Pathogenesis and risk factors of cerebrovascular accidents 1&2

Editing File

Color index :
Main text (black)
Female Slides (Pink)
Male Slides (Blue)
Important (Red)
Dr's note (Green)
Extra Info (Grey)

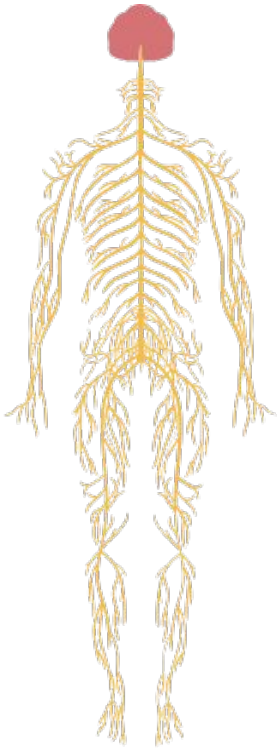
Objectives

- Explain the concepts of brain “hypoxia”, “ischemia” and “infarction”.
- Understand the pathogenesis of thrombotic and embolic strokes and be able to identify the clinical risk factors.
- Identify the causes and consequences of subarachnoid and intracerebral hemorrhage.
- Build a list of the different causes that can lead to a cerebrovascular accident.

If you want to read the lecture from Robbins click here

If you want to listen to ninja nerd podcast click here

Very useful to review it while you are doing other activities



Cerebrovascular Disease

Osmosis Vid

Introduction

01

Definition: they are brain disorders caused by pathologic processes involving blood vessels.

02

- Cerebrovascular disease is the third leading cause of death (after heart disease and cancer) in the United States
-They are a major cause of death in the developed world and are the most prevalent cause of neurologic morbidity.

03

It is also the most prevalent neurological disorder in terms of both morbidity and mortality

The three main pathogenic mechanisms are:

1 Thrombotic occlusion

2 Embolic occlusion

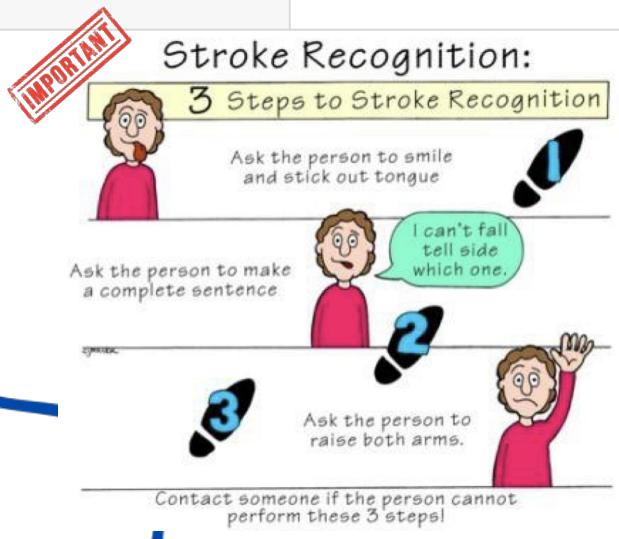
3 Vascular rupture

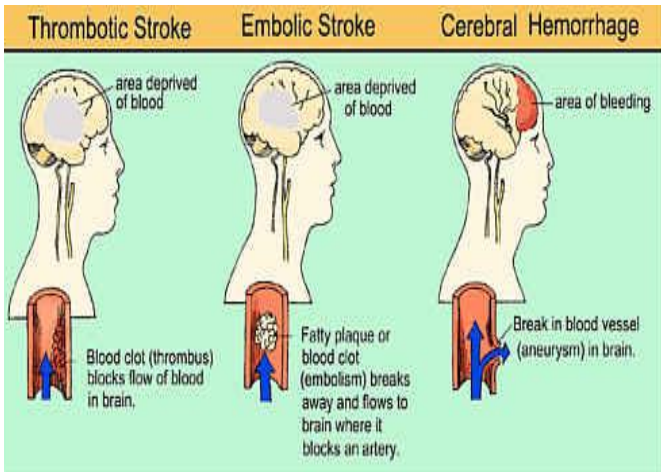
IMPORTANT

The brain may be deprived of oxygen by any of several mechanisms including	
Functional hypoxia	Ischemia either transient or permanent
1-low partial pressure of oxygen (e.g. High Altitude) 2-Impaired oxygen-carrying capacity (e.g. severe anemia, Carbon monoxide poisoning) 3-Inhibition of oxygen use by tissue (e.g. cyanide poisoning)	Due to tissue hypoperfusion: 1- reduction in perfusion pressure, as in hypotension 2-Vascular obstruction. 3-Both.

Cerebrovascular Disease

Definitions	
Stroke	- It is the clinical term for a disease with acute onset of a neurologic deficit as the result of vascular lesions - either hemorrhage or loss of blood supply. - Stroke is the clinical designation applied to all of these conditions when symptoms begin acutely.
Thrombosis Embolism	- Thrombosis & embolism have similar consequences for the brain: loss of oxygen & metabolic substrates, resulting in infarction or ischemic injury of regions supplied by the affected vessel. (More common) - Infarction is complete loss of perfusion, hypoxemia (hypovolemic shock) or hypoglycemia
Hemorrhage	Hemorrhage accompanies rupture of vessels and leads to direct tissue damage as well as secondary ischemic injury (aneurysm or trauma)





Clinical Presentation of Stroke

Strokes can be asymptomatic or painless, however they may also present with symptoms depending on which part of the brain is injured, and how severely it is injured.	It is very important to recognize the warning signs of a stroke and to get immediate medical attention if they occur.	If the brain damage sustained has been slight, there is usually complete recovery, but most survivors of stroke require extensive rehabilitation.

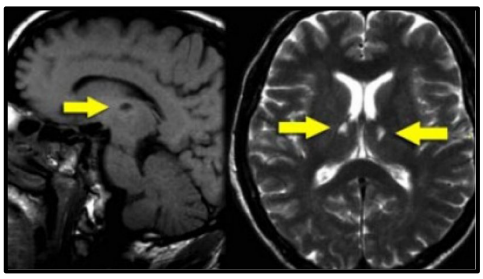
Symptoms

- Sudden.
- Headache
- The most common is weakness or paralysis of one side of the body with partial or complete loss of voluntary movement or sensation in a leg or arm.
- Speech problems and weak face muscles, causing drooling.
- Numbness or tingling is very common.
- Can affect balance, vision, swallowing, breathing and even unconsciousness. Due to involvement of the base of the brain (brainstem).
- In cases of severe brain damage, there may be deep coma, paralysis of one side of the body, and loss of speech, followed by death or permanent neurological disturbances after recovery.

Ischemic Stroke

Osmosis Vid

IMPORTANT

	Thrombotic stroke	Embolic stroke
Definition	- Formation of blood clot (thrombus) inside a blood vessel, obstructing the flow of blood. We know that you know it, but a reminder	- Intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin.
Overview	- The majority of thrombotic occlusions causing cerebral infarctions are due to **atherosclerosis** - Thrombotic occlusions are usually superimposed on atherosclerotic plaques, accompanied by anterograde extension, fragmentation, and distal embolization. - Thrombotic occlusions causing small infarcts of only a few mi"lacunar infarcts", occurs when small penetrating arteries are occluded.	-Embolic infarction are more common than thrombotic infarction
The most common sites	- The carotid bifurcation - The origin of the middle cerebral artery - At either end of the basilar artery	-The territory of distribution of the **middle cerebral arteries** (branches from the internal carotid arteries) most frequently affected by embolic infarction. - Emboli tend to lodge where vessels branch or at stenotic areas caused by atherosclerosis
Sources in case of embolic		1- Cardiac mural thrombi (frequent): A. Myocardial infarction, B. Valvular disease, C. Atrial fibrillation. 2- Paradoxical (unexpected) emboli : particularly in children with cardiac anomalies (e.g. patent foramen ovale) 3-Arteries :(often atheromatous plaques within the carotid arteries or the aortic arch) 4- Emboli associated with cardiac surgeries. 5 -Emboli of other material (tumor, fat, or air).

Global cerebral ischemia

1

Widespread ischemic/hypoxic injury occurs when there is a generalized reduction of cerebral perfusion (all the brain), usually below **systolic pressures of 50 mmHg.**

2

Causes include :

- **Cardiac arrest.**
- **Severe hypotension or shock.**

3

The clinical outcome varies with the severity of the insult.

4

If the patient survives the severe form, he could suffer either :

- **Severe** global cerebral ischemia → widespread neuronal death, irrespective of regional vulnerability, occurs and patient who survive often remain severely impaired neurologically & in a persistent vegetative state.
  - Total paralysis
  - Can't communicate

  But normal heart beat & can breath (unlike brain dead patients)
- **Mild**→ transient postischemic confusional state, with eventual complete recovery

Persistent vegetative state	Respirator brain
Severely neurologically impaired.	Meet the clinical criteria for "brain death" including evidence of : • Diffuse cortical injury (isoelectric or flat electroencephalogram EEG). • Brain stem damage, including absent reflexes and respiratory drive.
Deeply comatose.	When patients with this irreversible form of injury are maintained on mechanical ventilation, the brain gradually undergoes an **autolytic process resulting in the so -called "respirator brain"** **(When you do CT scan the skull will be empty).**

IMPORTANT

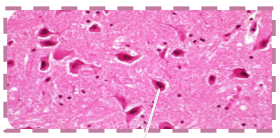
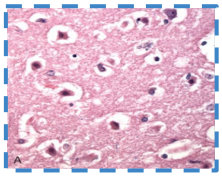
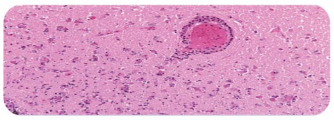
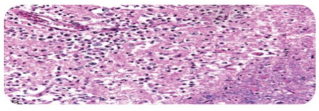
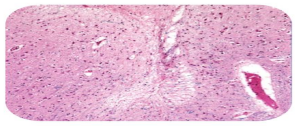
Sensitivity to ischemia (Histopathological features)

1-**Neurons** are more susceptible to hypoxia than glial cells.	2-The most susceptible to ischemia of short duration are :		
	A-pyramidal cells of the Sommer sector (CA1) of the hippocampus.	B-Purkinje cells of the cerebellum.	c-pyramidal neurons in the neocortex.

* Macroscopic changes next slide

IMPORTANT

Histopathological features

Macroscopic features (Gross pathology)	• The brain is **swollen** (edema), with wide gyri and narrowed sulci. • The cut surface shows **poor demarcation** between gray and white matter .		
Microscopic Features **FEMALES SLIDES**	**Early changes (12-24 H)**	**Subacute changes (24H- 2 W)**	**Repair (after 2 W)**
	▪ **Red neurons :** characterized initially by microvacuolization, cytoplasmic eosinophilia, and later nuclear pyknosis and karyorrhexis. ▪ Similar changes occur later in glial cells.	▪ The reaction to tissue damage begins with the **infiltration by neutrophils.** ▪ Necrosis of tissue, influx of macrophages, vascular proliferation and reactive gliosis.	▪ Removal of all necrotic tissue. ▪ Loss of the organized CNS structure. ▪ **Gliosis.** Remember here is no recovery of brain tissue
	 Red neurons 	 Infiltration of a cerebral infarction by neutrophils begins at the edges of the lesion where the vascular supply is intact.  By day 10, an area of infarction shows the presence of macrophages and surrounding reactive gliosis.	 Old intracortical infarcts are seen as areas of tissue loss with a modest amount of residual gliosis. A lot of macrophage

Focal cerebral ischemia

01

Cerebral arterial occlusion or cerebral hemorrhage lead first to focal ischemia then an infarction in the distribution of the **compromised vessels.** (not caused by hypotension or something affect heart).

02

The size, location and shape of the infarct and the extent of tissue damage may be modified by **collateral blood flow.**

03

The major source of collateral flow is the **circle of Willis.**

04

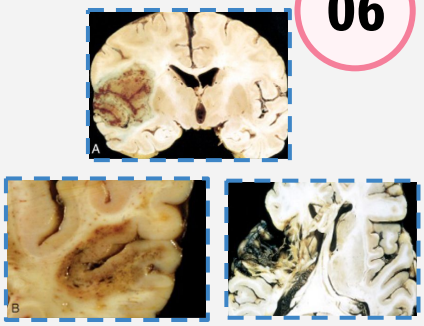
Partial collateralization is also provided over the **surface** of the brain through **cortical-leptomeningeal anastomoses.**

05

In contrast, there is **little** if any collateral flow for the deep penetrating vessels supplying structures such as :

- **Thalamus.**
- **Basal ganglia.**
- **Deep white matter.**

06



Deep Focus Question

What is the most common type of stroke?

A. Ischemic stroke
B. Subdural hematoma
C. Epidural hematoma
D. Subarachnoid hemorrhage
E. Intraparenchymal bleed

Answer: A

Deep Focus Question

Which of the following are the risk factors for stroke?

A. Fibrillin-1 mutation and smoking history
B. Family history and wingspan-to-height ratio
C. Race and age
D. Diabetes and hyperlipidemia

Answer: D

Infarcts

Infarcts can be divided into two broad groups based on their **macroscopic** and corresponding **radiologic appearance :**

Non hemorrhagic infarcts

which result from acute vascular occlusions.

Hemorrhagic infarcts

May evolve from non hemorrhagic infarcts when there is reperfusion of ischemic tissue, either through collaterals or after dissolution of emboli, and often produce multiple, sometimes confluent petechial hemorrhages.

IMPORTANT

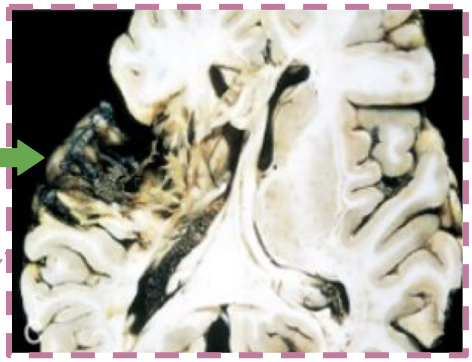
Histopathological (Gross) Features of Non-Hemorrhagic Infarcts macroscopically	**First 6 hours**	the tissue is unchanged in appearance, little can be observed.
	By 48 hours	the tissue becomes **pales soft and swollen**, and the corticomedullary junction becomes indistinct
	2-10 days	the tissue becomes **gelatinous, friable** and the previously ill-defined boundary between normal and abnormal tissue becomes more **distinct** as edema resolves in the adjacent viable tissue that has survived.
	Day 10- week 3	The tissue liquifies leaving a **fluid-filled cavity** (lined by dark gray tissue) which gradually expands as dead tissue is resorbed/removed.

Extra INFO

usually manifest as multiple, sometimes confluent, petechial hemorrhages The microscopic picture and evolution of hemorrhagic infarction parallel those of ischemic infarction

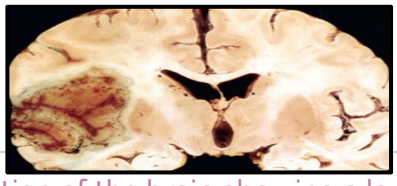
Cavity

Old cystic infarcts shows destruction of cortex and surrounding gliosis



Microscopic Features

IMPORTANT

<table>
<tr><td rowspan="5">Non-Hemorrhagic Infarcts</td><td colspan="4">After the first 12 hours :</td></tr>
<tr><td>▪ Ischemic neuronal change (red neurons) and edema both vasogenic and cytotoxic.</td><td>▪ Endothelial & glial cells mainly astrocytes swell & myelinated fibers begin to disintegrate.</td><td>▪ Loss of the usual characteristics of white and gray matter structures.</td><td>▪ During the first several days neutrophils infiltrate the area of injury.</td></tr>
<tr><td colspan="4">Until 48 hours :</td></tr>
<tr><td>▪ There is some neutrophil emigration followed by mononuclear phagocytic cells in the ensuing 2 to 3 weeks.</td><td>▪ Macrophages containing myelin breakdown products or blood may persist in the lesion for months to years.</td><td colspan="2">▪ As the process of phagocytosis and liquefaction proceeds, astrocytes at the edges of the lesion progressively enlarge, divide, and develop a prominent network of protoplasmic extensions.</td></tr>
<tr><td colspan="4">After several months :</td></tr>
<tr><td>▪ The striking astrocytic nuclear and cytoplasmic enlargement Recedes or disappears.</td><td>▪ In the wall of the cavity, astrocyte processes form a dense feltwork of glial fibers admixed with new capillaries and a few perivascular connective tissue fibers.</td><td>▪ In the cerebral cortex the cavity is delimited from the meninges and subarachnoid space by a gliotic layer of tissue, derived from the molecular layer of the cortex.</td><td>▪ The pia and arachnoid are not affected and do not contribute to the healing process.</td></tr>
<tr><td rowspan="5">Hemorrhagic Infarcts</td><td colspan="4">Hemorrhagic infarcts usually manifest as multiple, sometimes confluent petechial hemorrhages.</td></tr>
<tr><td colspan="4">The microscopic picture and evolution of hemorrhagic infarction parallel those of ischemic infarction, with the addition of blood extravasation & resorption (RBC in tissue).</td></tr>
<tr><td colspan="4">In individuals with coagulopathies or receiving anticoagulants, hemorrhagic infarcts may be associated with extensive intracerebral hematomas.</td></tr>
<tr><td colspan="2">  </td><td colspan="2">  </td></tr>
<tr><td colspan="2">An infarct with punctate hemorrhages, consistent with ischemia-reperfusion injury, is present in the temporal lobe.</td><td colspan="2">A section of the brain showing a large discolored focally hemorrhagic region in the left middle cerebral artery distribution (hemorrhagic or red infarction).</td></tr>
</table>

Border Zone (Watershed) infracts

It's not a disease , a term to describe an infarction

Wedge-shaped areas of infarction that occur in those regions of the brain and spinal cord that lie at the most distal fields of arterial perfusion

It is usually seen after **hypotensive** episodes

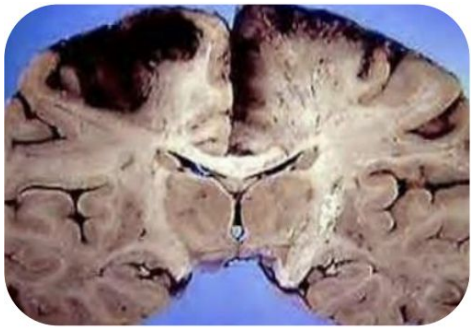
-In the cerebral hemispheres, the border zone between the anterior and the middle cerebral artery distributions is at greatest risk

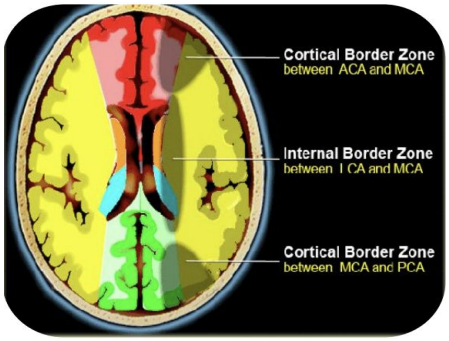
-Damage to this region produces a wedge shaped band of necrosis over the cerebral convexity a few centimeters lateral to the interhemispheric fissure

Clinical Note

Patients with stroke often present with :

- Rapid onset of focal central nervous system (CNS) signs and symptoms related to the distribution of the affected artery (stroke, cerebrovascular accident).
- The majority involve the territory of the middle cerebral artery of a cerebral hemisphere, resulting in varying degrees of contralateral hemiplegia and hemiparesis, homonymous hemianopia, and dysphasia.

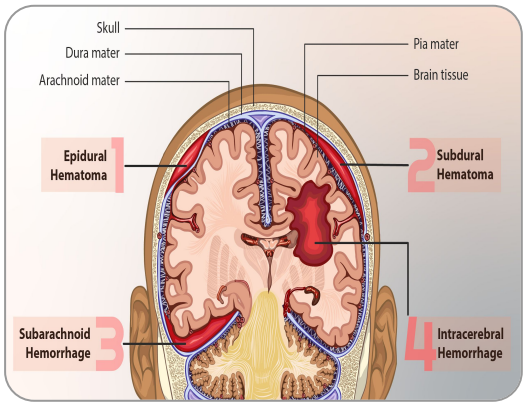




Types of Brain hemorrhage

Types

- Intracranial hemorrhage
- Subarachnoid Hemorrhage



Intracerebral Hemorrhage

- Hemorrhages within the brain tissue (intracerebral) can occur secondary to :

01 Hypertension

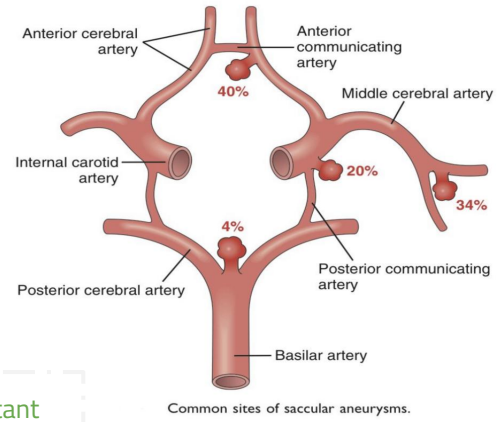
02 Other forms of vascular wall injury (e.g. vasculitis)

03 Arteriovenous malformation

04 An intraparenchymal tumor

05 Cerebral amyloid angiopathy

- Subdural epidural hemorrhages usually associated with trauma.
- Hemorrhages associated with the dura (in either subdural or epidural spaces) make up a pattern associated with trauma (discussed in another lecture)



Common sites of saccular aneurysms.

The most common site is branching point of **anterior communicating artery**

Dr. Note: This is important
Memorize the distribution with percentage

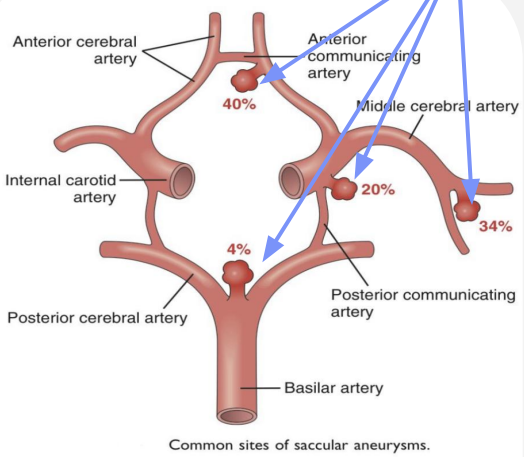
Subarachnoid Hemorrhage

Causes

	Rupture of a saccular (berry) aneurysm (The most frequent clinically significant cause).
	Vascular malformation
	Trauma (in which case it is usually associated with other signs of the injury)
	Rupture of an intracerebral hemorrhage into the ventricular system
	Hematologic disturbances
	Tumors

IMPORTANT

Berry aneurysm



Common sites of saccular aneurysms.

The most common site is branching point of **anterior communicating artery**

"This is very important subject especially in young people it's called **Berry aneurysm:**
It is an aneurysm around circle of willis , the bleeding will happen in the subarachnoid layer , it's congenital (but not from birth) It takes time to grow,
can lead to rupture and severe hemorrhage

MEMORIZE THE DISTRIBUTION.

Subarachnoid Hemorrhage

General info

Rupture can occur at any time, but in about one-third of cases it is associated with acute increases in intracranial pressure,such as with straining at stool or sexual orgasm.

Symptoms

Blood under arterial pressure is forced into the subarachnoid space, The patient experiences a sudden, excruciating headache and rapidly lose consciousness.

Location

About 90% of saccular aneurysms occur in the anterior circulation near major arterial branch points.

Source

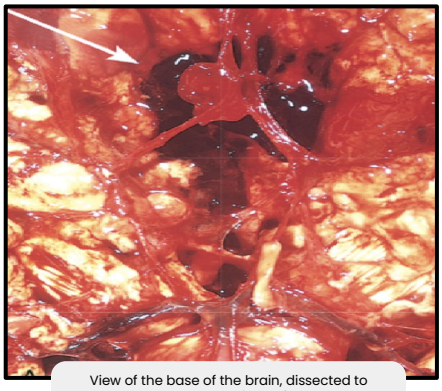
Multiple aneurysms exist in 20% to 30% of cases. Although they are sometimes referred to as *congenital,* they are not present at birth but develop over time because of underlying defects in the vessel media.

Risks

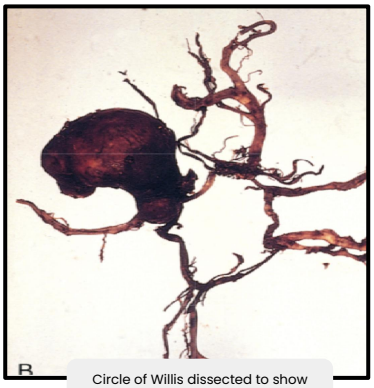
- The probability of aneurysm rupture increases with the size of the lesion, such that aneurysms greater than 10 mm have a roughly 50% risk of bleeding per year.
- In the early period after a subarachnoid hemorrhage, there is a risk of additional ischemic injury from vasospasm involving other vessels.

Subarachnoid Hemorrhage

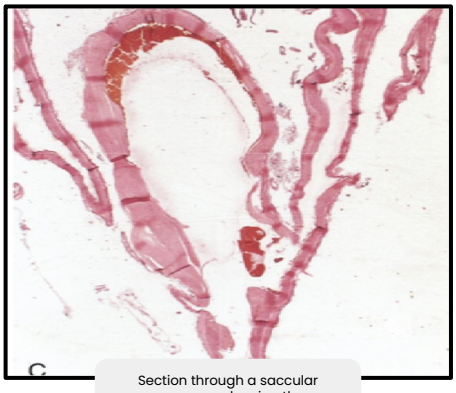
Recurring bleeding and prognosis	Healing phase
• Between 25% and 50% of individuals die with the first rupture, although those who survive typically improve and recover consciousness in minutes • Recurring bleeding is common in survivors; it is currently not possible to predict which individuals will have recurrences of bleeding • The prognosis worsens with each episode of bleeding	In the healing phase of subarachnoid hemorrhage: 1-meningeal fibrosis and scarring occur 2-sometimes leading to obstruction of CSF flow 3-interruption of the normal pathways of CSF resorption, Lead to secondary hydrocephalus.



View of the base of the brain, dissected to show the circle of Willis with an aneurysm of the anterior cerebral artery(arrow).



Circle of Willis dissected to show the large aneurysm.



Section through a saccular aneurysm showing the hyalinized fibrous vessel wall

Deep Focus Question

How does atrial fibrillation increase the risk of stroke?

A. Atrial fibrillation can cause hypertension, leading to a stroke.
B. Inflammation of the blood vessels occurs with atrial fibrillation, resulting in a stroke.
C. Rapid heart rate can lead to hypotension, causing a stroke.
D. Atrial fibrillation increases the risk of bleeding and can cause a hemorrhagic stroke.
E. Thrombi can form in the left atrial appendage and then embolize into the cerebral circulation, causing a stroke.

Answer: E

Hypertensive Cerebrovascular Disease

Hypertension affects the deep penetrating arteries and arterioles that supply the basal ganglia and hemispheric white matter and the brain stem.

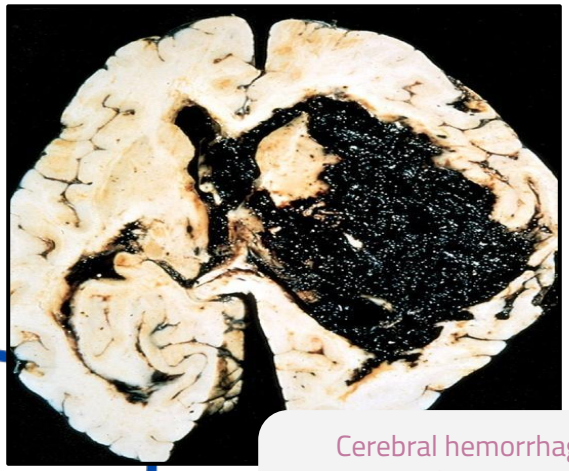
It causes several changes, including hyaline arteriolar sclerosis in arterioles which lead to weaker than normal vessels and are more vulnerable to rupture.

In some instances, chronic hypertension is associated with the development of minute aneurysms in vessels that are less than 300 µm in diameter that are termed as Charcot-Bouchard microaneurysms (lots of tiny aneurysms), which can rupture.

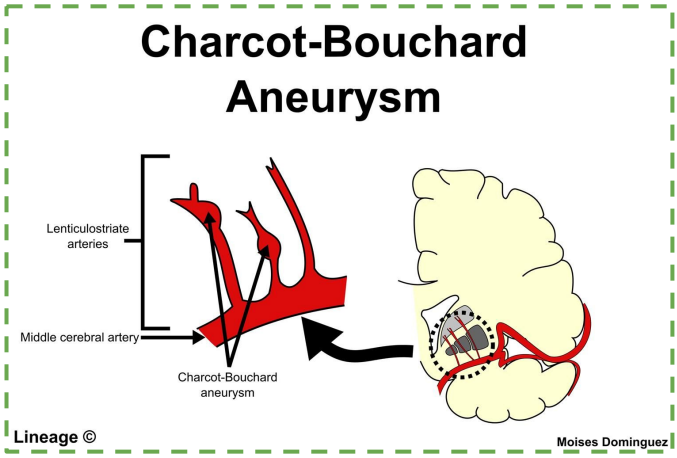
The most important effects of hypertension on the brain include:

- Massive hypertensive intracerebral hemorrhage (most important)
- Lacunar infarcts
- Slit hemorrhages
- Hypertensive encephalopathy

effects the entirety of brain function



Cerebral hemorrhage. Massive hypertensive hemorrhage rupturing into a lateral ventricle.



Hypertensive Cerebrovascular Disease

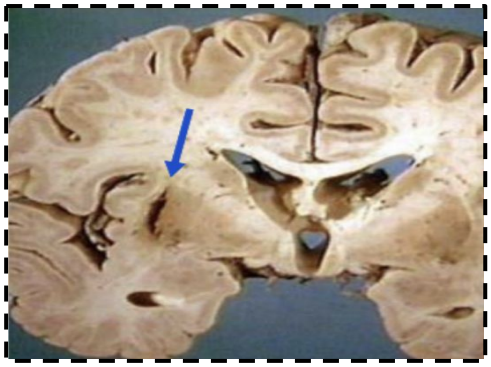
Lacunar infarcts

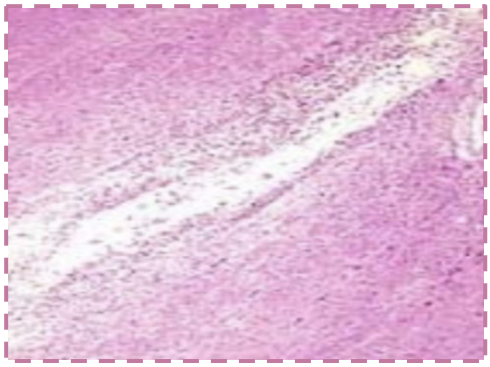
1. Small cavitary infarcts
2. They are most commonly in the deep gray matter (basal ganglia and thalamus), internal capsule, deep white matter, and pons. (locations are important)
3. Lacunar infarcts consist of cavities of tissue loss with scattered lipid-laden macrophages and surrounding gliosis.
4. Depending on their location in the CNS, lacunas can either be clinically silent or cause significant neurologic impairments.

Slit hemorrhages

- They are the rupture of small-caliber penetrating vessels and the development of small hemorrhages.
- In time, these hemorrhages are resorbed, leaving behind a slit-like cavity surrounded by brownish discoloration.

a common site for slit hemorrhages is the eye's retina





Hypertensive Cerebrovascular Disease

Acute hypertensive encephalopathy

A clinicopathologic Syndrome

Diffuse cerebral dysfunction, including headaches, confusion, vomiting, and convulsions, sometimes leading to coma.

Does not usually remit spontaneously.

May be associated with an edematous brain, with or without transtentorial or tonsillar herniation.

Petechiae and fibrinoid necrosis of arterioles in the gray and white matter may be seen microscopically.

Extra INFO

Transtentorial Herniation:
occurs when brain tissue is forced downward through the opening in the tentorium cerebelli, a structure that separates the cerebrum from the cerebellum within the skull. This type of herniation can exert pressure on vital brain structures, potentially leading to altered consciousness, compression of the brainstem, and life-threatening consequences.

Tonsillar Herniation:
when the lower part of the cerebellum, called the cerebellar tonsils, is pushed downward through the foramen magnum, the opening at the base of the skull. This can compress the brainstem and interfere with vital functions, such as respiration and heart rate regulation, and it is a potentially life-threatening condition.

Vasculitis

Definition

Infectious arteritis of small and large vessels

Previously seen in association with syphilis and tuberculosis.

Now more commonly occurs in the setting of immunosuppression and opportunistic infections (such as toxoplasmosis, aspergillosis, and CMV encephalitis).

Systemic forms of vasculitis, such as polyarteritis nodosa, may involve cerebral vessels and cause single or multiple infarcts throughout the brain.

Primary angiitis of the CNS:

Affected individuals manifest a diffuse encephalopathic clinical picture, often with cognitive dysfunction.

01 An inflammatory disorder that involves multiple small to medium-sized parenchymal and subarachnoid vessels.

02

03 Improvement occurs with steroid and immunosuppressive treatment.

FEMALES SLIDES

Vascular Malformations

They are classified into four principal types based on the nature of the abnormal vessels:

- Arteriovenous Malformations (AVMs) — AVMs, the most common of these, and affect males twice as frequently as females
- Cavernous Malformations
- Capillary Telangiectasias
- Venous Angiomas

They most commonly manifest between the ages of 10 and 30 years with seizures, an intracerebral a subarachnoid hemorrhage.

The risk of bleeding makes AVM the **most dangerous** type of vascular malformation.

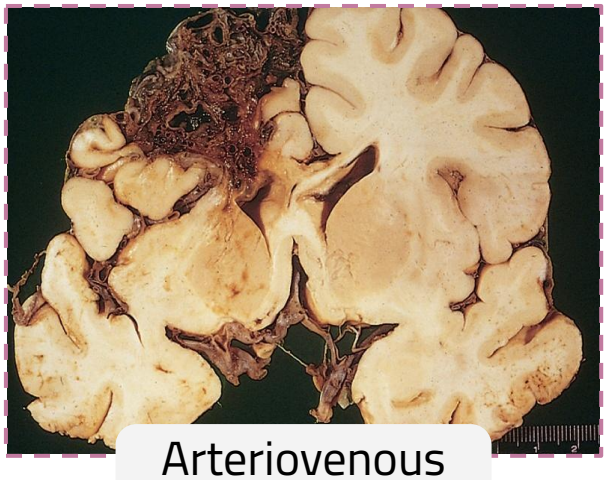
Multiple AVMs can be seen in the setting of hereditary hemorrhagic telangiectasia, an autosomal dominant condition often associated with mutations affecting the TGFβ pathway.

Deep Focus Question

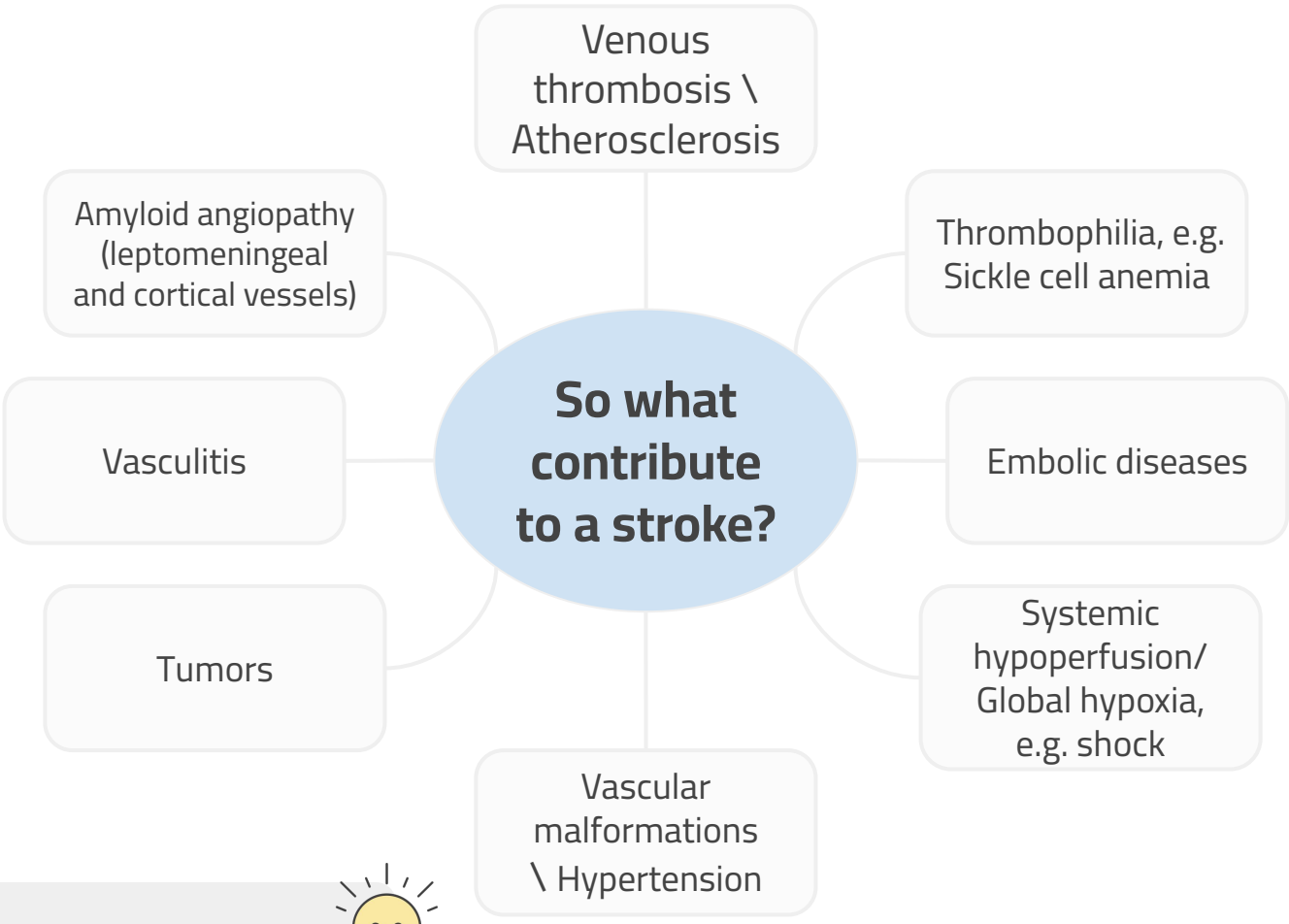
What is a cause of a focal ischemic stroke due to inflammation of a blood vessel?

A. Atrial fibrillation
B. Temporal arteritis
C. Hypertension
D. Heart valve disease

Answer: B



Arteriovenous Malformation



Did you know!!

Brain tissue ceases to function if deprived of oxygen for more than 60 to 90 seconds and after approximately three hours, will suffer irreversible injury possibly leading to death of the tissue

Take Home Messages

- Stroke is the clinical term for a disease with acute onset of a neurologic deficit as the result of vascular lesions, either hemorrhage or loss of blood supply.
- Cerebral infarction follows loss of blood supply and can be widespread, focal or affect regions with the least robust vascular supply ("watershed" infarcts).
- Focal cerebral infarcts are most commonly embolic; if there is subsequent fragmentation of an embolism, a non-hemorrhagic infarct can become hemorrhagic.
- Primary intraparenchymal hemorrhages are typically due to either hypertension (most commonly in white matter, deep gray matter, or posterior fossa contents) or cerebral amyloid angiopathy.
- Spontaneous subarachnoid hemorrhage is usually caused by a structural vascular abnormality, such as an aneurysm or arteriovenous malformation.

Keywords

Ischemic stroke	Thrombotic	• atherosclerosis • In carotid bifurcation , MCA , basilar artery
	Embolic	• in the MCA • due to Cardiac mural thrombi (Atrial fibrillation) • Paradoxical emboli
Global cerebral ischemia	• Widespread • Due to hypotension or Cardiac arrest • Progress to coma or brain death " respirator brain"	
	12h- 24h	• Red neurons: micro vacuolization & nuclear pyknosis & karyorrhexis
	24 h - 2w	• neutrophils infiltration • Necrosis • macrophages Influx • reactive gliosis
	Repair > 2w	• Gliosis
Focal cerebral ischemia	• Sites without collateral supply eg . deep penetrating vessels of : Thalamus & Basal ganglia & Deep white matter	
Non-Hemorrhagic Infarcts	Grossly	• first 6h : no change • 48 h : pales soft & swollen tissue • 2-10 d : gelatinous, friable tissue & edema resolve • 10 d - 3W : fluid-filled cavity
	Microscopically	• >12 h : red neurons & edema • 48h : macrophages influx • Months : gliosis
Hemorrhagic Infarcts	Microscopically	• petechial hemorrhages • addition of blood extravasation & resorption.
Border Zone (Watershed) infarcts	• Wedge-shaped areas • distal fields of arterial perfusion • after hypotensive episodes • Zone between ACA & MCA	

Keywords

brain hemorrhage	Intracerebral	• Hypertension , vasculitis , tumors • Subdural or epidural hemorrhages associated with trauma. In bridging veins
	Subarachnoid	• rupture of a saccular (berry) aneurysm • Causes acute increase in ICP • headache & rapid lose consciousness • In anterior circulation
Hypertensive Cerebrovascular Disease	• Effect arteries supplying basal ganglia & hemispheric white matter and the brain stem. • hyaline arteriolar sclerosis • Charcot-Bouchard microaneurysms	
	Lacunar infarct	• In deep gray matter (basal ganglia and thalamus), internal capsule, deep white matter, and pons. • Has lipid-laden macrophage
	Slit haemorrhage	• Rapture of small caliber penetrating vessels • slit-like cavity surrounded by brownish discoloration
	Acute hypertensive encephalopathy	• Petechiae and fibrinoid necrosis of arterioles • edematous brain
Vasculitis	• Infection with : TB & syphilis Previously • Infection with : toxoplasmosis, aspergillosis, and CMV encephalitis in immunosuppression patients	
Vascular Malformation	arteriovenous malformations	• most dangerous • setting of hereditary hemorrhagic telangiectasia • autosomal dominant condition associated with TGFβ • pathway mutation
	Other	• cavernous malformations. • capillary telangiectasias. • venous angiomas.

MCQ

Most common site of berry (saccular) aneurysm?

A- Anterior cerebral artery

B- Middle cerebral artery

C- Posterior cerebral artery

D- Internal cerebral artery

Which of the following is the pathogenesis of WATERSHED strokes?

A- Fat embolism

B- Severe hypertension

C- Prolonged hypotension

D- Vasculitis

What is the most likely cause of lacunar infarction?

A- Hypertension

B- Multiple sclerosis

C- Berry aneurysm

D- Vascular malformation

The majority of thrombotic occlusions causing cerebral infarctions are due to?

A- Atherosclerosis

B- Hypertension

C- Rupture of berry aneurysm

D - Embolic stroke

1-A / 2-C / 3-A / 4-A

MCQ

Which of the following is the most common site of embolic thrombi in patient with atherosclerosis that cause embolic infarction?

A- External carotid artery

B- Internal carotid artery

C- Middle cerebral artery

D- Basilar artery

Chronic hypertension in hypertensive cerebrovascular disease is characterised by?

A- fibrinoid necrosis

B- opportunistic infections

C- Charcot- Bouchard microaneurysm

D- seizures

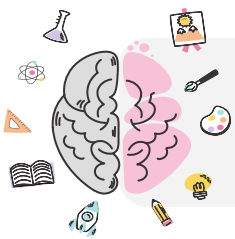
80 years old male came to the ER after being found collapsed on the ground. Unfortunately he died in the hospital from an infarction. A biopsy was done, which of the following microscopic features would you find in an early period of infraction?

A- Neutrophilic infiltration

B- Macrophages infiltration

C- Liquefactive Necrosis

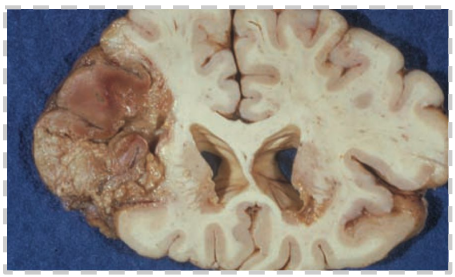
D- No major manifestation



Need a SUMMARY ? Click here

1-C / 2-C / 3- A

Cases

1.A 68-year-old obese woman (BMI = 34 kg/m2) suffers a stroke and expires. Histologic examination of the brain at autopsy reveals extensive arteriolar lipohyalinosis and numerous Charcot-Bouchard aneurysms. Which of the following best accounts for the pathogenesis of these autopsy findings?			
A.Atherosclerosis	B.Autoimmunity	C.Diabetes	D.Hypertension
2.A 56-year-old man is rushed to the emergency room after collapsing while shoveling snow. The patient has no pulse on admission but is resuscitated. Laboratory studies show elevated serum levels of cardiac-specific proteins, and ECG confirms a transmural infarct of the left ventricle. The patient expires 2 weeks later of cardiac tamponade. Examination of the patient's brain at autopsy would most likely reveal necrosis of Purkinje cells and selective loss of neurons in which of the following regions?			
A.Frontal lobes	B.Hippocampus	C.Hypothalamus	D.Thalamus
3.A 30-year-old woman suffers massive trauma in an automobile accident and suffered from oblique displacement fracture of femur and expires 4 days later of respiratory insufficiency. A horizontal section of the patient's brain at autopsy reveals numerous petechiae scattered throughout the white matter. Which of the following is the most likely explanation for this pathologic finding?			
A.Fat embolism	B.Sepsis	C.Uremia	D.Global ischemia
4.A 67-year-old man with a history of ischemic heart disease is rushed to the emergency room after collapsing in his garden. A CT scan demonstrates a large infarct of the left frontal lobe. The patient dies, and the brain is examined at autopsy (show in the image). This lesion was caused by thrombosis of which of the following blood vessels? 			
A.External carotid artery	B.Internal carotid artery	C.Middle cerebral artery	D.vertebral artery

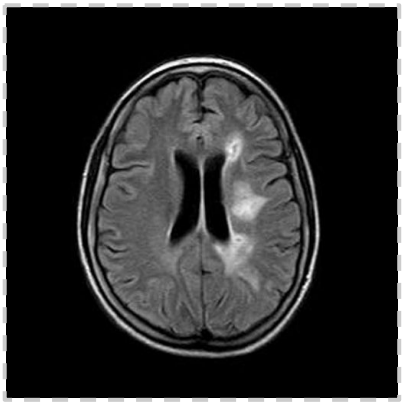
1-B / 2-B / 3-A / 4-C

NEED EXPLANATION ? CLICK HERE

Cases

EXTRA CASES REQUIRE EXTRA INFO

1.A 22-year-old man is brought into the emergency department by emergency medical services after being found face down on the sidewalk. The patient has a temperature of 36°C (96.8°F), pulse of 62/min and regular, blood pressure of 102/72 mmHg and respiratory rate of 7/min, and O2 saturation of 75% on room air. He is stuporous, and pupils are pinpoint. He is subsequently given naloxone. Repeat vital signs are temperature of 36°C (96.8°F), pulse of 72/min, blood pressure of 107/71 mmHg and respiratory rate of 14/min, and O2 saturation of 95% on room air. The patient remains stuporous following administration of this medication. The patient is subsequently intubated and admitted to the intensive care unit, and an MRI of the head reveals the following image:



Which of the following is the most likely etiology of this patient's clinical condition?

A.Prolonged cerebral hypoperfusion	B.Rupture of bridging veins	C.Middle meningeal artery tear	D.Small-vessel lipohyalinosis

2.A 78-year-old right-handed man is brought to the emergency department following sudden-onset weakness in his right arm and inability to speak for twenty four hours. The patient's daughter states she initially became concerned when he dropped his cup of coffee while walking to the kitchen table last night. The daughter states he has had a similar episode in the past that resolved spontaneously. He has a history of hypertension, for which he takes lisinopril. His temperature is 37°C (98.6°F), pulse is 92/min, and blood pressure is 158/104 mmHg. The patient is alert and visibly frustrated by not being able to speak. Physical examination reveals 2/5 strength in the right upper extremity and 4/5 strength in the right lower extremity. He follows written and verbal commands but is unable to speak or write. MRI of the head demonstrates ischemic changes in the cerebral territory supplied by the left middle cerebral artery. Which of the following histopathological findings are most likely to be observed in this patient's brain at the present time?

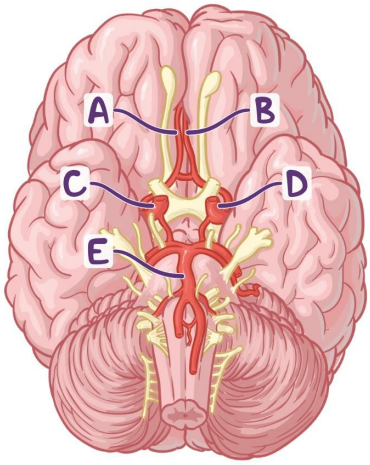
A.Astrocytic and vascular proliferation	B.Glial scar formation	C.No observable histopathological changes	D.Eosinophilic neuronal cytoplasm and pyknotic nuclei

1-A / 2-D

Cases

EXTRA CASES REQUIRE EXTRA INFO

3.An 81-year-old right-handed woman is brought to the emergency department for evaluation of sudden onset weakness in the right upper and right lower extremity. The patient's partner states she fell while walking to the kitchen and several minutes later noticed that the patient urinated on herself. The partner denies shaking of the limbs or head trauma. The patient denies confusion, loss of consciousness, or tongue biting. Her past medical history includes hypertension and hyperlipidemia for which she is taking lisinopril and atorvastatin. Temperature is 37°C (98.6°F), pulse is 92/min, and blood pressure is 158/104 mmHg. The patient is alert and oriented. Physical examination reveals full visual fields with no defects. There is 4/5 strength in the right upper extremity and 2/5 strength in the right lower extremity. Finger-to-nose testing is normal and without dysdiadochokinesia. Which of the following vessels labeled in the diagram below is most likely implicated in this patient's clinical condition?



A	B	C	D

3- B

PATHOLOGY TEAM

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- ساره العجاجي
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- فيصل الشويعر
- رغد المصلح
- عروب المحمود
- يزيد ال طلحه
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- الحوراء العوامي
- لؤي الحديثي
- يزيد المطيري

- هياء العجمي
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- محمد معشي
- أفنان الأحمري
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