Medical Academy named after S.I. Georgievsky of V.I. Vernadsky CFU
Department of Neurology and Neurosurgery

Class 9

Cerebrovascular diseases. Ischemic Stroke.

1. Goals:

1.1. To study the Anatomical fundamentals of the Brain Blood Circulation.

1.2. To study the Neurological fundamentals of the Ischemic Stroke.

2. Basic questions:

2.1. Ischemic Stroke:

2.1.1. *Etiology.*

2.1.2. *Pathogenesis.*

2.1.3. *Clinical Features.*

2.1.4. *Diagnostic evaluation.*

2.1.5. *Treatment.*

2.1.6. *Prophylaxis. Prognosis.*

Literature:

Mark Mumenthaler, M.D., Heinrich Mattle, M.D. Fundamentals of Neurology. - 2006 - P.98-109.

The term ***"stroke"*** encompasses both ischemic and hemorrhagic disturbances of the cerebral circulation producing central neurological deficits of acute or subacute onset. Ischemia accounts for 80 to 85% of stroke, hemorrhage for 15 to 20%.

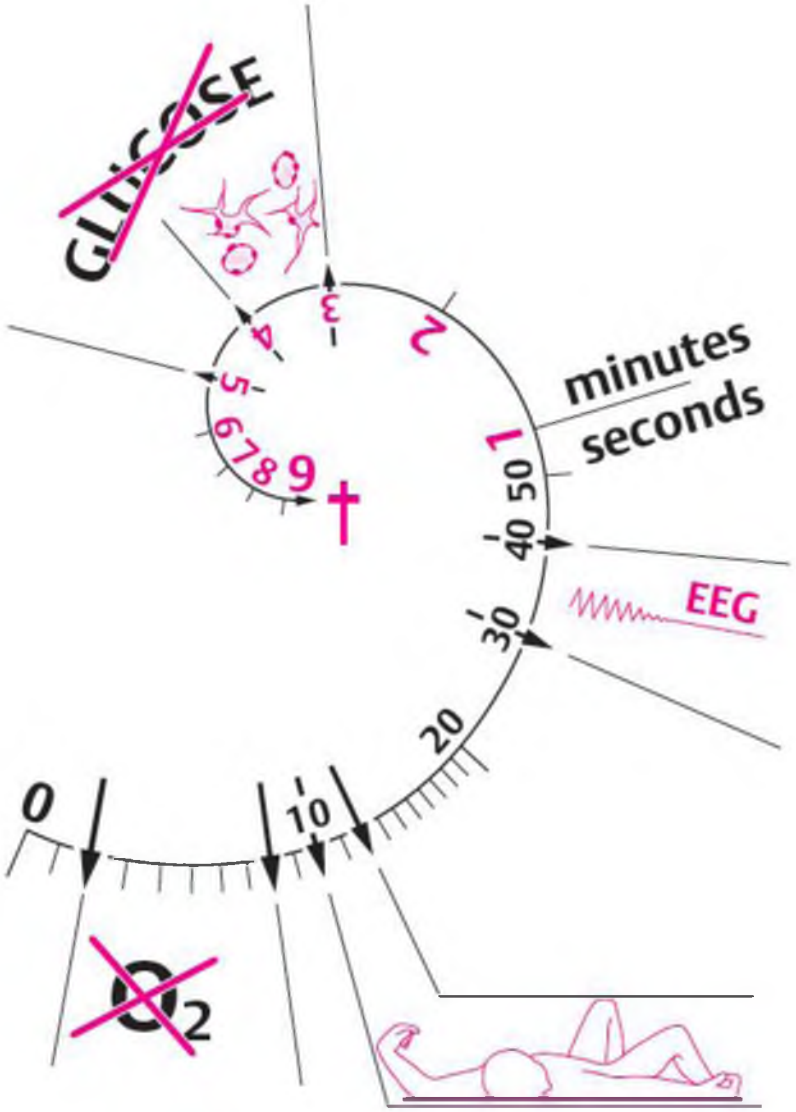
Cerebral Ischemia

Ischemia causes **critical hypoperfusion** in an area of the brain. Depending on its extent and duration, hypoperfusion can induce neurological deficits that may be either **transient** (TIA, RIND) or **permanent** (completed stroke, infarction). The more common causes of ischemia are **blockage of the arterial blood supply** by arteriosclerotic processes of both larger and smaller blood vessels (**macroangiopathic and microangiopathic processes**) and **embolic events** (arterio-arterial and cardiogenic embolization). A less common cause is obstruction of **venous outflow** (e. g., venous sinus thrombosis). Every ischemic event should prompt thorough diagnostic evaluation to identify its etiology, so that effective measures can be taken to prevent a recurrence.

Nontraumatic Intracranial Hemorrhage

Regulation of cerebral perfusion. Glucose is the brain's nearly exclusive source of energy. The brain accounts for only about 2% of body weight, but it receives about 15% of the cardiac output. *Regulatory mechanisms* ensure that the cerebral perfusion remains constant despite fluctuations in the arterial blood pressure, as long as the latter remains within a certain range. Thus, if the arterial blood pressure should fall, a compensatory dilatation of the cerebral arteries occurs to maintain cerebral perfusion, which is significantly reduced only when the systolic blood pressure falls below 70mmHg (or below 70% of the baseline value in hypertensive individuals). Hyperventilation and elevated intracranial pressure reduce cerebral perfusion, while hypoventilation (i. e., an elevated partial pressure of CO2) increases it.

Consequences of ischemia. Normal *cerebral perfusion* is ca. 58 mL per 100 g brain tissue per minute. Signs and symptoms of ischemia begin to appear when perfusion falls below 22 mL per 100 g per min. In this stage of **relative ischemia**, the functional metabolism of the affected
brain tissue is impaired, but the *infarction threshold* has not yet been reached and the tissue can regain its normal function as soon as the perfusion renormalizes. The longer relative ischemia lasts, however, the less likely it is that normal function will be regained. The zone of tissue in which the local cerebral perfusion lies between the functional threshold and the infarction threshold is called the **ischemic penumbra** ("partial shadow"). Total ischemia causes irreversible structural damage
of the affected region of the brain. If the blood supply of the entire brain is cut off, unconsciousness ensues in 10 to 12 seconds and cerebral electrical activity, as demonstrated by EEG, ceases in 30 to 40 seconds (See the Figure below).

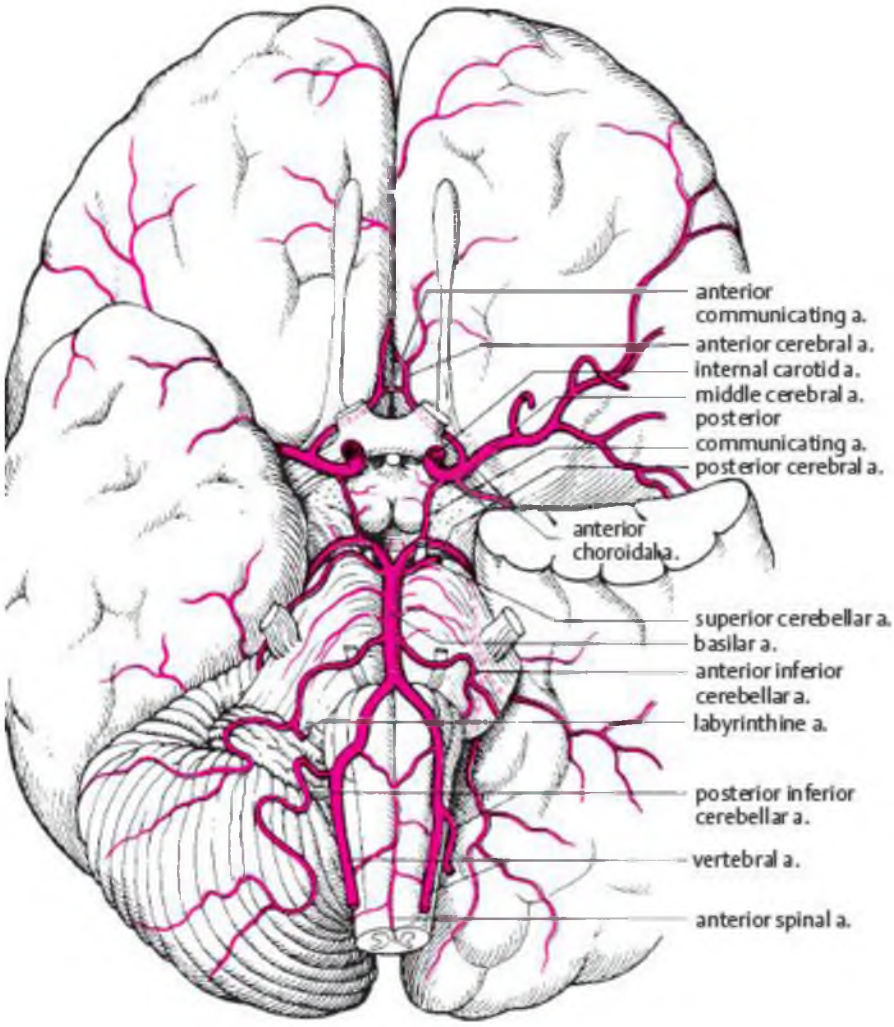


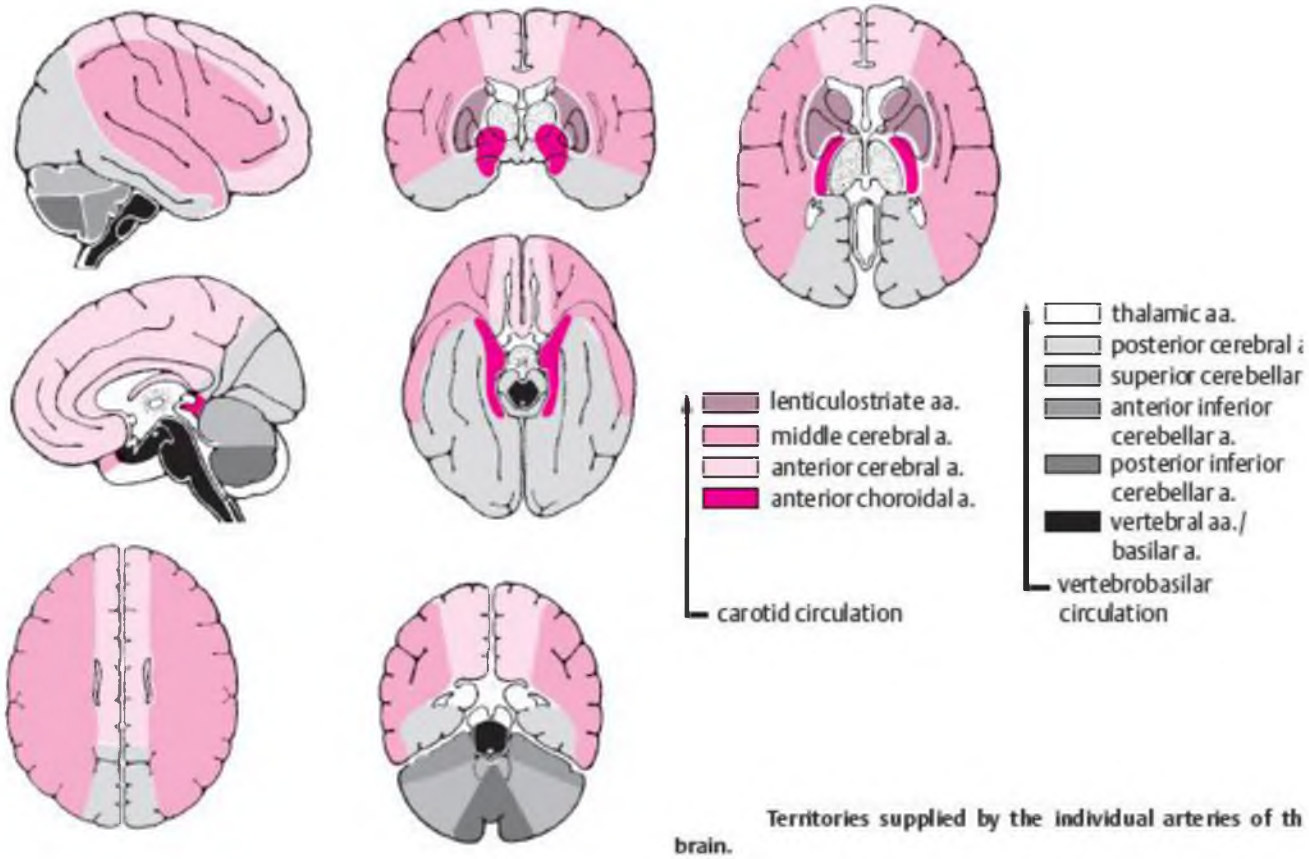
Time course of cerebral ischemia. Diagram of the effect of sudden total deprivation of blood supply to the brain on tissue metabolism, consciousness, the EEG, neuronal morphology, and tissue glucose concentration.

Cellular metabolism collapses, the sodium/potassium pump ceases to function, and interstitial fluid—i. e., sodium and water—flows into the cells. The resulting cellular swelling is called **cytotoxic cerebral edema**. Later, when the blood–CSF barrier collapses, further plasma components, including osmotically active substances, enter the brain tissue; a net flow of fluid from the intravascular space into the intercellular and intracellular spaces then produces **vasogenic cerebral edema**. In a vicious circle, these two varieties of edema lead to additional compression of brain tissue, thereby impairing the cerebral perfusion still further.

Dynamic time course of cerebral ischemia. Cerebral perfusion can cause a wide variety of clinical manifestations. In clinical practice, these are often classified by their temporal course and their degree of reversibility or irreversibility. Although classification in this way is useful, it says nothing about the underlying etiology of the ischemic events. Moreover, the boundaries between the listed entities (e. g., TIA and RIND) are not sharp.

Arterial blood supply of the brain. To understand how the localization and extent of cerebral infarcts depends on the particular artery that is occluded, one must know the anatomy of the territories of the individual vessels, as well as their numerous anastomoses. The anastomotic arterial *circle of Willis*, at the base of the brain, provides a connection between the carotid and vertebral circulations and between the blood supplies of the right and left cerebral hemispheres (See the Figure below). The territories of the major cerebral arteries are shown in Fig below.





Territories supplied by the individual arteries of th brain.

Ischemic Stroke

Ischemic stroke occurs when persistent ischemia or a complete interruption of the blood supply to a particular area of the brain produces **irreversible destruction of brain tissue**. The resulting neurological deficits usually arise quite suddenly (whence the term "stroke") but can sometimes progress over a longer period of time ("stroke in evolution"). They are irreversible, or at most only partly reversible.

Etiology.

Ischemic stroke has many causes.

Embolic events and **vascular stenosis due to atherosclerosis** play important roles, as do hypertensive atherosclerotic changes of medium-caliber or small cerebral arteries.

! The most important risk factor for stroke is arterial hypertension.

The major etiologies of ischemic stroke are summarized in Table below.

Classification of ischemic stroke by etiology

I.	**Atherosclerosis of large extra- and intracranial vessels,** leading to: ● thrombosis in the region of an atherosclerotic plaque, ● hemodynamic insufficiency in the poststenotic circulation, or ● arterio-arterial embolism
II.	**Cardiogenic and aortogenic embolism**
III.	**Cerebral small vessel disease/arteriolosclerosis,** usually due to hypertension
IV.	**Other etiologies,** e. g.: ● vasculopathies ● coagulopathies
V.	**Undetermined etiology**

The acute symptoms are sometimes produced by a sudden drop in blood pressure. The major risk factors for atherosclerosis and ischemic stroke are listed in Table below.

Risk factors for atherosclerosis and ischemic stroke

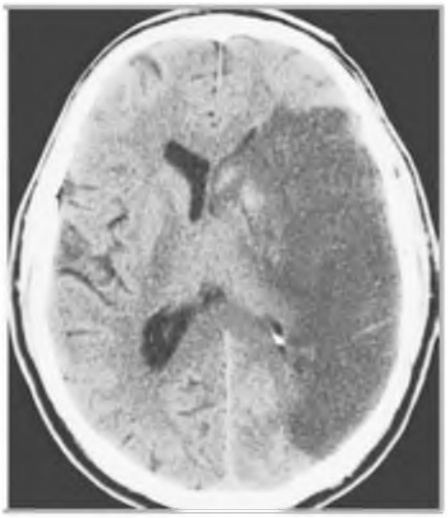
Positive family history of early onset of atherosclerotic disease (< 55 years of age)
Arterial hypertension
Cigarette smoking
Truncal obesity, hypercholesterolemia
Diabetes mellitus
Sleep apnea syndrome
Past history of cardio- or cerebrovascular disease or occlusive peripheral vascular disease

Pathogenesis of the ischemic stroke.

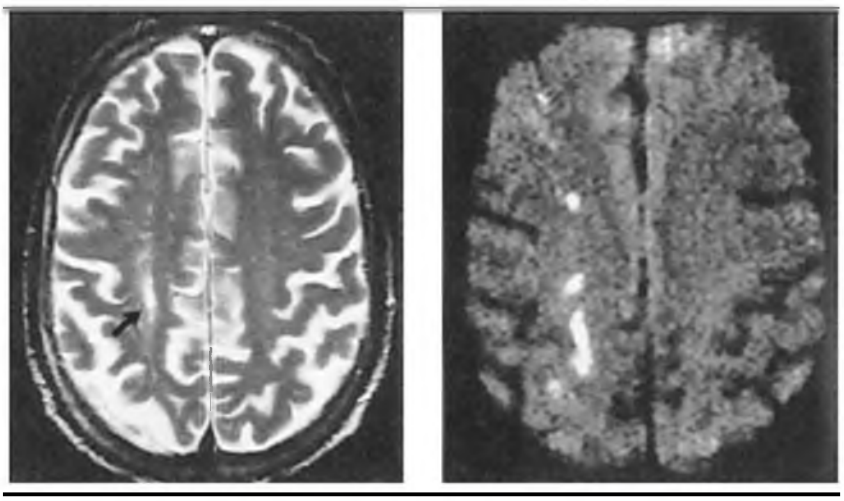
The Ischemic Cascade

1. Oxygen deficiency prevents ATP production
2. Loss of ATP leads to rapid loss of potassium
3. Resultant depolarization opens voltage-sensitive calcium channels and glutamate release
4. Overstimulation of glutamate receptors leads to uncontrolled calcium entry into neurons, further decreasing ATP
5. Activation of NOS leads to free radical formation
6. Free radical formation leads to cell death

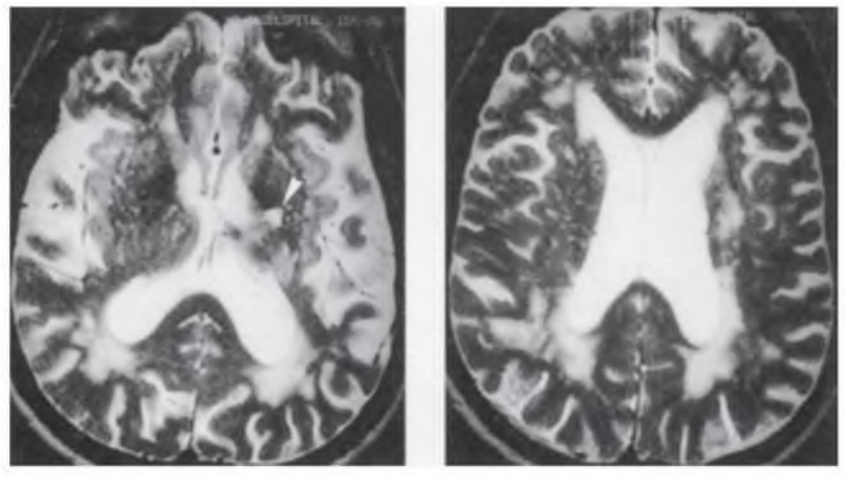
Types of infarct. There are *three basic types of brain infarct*, distinguished from each other by the caliber of the occluded arteries:



Territorial infarcts are mainly produced by *occlusions of the main trunks or major branches of cerebral arteries* (cerebral macroangiopathy), which may be due to thrombosis, embolism, or other causes. The infarct includes both cortex and subcortical white matter and sometimes the basal ganglia and thalamus (see the Figure on the left). It is usually possible to infer which vessel has been occluded from the pattern of neurological deficits that are produced.



Watershed infarcts (border zone infarcts) are infarcts of hemodynamic origin that are likewise due to microangiopathic processes. Narrowing of small vessels *impairs perfusion in the vulnerable regions at the borders between the territories of two or more arteries* (Figure). If the perfusion pressure is inadequate, infarction ensues.

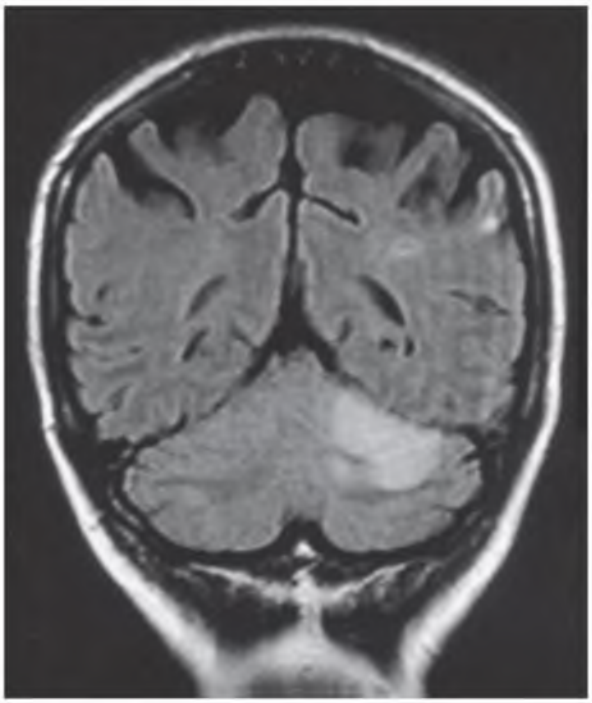


Lacunar infarcts are caused by microangiopathy (usually atherosclerosis of small vessels due to hypertension). The infarcts (lacunes) are less than 1.5 cm in diameter and often multiple. They are found mainly in the basal ganglia, thalamus, and brainstem, and sometimes in the cerebral cortex and subcortical white matter. Their clinical presentation depends on their number and localization. Multiple subcortical infarcts due to hypertension are the hallmark of *subcortical arteriosclerotic encephalopathy*, also called Binswanger disease.

Signs and Symptoms of Ischemic Stroke.

The neurological deficits produced by ischemic stroke depend on the area of the brain that is ischemic or infarcted.

Ophthalmic a.	Transient ischemia in the territory of this vessel produces **amaurosis fugax** (transient monocular blindness), while longer-lasting ischemia causes retinal infarction. Retinal ischemia is often due to embolism of cholesterol crystals from ulcerating plaques in the internal carotid a. into the ophthalmic a. Embolized crystals within the arteries of the retina can occasionally be seen by ophthalmoscopy.
Internal carotid a.	Stenosis or occlusion of this vessel can cause simultaneous ischemia of the eye with monocular visual loss (see above) and contralateral hemiparesis, in combination with neuropsychological deficits.
Middle cerebral a. (MCA).	The site of occlusion (main trunk vs. branch of the middle cerebral a.) determines the clinical manifestations. As a rule, a mainly brachiofacial hemiparesis and hemisensory deficit are found, often accompanied by *homonymous hemi- or quadrantanopsia* and, in the initial phase, a *horizontal gaze palsy* toward the side of the hemiparesis. An MCA occlusion on the language-dominant (usually left) side additionally produces *aphasia* and *apraxia*, while one on the nondominant side produces *impairment of spatial orientation.* An occlusion of the main stem of the MCA causes ischemia not only of the cortex, but also of the basal ganglia and internal capsule, producing a more severe contralateral hemiparesis. If the hemiparesis fails to improve over time, or does so only partially, a typical, permanent impairment of gait results: circumduction of the spastically extended lower limb, flexion of the paretic upper limb at the wrist and elbow, and absence of arm swing on the affected side (***Wernicke–Mann gait***) (see Figure below).  **Typical gait disturbance of a hemiplegic patient.** Circumduction of the spastically paretic leg with predominant extensor tone, and flexion of the spastically paretic arm at the elbow because of predominant flexor tone.
Anterior choroidal a.	Ischemia in the territory of this vessel causes a homonymous visual field defect, a hemisensory deficit and hemiparesis. The clinical manifestations resemble those of occlusion of the lenticulostriate aa. (branches of the middle cerebral a. supplying the

	basal ganglia and internal capsule). There may also be extrapyramidal motor signs, such as hemiballism.
Anterior cerebral a.	An infarct in the territory of this artery causes contralateral hemiparesis mainly affecting the lower limb, sometimes accompanied by contralateral ataxia and, if the lesion is left-sided, by apraxia. Occasionally there may be apathy, abulia (pathological lack of drive and motivation), and urinary incontinence.
Posterior cerebral a.	Occlusion of this artery can produce infarction in the cerebral peduncle, the thalamus, mediobasal portions of the temporal lobe, and the occipital lobe. The most prominent clinical sign of a distal occlusion (beyond the origin of the posterior communicating a.) is contralateral homonymous hemianopsia, possibly combined with neuropsychological deficits and contralateral hemianesthesia.
Basilar a.	Occlusion of the main stem or of a branch of the basilar a. causes brainstem, cerebellar, and thalamic signs. Thalamic infarction results from occlusion of one of the arteries supplying the thalamus. A total occlusion can occur in the lower or middle third and involves a large number of bilateral structures. The complete syndrome includes: -Bilateral sensory and motor deficits -Variable cerebellar and cranial nerve deficits -Some patients are comatose due to the involvement of the reticular activating system -Other patients are quadriplegic, mute but conscious, the only eye movements present are vertical and it is their only means of communication ("locked-in syndrome")
Posterior inferior cerebellar a. (PICA).	*Wallenberg syndrome* -Inferior Cerebellar peduncle is affected -Ipsilateral ataxia: Inferior Cerebellar peduncle deficit -Vertigo, horizontal or rotatory nystagmus,nausea and vomiting (Vestibular deficits) -Decrease or absent temperature, pain and touch sensation from the *ipsilateral* face and *contralateral* body -Sometimes pain and paresthesia on ipsilateral face -Horner's syndrome -Hoarseness, dysphagia and decreased of gag reflex (nucleus ambiguus) -Decreased taste sensation on ipsilateral tongue (solitary nucleus)
Vertebro-basilar aa.	Cerebellar infarction presents with vertigo, nausea, unsteady gait, dysarthria, and often acute headache and progressive impairment of consciousness. The neurological examination reveals ataxia, dysmetria, and nystagmus.  Acute infarct in the left cerebellar hemisphere

Diagnostic Evaluation of Ischemic Stroke.

Diagnostic evaluation in the acute phase is focused on the determination of the *anatomic site and extent* of cerebral ischemia and, above all, its *etiology.*

Acute diagnostic evaluation. In pursuit of these goals, the initial workup should always begin with the following:

1) *precise history taking* concerning not only the present illness, but also the past medical history, with special attention to risk factors and systemic illnesses;
2) *a thorough clinical neurological examination* enabling localization of the lesion;
3) *examination of the cardiovascular system* (measurement of pulse and blood pressure and auscultation of the heart, the carotid aa., and perhaps other vessels, depending on the clinical situation; particular attention should be paid to bruits and to any irregularities of the pulse that suggest arrhythmia).

Ancillary testing in the acute phase. The following ancillary tests should also be performed on all stroke patients in the acute phase:

1) *Laboratory tests*, mainly for the identification of risk factors, infectious/inflammatory disorders, and coagulopathies (erythrocyte sedimentation rate, blood sugar, lipid profile, complete blood count and hemoglobin, coagulation profile, and sometimes protein C, antiphospholipid antibodies, syphilis serology, etc.).
2) *Imaging studies.* Even before these are performed, any central neurological deficit of acute onset is very likely to be due to a cerebrovascular accident, of which ischemic stroke is the most common type; yet neuroimaging is still indicated for definitive confirmation of the diagnosis. Any patient thought to be suffering from acute ischemic stroke should undergo **CT as soon as possible**, as this will have important implications for the course of treatment, even though areas of ischemia usually cannot be seen by CT till several hours after the onset of symptoms. Early CT does, however, reveal acute brain hemorrhage, if present. MRI can also be performed, if available. MRI reveals the infarct zone and perifocal edema as soon as the patient begins to experience symptoms and it displays brainstem and cerebellar infarcts more clearly than CT.
3) *Doppler ultrasonography* of the extra- and intracranial vessels to detect vascular stenosis, occlusion, and vascular collateralization.
4) An *electrocardiogram* (arrhythmia pointing to a likely cardioembolic event, old or acutemyocardial infarction, evidence of regional cardiac wall motion abnormalities, creating a danger of intracardiac thrombosis and embolism).

! When an ischemic stroke is suspected, the most important immediate question in the differential diagnosis is whether the patient is not, in fact, suffering from an intracerebral hemorrhage, rather than from cerebral ischemia. The history and physical examination alone cannot provide a reliable answer; therefore, a neuroimaging study must be performed.

Further diagnostic tests after the acute phase. Depending on the clinical situation, the following tests can also be performed after the acute phase:

1) *angio-MRI* to reveal stenosis of the carotid or vertebral a.;
2) transthoracic or transesophageal *echocardiography* to reveal potential sources of emboli in the heart and aortic arch, as well as any dysfunction of the heart valves;
3) *cerebral angiography* to reveal stenosis or occlusion of the cerebral blood vessels (also performed in the acute phase as a part of thrombolytic treatment);
4) *SPECT* to demonstrate impaired perfusion.

Treatment of Ischemic Stroke.

Once the diagnosis of ischemic stroke has been made and an intracerebral hemorrhage has been excluded, the initial goal of treatment is to minimize the amount of brain tissue that will be irreversibly damaged. Brain tissue in the zone of relative ischemia (the ischemic penumbra) can be "salvaged" by prompt restoration of its obstructed blood supply.

! Patients with suspected stroke should be immediately transported to an acute care hospital and admitted. Inpatient treatment markedly improves prognosis.

In parallel with the acute measures already discussed, a further treatment strategy should also be settled upon for long-term prevention of recurrent stroke. The appropriate strategy depends on the etiology of the infarct. General treatment strategies for ischemic stroke are as follows:

_ *keeping the blood pressure relatively high* (values up to 200−220 mmHg systolic and 110 mmHg diastolic are tolerable);

_ *stabilization of cardiovascular function* (adequate hydration, treatment of heart failure and/or arrhythmia, if present);

_ *treatment of cerebral edema*, if present; and

_ *in some patients*, *intravenous thrombolysis* within three hours of the onset of symptoms, or *intra-arterial thrombolysis* within six hours; if thrombolysis is contraindicated, aspirin is the drug of choice.

Optimization of oxygen and substrate delivery to the ischemic zone:

_ *monitoring of respiratory function* (with blood gas analyses, if necessary, and prophylaxis and treatment of pneumonia);

_ *treatment of pathological metabolic processes that elevate the demand for oxygen and energy* (e. g., treatment of fever, suppression of epileptic seizures); and

_ *optimal blood sugar management*, with prevention and, if necessary, treatment of hyper- or hypoglycemia.

Further therapeutic measures include rehabilitation and prophylactic measures against recurrent stroke:

_ Early rehabilitation: mobilization (decubitus prophylaxis), physical and occupational therapy, and, if needed, speech therapy.

Prevention of recurrent stroke:

_ *General medical treatment*: minimization of vascular risk profile (optimal treatment of hypertension, diabetes mellitus, hypercholesterolemia, or sleep apnea syndrome, if present, and cessation of smoking); treatment of heart failure and/or arrhythmia.

_ *Antithrombotic therapy*: the type to be given depends on the etiology of the initial stroke. The following options are available:

_ *inhibition of platelet aggregation* (mainly aspirin, but also clopidogrel or aspirin with dipyridamole);

_ *full heparinization* and *oral anticoagulation* (mainly after cardio- or aortoembolic stroke, basilar artery thrombosis, stroke in evolution, venous thrombosis, or venous sinus thrombosis; there is no consensus on other potential indications);

_ *surgical therapy*: endarterectomy for high-grade carotid stenosis, or insertion of an intravascular stent.

Nontraumatic Intracranial Hemorrhage

Nontraumatic intracranial hemorrhage is defined as a spontaneous hemorrhage into the brain parenchyma (**intracerebral hemorrhage**) or the cerebrospinal fluid space (**subarachnoid hemorrhage**).

Intracerebral hemorrhages cause acute signs and symptoms resembling those of cerebral ischemia and account for about 10% of strokes. One of the more common forms of intracerebral hemorrhage is hypertensive hemorrhage. The main symptom of subarachnoid hemorrhage is headache; its most common source is a ruptured aneurysm.

General manifestations of intracranial hemorrhage.

Though the manifestations of intracranial hemorrhage and cerebral ischemia are similar, generally speaking (sudden onset of focal neurological deficits), there are a number of clinical signs and symptoms that are more characteristic of hemorrhage than of ischemia. These include: acute *headache*, often accompanied by *vomiting*;

_ rapidly or very rapidly *progressive neurological deficits* (whose type depends on the site of hemorrhage);

_ progressive *impairment of consciousness*, perhaps leading to coma;

_ in many patients, *epileptic seizures.*

If these manifestations are present, an intracranial hemorrhage is the probable cause. The definitive diagnosis, however, can only be made with neuroradiological methods.

Score on number repeated:

0 = 5

1 = 4

2 = 2

3 = 0

Fluency _____

Score according to patient's spontaneous speech fluency, or

Ask patient to name as many words as he can within one minute beginning with the letter 'A' (excluding proper names)

Score as:

5 = Essentially no verbal output

3 = Moderately loss; inability to recognize stationary finger, sees moving finger 1 = Mild loss; defect to double simultaneous stimulation

0 = Normal

OTHER CORTICAL FUNCTIONS AND CRANIAL NERVES

Visual fields _____

Test clinically and score hemi-field loss as:

3 = Severe loss; inability to recognize moving hand, no response to threat

2 = Moderate loss; inability to recognize stationary finger, sees moving finger

1 = Mild loss: defect to double simultaneous stimulation

0 = Normal

Gaze _____

Score eye movements:

2 = Gaze play, or persistent deviation

1 = Gaze preference, or difficulty with far lateral gaze

0 = Normal

Facial expression _____

Score movement:

3 = Severe weakness; drooling

2 = Moderate loss; asymmetry at rest

1 = Mild weakness; asymmetry on smiling

0 = Normal

Dysarthria _____

Score talking:

2 = Severe dysarthria

1 = Moderate dysarthria

0 = Normal

Dysphagia _____

Score swallow of glass water:

2 = Severe dysphagia

1 = Moderate dysphagia

0 = Normal

Neglect syndrome _____

Ask about weak limbs, and ask to bisect a line 7 inches (20 cm) long on piece of paper in visual midline

Score:

2 = Anosoagnosia, or denial of body part

1 = Consistently bisects line towards 'good' side of body

0 = Bisects line in middle

Visual construction _____

Ask patient to copy three figure given, and score:

3 = Unable to copy any figure

2 = Can copy a square

1 = Can copy a 'Greek Cross' ('Cross of St. George')

0 = Can copy 3D drawing of cube

MOTOR FUNCTION

Arm, proximal _____

Arm, distal _____

Leg, proximal _____

Leg, distal _____

All scored 0-7 as:

7 = No movement (MRC 0)

6 = Trace movement only (MRC 1)

5 = Motion without gravity only (MRC 2)

4 = Moves against gravity but not against resistance (MRC 3)

3 = Moderate weakness (MRC 4 -)

2 = Mild weakness (MRC 4)

1 = Positive drift of arm/leg (MRC 4 +)

0 = Normal (MRC 5)

Deep tendon reflexes _____

2 = Hypoactive or hyperactive

0 = Normal

Pathologic reflexes _____

2 = Babinski (plantar) and another abnormal

1 = Babinski (plantar) or another abnormal

0 = Normal

Muscle tone _____

2 = Increased or decreased

0 = Normal

Gait _____

Test ability to stand and walk, and score:

6 = Unable to stand unsupported or cannot evaluate

5 = Can stand with support but cannot walk

4 = Severely abnormal; walking distance limited even with support (from aid or person)

3 = Moderately abnormal; no assistance required (apart from a stick/cane), but distance limited

2 = Mildly abnormal (weak, uncoordinated); can walk independently but slowly

1 = Minimally abnormal, no reduction in speed or distance

0 = Normal

SENSORY

Primary modalities (of affected side only), arm _____

Test touch, pain and score as:

4 = Anaesthesia

3 = Severe hypaesthesia

2 = Moderate hypaesthesia or deficit only; or extinction to double simultaneous stimulation

1 = Mild hypaesthesia or dysaesthesia

0 = Normal

Stereognosis _____

Test ability to distinguish two coins and a key, and score: 3 = Unable to achieve any distinctions

2 = Can distinguish a coin from a key
1 = Can distinguish between two very different sized coins (penny and ten-pence piece, penny and quarter)
0 = Can distinguish between two similar sized coins (penny and nickel, or two-pence piece and ten-pence piece)

OVERALL TOTAL _____

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the Barthel Index

Activity **Score**

FEEDING
0 = unable
5 = needs help cutting, spreading butter, etc., or requires modified diet
10 = independent

BATHING
0 = dependent
5 = independent (or in shower)

GROOMING
0 = needs to help with personal care
5 = independent face/hair/teeth/shaving (implements provided)

DRESSING
0 = dependent
5 = needs help but can do about half unaided
10 = independent (including buttons, zips, laces, etc.)

BOWELS
0 = incontinent (or needs to be given enemas) 5
= occasional accident
10 = continent

BLADDER
0 = incontinent, or catheterized and unable to manage alone
5 = occasional accident
10 = continent

TOILET USE
0 = dependent
5 = needs some help, but can do something alone 10
= independent (on and off, dressing, wiping)

TRANSFERS (BED TO CHAIR AND BACK)
0 = unable, no sitting balance
5 = major help (one or two people, physical), can sit
10 = minor help (verbal or physical)
15 = independent

MOBILITY (ON LEVEL SURFACES)
0 = immobile or < 50 yards
5 = wheelchair independent, including corners, > 50 yards
10 = walks with help of one person (verbal or physical) > 50 yards
15 = independent (but may use any aid; for example, stick) > 50 yards

STAIRS
0 = unable
5 = needs help (verbal, physical, carrying aid)
10 = independent

TOTAL (0–100): _____

Glasgow Coma Scale

Activity		Score
EYE OPENING		
None	1 = Even to supra-orbital pressure	
To pain	2 = Pain from sternum/limb/supra-orbital pressure	
To speech	3 = Non-specific response, not necessarily to command	
Spontaneous	4 = Eyes open, not necessarily aware	_____
MOTOR RESPONSE		
None	1 = To any pain; limbs remain flaccid	
Extension	2 = Shoulder adducted and shoulder and forearm internally rotated	
Flexor response	3 = Withdrawal response or assumption of hemiplegic posture	
Withdrawal	4 = Arm withdraws to pain, shoulder abducts	
Localizes pain	5 = Arm attempts to remove supra-orbital/chest pressure	
Obeys commands	6 = Follows simple commands	_____
VERBAL RESPONSE		
None	1 = No verbalization of any type	
Incomprehensible	2 = Moans/groans, no speech	
Inappropriate	3 = Intelligible, no sustained sentences	
Confused	4 = Converses but confused, disoriented	
Oriented	5 = Converses and oriented	_____

TOTAL (3–15): _____

QUESTIONS FOR SELF-EDUCATION

1. Which of the following is true about stroke? Select applicable:

1. Sudden focal neurological deficit
2. Produced by an acute episode of ischemia of the brain tissue
3. Lasts longer than 24 hours
4. Only vascular cause
5. Hemorrhagic stroke: rupture of a blood vessel
6. Sudden global neurological deficit
7. Usually lasts less than 1 hour
8. May have posttraumatic origin (posttraumatic hemorrhage)
9. Subacute or chronic onset of focal neurological deficit
10. Ischemic stroke: artery obstruction or severe hypotension

2. Select modifiable stroke risk factors:

1. Hypertension
2. Age
3. Smoking
4. TIAs
5. Gender

6. Heart disease
7. Diabetes mellitus
8. Race
9. Prior history of stroke
10. Atrial fibrillation
11. Obesity
12. Hereditary
13. Physical inactivity

3. Select non-modifiable stroke risk factors:

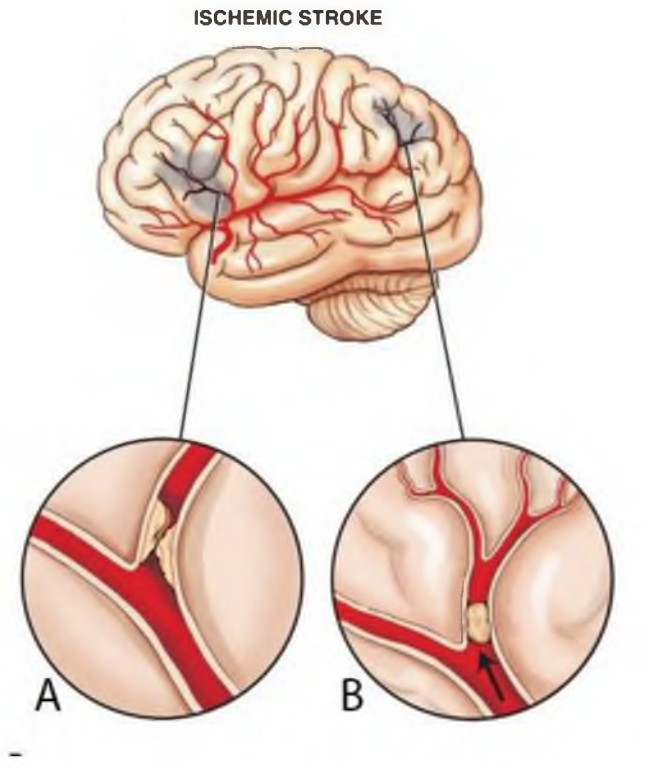
1. Hypertension
2. Age
3. Smoking
4. TIAs
5. Gender
6. Heart disease
7. Diabetes mellitus
8. Race
9. Prior history of stroke
10. Atrial fibrillation
11. Obesity
12. Hereditary
13. Physical inactivity

4. Differential diagnosis considerations for acute ischemic stroke and TIA (select applicable):

1. Traumatic (subdural, brain contusion)
2. Viral meningitis
3. Post-ictal from focal-onset seizure (Todd's phenomenon, Todd's paralysis)
4. Dementia (Alzheimer's disease)
5. Multiple sclerosis
6. Parkinson's disease
7. Mononeuropathy or radiculopathy
8. Bacterial meningitis
9. Brain abscess or cerebritis
10. Migraine
11. Brain tumor
12. Hypoglycemia

5. What is the penumbra?

1. Focal CNS ischemia with infarction, including cerebral, spinal cord, and retinal infarctions.
2. A transient episode of neurologic dysfunction caused by focal CNS ischemia without Infarction. It is a warning sign that there is an increased risk of stroke.
3. This is a zone of reversible ischemia around a core of irreversible infarction during the first few hours after ischemic stroke.
4. Temporary interruption of blood flow for a few seconds up to 24 hours, not significant enough to cause significant tissue damage (precursor to major stroke, "warning signal")
5. Sudden numbness or weakness in the face, arm or leg, especially on one side of the body; sudden confusion or trouble speaking or understanding; sudden trouble seeing on one or both eyes; sudden trouble walking, dizziness or loss of balance/coordination; sudden severe headache with no known cause.



6. Select type of the ischemic stroke indicated with letter A:

1. Lacunar
2. Subcortical
3. Embolic
4. Subarachnoid
5. Aneurysm
6. Parenchymal
7. Cortical
8. Atherothrombotic
9. Arteriovenous malformation
10. Superficial

7. Select type of the ischemic stroke indicated with letter B:

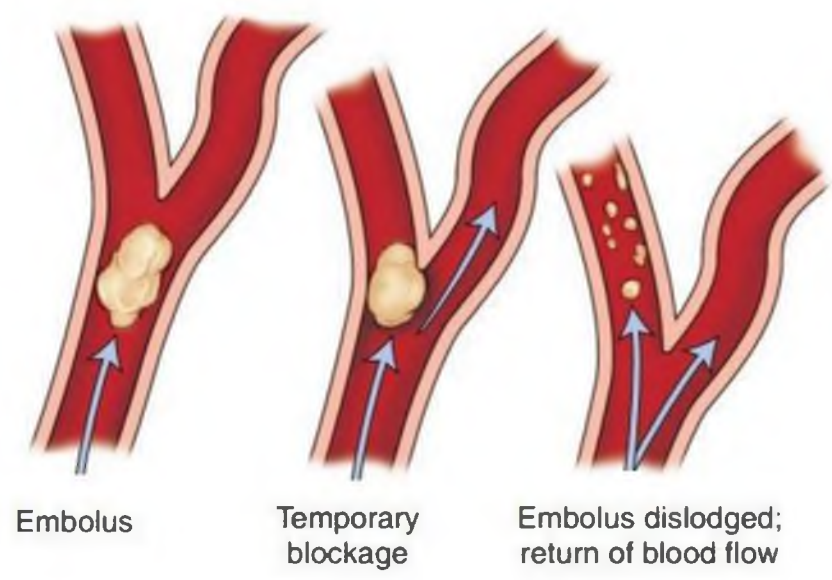
1. Lacunar
2. Subcortical
3. Embolic
4. Subarachnoid
5. Aneurysm
6. Parenchymal
7. Cortical
8. Atherothrombotic
9. Arteriovenous malformation
10. Superficial

8. The zone of compromised neuronal cells unable to function during a stroke, but remain viable is called:

1. Lacunar infarction (lacuna)
2. Dead tissue (necrosis)
3. Watershed infarction (border zone infarct)
4. Partial shadow (penumbra)
5. Territorial infarct
6. Subarachnoid hemorrhage (SAH)
7. Transient ischemic attack (TIA)
8. Intracranial vasospasm
9. Hemorrhagic transformation
10. Reperfusion injury

9. This diagnostic scale assesses ability to speak, obey commands, and open eyes when verbal or painful stimulus is applied; evaluates opening of eyes, best verbal response, and best motor response (select applicable):

1. National Institutes of Health Stroke Scale (NIHSS)
2. Scandinavian Stroke Scale (SSS)
3. Glasgow Outcome Scale (GOS)
4. Hunt & Hess Scale
5. Hemispheric Stroke Scale (HSS)
6. Glasgow Coma Scale (GCS)
7. Barthel Index
8. Modified Rankin Scale (MRS)



10. Using the figure from the left indicate pathological condition:

1. Ischemic stroke
2. Subarachnoid hemorrhage
3. Lacunar stroke
4. Transient ischemic attack
5. Stroke in progress
6. Normal bloodflow

11. A 67-year-old woman with a history of type II diabetes mellitus and atrial fibrillation presents to the emergency room with right body weakness and slurred speech. The onset was sudden while she was brushing her teeth 1 h ago, and she was brought immediately to the emergency room. She has no complaints of word-finding difficulties, no dysesthesia, and no headache. She is taking warfarin. Physical exam findings include blood pressure of 205/90 and irregularly irregular heart beat. There is left side neglect with slurred speech. There is a corticospinal pattern of weakness of the right body, with the face and upper extremity worse than the lower extremity. Routine chemistries and cell counts are normal. Her INR is 1.8. Which of the following should be done next?

1. Administer tissue plasminogen activator
2. Call a vascular surgery consult for possible endarterectomy
3. Order a brain CT
4. Order a cerebral angiogram
5. Start heparin

12. A 62-year-old man with a history of myocardial infarction awakens with a dense right-sided hemiplegia. He appears to be alert and responds to pain on the left side of his body. His speech is unintelligible and nonfluent, and he follows no instructions. Efforts to get him to repeat simple phrases consistently fail. Select applicable:

1. Global aphasia
2. Transcortical sensory aphasia
3. Wernicke's aphasia
4. Transcortical motor aphasia
5. Anomic (amnestic) aphasia
6. Broca's aphasia
7. Conduction aphasia
8. Mixed transcortical aphasia

13. You are taking care of a 59 year old male who has a history of smoking and hypertension. Which of the following is the biggest risk factor for stroke in this subject? Select applicable:

1. Age
2. Gender
3. Hypertension
4. Smoking

14. Atrial fibrillation is a risk factor for what type of stroke and why? Select applicable:

1. Hemorrhagic, increased pressure on cerebral blood vessels due to low atrial ejection fraction and backflow of blood
2. Ischemic, embolus due to stasis of blood in heart, blood clot formation, and release of clot into brain through internal jugular arteries
3. Ischemic, thrombus formation due to stasis of blood in heart, blood clot formation, and release of clot

into brain through the carotid arteries
4. Ischemic, embolus due to stasis of blood in heart, blood clot formation, and release of clot into brain through carotid arteries.

15. Which of the following describes the pathophysiology behind ischemic strokes caused by thrombi? Select applicable:

1. Clot forms in another area of body and travels to brain causing an occluded vessel
2. Plaque builds up in artery causing occlusion of the artery
3. Endothelial injury occurs in cerebral artery, fatty streak and plaque formation occur, plaque ruptures and clot forms occluding artery
4. Vasospasm of cerebral vessel occludes vessel

16. Which of the following is correctly paired with its cause? Select applicable:

1. Subarachnoid hemorrhage- hypertension causes blood vessel in brain to rupture
2. Intracerebral hematoma- ruptured aneurysm causes bleeding into space between arachnoid mater and the pia mater.
3. Subarachnoid hemorrhage- ruptured aneurysm causes bleeding into space between arachnoid mater and the pia mater.
4. Intracerebral hematoma- hypertension causes blood vessel in brain to rupture

17. A patient has just arrived to the emergency department via ambulance for a possible stroke. The neurologist knows that there are certain things that need to be done within certain time frames upon arrival to the emergency department. The first thing that has to be done upon arrival is? Select applicable:

1. Start an intravenous thrombolysis
2. Obtain CT of the head
3. Obtain vitals and assess airway, breathing, circulation
4. Obtain 12 lead ECG

18. Which of the following are typical signs/symptoms of the early stages (grade I & II) of a subarachnoid hemorrhage? Select applicable:

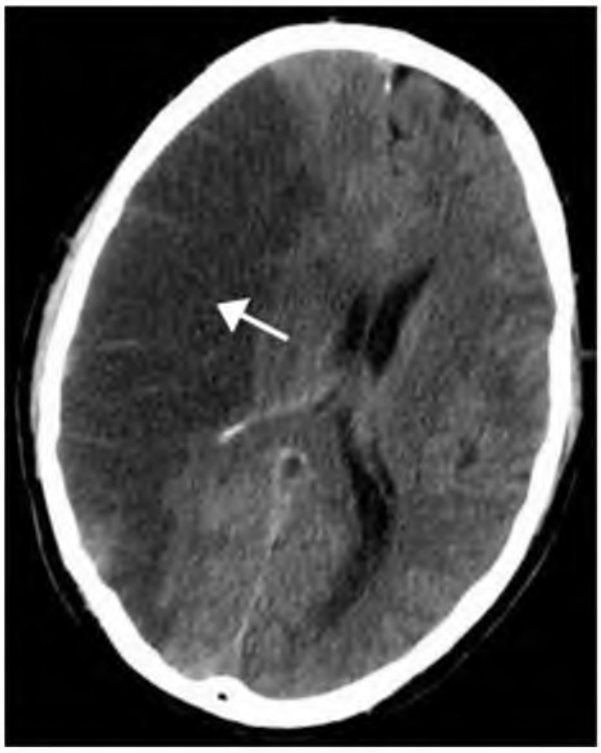
1. Patient with focal neurological deficits
2. Patient with acute confusion
3. Patient with "worst headache of my life"
4. Patient with photophobia and stiff neck

19. What is the main use for nimodipine in the patient with subarachnoid hemorrhage?

1. Blood pressure control
2. Angiography studies
3. Prevention/treatment of vasospasms
4. Prevention/treatment of hydrocephalus

20. The stroke guidelines recommend to not lower arterial blood pressure in patients (not candidates for thrombolytic therapy) unless the systolic pressure exceeds __?_ or diastolic exceeds __?_ and then to lower blood pressure only by 10-15% within the first 24 hrs. Select applicable:

1. 180 mmHg / 90 mmHg
2. 145 mmHg / 85 mmHg
3. 160 mmHg / 100 mmHg
4. 170 mmHg / 110 mmHg
5. 220 mmHg / 120 mmHg
6. 190 mmHg / 100 mmHg

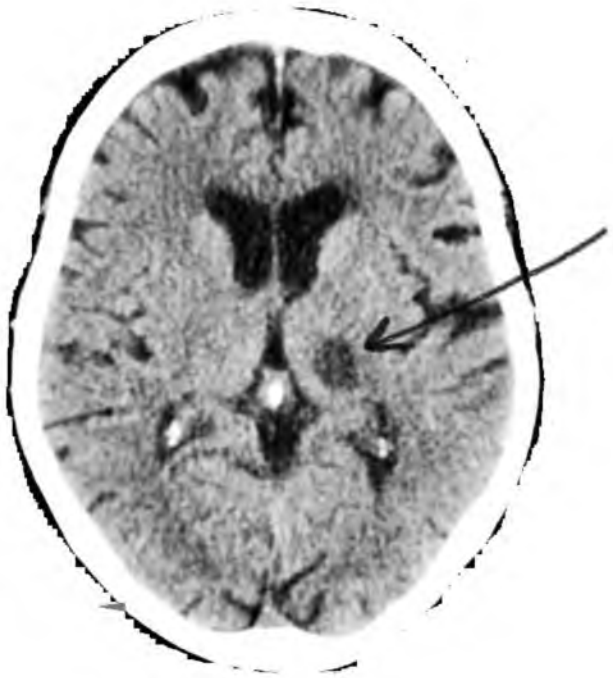


21. Using the figure from the left indicate stroke type? Select applicable:

1. Subarachnoid hemorrhage
2. Lacunar infarct
3. Watershed zone infarct
4. Intracranial hemorrhage
5. Territorial infarct
6. Unchanged brain tissue
7. Poststroke gliosis
8. Parenchymal hemorrhage

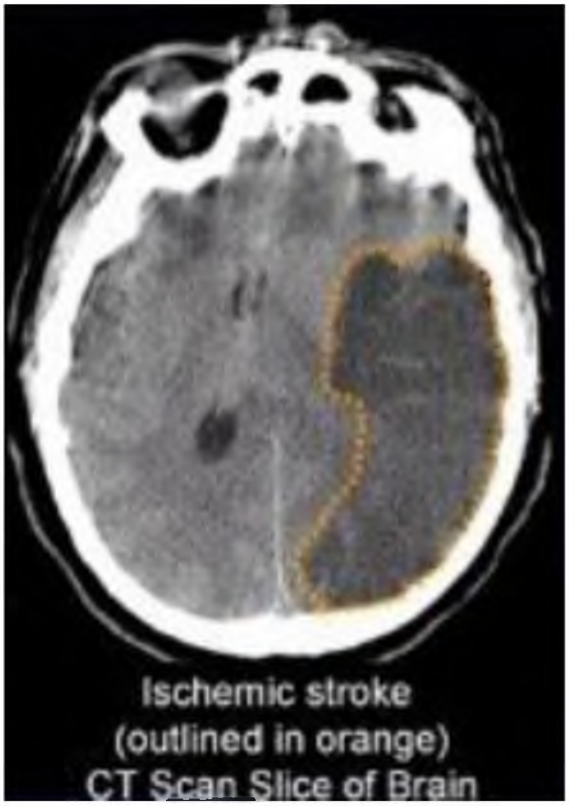
22. What is the window ('golden hour') for treatment of ischemic stroke with thrombolytics?

1. 3 hours
2. 6 hours
3. 9 hours
4. 12 hours
5. 24 hours



23. Using the figure from the left indicate stroke type? Select applicable:

1. Subarachnoid hemorrhage
2. Lacunar infarct
3. Watershed zone infarct
4. Intracranial hemorrhage
5. Territorial infarct
6. Unchanged brain tissue
7. Poststroke gliosis
8. Parenchymal hemorrhage



24. Using the figure from the left (which reveals ischemia-induced infarction) indicate, what shows up as darkened areas on CT?

1. Cerebral hemorrhage
2. Cerebral hyperperfusion
3. Normal cerebral perfusion
4. Cerebral hypoperfusion
5. Subarachnoid hemorrhage

25. Indicate symptoms of ischemic stroke in the brain area supplied from Anterior Cerbral Artery. Select applicable:

1. Contralateral lower extremity weakness
2. Cranial nerve abnormalities
3. Vertigo
4. Contralateral hemianesthesia
5. Contralateral homonymous hemianopsia
6. Aphasia (if dominant hemisphere)
7. Neglect (if nondominant hemisphere)
8. Apraxia (if dominant hemisphere)
9. Dysphagia and dysarthria
10. Neuropsychological deficit
11. “pure stroke”: focal motor or sensory deficit
12. Dysarthria-clumsy hand syndrome
13. Cognitive or personality changes
14. Contralateral face and upper extremity weakness
15. Blindness (if bilateral involvement)

26. Indicate symptoms of ischemic stroke in the brain area supplied from Middle Cerbral Artery. Select applicable:

1. Contralateral lower extremity weakness
2. Cranial nerve abnormalities
3. Vertigo
4. Contralateral hemianesthesia
5. Contralateral homonymous hemianopsia
6. Aphasia (if dominant hemisphere)
7. Neglect (if nondominant hemisphere)
8. Apraxia (if dominant hemisphere)
9. Dysphagia and dysarthria
10. Neuropsychological deficit
11. “pure stroke”: focal motor or sensory deficit
12. Dysarthria-clumsy hand syndrome

13. Cognitive or personality changes
14. Contralateral face and upper extremity weakness
15. Blindness (if bilateral involvement)

27. Indicate symptoms of ischemic stroke in the brain area supplied from Posterior Cerbral Artery. Select applicable:

1. Contralateral lower extremity weakness
2. Cranial nerve abnormalities
3. Vertigo
4. Contralateral hemianesthesia
5. Contralateral homonymous hemianopsia
6. Aphasia (if dominant hemisphere)
7. Neglect (if nondominant hemisphere)
8. Apraxia (if dominant hemisphere)
9. Dysphagia and dysarthria
10. Neuropsychological deficit
11. "pure stroke": focal motor or sensory deficit
12. Dysarthria-clumsy hand syndrome
13. Cognitive or personality changes
14. Contralateral face and upper extremity weakness
15. Blindness (if bilateral involvement)

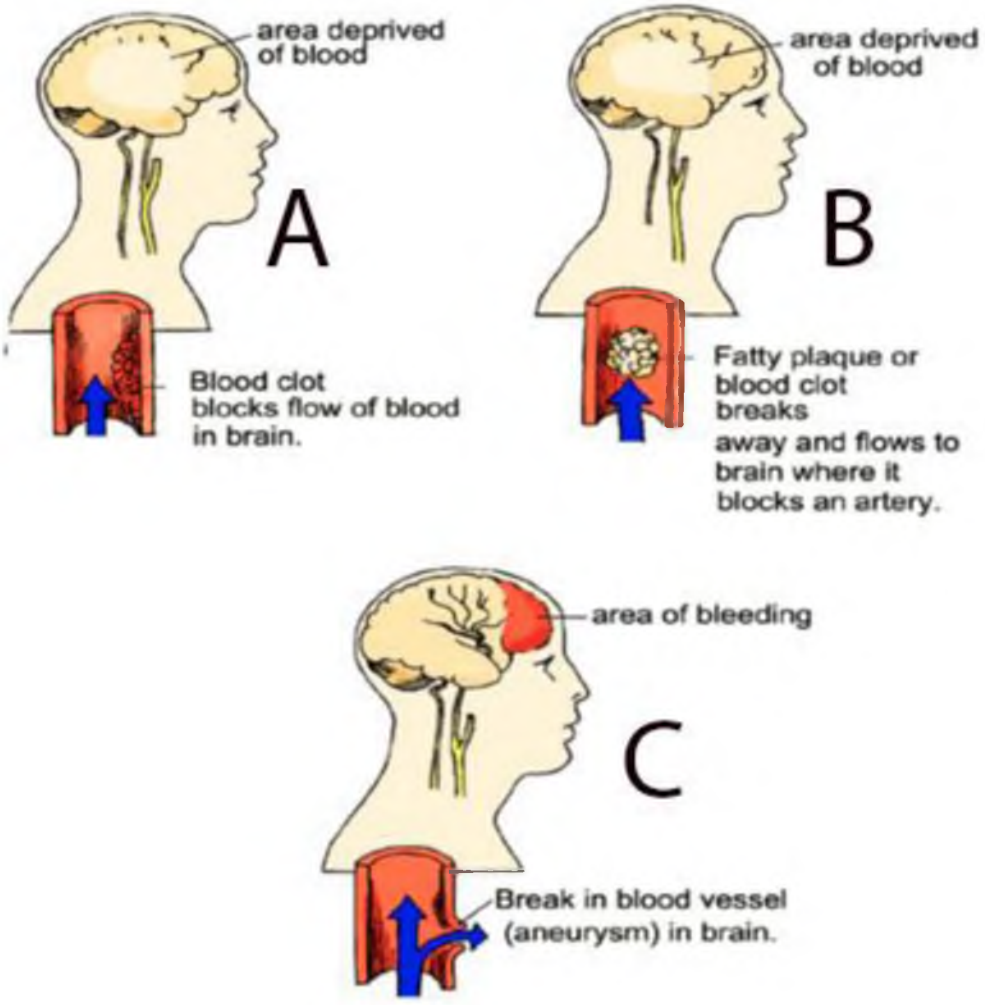
28. Indicate symptoms of ischemic stroke in the brain area supplied from Basilar Artery. Select applicable:

1. Contralateral lower extremity weakness
2. Cranial nerve abnormalities
3. Vertigo
4. Contralateral hemianesthesia
5. Contralateral homonymous hemianopsia
6. Aphasia (if dominant hemisphere)
7. Neglect (if nondominant hemisphere)
8. Apraxia (if dominant hemisphere)
9. Dysphagia and dysarthria
10. Neuropsychological deficit
11. "pure stroke": focal motor or sensory deficit
12. Dysarthria-clumsy hand syndrome
13. Bilateral sensory and motor deficits
14. Contralateral face and upper extremity weakness
15. Blindness (if bilateral involvement)

29. Indicate symptoms of Lacunar strokes. Select applicable:

1. Contralateral lower extremity weakness
2. Cranial nerve abnormalities
3. Vertigo
4. Contralateral hemianesthesia
5. Contralateral homonymous hemianopsia
6. Aphasia (if dominant hemisphere)
7. Neglect (if nondominant hemisphere)
8. Apraxia (if dominant hemisphere)
9. Dysphagia and dysarthria
10. Neuropsychological deficit
11. "pure stroke": focal motor or sensory deficit
12. Dysarthria-clumsy hand syndrome

13. Cognitive or personality changes
14. Contralateral face and upper extremity weakness
15. Blindness (if bilateral involvement)



30. Select type of the stroke indicated with letter B:

1. Lacunar
2. Subcortical
3. Embolic
4. Subarachnoid
5. Aneurysm
6. Parenchymal
7. Cortical
8. Atherothrombotic
9. Arteriovenous malformation
10. Superficial