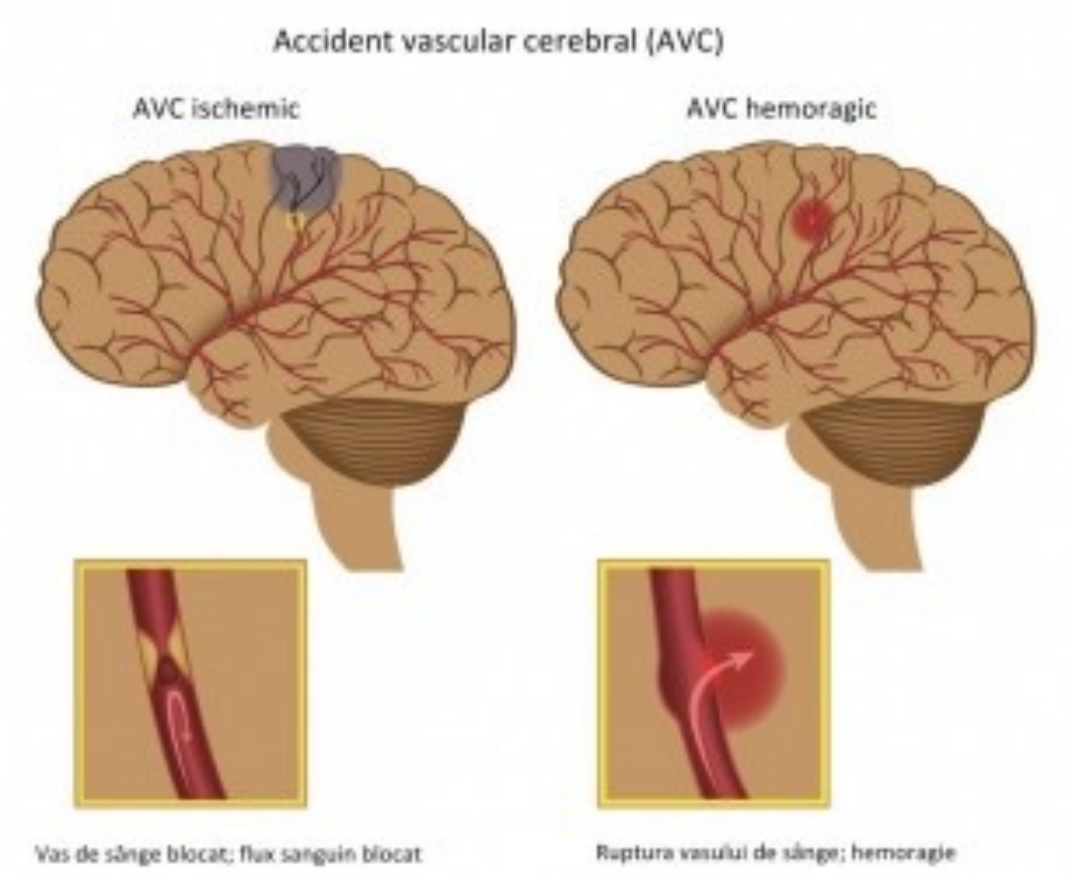
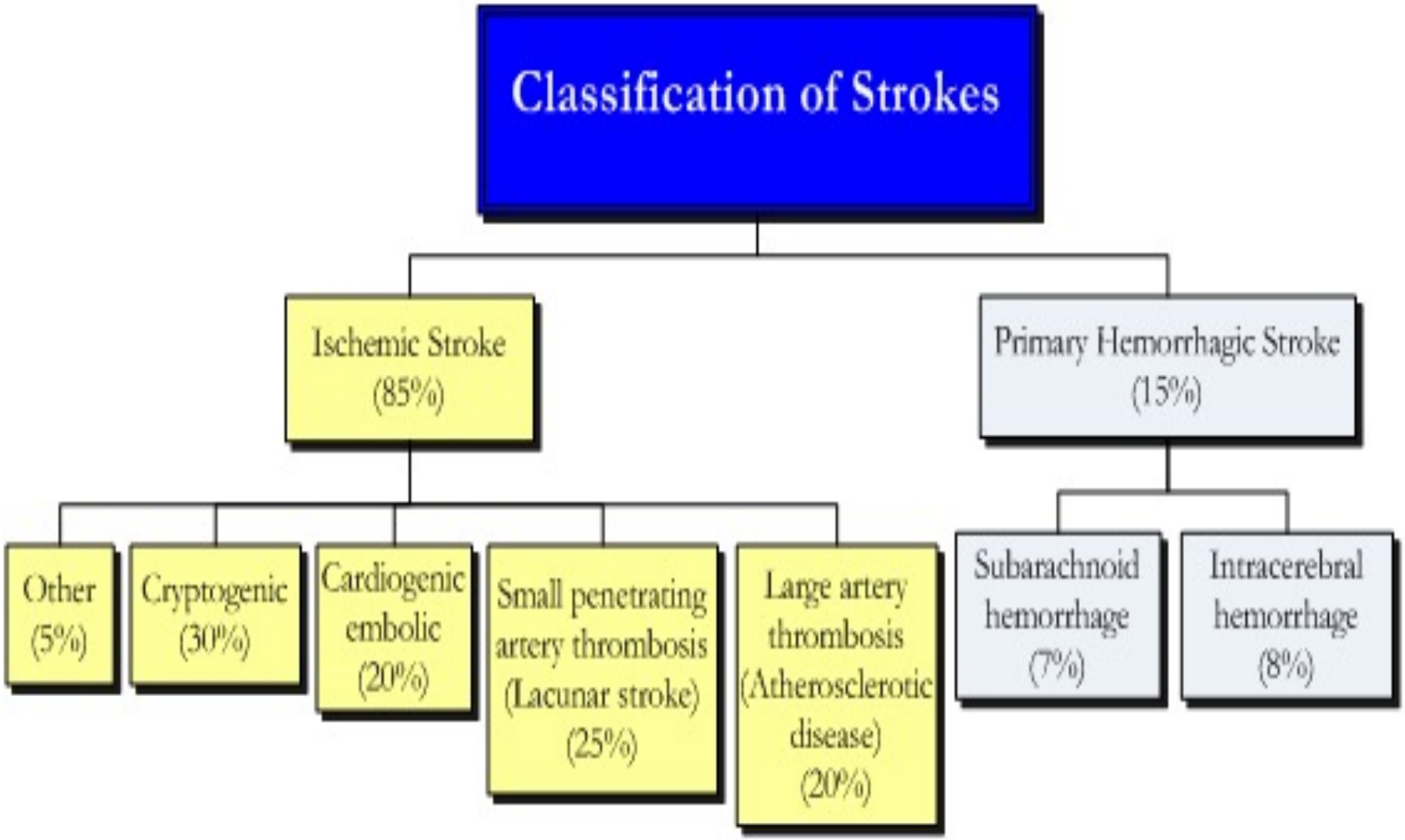
HEMORRHAGIC STROKE

Mihail GAVRILIUC

CEREBROVASCULAR DISEASES

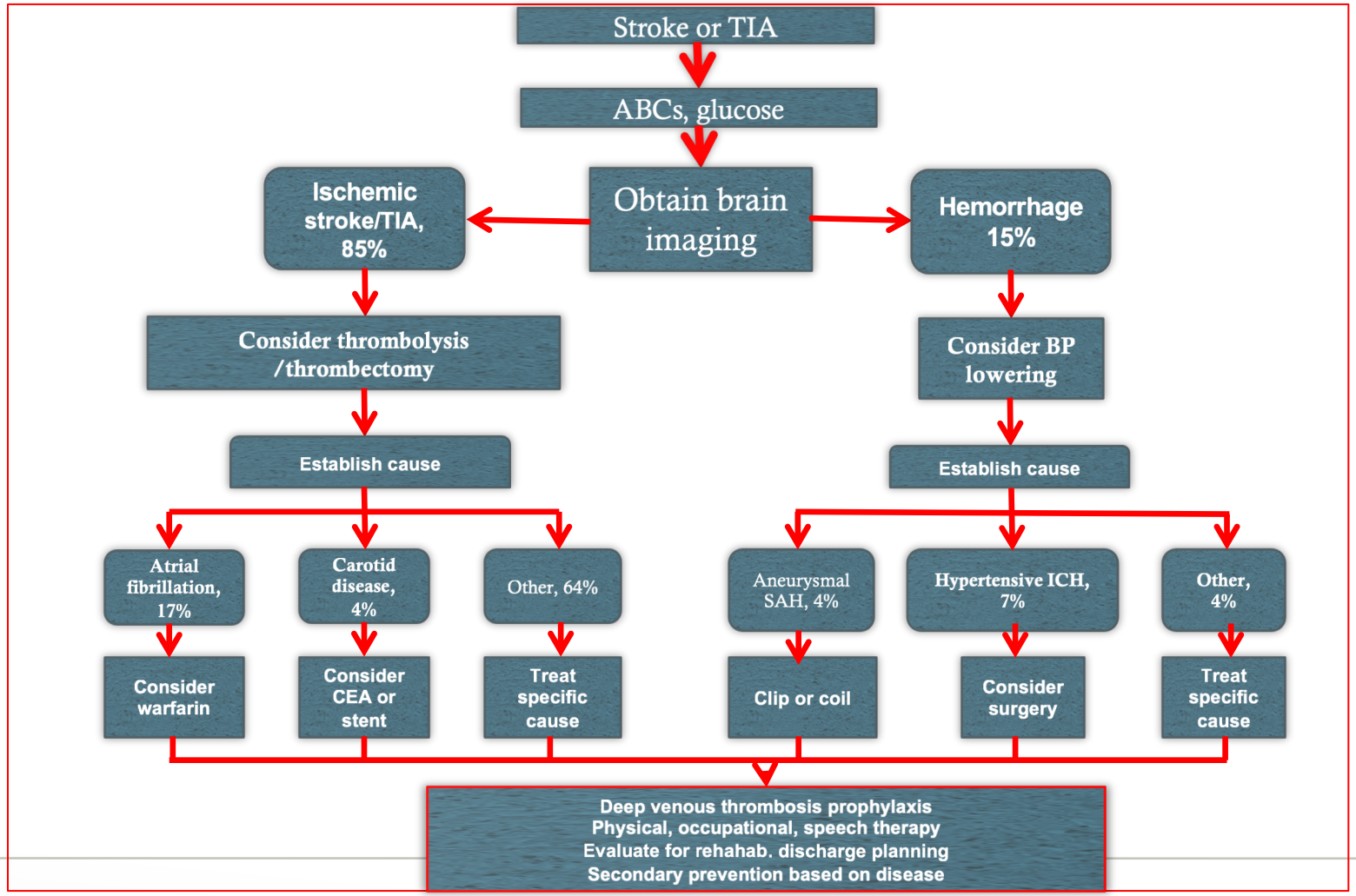
These are of two main types **- ischemia**, with or without infarction, and **hemorrhage** - and unless one or the other occurs, the vascular lesion usually remains silent.



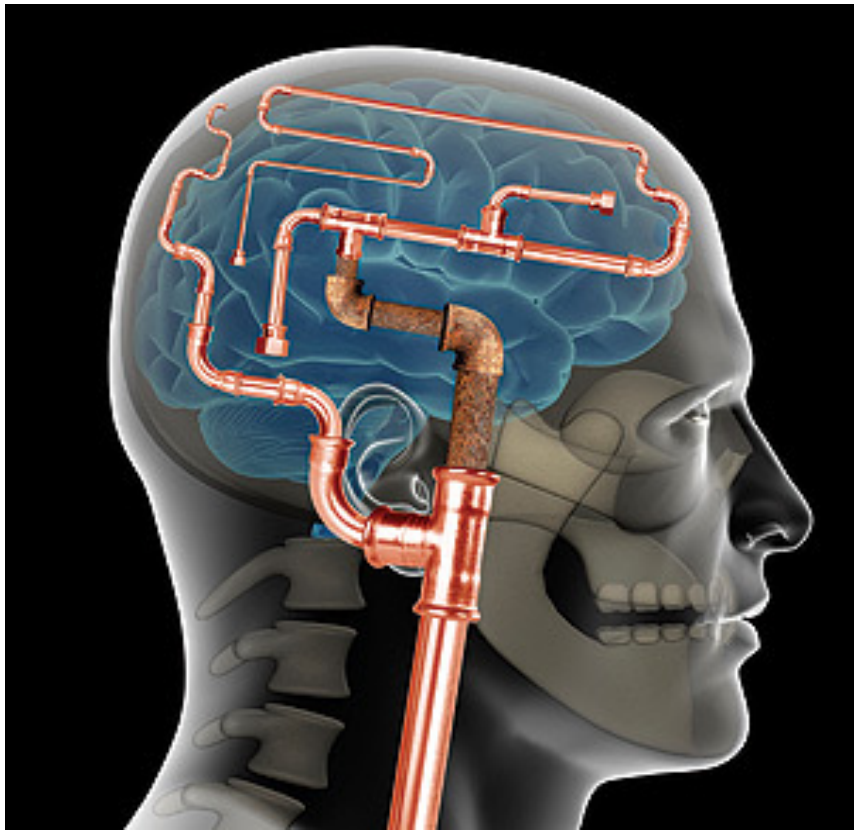

Classification of Strokes
Ischemic Stroke (85%)
Primary Hemorrhagic Stroke (15%)
Other (5%)
Cryptogenic (30%)
Cardiogenic embolic (20%)
Small penetrating artery thrombosis (Lacunar stroke) (25%)
Large artery thrombosis (Atherosclerotic disease) (20%)
Subarachnoid hemorrhage (7%)
Intracerebral hemorrhage (8%)

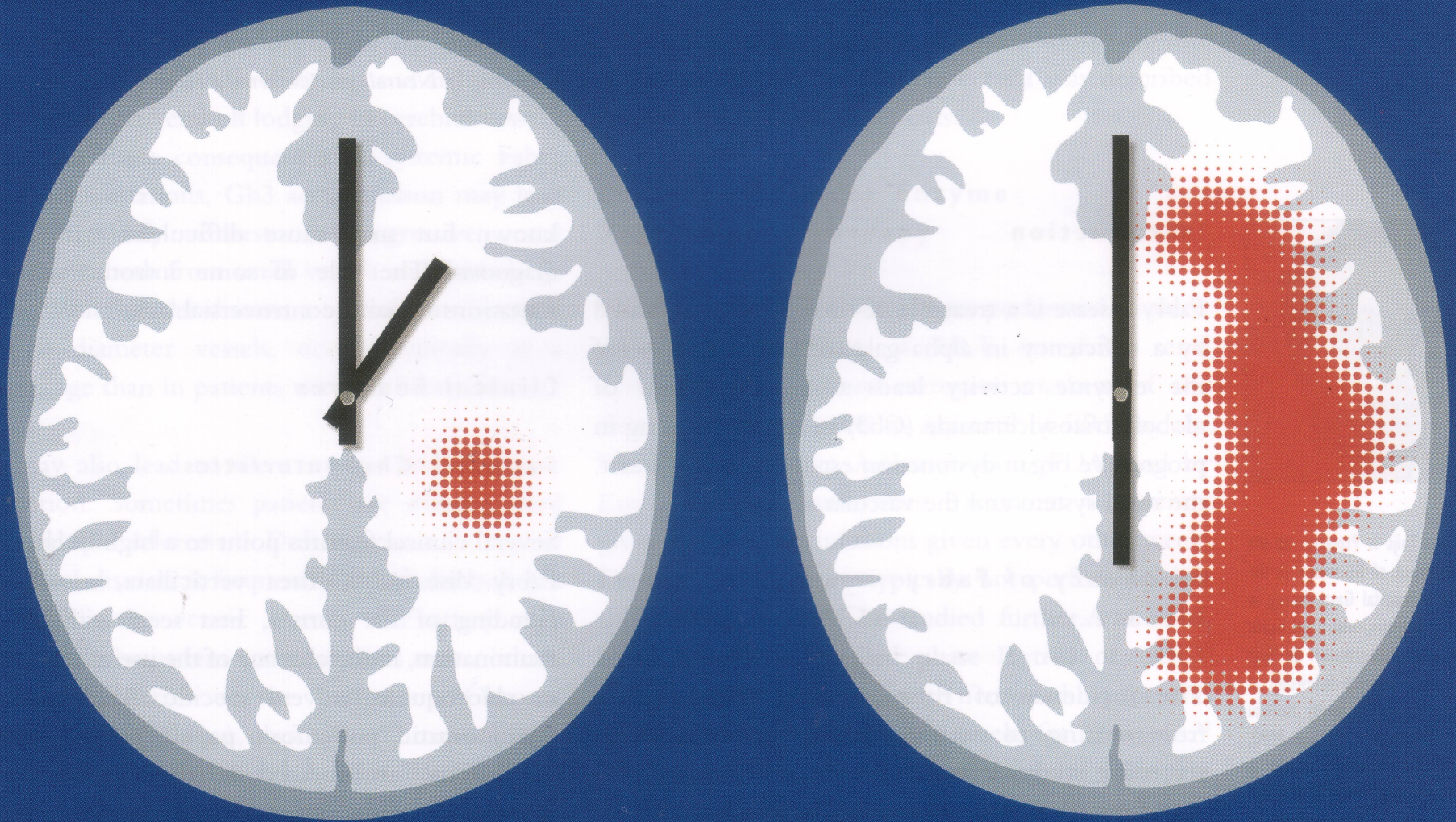
Algorithm for Stroke and TIA Management

Rounded boxes are diagnoses; rectangles are interventions. Numbers are percentages of stroke overall. TIA, transient ischemic attack; ABCs, airway, breathing, circulation; BP, blood pressure; CEA, carotid endarterectomy; SAH, subarachnoid hemorrhage; ICH, intracerebral hemorrhage.



Vascular lesion – hemorrhagic STROKE

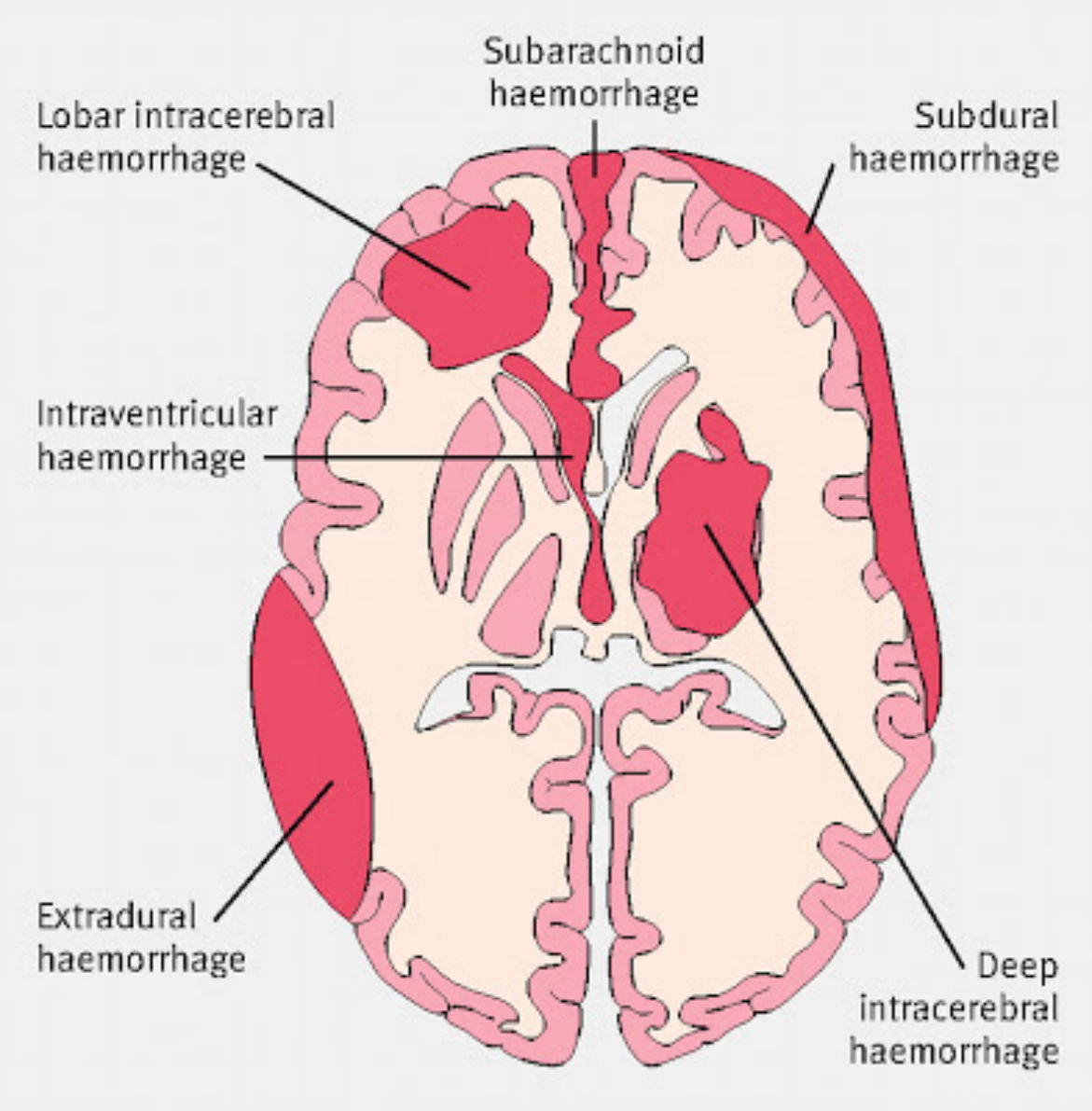




INTRACRANIAL HEMORRHAGE

is caused by bleeding directly into or around the brain; it produces neurologic symptoms by producing a mass effect on neural structures, from the toxic effects of blood itself, or by increasing intracranial pressure

INTRACRANIAL HEMORRHAGE



CAUSES OF INTRACRANIAL HEMORRHAGE

CAUSE	LOCATION	COMMENTS
Head trauma	Intraparenchymal: frontal lobes, anterior temporal lobes; subarachnoid	Coup and contracoup injury during brain deceleration
Hypertensive hemorrhage	Putamen, globus pallidus, thalamus, cerebellar hemisphere, pons	Chronic hypertension produces hemorrhage from small (~100 μm) vessels in these regions
Transformation of prior ischemic infarction	Basal ganglion, subcortical regions, lobar	Occurs in 1-6% of ischemic strokes with predilection for large hemispheric infarctions
Metastatic brain tumor	Lobar	Lung, choriocarcinoma, melanoma, renal cell carcinoma, thyroid, atrial myxoma
Coagulopathy	Any	Uncommon cause; often associated with prior stroke or underlying vascular anomaly
Drug	Lobar, subarachnoid	Cocaine, amphetamine, phenylpropranolamine
Arteriovenous malformation	Lobar, intraventricular, subarachnoid	Risk is ~2-4% per year for bleeding
Aneurysm	Subarachnoid, intraparenchymal, rarely subdural	Mycotic and nonmycotic forms of aneurysms
Amyloid angiopathy	Lobar	Degenerative disease of intracranial vessels; linkage to Alzheimer's disease, rare in patients <60
Cavernous angioma	Intraparenchymal	Multiple cavernous angiomas linked to mutations in KRIT1, CCM2, and PDCD10 genes
Dural arteriovenous fistula	Lobar, subarachnoid	Produces bleeding by venous hypertension
Capillary telangiectasias	Usually brainstem	Rare cause of hemorrhage

INTRACRANIAL HEMORRHAGE

A cerebral hemorrhage can take several forms:

Intracerebral hemorrhages. This is bleeding inside the brain. The symptoms and prognosis of an intracerebral bleed vary depending on the size and location of the bleed.

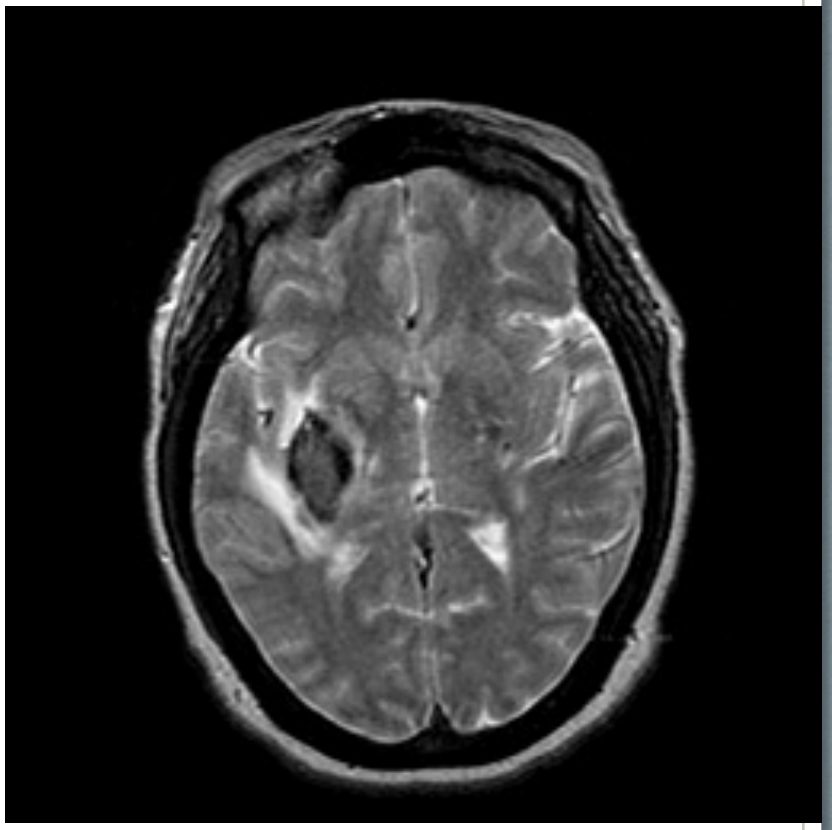
Subarachnoid hemorrhages. This is bleeding between the brain and the membranes that cover the brain.

Subdural hemorrhages. This is bleeding between the layers of the brain's covering (the meninges).

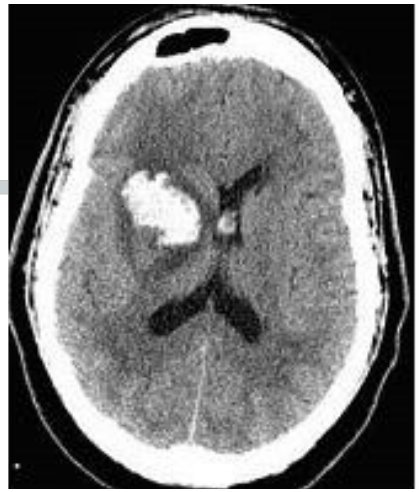
Epidural hemorrhages. This is bleeding between the skull and the covering of the brain.

CEREBROVASCULAR DISEASES

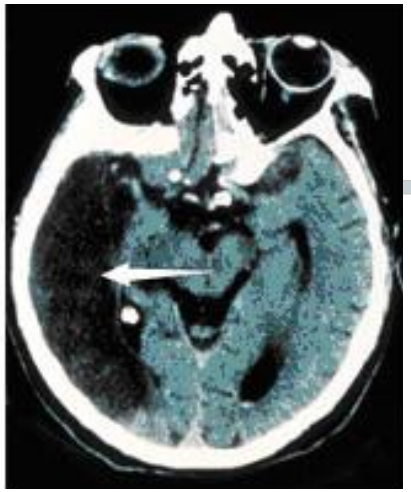
In cerebral hemorrhage, blood leaks from the vessel (usually a small artery) directly into the brain forming a hematoma in the brain substance, and spreading into the ventricles and subarachnoid space. Once the leakage is arrested, the blood slowly disintegrates and is absorbed over a period of weeks and months. The mass of clotted blood causes physical disruption of the tissue and pressure on the surrounding brain. Blood within the subarachnoid space, mainly the result of aneurysmal rupture, may cause cerebral ischemia through a mechanism of constriction of the vessels of the circle of Willis and their primary branches (vasospasm).



STROKE: CT PICTURES

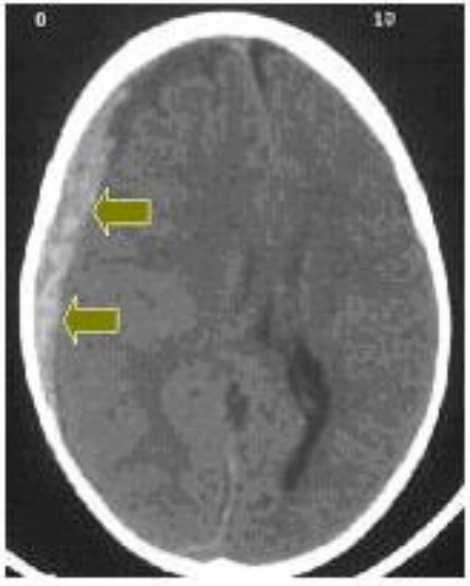


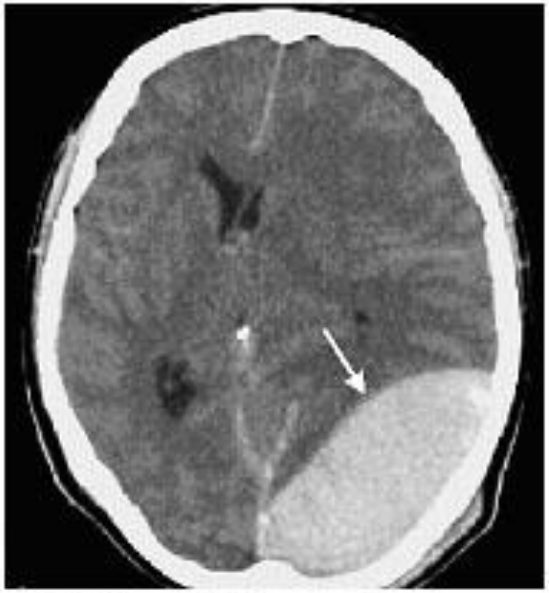
HEMORAGIE



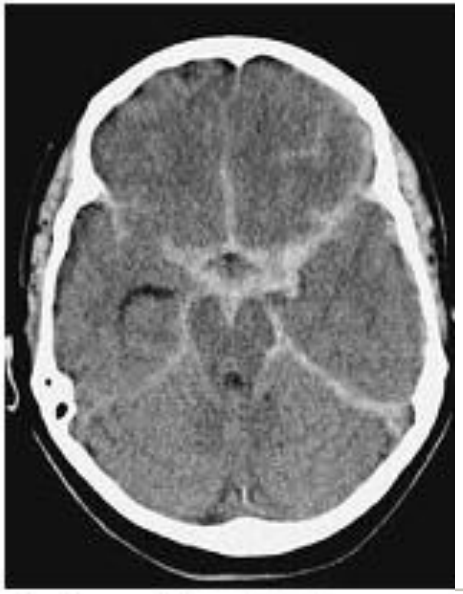
ISCHEMIE

Hematom subdural





Hematom epidural



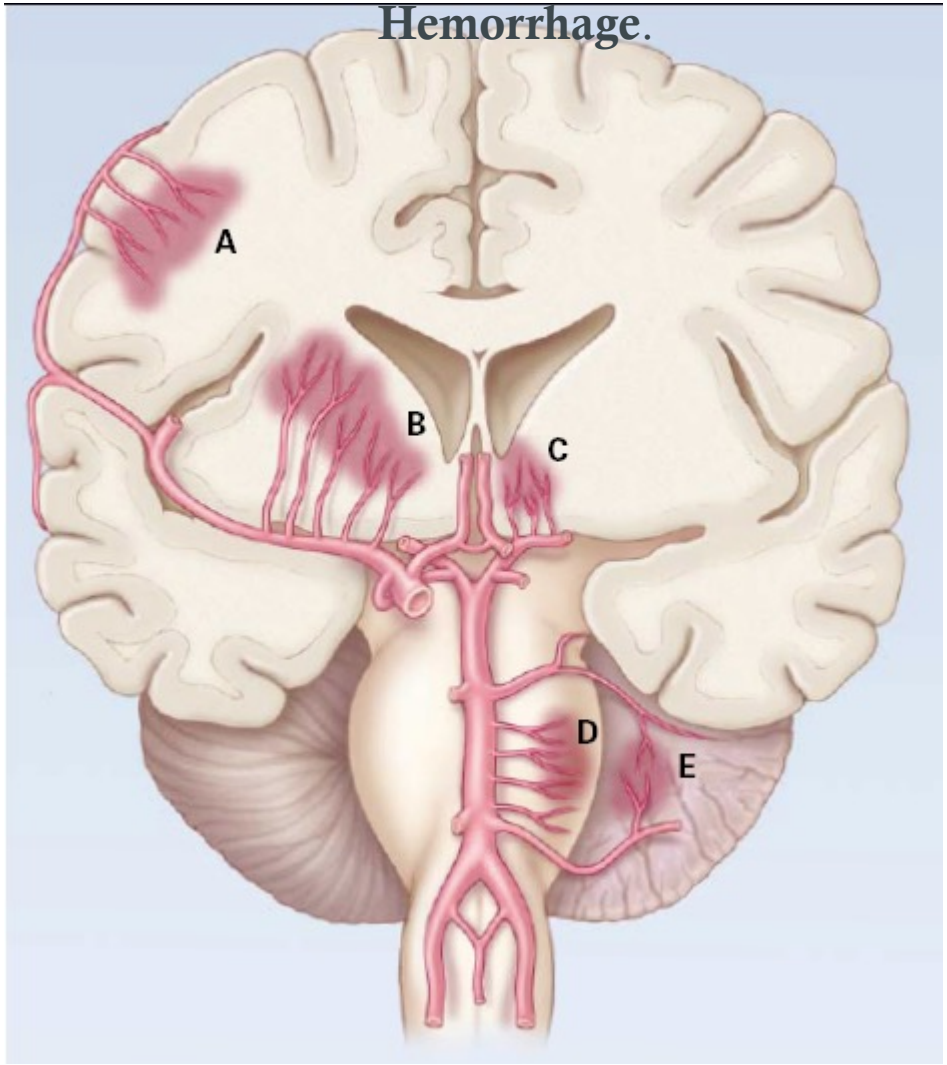
Hemoragie subarahnoidiană

Most Common Sites and Sources of Intracerebral Hemorrhage.

Intracerebral hemorrhages most commonly involve cerebral lobes, originating from:

penetrating cortical branches of the anterior, middle, or posterior cerebral arteries (A);

basal ganglia, originating from ascending lenticulostriate branches of the middle cerebral artery (B);



the thalamus, originating from ascending thalmogeniculate branches of the posterior cerebral artery (C);

and the cerebellum, originating from penetrating branches of the posterior inferior, anterior inferior, or superior cerebellar arteries (E).

the pons, originating from paramedian branches of the basilar artery (D);

INTRACRANIAL HEMORRHAGE

Causes

There are several causes of bleeding inside the skull, including:

Head injuries. For people under the age of 50, this is the most common cause of hemorrhage inside the skull. In the elderly, subdural hematoma after relatively minor head injury is not uncommon.

Arteriovenous malformation (AVM). This is an anatomical abnormality in the arteries or veins in or around the brain. Such an abnormality may be present from birth, but it is only detected if symptoms develop. Symptoms resulting from bleeding of an AVM vary, depending on size and location.

Aneurysm. This is a weakening in a blood vessel wall that swells. The thin walls of an aneurysm can burst and cause bleeding into the subarachnoid space and the brain, leading to hemorrhagic stroke.

Hypertension. Poorly controlled hypertension over a long period of time can weaken blood vessel walls.

INTRACRANIAL HEMORRHAGE

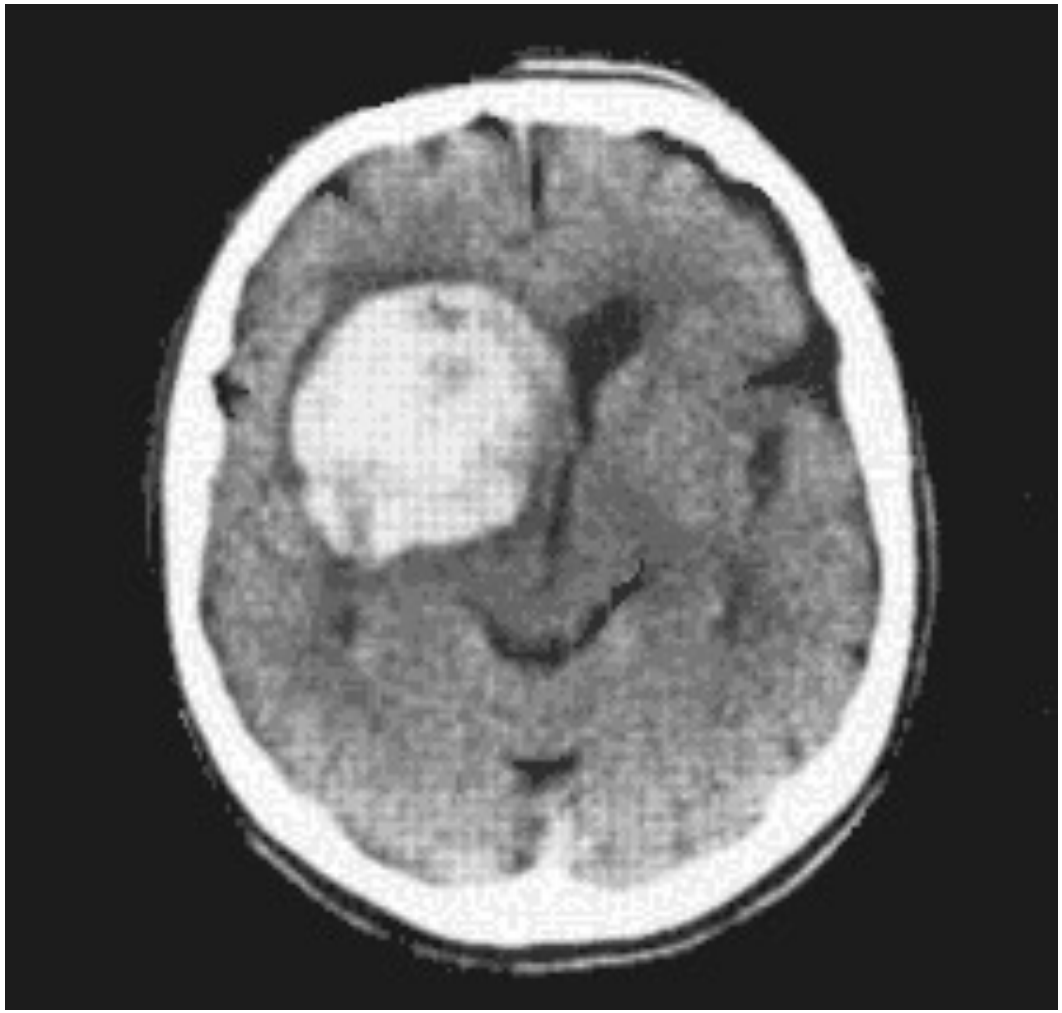
Primary (Hypertensive) Intracerebral Hemorrhage

This is the common, well-known "spontaneous" brain hemorrhage. It is due predominantly to chronic hypertension and degenerative changes in cerebral arteries. In recent decades, with increased awareness of the need to control blood pressure, the proportion of cases attributable to hypertension has been greatly reduced; more than one-third such hemorrhages are in normotensives, and the hemorrhages more often than previously arise in locations that are not typical for hypertension.

Nevertheless, the hypertensive cerebral hemorrhage serves as a paradigm for understanding and managing all the other types.

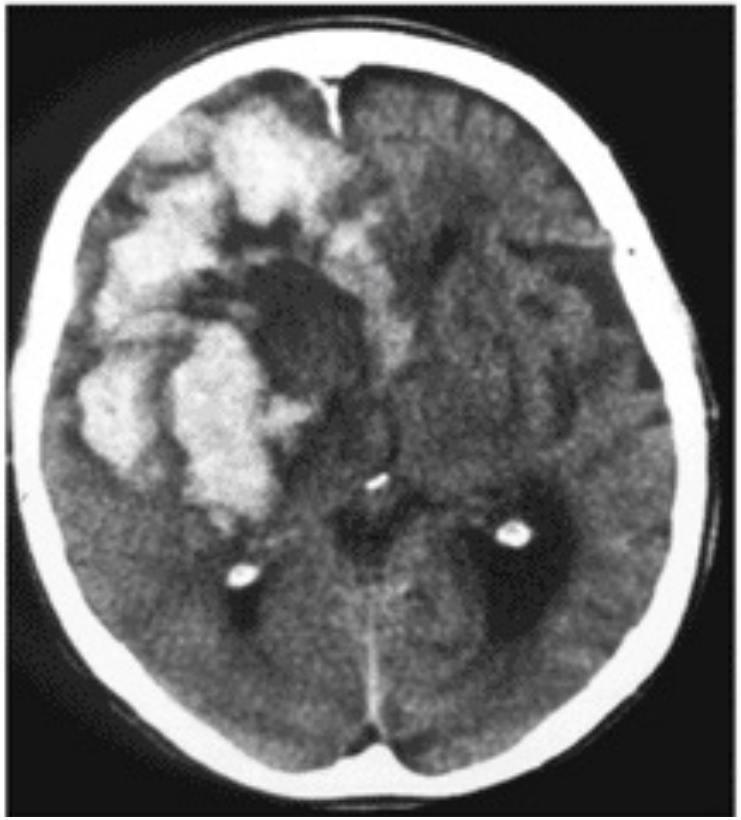
INTRACRANIAL HEMORRHAGE

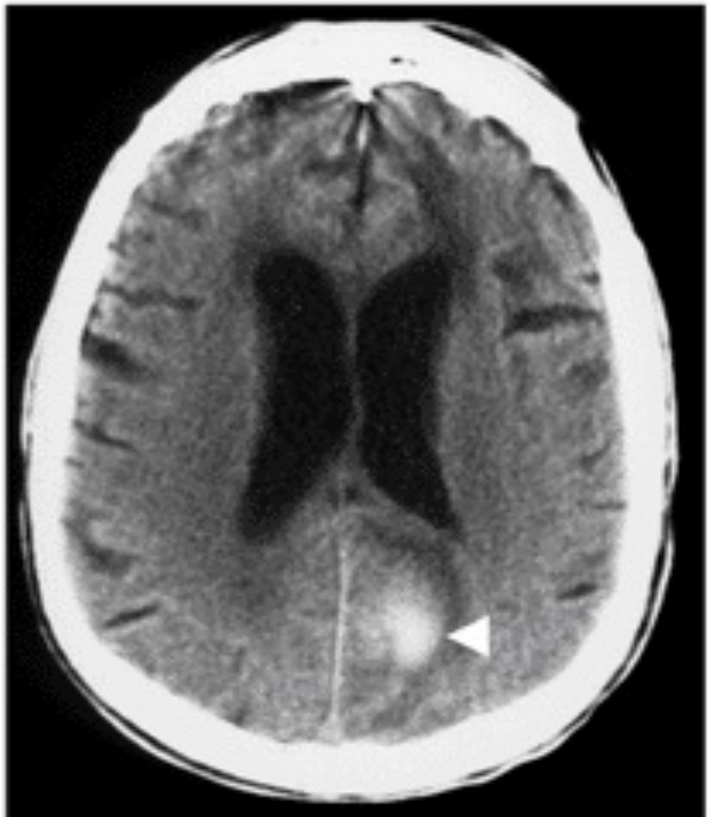
The bleeding occurs within brain tissue, and rupture of arteries lying in the subarachnoid space is practically unknown apart from aneurysm. The extravasation forms a roughly circular or oval mass that disrupts the tissue and grows in volume as the bleeding continues.



An unenhanced CT scan showing the typical picture of a massive primary (hypertensive) hemorrhage in the basal ganglia. The third ventricle and opposite lateral ventricle are compressed and displaced by the expanding mass (12 h after onset of stroke).

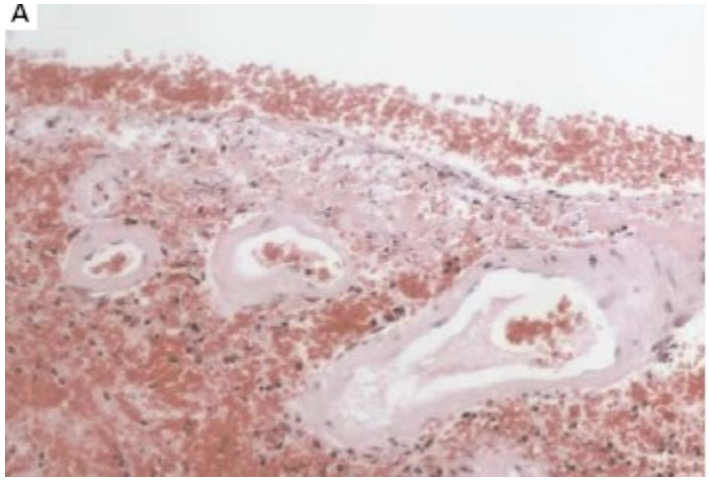
Amyloid cerebral angiopathy

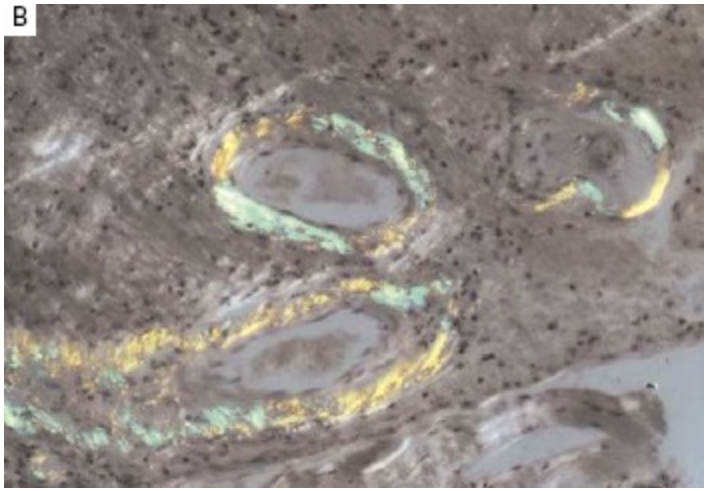




INTRACRANIAL HEMORRHAGE

Pathogenesis The nature of the hypertensive vascular lesion that leads to arterial rupture is not fully known, but in the few cases studied by serial sections, the hemorrhage appeared to arise from an arterial wall altered by the effects of hypertension, i.e., the change referred to in a preceding section as segmental lipohyalinosis and the false aneurysm (microaneurysm) of Charcot-Bouchard. Ross Russell has affirmed the relationship of these aneurysms to hypertension and hypertensive hemorrhage, and their frequent localization on penetrating small arteries and arterioles of the basal ganglia, thalamus, pons, and subcortical white matter.





INTRACRANIAL HEMORRHAGE

Clinical Picture Of all the cerebrovascular diseases, brain hemorrhage is the most dramatic and from ancient times has been surrounded by "an aura of mystery and inevitability." It has been given its own name, "apoplexy." The prototype is an obese, plethoric, hypertensive male who, while sane and sound, falls senseless to the ground - impervious to shouts, shaking, and pinching - breathes stertorously, and dies in a few hours. A massive blood clot escapes from the brain as it is removed postmortem.

With smaller hemorrhages, the clinical picture conforms more closely to the usual temporal profile of a **stroke**, i.e., an abrupt onset of symptoms that evolve gradually and steadily over minutes, hours, or a day or two, depending on the size of the ruptured artery and the speed of bleeding.

INTRACRANIAL HEMORRHAGE

Laboratory Findings.

Among laboratory methods for the diagnosis of intracerebral hemorrhage, the **CT scan** occupies the foremost position. This procedure has proved totally reliable in the detection of hemorrhages that are 1.0 cm or more in diameter.

Smaller pontine hemorrhages are visualized with less certainty.

The CT scan is particularly useful in the diagnosis of brain hemorrhages that do not spill blood into the CSF and were heretofore clinically unrecognizable.

At the same time, coexisting hydrocephalus, tumor, cerebral swelling, and displacement of the intracranial contents are readily appreciated.

MRI is particularly useful for demonstrating brainstem hemorrhages and residual hemorrhages, which remain visible long after they can no longer be seen by the CT scan (after 4 to 5 weeks).

Hemosiderin and iron pigment have their own characteristic appearances.

INTRACRANIAL HEMORRHAGE

Treatment. The general medical management of the comatose patient with intracerebral hemorrhage is the same as that of patients with ischemic or embolic infarction. The management of patients with large intracerebral hemorrhages and coma includes the maintenance of adequate ventilation, use of controlled hyperventilation to a PCO_2 of 25 to 30 mmHg, monitoring of intracranial pressure and its control by the use of tissue-dehydrating agents such as mannitol (osmolality kept at 295 to 305 mosmol/L and Na at 145 to 150 meq), and limiting fluid intake to 1200 mL/day, given as intravenous infusions of normal saline.

INTRACRANIAL HEMORRHAGE

Treatment Rapid reduction in blood pressure, in the hope of reducing further bleeding, is not recommended, since it risks compromising cerebral perfusion in cases of raised intracranial pressure. On the other hand, sustained mean blood pressures of greater than 110 mmHg may exaggerate cerebral edema and risk further bleeding. It is at approximately this level of acute hypertension that the use of beta-blocking (esmolol, labetalol), or angiotensin-converting enzyme inhibitory drugs is recommended.

Diuretics are helpful in combination with any of the antihypertensive medications. More rapidly acting and titratable agents such as nitroprusside may be used in extreme situations, recognizing that they may further raise intracranial pressure.

INTRACRANIAL HEMORRHAGE

Treatment Surgical removal of the clot in the acute stage, either by evacuation or aspiration, may occasionally be lifesaving, and we have referred numerous patients, in whom hemispheral hemorrhages were more than 3 cm in diameter and whose clinical state was deteriorating, for surgical treatment.

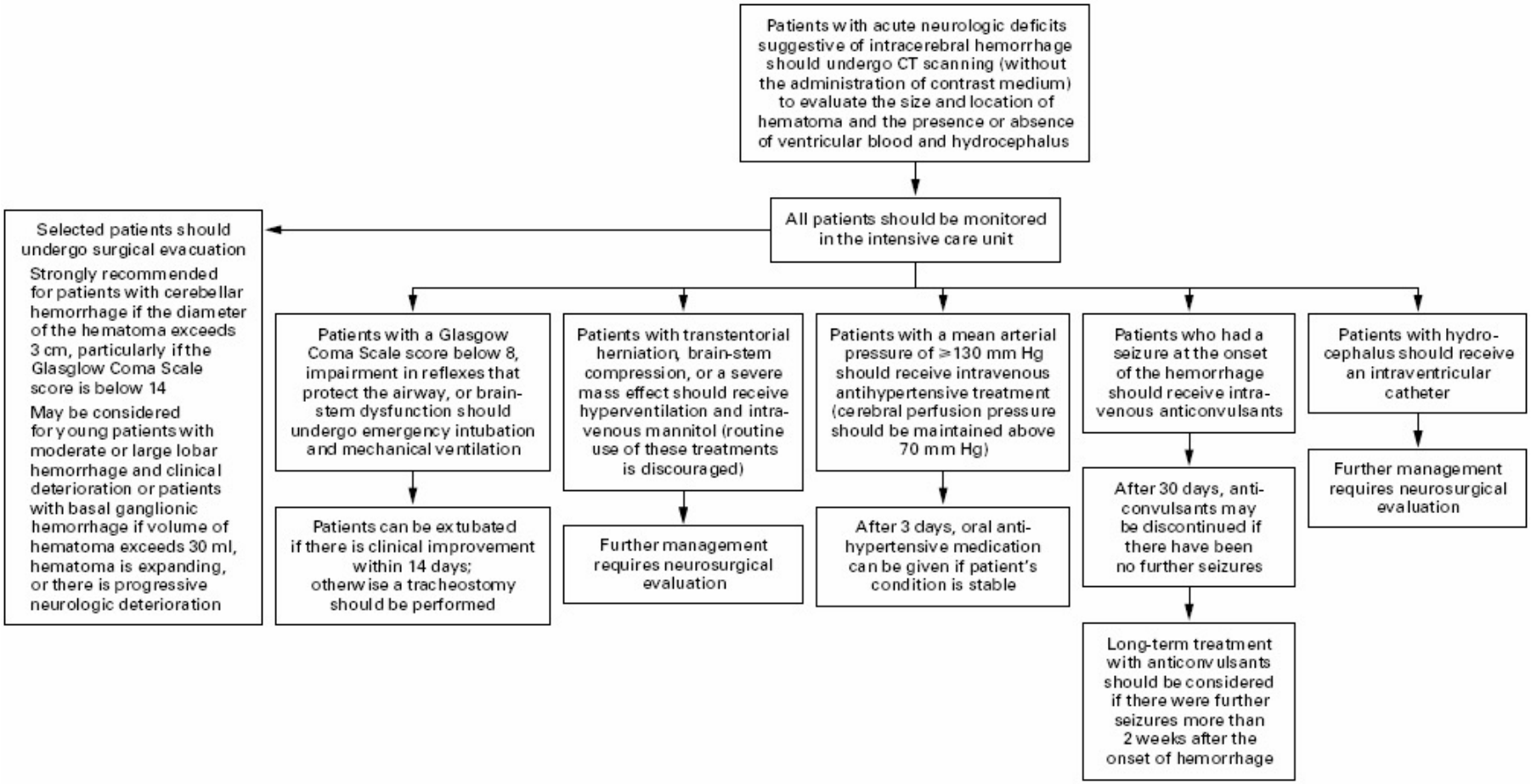
The most successful surgical results have been in patients with lobar or putaminal hemorrhages.

Although selected patients may be saved from progression to brain death, the underlying focal neurologic deficit is not altered.

Moreover, even this degree of success requires that operation be carried out before or very soon after coma supervenes.

Once the patient becomes deeply comatose, with dilated fixed pupils, the chances of recovery are negligible.

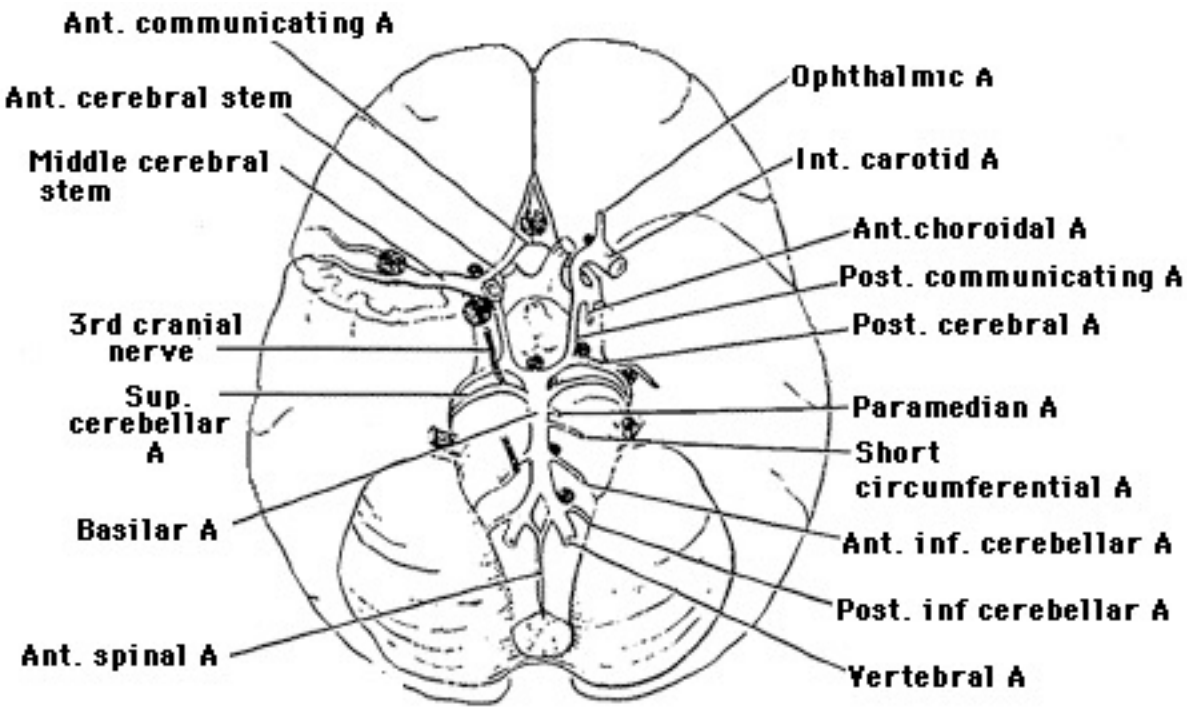
Algorithm for the Management of Intracerebral Hemorrhage.



INTRACRANIAL HEMORRHAGE

Spontaneous Subarachnoid Hemorrhage (Ruptured Saccular Aneurysm)

This is the fourth most frequent cerebrovascular disorder - following atherothrombosis, embolism, and primary intracerebral hemorrhage. Saccular aneurysms are also called "berry" aneurysms, as they take the form of small, thin-walled blisters protruding from arteries of the circle of Willis or its major branches. Their rupture causes a flooding of the subarachnoid space by blood under high pressure. As a rule, the aneurysms are located at bifurcations and branchings.



The principal sites of saccular aneurysms. Approximately 90 percent of aneurysms are on the anterior half of the circle of Willis.

Spontaneous (non-traumatic) Subarachnoid Hemorrhage

- The average annual incidence reaches 9.1 per 100,000 inhabitants
- Diagnostic and therapeutic urgency!

INTRACRANIAL HEMORRHAGE

Spontaneous Subarachnoid Hemorrhage (Ruptured Saccular Aneurysm)

Clinical Picture Prior to rupture, saccular aneurysms are usually asymptomatic. Exceptionally, if sufficiently large to compress pain-sensitive structures, they may cause localized cranial pain. With a cavernous or anterolaterally situated aneurysm on the first part of the middle cerebral artery, the pain may be localized to the orbit. An aneurysm on the posterior-inferior or anterior-inferior cerebellar artery may cause unilateral occipital or cervical pain. The presence of a partial oculomotor palsy with dilated pupil may be indicative of an aneurysm of the posterior communicating-internal carotid junction (rarely posterior communicating-posterior cerebral junction). Occasionally, large aneurysms just anterior to the cavernous sinus may compress the optic nerves or chiasm, third nerve, hypothalamus, or pituitary gland. In the cavernous sinus they may compress the third, fourth, or sixth nerves, or the ophthalmic division of the fifth nerve. A monocular visual field defect may also develop with a supraclinoid aneurysm near the anterior and middle cerebral bifurcation or the ophthalmic-carotid bifurcation.

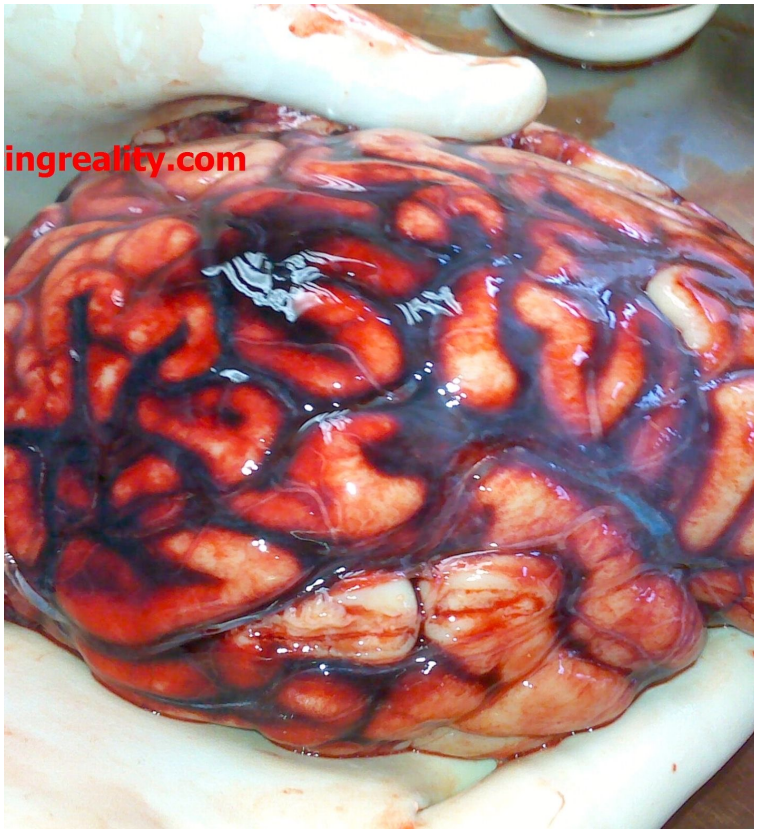
INTRACRANIAL HEMORRHAGE

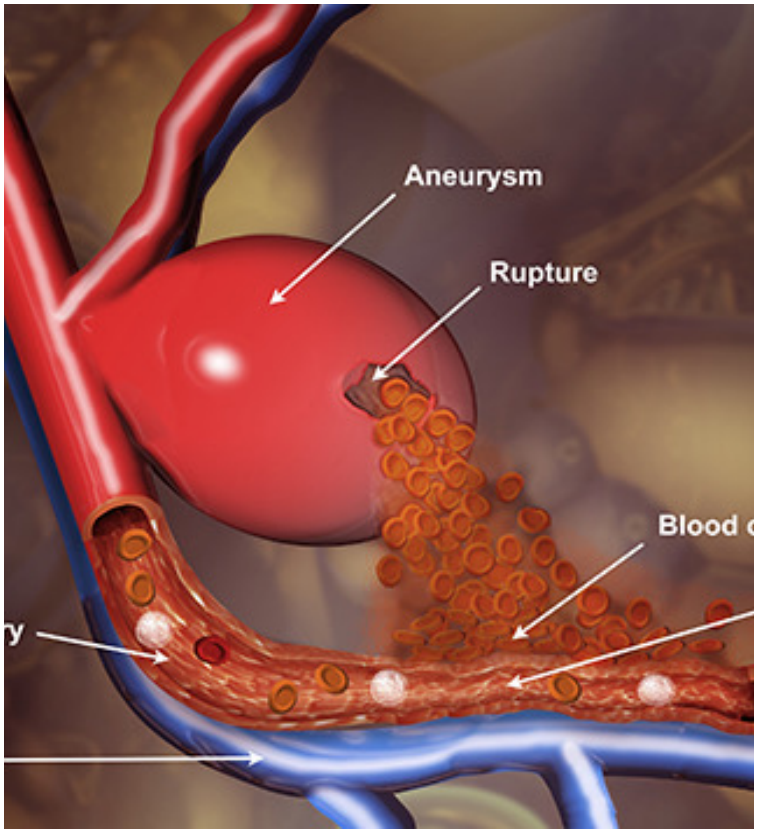
Spontaneous Subarachnoid Hemorrhage (Ruptured Saccular Aneurysm)

In summary, the clinical sequence of sudden severe headache, collapse, relative preservation of consciousness with a paucity of lateralizing signs, and neck stiffness is diagnostic of subarachnoid hemorrhage due to a ruptured saccular aneurysm.

Almost all patients are hypertensive for one or several days following the bleed, but preceding hypertension is not more common than in the general population. Levels of 200 mmHg systolic are seen occasionally just after rupture but usually the pressure is elevated only moderately and fluctuates with the degree of head pain. Spontaneous intracranial bleeding with normal blood pressure should always suggest ruptured aneurysm or arteriovenous malformation, a bleeding diathesis, and, rarely, hemorrhage into a cerebral tumor. Nuchal rigidity is usually present but occasionally absent, and the main complaint of pain may be referable to the interscapular region or even the low back rather than to the head. Examination of the fundi frequently reveals smooth-surfaced, sharply outlined collections of blood that cover the retinal vessels¾the so-called preretinal or subhyaloid hemorrhages; Roth spots are seen occasionally. Bilateral Babinski signs are found in the first few days following rupture. Fever to 39°C may be seen in the first week. Rarely, escaping blood enters the subdural space and produces a hematoma, evacuation of which may be lifesaving

SAH: medical emergency!

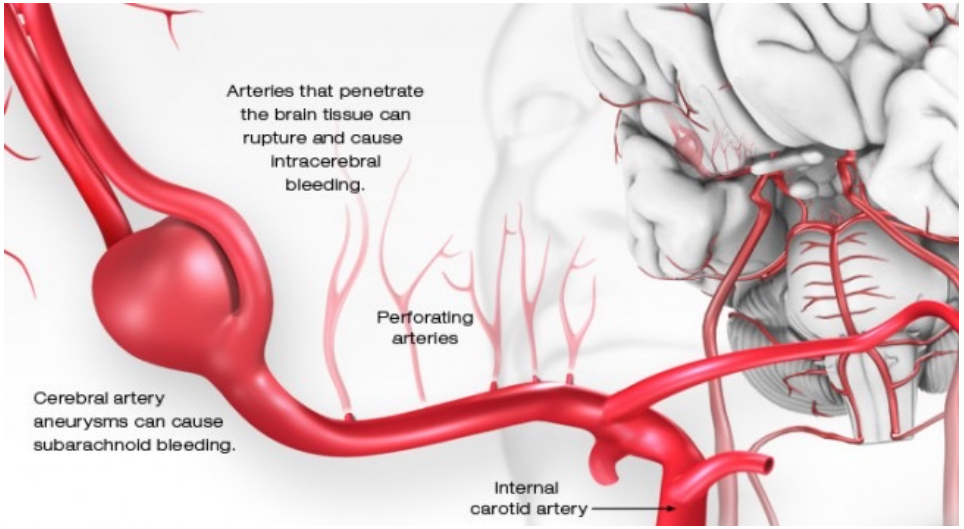


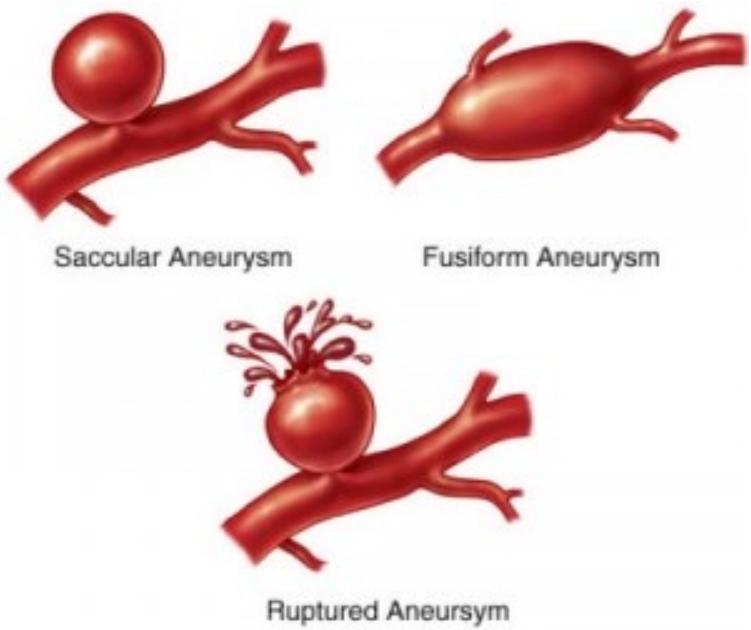


SAH etiology

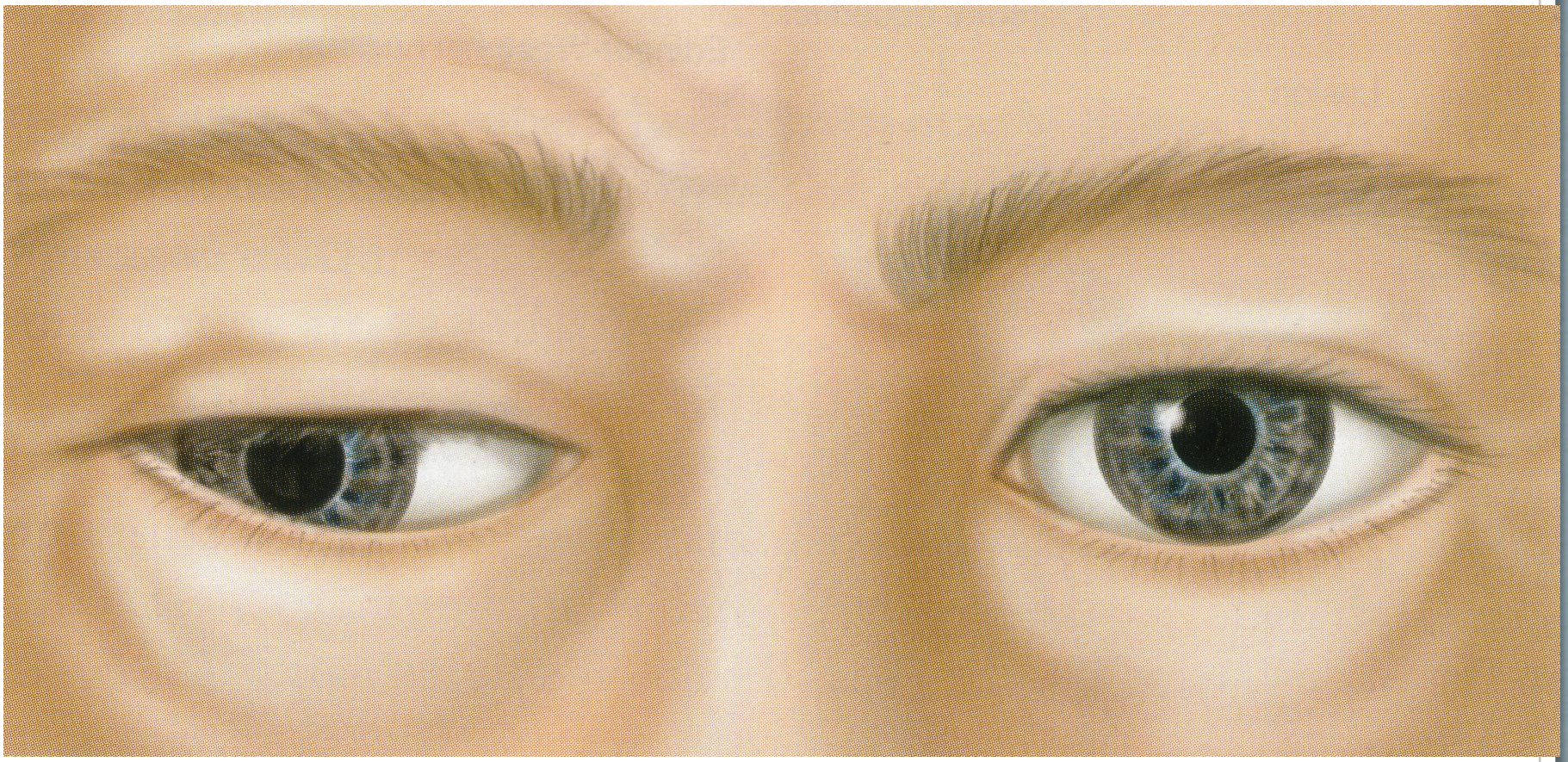
- Ruptured aneurysm (80%)
- Unspecified (15%)
- Others (5%): non-inflammatory lesions of the cerebral arteries, inflammatory lesions of the cerebral arteries, spinal cord injuries, tumors, acquired or constitutional coagulopathy, toxic.

ANEVRISM


Arteries that penetrate the brain tissue can rupture and cause intracerebral bleeding.
Perforating arteries
Cerebral artery aneurysms can cause subarachnoid bleeding.
Internal carotid artery


Saccular Aneurysm
Fusiform Aneurysm
Ruptured Aneursym

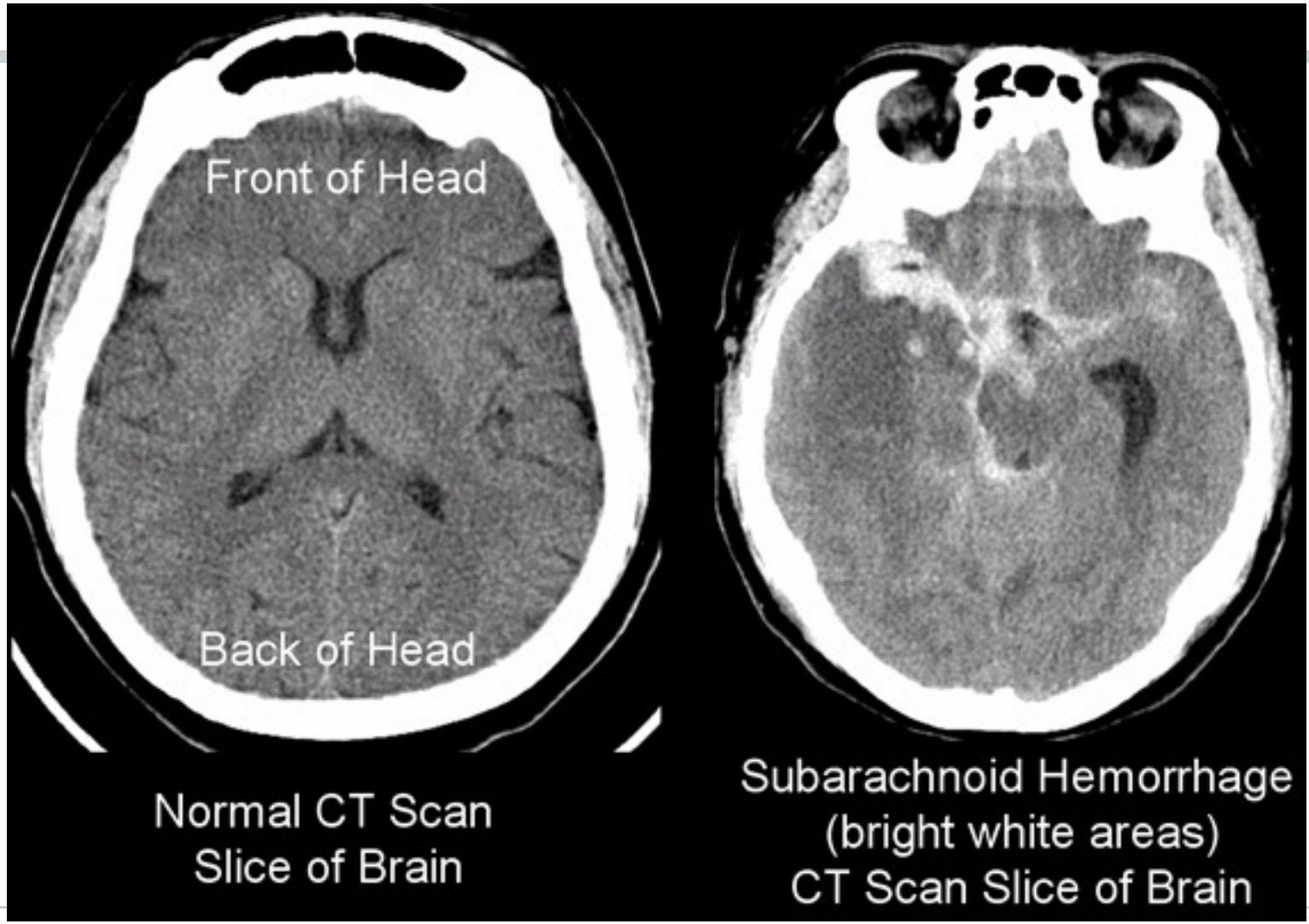
Intrinsic and extrinsic III nerve palsy



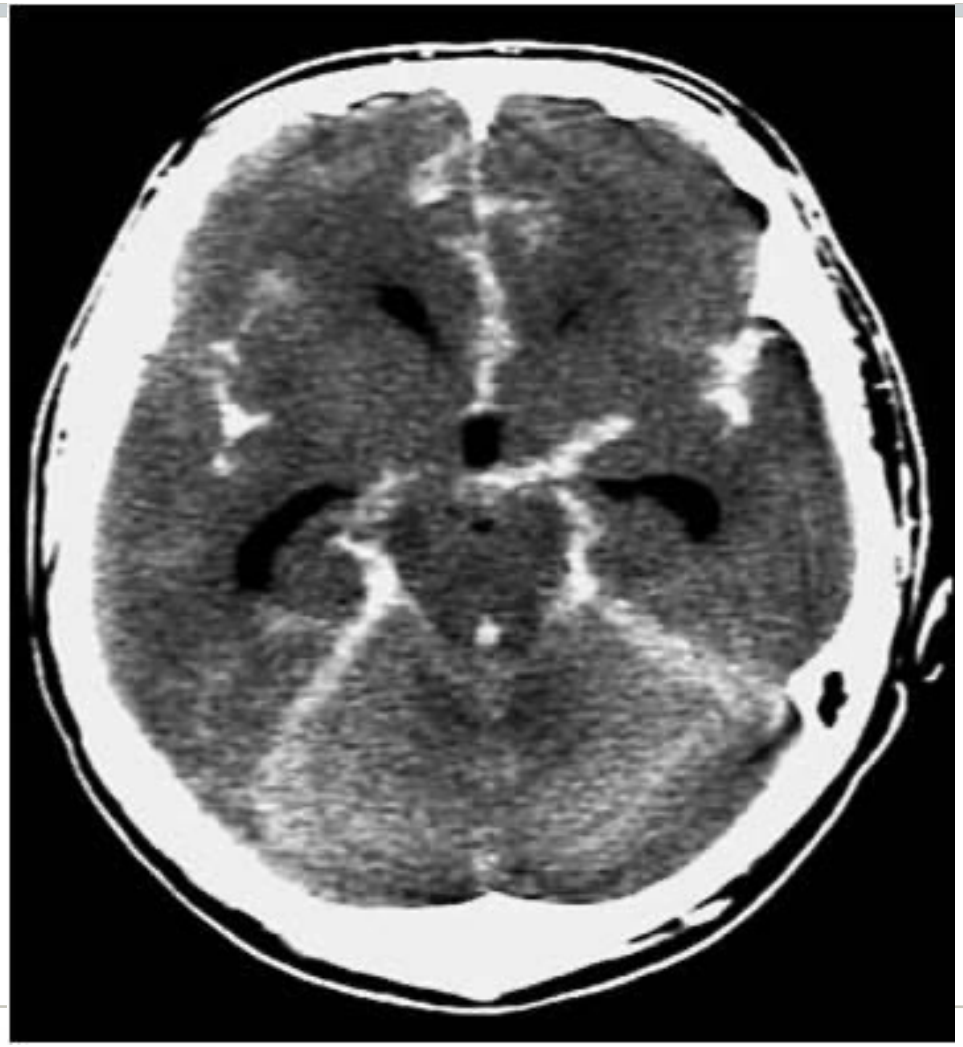
WFNS scale for SAH

WFNS degree	Glasgow score	Motor deficiency
Degree I	15	-
Degree II	14-13	-
Degree III	14-13	+
Degree IV	12-7	+ / -
Degree V	6-3	+ / -

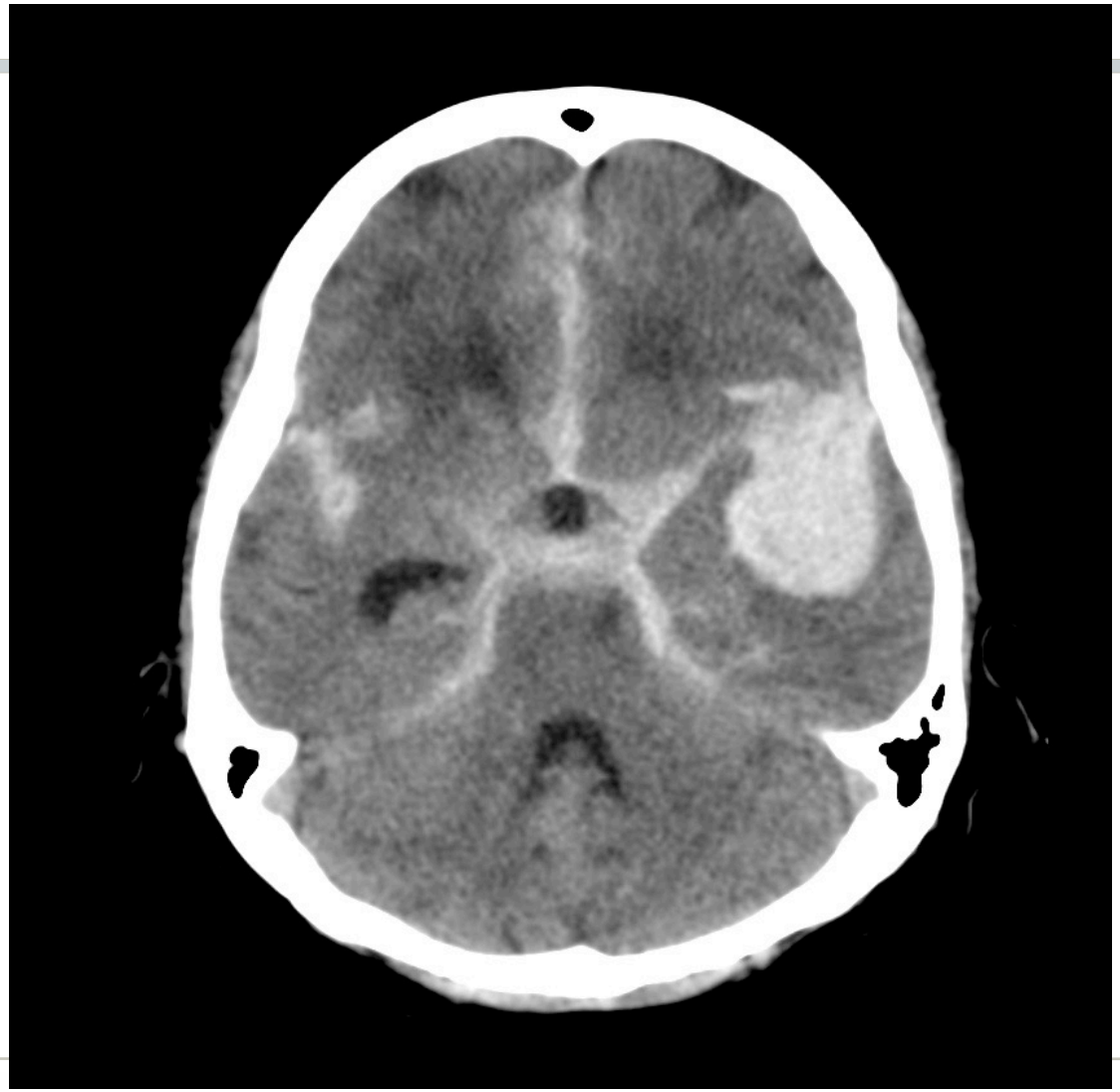
Key exam: CT!


Front of Head
Back of Head
Normal CT Scan
Slice of Brain
Subarachnoid Hemorrhage
(bright white areas)
CT Scan Slice of Brain

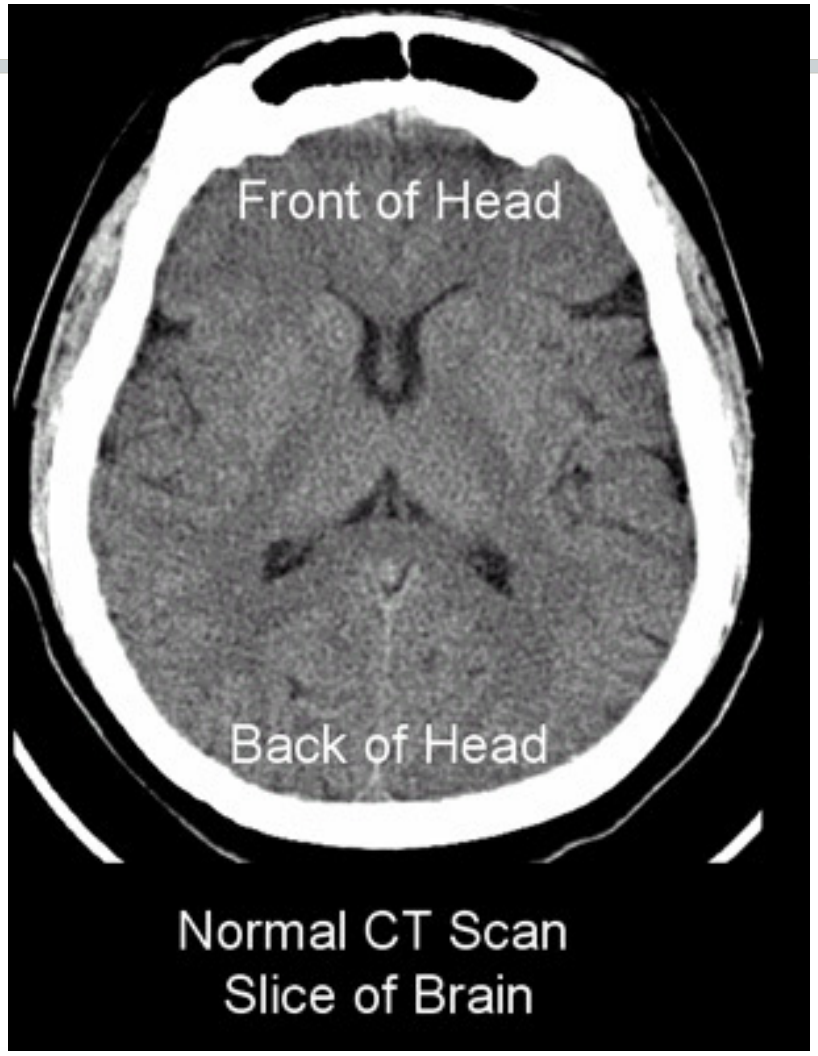
Key exam: CT!



Key exam: CT!



CT examination is normal (20% cases):



1. discrete hemorrhage

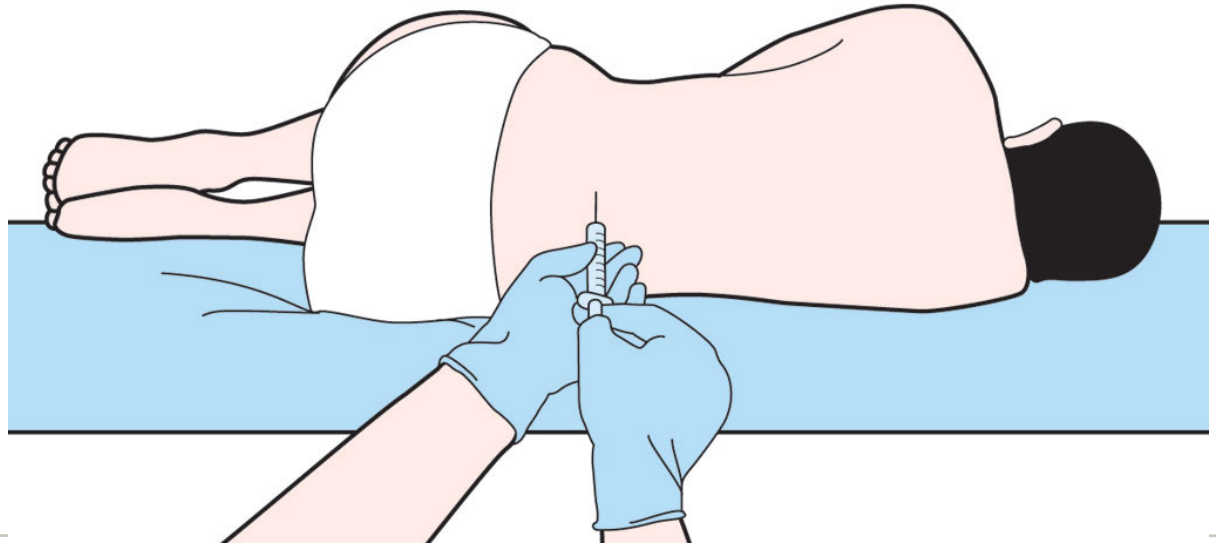
2. chronic bleeding

WHAT ARE WE DOING?

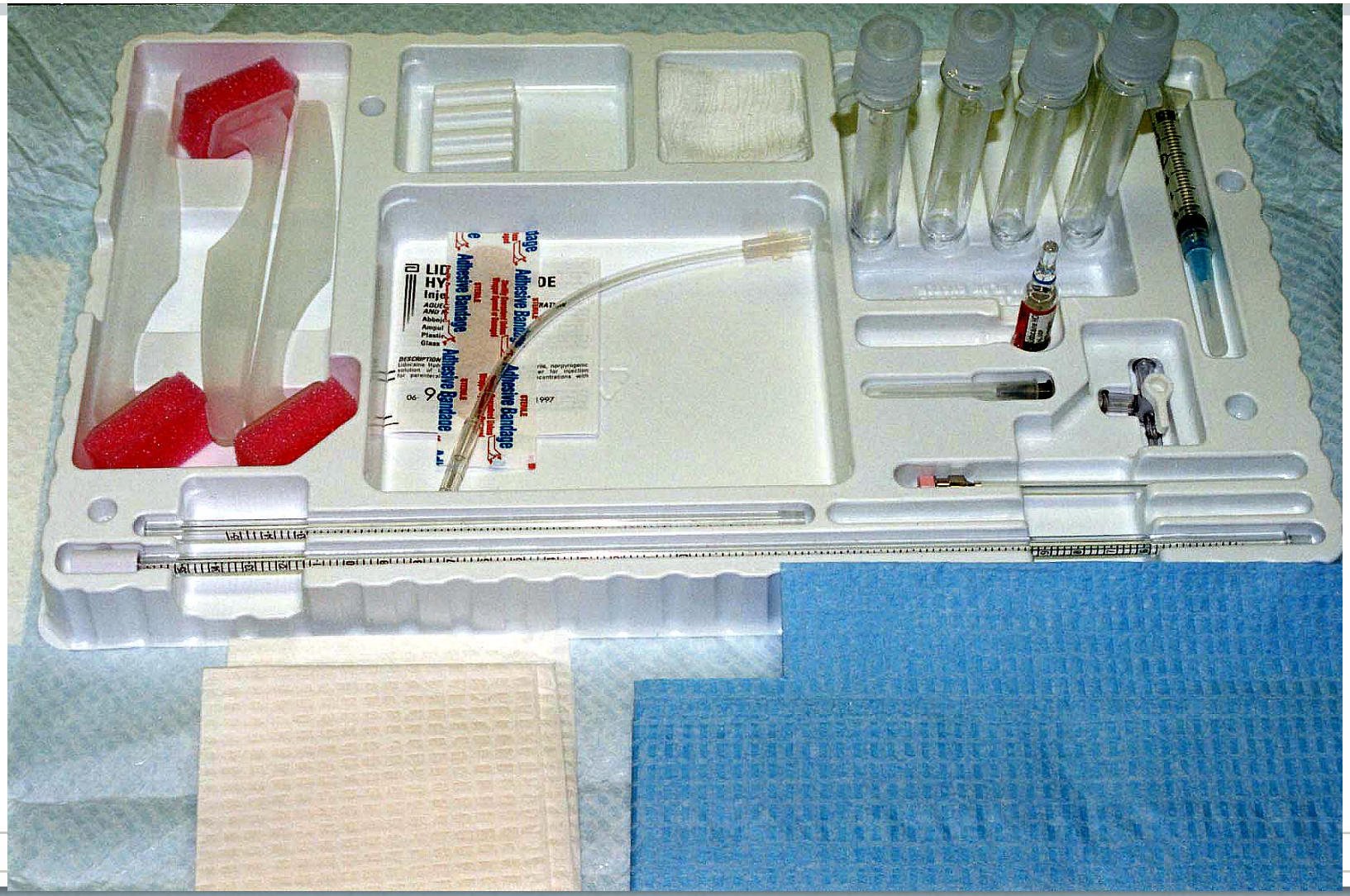
LUMBAR PUNCTURE!

LUMBAR PUNCTURE:

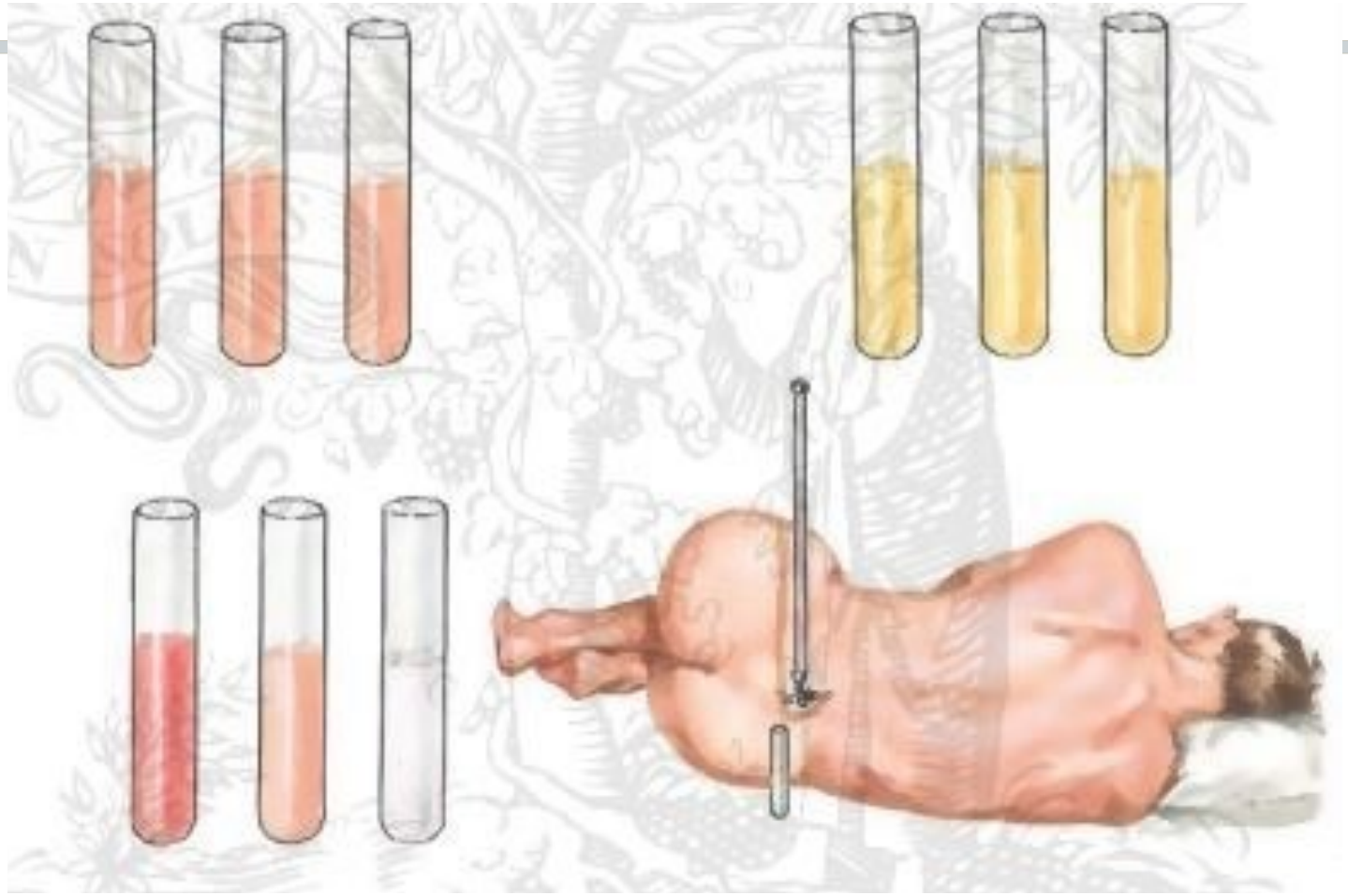
- Normal CT exam
- It is contraindicated in intracranial hypertension syndrome
- Preceded by ophthalmoscopy
- Informed consent (elimination of myths)



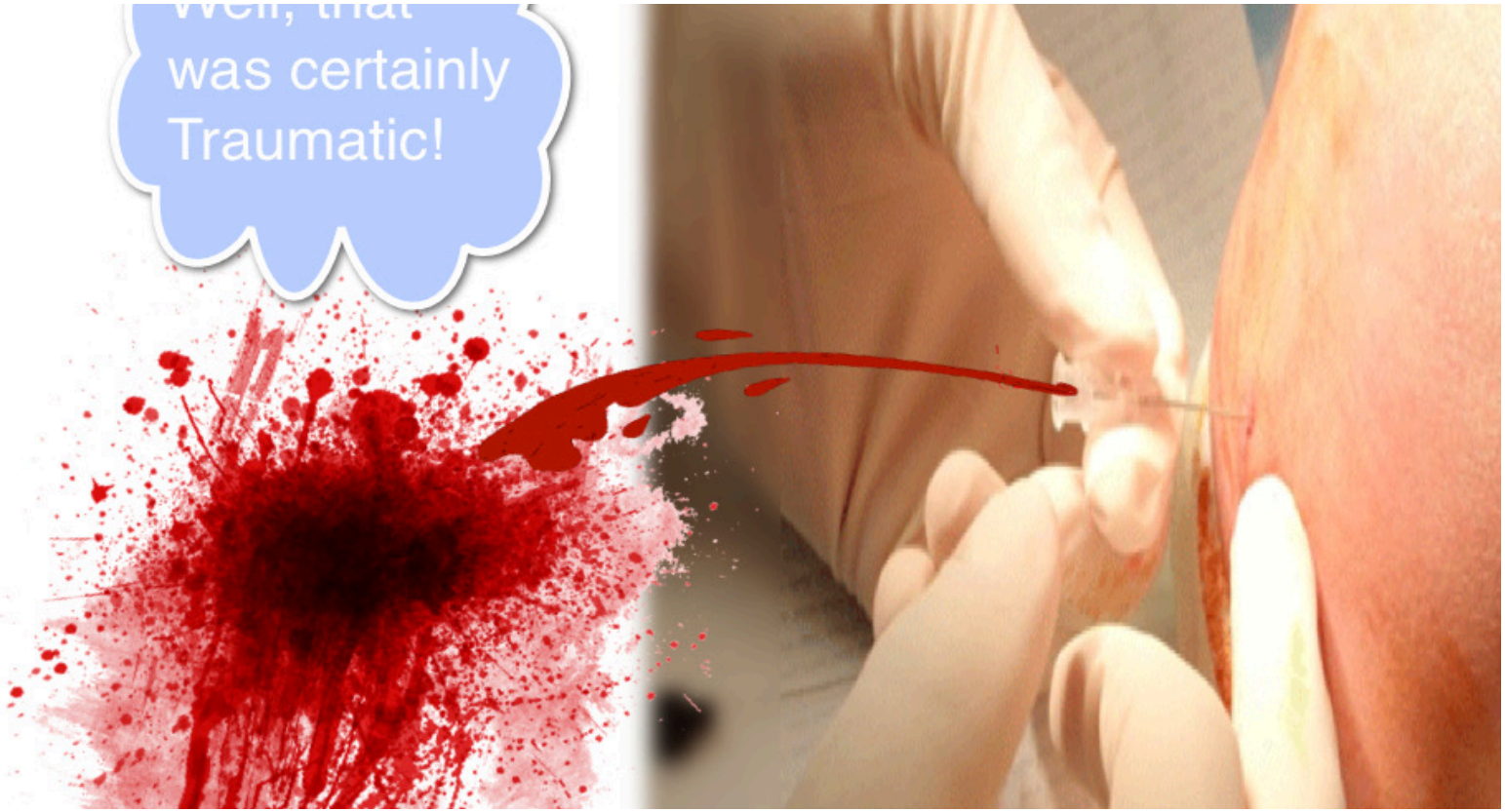
Lumbar puncture kit



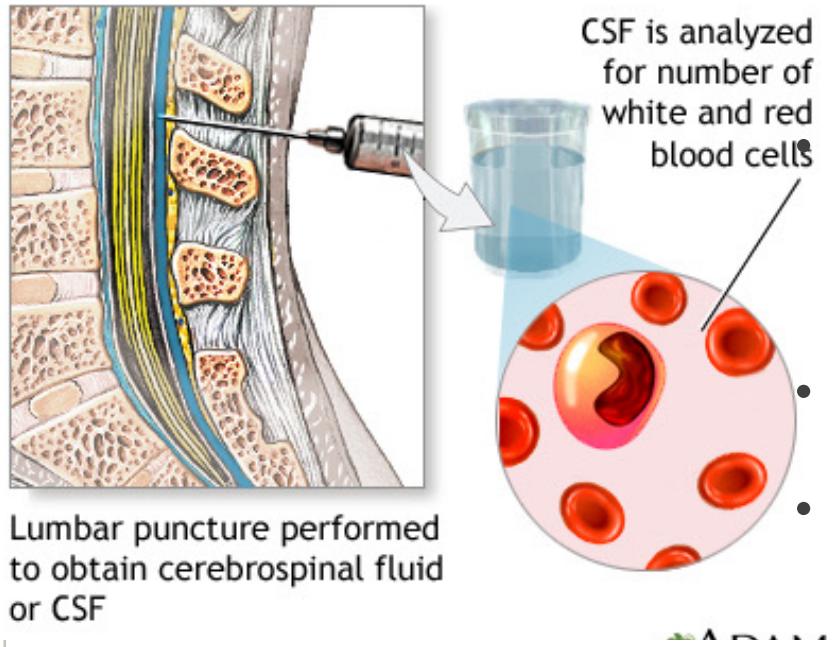
Lumbar puncture in SAH: the test of the 3 tubes



associated blood?

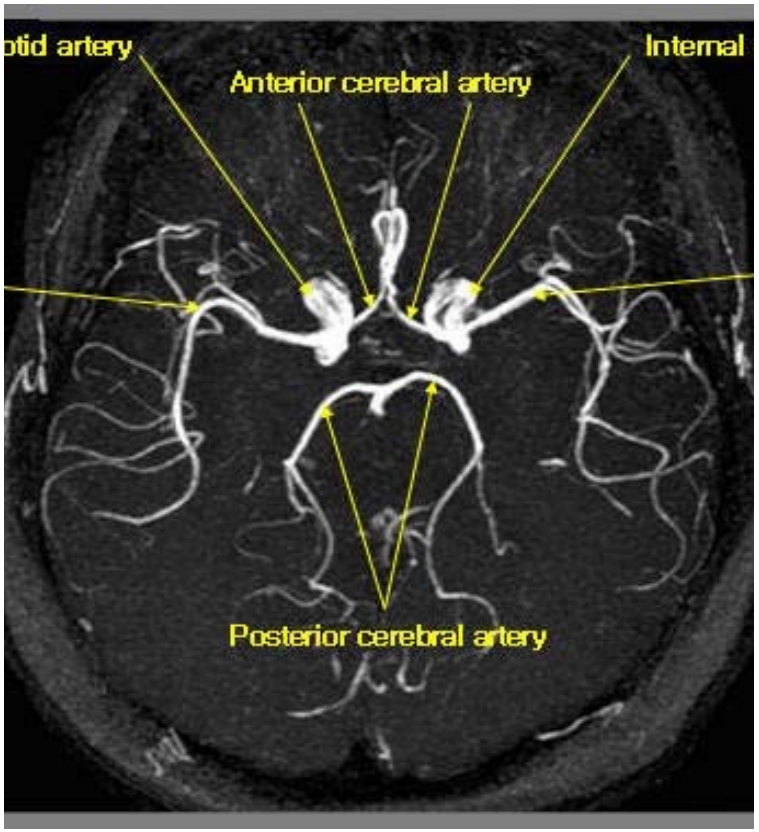


SAH: abnormalities of CSF



- Red (pink) uniform color, incoagulable in three tubes
- Xanthochromic supernatant after centrifugation, with blood pigmentation (starting at 12 o'clock)
- Increased number of red blood cells
- Increased pressure

Investigations to detect the etiology of SAH

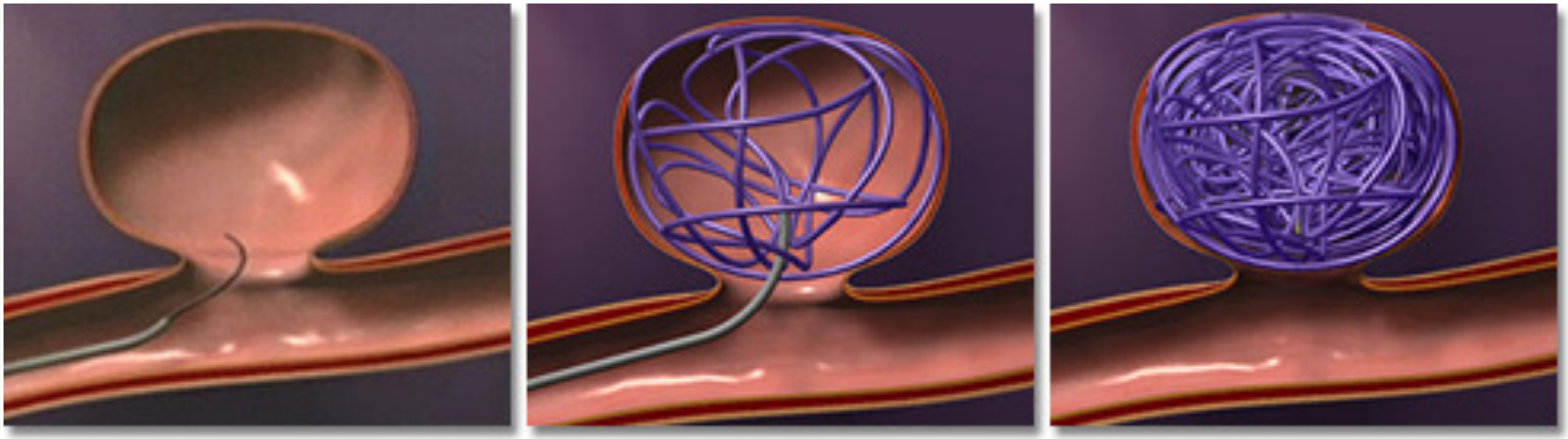


- CT angiography with 3D reconstruction
- Angiography with digital subtraction (if the angio-CT scan is negative)
- Other examinations as appropriate (hemocultures - mycotics; accordin to the context.)

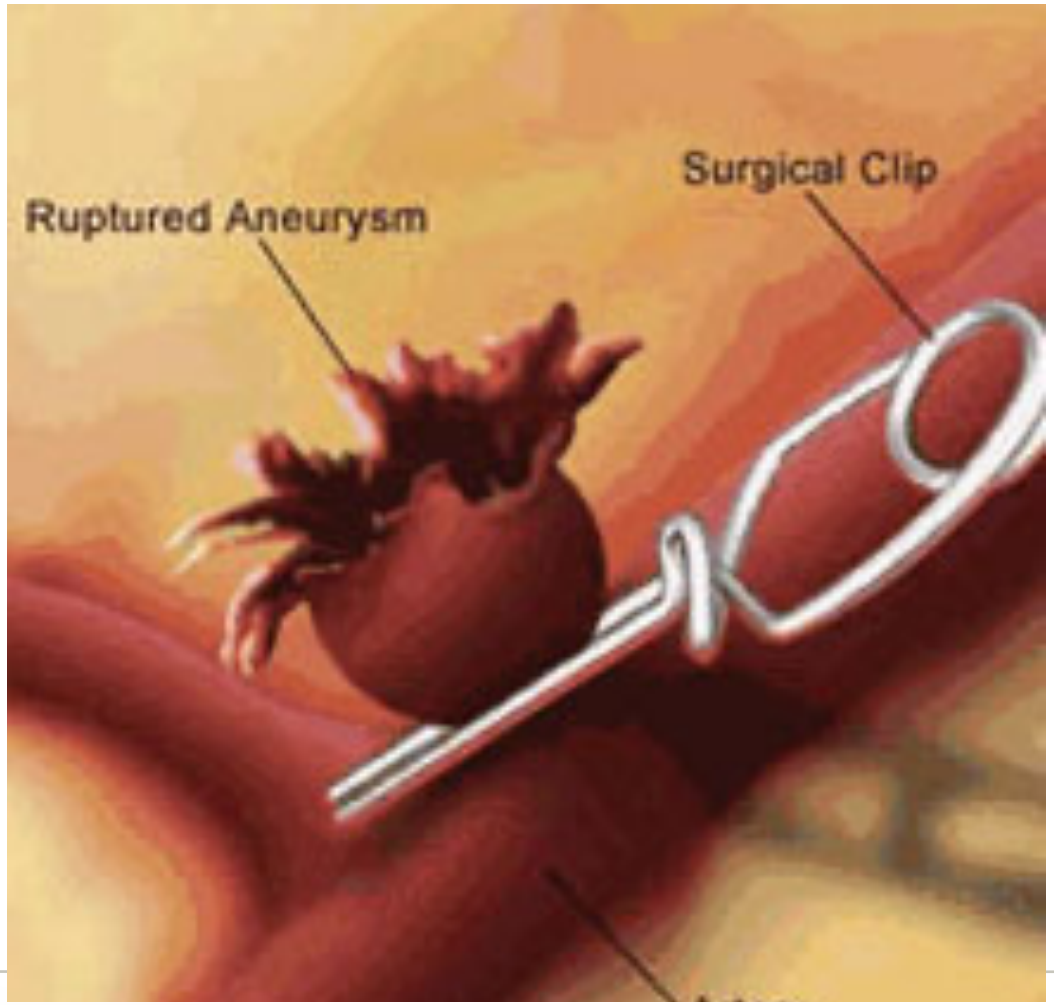
SAH: treatment

- Immediate transfer to the neurosurgery
- Treatament of the pain of acute meningeal syndrome
- Suppression of the cause of bleeding
- Prevention and treatment of possible complications

Endovascular SAH treatment:
COILING of aneurysm



Endovascular HSA treatment: aneurysm cliping



SAH: complications

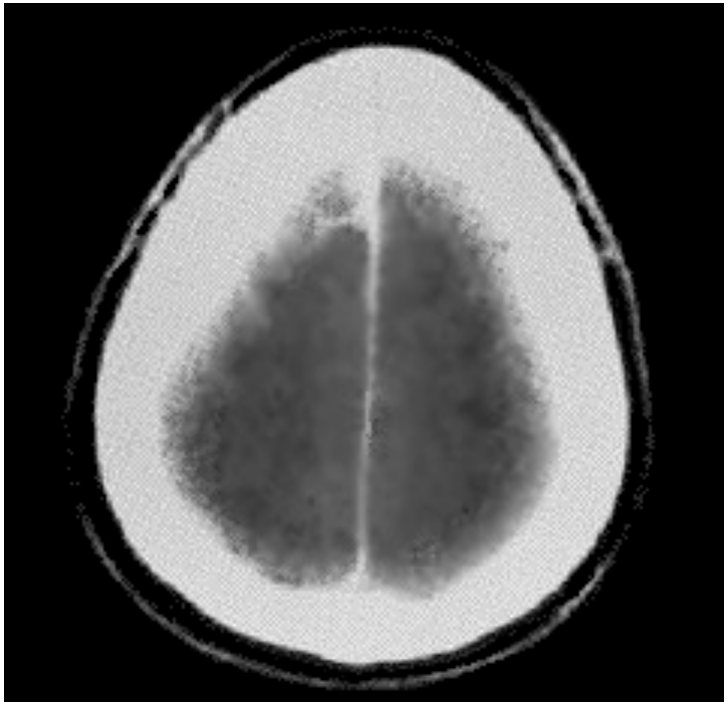
- ICH syndrome
- Early acute hydrocephalus
- Hemorrhagic recurrence
- Cerebral vasospasm (nimodipine!)
- Other early complications (hyponatremia by hypersecretion of the natriuretic factor; repolarization and heart rhythm disorders; hyperthermia, resuscitation complications, etc.)
- Late complications (chronic adult hydrocephalus and late hemorrhagic relapse)

PREVENTION of REBLEEDING

INTRACRANIAL HEMORRHAGE

Spontaneous Subarachnoid Hemorrhage (Ruptured Saccular Aneurysm)

Laboratory Findings A CT scan will detect blood locally or diffusely in the subarachnoid spaces or within the brain or ventricular system. This should be the initial investigative procedure, since it confirms a subarachnoid hemorrhage in more than 95 percent of cases. The blood may appear as a subtle shadow along the tentorium, difficult to distinguish from the veins in this area, or in the sylvian or adjacent fissures. A large localized collection of subarachnoid blood may indicate the location of the aneurysm and the region of subsequent vasospasm as already noted. When two or more aneurysms are visualized by arteriography, the CT scan may identify the one that had ruptured by the clot that surrounds it. Also, a coexistent hydrocephalus will be demonstrable. If the CT scan documents subarachnoid blood with certainty, a spinal tap is not necessary.



INTRACRANIAL HEMORRHAGE

Spontaneous Subarachnoid Hemorrhage (Ruptured Saccular Aneurysm)

Laboratory Findings In all other cases a lumbar puncture should be undertaken when the clinical features suggest a subarachnoid hemorrhage. Usually the CSF is grossly bloody, with RBC counts up to 1 million per cubic millimeter or even higher. With a relatively mild hemorrhage, there may be only a few thousand cells. It is unlikely that an aneurysm can rupture entirely into brain tissue without some leakage of blood into the subarachnoid fluid, so that the diagnosis of ruptured saccular aneurysm (by lumbar puncture) cannot be confirmed unless blood is present in the CSF. Usually deep xanthochromia is found after centrifugation if several hours have elapsed.

Carotid and vertebral angiography is the only certain means of demonstrating an aneurysm and does so in some 85 percent of patients in whom the correct diagnosis of spontaneous subarachnoid hemorrhage is made on clinical grounds. MRI and MRA detect most aneurysms of the basal vessels but are as yet of insufficient sensitivity to replace conventional angiography.

INTRACRANIAL HEMORRHAGE

Spontaneous Subarachnoid Hemorrhage (Ruptured Saccular Aneurysm)

Treatment This is influenced by the neurologic and general medical state of the patient as well as by the location and morphology of the aneurysm. Ideally, all patients should have the aneurysmal sac surgically obliterated, but the mortality is high if the patient is stuporous or comatose (grade IV or V, see below). Before deciding on a course of action, it has been useful to assess the patient with reference to the widely employed scale introduced by Botterell and by Hunt and Hess, as follows:

Grade I. Asymptomatic or with slight headache and stiff neck

Grade II. Moderate to severe headache and nuchal rigidity but no focal or lateralizing neurologic signs

Grade III. Drowsiness, confusion, and mild focal deficit

Grade IV. Persistent stupor or semicoma, early decerebrate rigidity and vegetative disturbances

Grade V. Deep coma and decerebrate rigidity.

INTRACRANIAL HEMORRHAGE

Spontaneous Subarachnoid Hemorrhage (Ruptured Saccular Aneurysm)

Treatment The general medical management in the acute stage includes the following, in all or part: bed rest, fluid administration to maintain above-normal circulating volume and venous pressure, use of elastic stockings and stool softeners; administration of propranolol, intravenous nitroprusside, or other medication - to reduce greatly elevated blood pressure and then maintain systolic blood pressure at 150 mmHg or less; and pain-relieving medication for headache (this alone will often reduce the hypertension).

The prevention of systemic venous thrombosis is critical, usually through the use of cyclically inflated whole leg compression boots.

The use of anticonvulsants is controversial; many neurosurgeons administer them early, with a view of preventing a seizure-induced risk of rebleeding. We have generally avoided them unless a seizure has occurred.

INTRACRANIAL HEMORRHAGE

Spontaneous Subarachnoid Hemorrhage (Ruptured Saccular Aneurysm)

Treatment Calcium channel blockers are being used extensively to reduce the incidence of **stroke** from vasospasm. Nimodipine, 60 mg administered orally every 4 h, is currently favored. Although calcium channel blockers do not alter the incidence of angiographically demonstrated vasospasm, they have reduced the number of strokes in each of five randomized studies.

Patients with stupor or coma who have massive hydrocephalus often benefit from decompression of the ventricular system. This is accomplished initially by external drainage and may require permanent shunting if the hydrocephalus returns. The risk of infection of the external shunt tubing is high if it is left in place for much more than 3 days.

The timing of surgery for grade III patients is still controversial, but if their general medical condition allows, they probably also benefit from the same aggressive approach. In grade IV patients, the outcome is generally dismal, no matter what course is taken, but we have usually avoided early operation. The insertion of ventricular drains into both frontal horns has occasionally raised a patient with severe hydrocephalus to a better grade and prompted early operation. In the hands of experienced anesthesiologists and cerebrovascular surgeons using microdissection, the operative mortality, even in grades III and IV patients, has now been reduced to 2 to 3 percent.

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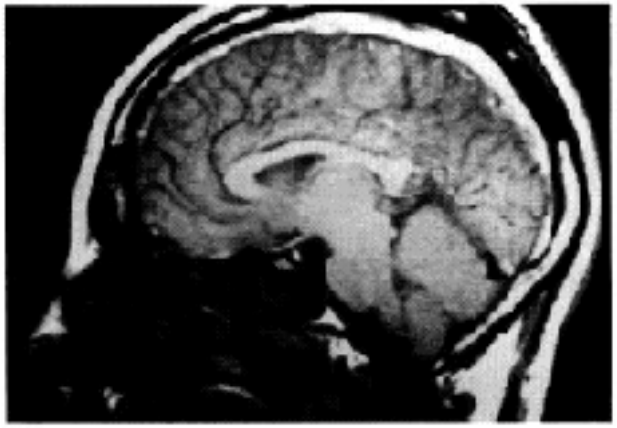
Thrombosis of Cerebral Veins and Venous Sinuses

Thrombosis of the venous sinuses, particularly of the superior sagittal or lateral sinus, usually develops in relation to infections of the ear and paranasal sinuses or to one of the hypercoagulable states.

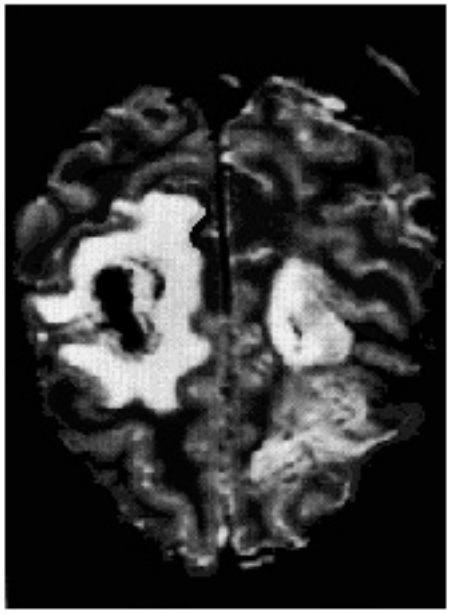
Occlusion of cortical veins that are tributaries of the dural sinuses takes the form of an venous infarctive **stroke**.
Diagnosis is difficult except in certain clinical settings known to favor the occurrence of venous thrombosis, such as the taking of birth control pills or postpartum and postoperative states.
Anticoagulant therapy beginning with heparin for several days, followed by warfarin, combined with antibiotics if the venous occlusion is inflammatory, has been lifesaving in some cases but the overall mortality rate remains high at 10 to 20 percent, in part due to large hemorrhagic venous infarctions.
The trial by Einhaupl and colleagues, however, has settled the question in favor of aggressive anticoagulation.

Thrombosis of Cerebral Veins and Venous Sinuses

(A) Sagittal T1 WI shows hyperintense signal in thrombosed superior longitudinal sinus. (B) Axial T2 WI shows frontal bilateral venous infarcts with hypointense central area and peripheral hyperintensity associated with perilesional edema. (C) Axial postgadolinium T1 WI shows hyperintensity in the superior longitudinal sinus and cortical veins. Hemorrhagic infarct with perilesional edema in the left frontal lobe is seen.



A



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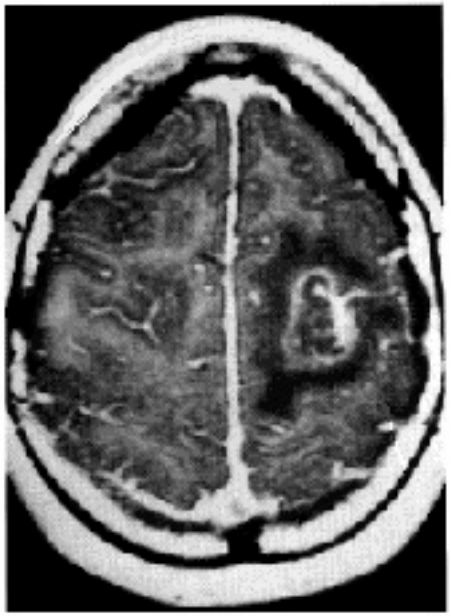


C

Thrombophlebitis of the superior longitudinal sinus and bilateral hemorrhagic infarct. 21-year-old woman with headache, bilateral hemiparesis, and stupor.

DIFFERENTIATION OF VASCULAR DISEASE FROM OTHER NEUROLOGIC ILLNESSES

The diagnosis of a vascular lesion rests essentially on recognition of the **stroke** syndrome; without evidence of this the diagnosis must always be in doubt. The three criteria by which the **stroke** is identified should be reemphasized: (1) the temporal profile of the clinical syndrome, (2) evidence of focal brain disease, and (3) the clinical setting. Definition of the temporal profile requires a clear history of the premonitory phenomena, the mode of onset, and the evolution of the neurologic disturbance in relation to the patient's medical status. If these data are lacking, the **stroke** profile may still be determined by extending the period of observation for a few days or weeks, thus invoking the clinical rule that the physician's best diagnostic tool is a second and third examination. An inadequate history is the most frequent cause of diagnostic error.

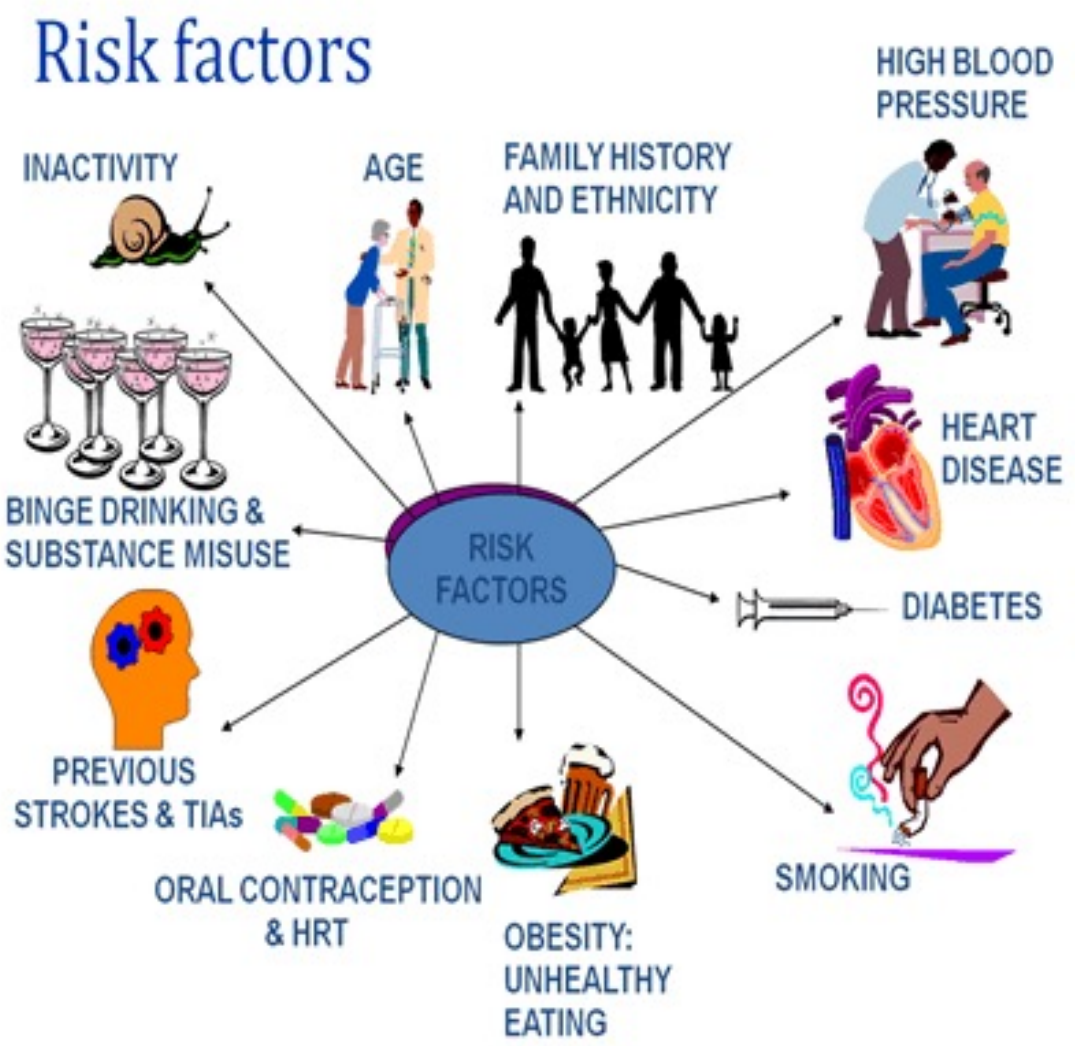
RISK FACTORS FOR STROKE

RISK FACTOR	RELATIVE RISK	RELATIVE RISK REDUCTION WITH TREATMENT	NUMBER NEEDED TO TREAT[α]	
			PRYMARY PREVENTION	SECONDARY PREVENTION
Hypertension	2 – 5	38%	100-300	50-100
Atrial fibrillation	1.8-2.9	68% warfarin, 21% aspirin	20-83	13
Diabetes	1.8-6	No proven effect		
Smoking	1.8	50% at 1 year, baseline risk at 5 years post cessation		
Hyperlipidemia	1.8-2.6	16-30%	560	230
Asymptomatic carotid stenosis	2.0	53%	85	N/A
Symptomatic carotid stenosis (70-99%)		65% at 2 years	N/A	12
Symptomatic carotid stenosis (50-69%)		29% at 5 years	N/A	77

[α] Number needed to treat to prevent one stroke annually. Prevention of other cardiovascular outcomes is not considered here.

Note: N/A, not applicable.

AVC: factori de risc



THE END

QUESTIONS ???