comprehensive, or slurred speech.
-Sensory loss in one side (not both sides).
-Headache with nausea and vomiting, but more with hemorrhagic stroke since they have increased ICP. However, headache and neck pain could happen in ischemic stroke if the cause was dissection, or maybe vasculitis.

A common approach we teach the public in assessing someone with stroke is FAST.
F: face, ask them to smile you'll see asymmetry.
A: arms, there will be a drift
S: speech, ask them to talk.
T: time, come to hospital ASAP.

Now they added BE FAST to it, where B is balance and E is eye since this covers posterior circulation strokes for cerebellum and occipital lobes.

In neurology, there are some conditions that present suddenly such as strokes, headache disorders, seizures, and trauma. So always ask about the onset of neurological symptoms for two reasons. One, to know this is a stroke. And two, so that you can intervene. For example, someone presenting one hour after the onset of his symptoms, this guy has a penumbra that can be salvaged unlike someone presenting after two days in which the area of brain is already infarcted and you cannot do anything about it and can only prevent complications.
Onset of symptoms starts from the last time patient was seen normal. For example, someone went to sleep and was normal, woke up with his symptoms, onset here starts from the time he went to sleep.

On physical examination, high blood pressure is a very usual finding in both types of stroke. Always check the pulse, it may be irregular (AFib). Check temperature (fever may indicate IE). Listen to the heart sounds and the carotids (murmurs and bruits may indicate VHD or carotid stenosis).

When you do neurological examination and you're assessing UMN lesions or corticospinal tract lesions by checking motor power of limbs, extensors of the upper limbs will be weaker than flexors. While in lower limbs, flexors will be weaker than extensors.

In clinical presentation, it depends on which artery is affected. Cortical signs will have cortical function loss + differentiated weakness (weakness in one part of the body is different from the other). In subcortical areas, when you have lacunar strokes, hemiparesis in all body parts will be in similar extent, with no other findings such as speech problems or neglect.. Brain stem infarcts may have cranial nerve injuries.
Remember an infarct in the non-dominant hemisphere may cause neglect, since one of its functions is to orient you to the body.

When taking history, always ask about medications. Anti-coagulation or aspirin may lead to hemorrhagic stroke, or someone taking aspirin and stopped it may develop ischemic stroke. Also NOACs are good and wonderful but when you stop them, they may cause paradoxical embolization and hence stroke.

When a patient comes to the ER with a presentation of stroke, the first thing you do is stabilize him and then do an urgent plain CT of the head. 1st, it will tell you whether the stroke is ischemic or hemorrhagic, and 2nd, it will show you if there is an area of infarction or not. If you see a normal CT with no infarction, that's good and it means there is room for quick intervention. However, if you see an infarcted area, this area is dead and you can't save it, but don't forget that there may be a penumbra that can be salvaged, which depends on time of onset.

MRI is more sensitive than CT, it may show the area of infarction 30 minutes after the onset but a CT may take hours to show you the infarcted area. That being said, it's not routinely used since it's time consuming and not readily available, and sometimes patients may have contraindications to MRI.

Intervention can be given with **IV Thrombolytics and up to 4 and ½ hours** from time of onset, the earlier the better (make sure patient doesn't have contraindications to Thrombolytics). You can also intervene with **intra-arterial mechanical thrombectomy and up to 6 hours** from time of onset. If you're going to intervene mechanically with a catheter, give the patient IV thrombolysis while you prepare the angio room since this may take time.

Now we have MR Perfusion and CT Perfusion, which is the future of stroke. You give contrast and it shows you area of infarction and area of decreased perfusion (penumbra). What is the benefit? Take this example:
If someone last seen normal when he went to sleep, woke up after 6 hours having his symptoms, came to the ER and you did a CT which was normal (no infarction), do you give him IV Thrombolytics or not? Guidelines say no more than 4 and ½ hours but CT is normal, here is the time where CT Perfusion comes in, you do it and see if there is a penumbra that can be saved or not. So now in stroke management we're moving from time-based to tissue-based. Problem it's not available everywhere. **For your exam: IV Thrombolysis up to 4 and ½ hours, IA thrombectomy up to 6 hours.**

Any stroke patient must be admitted to "stroke unit", just like an MI patient to CCU. This improves outcome. Don't touch the high BP in the first 24h since it's elevated to perfuse the brain, unless you will do thrombectomy you wanna lower it a little bit to prevent bleeding. Always try to prevent complications (SC heparin for DVT, move the patient to prevent bed sores, be aware of aspiration pneumonia).