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

Class 10

Cerebrovascular diseases. Hemorrhagic Stroke.

1. Goals:

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

1.2. To study the Neurological fundamentals of the Cerebral Hemorrhage.

2. Basic questions:

2.1. Cerebral Haemorrhage:

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.

2.1.7. Prognosis.

2.2.*SAH – Subarachnoid Haemorrhage.*

2.2.1. *Etiology.*

2.2.1. *Pathogenesis.*

2.2.1. *Clinical Features.*

2.2.1. *Diagnostic evaluation.*

2.2.1. *Treatment.*

2.2.1. *Prophylaxis.*

2.2.1. *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%.

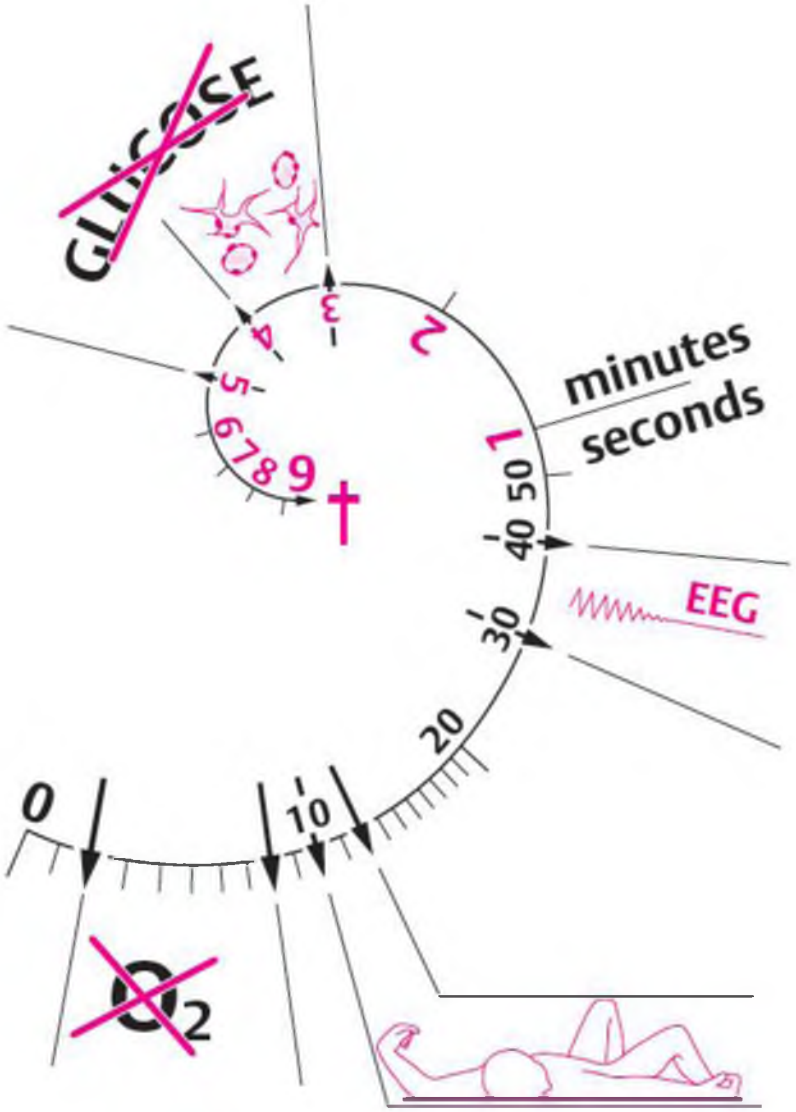
Cerebral Ischemia

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).

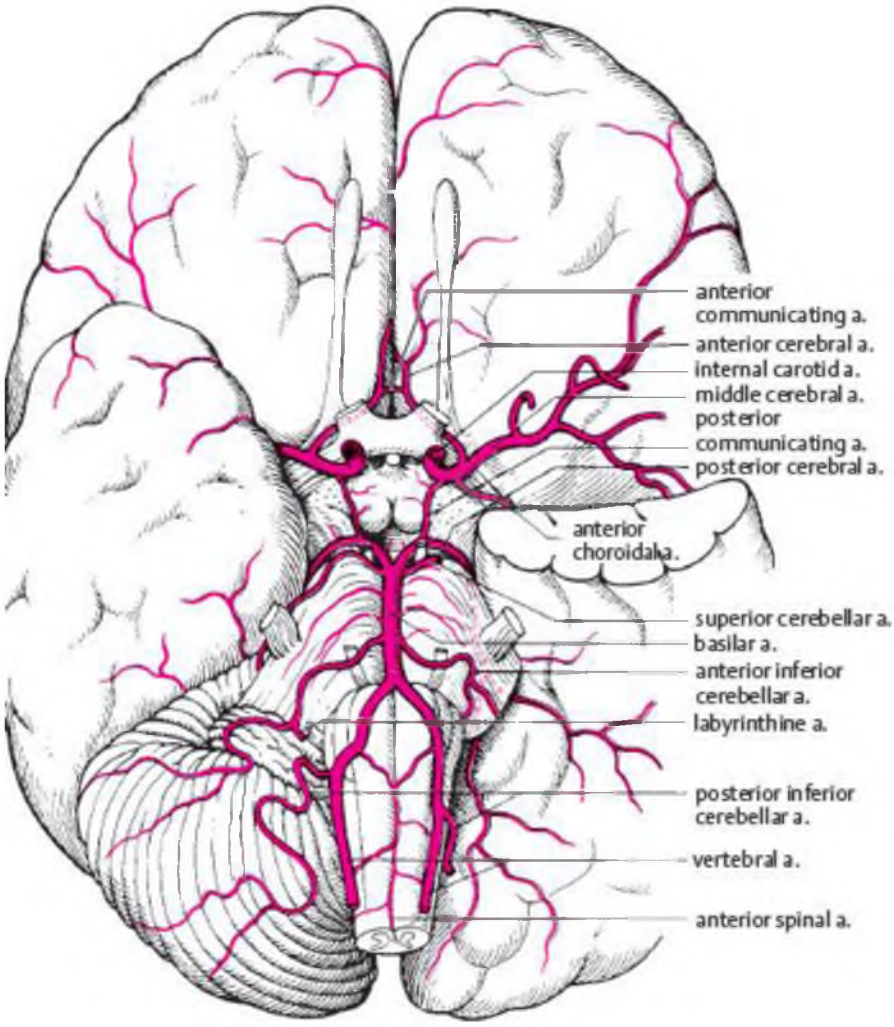


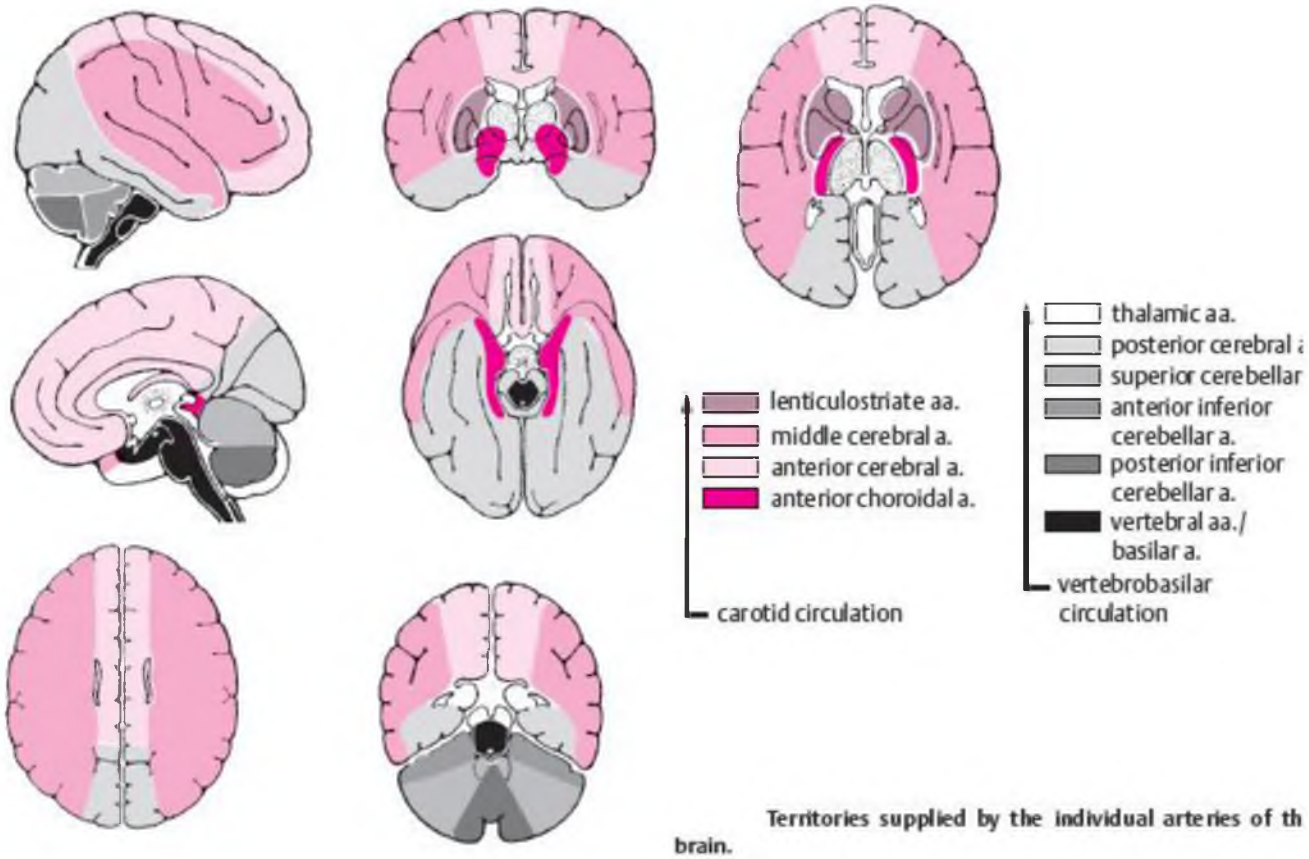
Time course of cerebral ischemia. Diagram of the effect of sudden total deprivation of blood supply to the brain on tissue metabolism, consciousness, the EEG, neuronal morphology, and tissue glucose concentration.

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.

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.

Intracerebral Hemorrhage

Etiology. Most cases of intracerebral hemorrhage are due to the rupture of *vascular lesions of hypertensive origin* ("rhexis hemorrhages" of pseudoaneurysms of lipohyalinotic arterioles), aneurysms, or arteriovenous malformations. Intracerebral hemorrhage may also be a complication of therapeutic (over-) anticoagulation. Smaller hemorrhages, particularly those that are near the cortical surface, are often due to amyloid angiopathy. There can also be bleeding into an infarct, a primary brain tumor, a metastasis, or a cavernoma. The more common etiologies of intracerebral hemorrhage are listed in Table below.

Causes of nontraumatic cerebral hemorrhage

Chronic arterial hypertension
Aneurysm rupture
Hemorrhage into a preexisting lesion (infarct, tumor)
Vascular malformation (cavernoma, arteriovenous malformation)
Vascular fragility due to vasculopathy, e. g., cranial arteritis, amyloid angiopathy
Bleeding diathesis due to hematologic disease or therapeutic anticoagulation
Cerebral venous thrombosis and venous sinus thrombosis
Rarely, in the setting of a hypertensive crisis or drug abuse (e. g., cocaine)

Clinical manifestations. The clinical picture mainly depends on the site and extent of the hemorrhage and to a much lesser extent on etiological factors. Certain aspects of the clinical course can, however, suggest that one etiology is more likely than another:

_ Chronic arterial hypertension and advanced age (typically 60–70) make a rhexis hemorrhage more likely. These hemorrhages are ultimately caused by hypertension and are usually very large. Common sites are the pallidum, the putamen, and the internal capsule, with the corresponding clinical manifestations: *contralateral*, *usually dense*, *hemiparesis* or hemiplegia, *horizontal gaze palsy*, and initially, in many cases, *dŭviation conjuguûe and deviation of the head to the side of the lesion.* Less common sites are the subcortical white matter, brainstem, thalamus (Fig. 6.**21**), and cerebellum. Very large hemorrhages, particularly if located in the posterior fossa, can rapidly elevate the intracranial pressure, causing brainstem compression and, in turn, impairment of consciousness and coma.

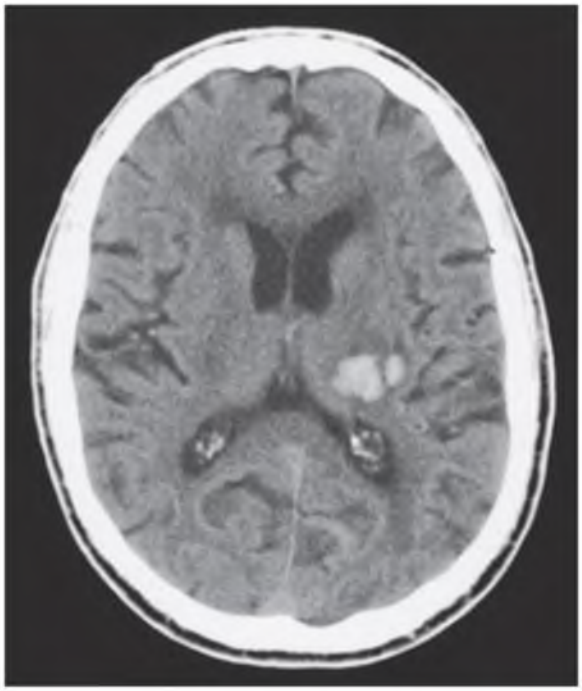


Fig. 6.**21** **Acute left thalamic hemorrhage** as seen by CT in a 76-year-old man with a right-sided hemisensory deficit of acute onset.

_ *Acute worsening* of more or less severe, *preexisting signs and symptoms*, perhaps accompanied by *additional impairment of consciousness*, suggests hemorrhage into an infarct or tumor.
_ *Focal or generalized epileptic seizures* preceding the onset of the acute event point toward a tumor, vascular malformation, or other structural lesion of the brain as the likely cause of hemorrhage.

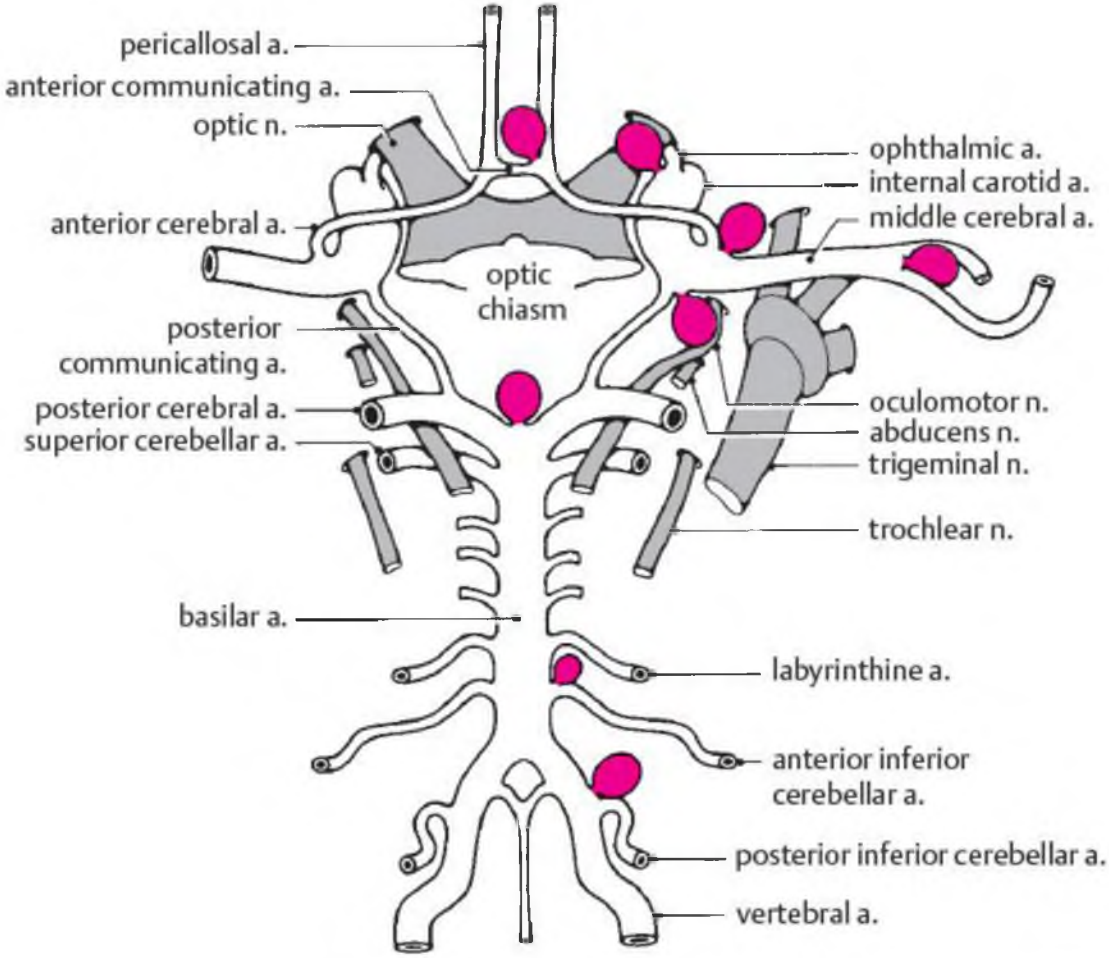
Diagnostic evaluation. The diagnosis of intracranial hemorrhage is suggested by the characteristic clinical findings and then definitively confirmed by the demonstration of blood on *CT* or *MRI*. When performed in the acute phase, these studies may fail to reveal an underlying vascular malformation, if present, which may be obscured by the hemorrhage; *angiography* may be necessary to complete the diagnostic work-up. The obtaining of a complete *coagulation profile* is indicated in some patients.

Treatment and prognosis. Patients suffering from an acute intracerebral hemorrhage require *close clinical observation*; in particular, signs of intracranial hypertension (vomiting, progressive impairment of consciousness, and sometimes anisocoria and papilledema) must be vigilantly watched for. Intracranial hypertension may be due to recurrent hemorrhage or to progressive brain swelling; in either case, it must be promptly detected and treated. In addition, *stabilization of vital functions* and the *treatment of epileptic seizures*, if present, are essential. In each case, the possible indication for *neurosurgical removal of the hematoma* should be carefully considered, in light of the neurological manifestations, site of the hemorrhage, and age and general condition of the patient. Cerebellar hemorrhage with mass effect generally confers a risk of impending brainstem compression and death and is often an indication for life-saving emergency surgery. Although about one-third of all patients with an intracerebral hemorrhage will die of it, while others go on to enjoy a more or less complete spontaneous recovery.

Subarachnoid Hemorrhage (SAH)

Nontraumatic subarachnoid hemorrhage, defined as spontaneous hemorrhage into the subarachnoid space, accounts for about 7% of all "strokes." It can occur at anyage, with peak incidence around age 50. Children are very rarely affected.

Etiology. Subarachnoid hemorrhage is usually due to the *spontaneous rupture of a saccular aneurysm on an artery at the base of the brain*, usually one of the arteries forming the circle of Willis. Common sites of saccular aneurysms are shown in Figure below. Less frequent causes of subarachnoid hemorrhage include arteriovenous malformations, vasculopathies, coagulopathies, and preceding trauma.



Clinical manifestations of subarachnoid hemorrhage are:

_ sudden, extremely intense **headache**, often described as the "worst headache of my life;" the headache may have been preceded by an earlier, transient episode of headache or other minor symptoms ("*premonitory headache*," "*warning leak*"); it is most commonly diffuse or bioccipital;

_ often, at first, a brief and transient **impairment of consciousness**, which may be followed, at some point in the following hours or days, by a recurrent impairment of consciousness or coma;

_ often, **nausea and vomiting**;

_ rarely, **cranial nerve palsies** (caused by aneurysms at particular sites) or other **focal neurological deficits**, caused, e. g., by additional hemorrhage into the brain parenchyma.

Diagnostic evaluation.

Physical evaluation reveals:

_ as the most prominent physical finding, *meningism*; and sometimes other clinical signs that may be useful for the localization of the lesion, e. g.:

_ *oculomotor nerve palsy* with aneurysms of the terminal segment of the internal carotid a. or the posterior communicating a.;

_ *abulia* with an aneurysm of the anterior communicating a.;

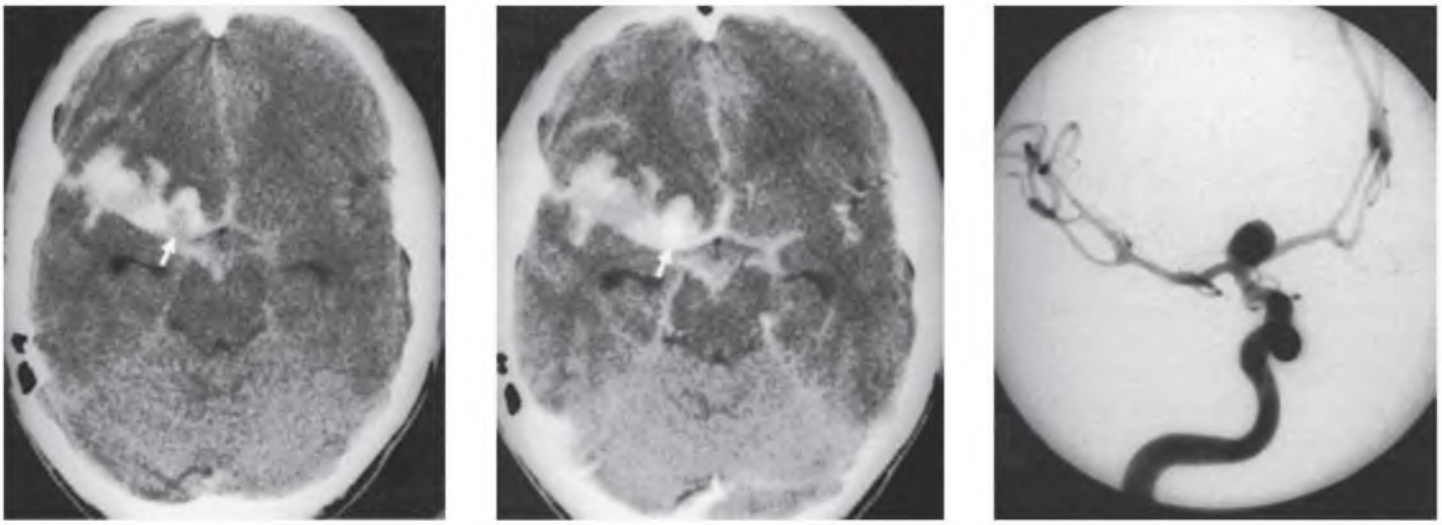
_ *hemiplegia* with an aneurysm of the middle cerebral a.;

_ *brainstem and cerebellar signs* with aneurysms of the basilar a.

The first scale of severity was described by Hunt and Hess in 1968:

Grade	Signs and symptoms	Survival
1	Asymptomatic or minimal headache and slight neck stiffness	70%
2	Moderate to severe headache; neck stiffness; no neurologic deficit except cranial nerve palsy	60%
3	Drowsy; minimal neurologic deficit	50%
4	Stuporous; moderate to severe hemiparesis; possibly early decerebrate rigidity and vegetative disturbances	20%
5	Deep coma; decerebrate rigidity; moribund	10%

Whenever subarachnoid hemorrhage is suspected on clinical grounds, **neuroimaging studies** should be per formed immediately. *CT* or *MRI with FLAIR sequences* can often demonstrate the presence of blood in the cerebrospinal fluid spaces on the day of the hemorrhage (Fig. below). These studies can sometimes also reveal the source of hemorrhage (aneurysm or other), though they often will not. If CT and MRI fail to demonstrate any hemorrhage in the face of clinical suspicion, a **lumbar puncture** must be performed. Bloody CSF is found in patients with acute subarachnoid hemorrhage, xanthochromic CSF in patients whose hemorrhage occurred a few hours of more before the LP.



a b c

Aneurysmal subarachnoid hemorrhage. a The nonenhanced CT reveals blood in the subarachnoid space, particularly along the course of the middle cerebral a. The aneurysm (arrow) is also seen. b The lumen of the aneurysm, dark in a, turns bright after the administration of intravascular contrast (arrow). c Carotid angiography shows the aneurysm at the bifurcation of the internal carotid a. into the anterior and middle cerebral aa.

! A negative CT or MRI does not rule out subarachnoid hemorrhage. If clinical suspicion remains, a lumbar puncture must be performed.

Once the diagnosis of subarachnoid hemorrhage is confirmed by imaging or lumbar puncture, **cerebral angiography** should be performed as soon as possible to determine the source of the hemorrhage, usually an aneurysm. Angiography is only indicated, however, if the patient is clinically stable enough to undergo an operation. Blood coming into contact with the outer walls of arteries that course through the subarachnoid space causes vasospasm, which can be detected with **transcranial Doppler or duplex ultrasonography**.

Treatment. Patients with aneurysmal subarachnoid hemorrhage must be immediately admitted or transferred to a hospital with a neurosurgical department. The goal of treatment is exclusion of the aneurysm from the circulation as soon as possible to prevent a potentially fatal recurrent hemorrhage. This is done either with neurosurgical clipping or, less often, with interventional neuroradiological techniques ("coiling" and others). In addition, **general measures** including strict bed rest, stabilization of cardiovascular functions, fluid and electrolyte administration, analgesia, sedation, and the administration of a calcium-channel blocker (*nimodipine*) to prevent vasospasm are indicated. The performance of transcranial ultrasonography at regular intervals enables prompt detection of vasospasm, which may require treatment.

Clinical course and long-term prognosis. The clinical course of subarachnoid hemorrhage is often dramatic. **Recurrent hemorrhage** after the initial bleed is particularly worrisome and often fatal. Without treatment, about 25% of patients die in the first 24 hours and 40% in the first three months. The course is often further complicated by vasospasm arising three to 14 days after the initial hemorrhage (usually in the first three to five days). This may cause transient ischemia or infarction in the distribution of the spastic artery. Vasospasm may not resolve until three or four weeks later. Another potential complication is **malresorptive hydrocephalus**, presumably caused by adhesions of the arachnoid villi obstructing the outflow of CSF. Patients who survive an initial aneurysmal subarachnoid hemorrhage without further treatment of the aneurysm have a long-term risk of recurrent hemorrhage of about 3% per year.

ABCD Score

Used to predict the risk of stroke during the first seven days after a TIA. Researchers found there to be over 30% risk of stroke in TIA patients with an 'ABCD score' of six, as compared to no strokes in those with a low ABCD score. Can be used in routine clinical practice to identify high-risk individuals who require emergency investigation and treatment.

A	**A**ge of patient	Age >/= 60	1
		Age < 60	0
B	**B**lood pressure at Assessment	SBP > 140 or DBP >/= 90	1
		Other	0
C	**C**linical Features presented with	Unilateral weakness	2
		Speech disturbance (no weakness)	1
		Other	0
D	**D**uration of TIA symptoms	>/= 60 minutes	2
		10-59 minutes	1
		<10 minutes	0
		TOTAL	**0-6**

Hemispheric Stroke Scale

Scored to give 0 (= good) to 100 (= bad)

Score

LEVEL OF CONSCIOUSNESS

15 – Glasgow Coma Scale Score _____

LANGUAGE

Comprehension _____

Give three commands:

'Stick out your tongue' or "Close your eyes'

'Point to the door'

'Place left/right hand on left/right ear and then on left/right knee (using unaffected side)

Score on number correctly followed:

0 = 5

1 = 4

2 = 2

3 = 0

Naming _____

Ask patient to name the following items:

Watch or Belt

Watch strap or Belt buckle

Index finger or Ring finger

Score on number correctly named:

0 = 5

1 = 4

2 = 2

3 = 0

Repetition _____

Ask the patient to repeat the following:

A single word, such as 'dog' or 'cat'

'The president lives in Washington'

'No ifs, ands, or buts'

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:

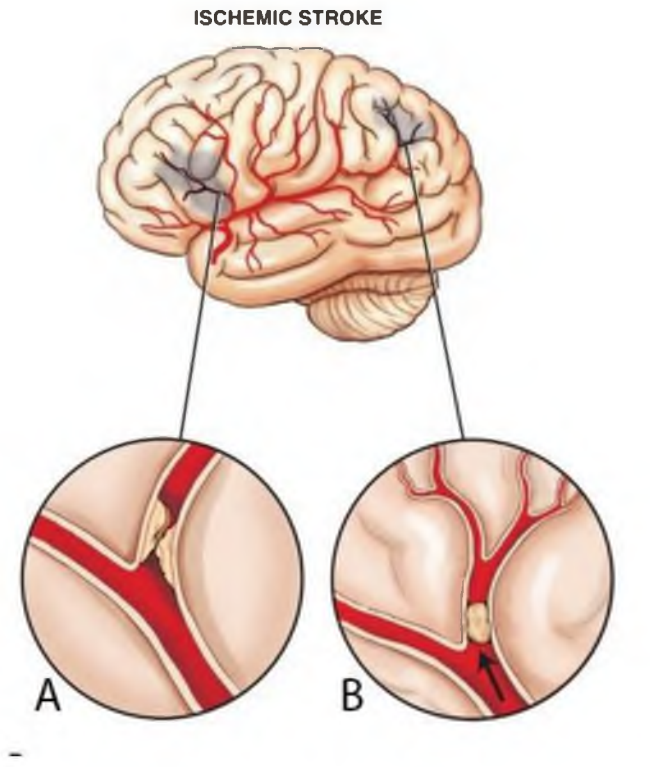
1. Hypertension
2. Age
3. Smoking
4. TIAs
5. Gender
6. Heart disease
7. Diabetes mellitus
8. Race
9. Prior history of stroke
10. Atrial fibrillation
11. Obesity
12. Hereditary
13. Physical inactivity

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:

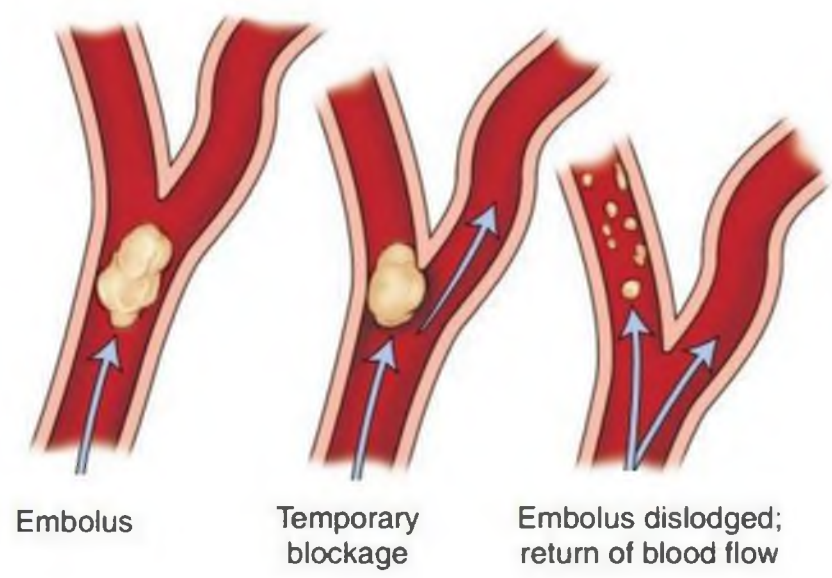
1. Lacunar
2. Subcortical
3. Embolic
4. Subarachnoid
5. Aneurysm
6. Parenchymal
7. Cortical
8. Atherothrombotic
9. Arteriovenous malformation
10. Superficial

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:

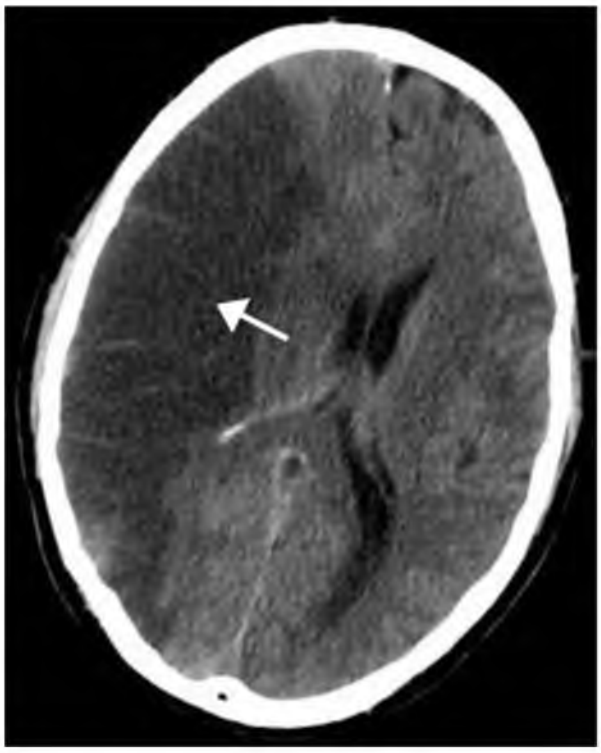
1. Patient with focal neurological deficits
2. Patient with acute confusion
3. Patient with "worst headache of my life"
4. Patient with photophobia and stiff neck

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

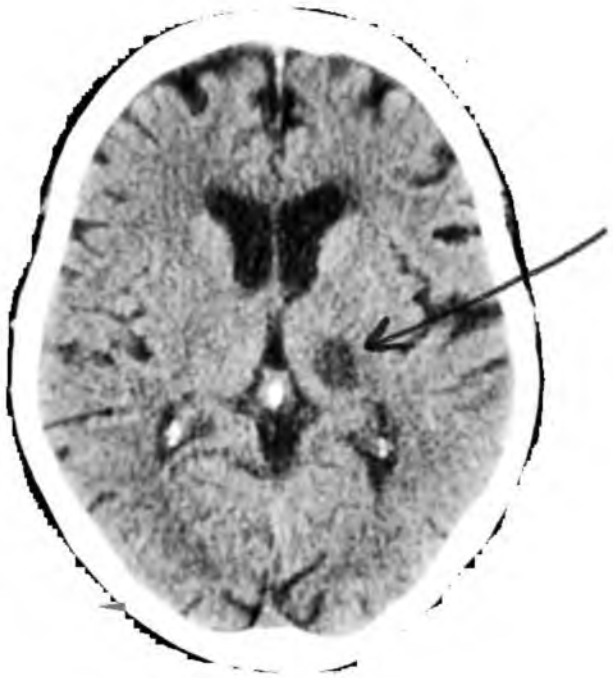


21. Using the figure from the left indicate stroke type? Select applicable:

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

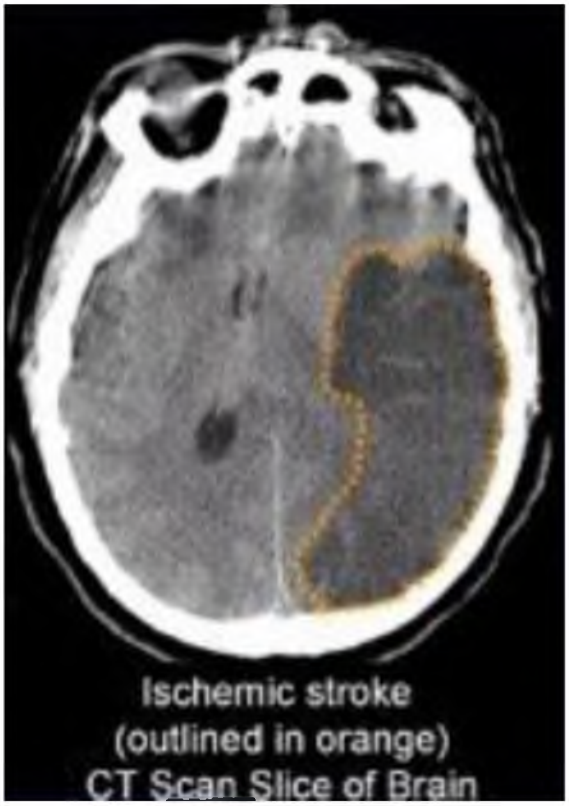
22. What is the window ('golden hour') for treatment of ischemic stroke with thrombolytics?

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)

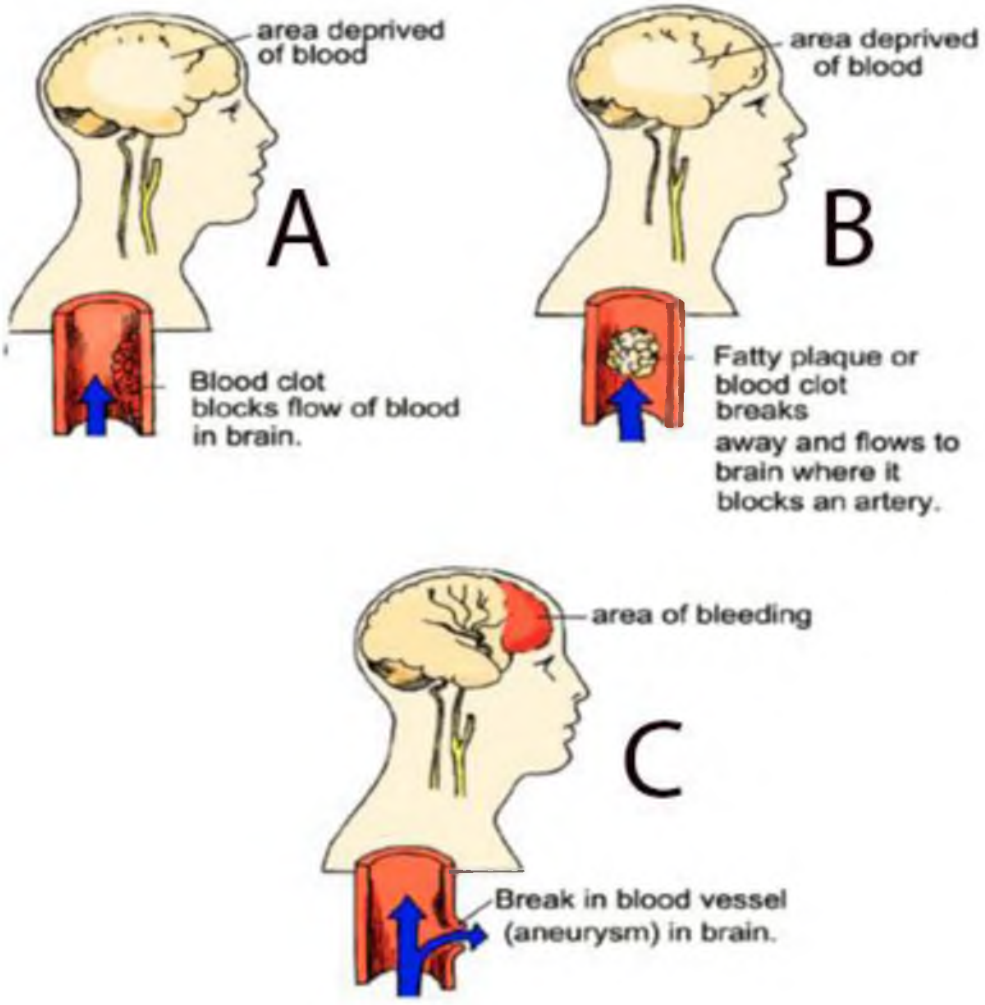
29. Indicate symptoms of Lacunar strokes. Select applicable:

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



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

Class 10

Cerebrovascular diseases. Hemorrhagic Stroke.

1. Goals:

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

1.2. To study the Neurological fundamentals of the Cerebral Hemorrhage.

2. Basic questions:

2.1. Cerebral Haemorrhage:

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.

2.1.7. Prognosis.

2.2.*SAH – Subarachnoid Haemorrhage.*

2.2.1. *Etiology.*

2.2.1. *Pathogenesis.*

2.2.1. *Clinical Features.*

2.2.1. *Diagnostic evaluation.*

2.2.1. *Treatment.*

2.2.1. *Prophylaxis.*

2.2.1. *Prognosis.*

Literature:

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