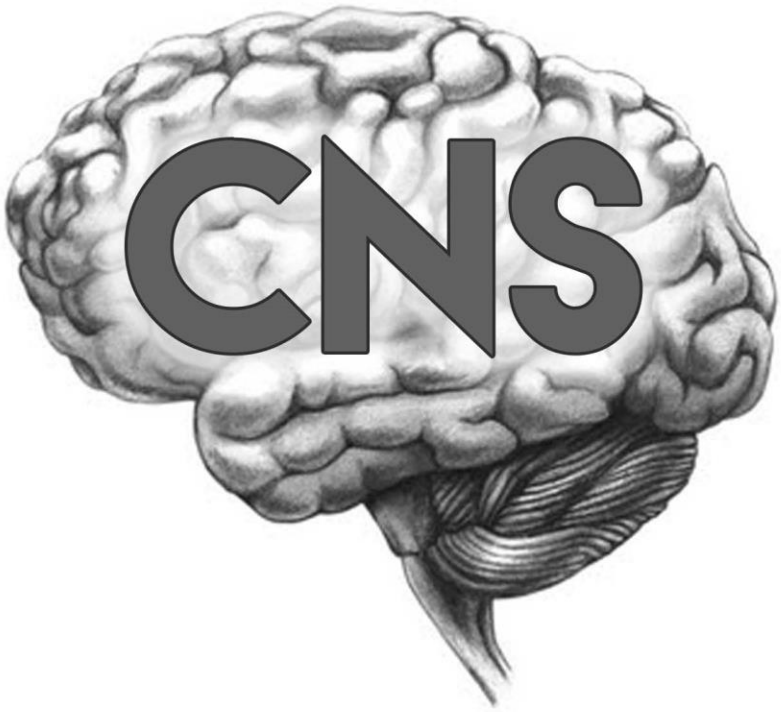

ANATOMY

☑ Sheet ○ Slide ○ Handout

Number

20

Subject

Callosal Syndrome, Olfaction and Blood supply

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Price:

The sheet was written according to section 3 recording.

In the previous lecture we talked about aphasias, and now we continue.

➔ **How do we examine a patient with aphasia?**

1- We listen to the speech output (how he speaks), is it fluent or nonfluent?

If fluent, the lesion is in the ***posterior part of the brain (Wernicke's aphasia)***, and usually it's not accompanied by paralysis; because the posterior area is a sensory area.

If nonfluent, the lesion is in the ***anterior part of the brain (Broca's aphasia or motor aphasia)***, commonly associated with right hemiplegia or monoplegia.

[Remember: whenever you see a patient with right hemiplegia or right monoplegia you should consider the presence or absence of aphasia].

2- Can the patient read or write without errors?

If he can read and write without errors, aphasia is not present.

3- Is there hemiparesis? If yes, the lesion is anterior and the aphasia is motor (nonfluent).

4- If he has fluent aphasia, we check whether he can repeat, comprehend and name. Remember that in **Wernicke's aphasia**, the patient **can't** comprehend, **can't** repeat and the naming is poor. In **conduction aphasia** (where the lesion is in the arcuate fasciculus, the connection between Wernicke's area and Broca's area) the patient **can** comprehend, but **can't** repeat and the naming is poor. In **anomic aphasia**, the patient **can** repeat and comprehend, but the naming is very difficult.

Also we started talking about ***Alexia (Dyslexia) and its subtypes***:

1- **Pure alexia without agraphia**: where there's a double lesion; one in the left primary visual area and another lesion in the splenium of corpus callosum. It's also called "***Pure word blindness***" –the patient can read but can't understand what's read, and he can write but can't understand what he has just written-.

➔ ***Pure word deafness*** (**auditory agnosia**): it's a completely different case where the patient can hear normally but can't understand what's heard. The patient has poor comprehension of spoken language and poor repetition, yet he **can normally** comprehend written language, name, write and do spontaneous speech. Results from either a lesion in the auditory association area, or due to disconnection between the auditory association area and Wernicke's area.

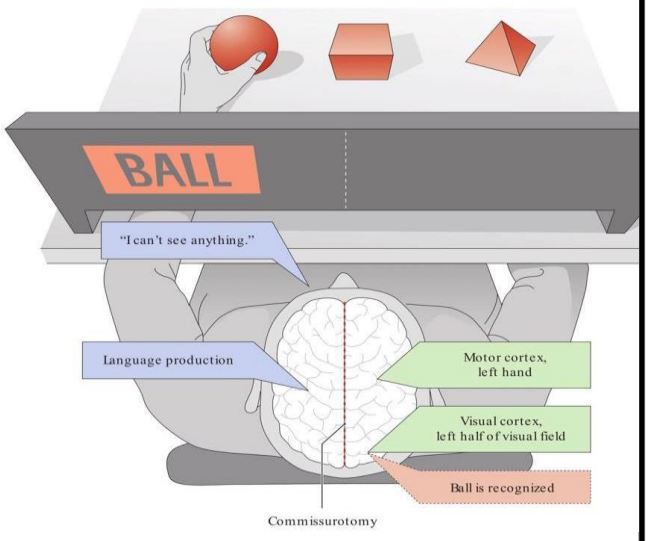
2- **Alexia with agraphia *(Parietal alexia)***: the patient has a defect in reading comprehension and writing.

Callosal syndrome: The two cerebral hemispheres are connected by the corpus callosum (Rostrum, Genu, Body and Splenium). The blood supply for most of the corpus callosum is by the ***anterior cerebral artery***, except for a **small part of the splenium** which is supplied by the ***posterior cerebral artery***.

In the past, they used to surgically cut the corpus callosum to relief the symptoms of Grand Mal epilepsy *(where there is an ectopic focus in one of the hemispheres firing action potentials that travel to the other hemisphere via the corpus callosum, and this causes convulsions in both sides of the body, biting of the tongue and cyanosis due to convulsions of diaphragmatic muscles)*. Nowadays, this surgical intervention is no longer done. But if a vascular lesion occurred and resulted in ischemia of the callosal fibers, the result will be callosal syndrome.

We said that **both hemispheres comprehend, but only the left hemisphere talks.

In this syndrome, if we put a key –for example- in the patient's left hand, stereognosis is transported via the dorsal column system and interpreted –in this case- in the sensory cortex of the right hemisphere. Then if we ask the patient to name the object he's holding, he won't be able to do so; why? Because in order for the patient to talk and name the object, impulses have to **travel from the right hemisphere** (where the interpretation of the sensory signal occurred) **to the left hemisphere** (to do the talking), and the corpus callosum is interrupted due to ischemia. This is known as "**Left tactile anomia**", inability to name an object held in the left hand. [If we place the key in the right hand, the sensory signal will be interpreted in the left hemisphere, which does the talking, and so the patient can easily name what he's holding]



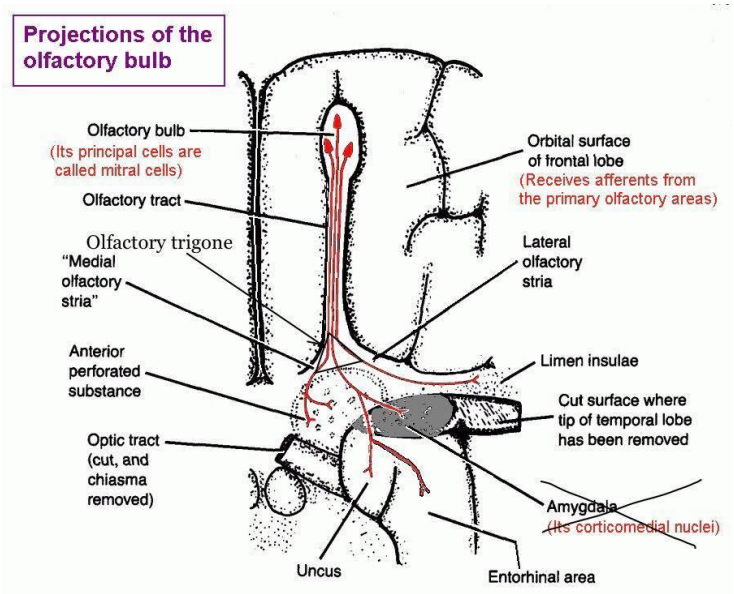
Now, if we asked the patient to read a written material in the left half of his visual field *(this is done by a Perimeter, an instrument found in hospitals that can view words and pictures in the left half of the visual field only)*, the visual signal will be interpreted in his right visual cortex. In order for the patient to understand what he reads, the signal must travel from the **right visual association cortex to the left angular gyrus (الفهّامة)**. In the case of callosal syndrome, the corpus callosum is interrupted, so the patient can't understand what he reads in his left visual field. This is known as "**Hemialexia**". [*Very Important*: Alexia is due to a lesion in the left angular gyrus, Hemialexia is due to an interruption of the corpus callosum]

The olfactory pathway:

The olfactory pathway starts from receptors in the olfactory mucosa, they represent the 1st order neurons that can generate an AP. These receptors are found in the olfactory epithelium in the upper part of roof of the nose. They have peripheral processes (cilia-like structures, that resemble the dendrites) and central processes that form the olfactory nerve and pierce the cribriform plate of ethmoid in the base of the skull.

A characteristic of the olfactory mucosa is that besides the 1st order neurons, it also contains **basal cells**, which are undifferentiated cells that can differentiate into neurons for regeneration. So the only place where neurons can regenerate is the olfactory mucosa.

Axons of 1st order neurons (that formed the olfactory nerve) then synapse with 2nd order neurons in the olfactory bulb. They are called **Mitral cells.** Their axons leave the olfactory bulb and form the olfactory tract, which will synapse with 3rd order neurons. If we look behind the olfactory tract (aka **olfactory trigone**), we find the **anterior perforated substance** which is perforated by the central branches of the anterior cerebral and the medial cerebral arteries.



The olfactory tract divides into: **medial olfactory stria** and **lateral olfactory stria**. The medial olfactory stria has no significance and we don't know exactly where it goes.

The lateral olfactory stria goes to 3 distinct areas which are collectively called "**the Pyriform cortex/Lobe**".

- The first area is the Uncus: an important area in the base of the brain close to the tentorial notch. If the intracranial pressure increases, it might result in **uncal herniation**.
- The second area is the Entorhinal area: the ventral part of the parahippocampal gyrus.

***Thought to be involved in emotions only, but now they say it has to do with olfaction.*

- The third area is the Limen Insulae.

These three areas are related to olfaction and are referred to as the pyriform cortex.

➔ **How do the receptors of the 1st order neurons in the olfactory mucosa react to odorant molecules?**

Any odorant molecule will activate the receptors → then activation of G-proteins → activation of adenylate cyclase → increased cAMP → formation of a receptor potential → depolarization of the central processes that form the olfactory nerve → if it reaches the threshold and action potential is formed → the AP travels through the olfactory nerve to the olfactory bulb then to the olfactory tract.

The above described pathway indicates that the olfactory signal doesn't reach the thalamus, it goes directly to the cortex, that's what all old books and some new books state. However, some American books state that part of the olfactory tract goes to the **dorsomedial nucleus of the thalamus**.

Applied anatomy: the olfactory bulb and tract are present above the cribriform plate of ethmoid. If the base of the skull and the cribriform plate are fractured, the olfactory bulb or the olfactory tract may get injured. The result is **Anosmia** (loss of the sense of smell). Patients with anosmia also suffer from **CSF rhinorrhea**; because the dura mater is attached to the base of the skull, if it fractures, the dura mater and the arachnoid mater are ruptured, the subarachnoid space is open and the CSF leaks. This is very dangerous because of the possibility to get an ascending infection through the nose to the subarachnoid space.

[CSF rhinorrhea → CSF leakage from the nose, due to fracture in the anterior cranial fossa, specifically the cribriform plate.

CSF otorrhea → CSF leakage through the ear, due to fracture in the middle cranial fossa, involving the petrous temporal bone.

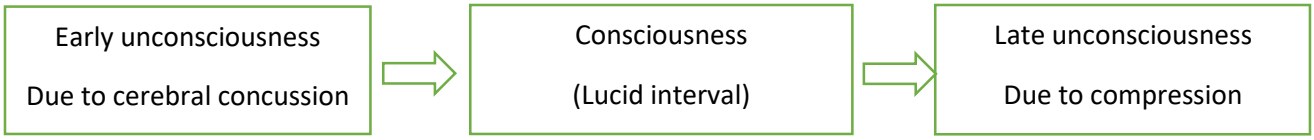
Now we go back the last part of handout 4 concerned with the blood supply:

➔ **Types of hemorrhage:**

1- Subarachnoid hemorrhage: bleeding is in the subarachnoid space, between the pia and arachnoid, which contains blood vessels and CSF.

It commonly results from a rupture of a congenital berry aneurysm. Since it's congenital, why doesn't it rupture early in life? Why does the hemorrhage manifest at the age of 50 or 60? Because one of the most important factors to cause the rupture is **severe hypertension**. Hypertension causes the aneurysm to rupture and causes subarachnoid hemorrhage where the blood is mixed with CSF, noticed when we perform a **lumbar puncture**.

2- Extradural (Epidural) hemorrhage: less common than the subdural hemorrhage, results from a trauma to the head that leads to fracture of the skull, especially the pterion part, and rupture of the **middle meningeal artery or vein**. Here the bleeding is outside the dura mater. **Hematoma formation takes long time** (hours – days) because the dura has to separate from the skull. Characterized by the presence of the so called "**Lucid interval**". Lucid interval means a phase of consciousness (ranges from hours to days) between an earlier unconsciousness that's due to the cerebral concussion (physiological paralysis of brain functions) and a later unconsciousness that's due to compression of the hematoma on the brain.



The middle meningeal artery lies opposite to the representative area of the face on area 4, so hematoma is first formed in this area, and the patient complains of paresis of facial muscles. As the hematoma enlarges and reaches higher areas, paresis spreads to the upper limb then to the lower limb ***(paresis or paralysis follows a sequence)***.

If we perform a lumbar puncture, no blood is found in the CSF.

3- Subdural hemorrhage: more common than the epidural hemorrhage, results from a rupture of the **superior cerebral <u>veins</u>** (which open in the superior sagittal sinus –figure 1) due to trauma or concussion. Here the **hematoma is formed easily** between the dura

and the arachnoid (a very big hematoma is formed within a very short time). No lucid interval. No sequence for paralysis (paralysis is haphazard).

When we perform a lumbar puncture blood can be found in CSF in case the trauma was intense and caused a rupture of the arachnoid mater, and blood passed from the subdural space to the subarachnoid space.

[Students were asked about this figure in last year's exam. The dr. pointed at the superior cerebral veins and asked whether rupture of these blood vessels causes epidural hemorrhage or causes lucid interval, which is false of course].

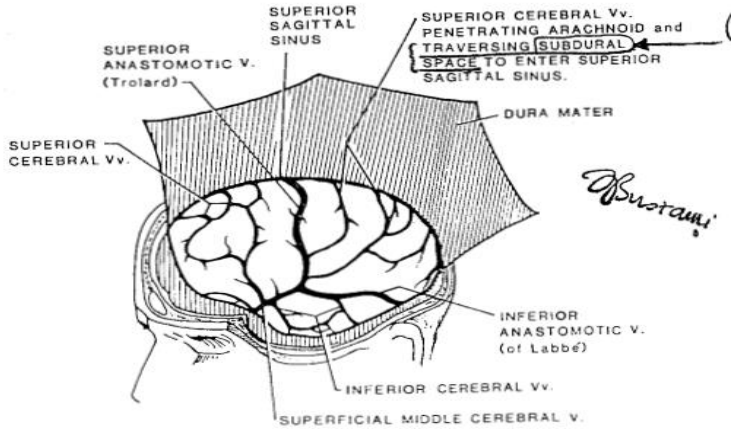


Figure 14A. Lateral View of the Superficial Cerebral Veins on the Surface of the Left Cerebral Hemisphere

Figure 1

The circulus arteriosus of Willis:

This is present in the base of the brain within the subarachnoid space. Any rupture of any artery in this circle leads to subarachnoid hemorrhage (diagnosed by a lumbar puncture).

➔ What forms the circulus arteriosus of Willis? –Please follow figure 2 as you go through the text-.

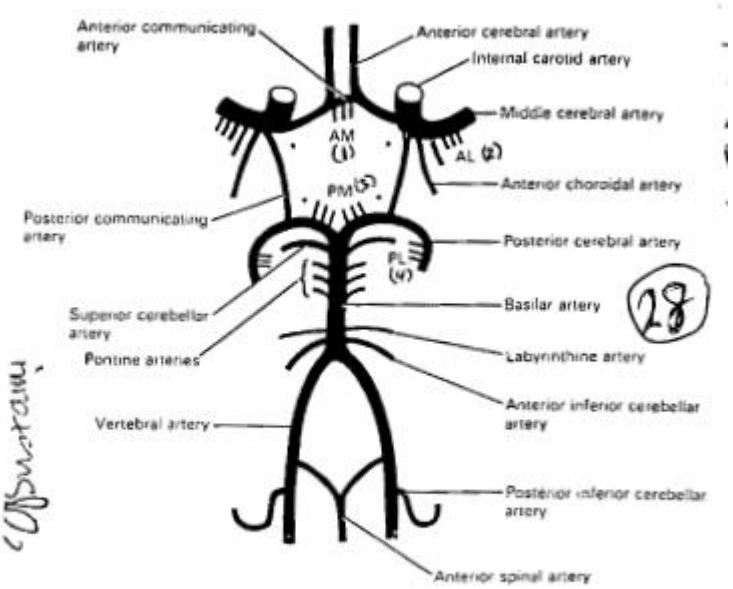


Fig. 14.1 Diagram of arteries at the base of the brain showing the circulus arteriosus*. The groups of central branches are anteromedial (AM), anterolateral (AL), posteromedial (PM) and posterolateral (PL).

Figure 2

1- **The internal carotid artery**: a branch of the common carotid artery. It gives no branches in the neck, it enters in the petrous temporal bone through the carotid canal, then it ascends and enters the cavernous sinus (accompanied here by the abducent nerve). Leaves the cavernous sinus in the base of the skull and divides into **middle cerebral artery** and **anterior cerebral artery**. And as we said any embolus that gets into the internal carotid it enters the middle cerebral artery more easily *(because its bigger and in-line with the internal carotid artery)*.

Another branch of the internal carotid is the **anterior choroidal artery**, which supplies an important part of the posterior limb of the internal capsule (where the motor pathways pass). Also it supplies the retrolentiform part of the internal capsule (where the optic radiation passes). Thus, if the anterior choroidal artery is occluded the result is hemiparesis and contralateral homonymous hemianopia.

[So a patient with a stroke in the internal capsule might end up with: contralateral hemiplegia -damage to pyramidal and extrapyramidal tracts-, contralateral hemiface -damage to corticobulbar tract0, **temporary** contralateral hemianesthesia –after a while, the patient restores the sensation of pain, temperature and simple touch **crudely**-, hemianopia –if the lesion is extensive enough to reach the retrolentiform part-, and impaired hearing –if it reaches the sublentiform part-].

2- **The vertebro-basilar system**: two vertebral arteries (which branch from the 1st part of the subclavian) enter through the foramen magnum, ascend in front of the medulla and at the lower border of pons they unite to form the basilar artery. The basilar artery gives the superior cerebellar arteries then divides into two posterior cerebral arteries.

[**Clinical correlate**: between the posterior cerebral artery and the superior cerebellar artery on one side, the oculomotor nerve emerges. If an aneurysm occurred in one of these arteries, it might compress the oculomotor and lead to the symptoms of oculomotor lesions].

3- The two anterior cerebral arteries are linked through the **anterior communicating artery**. The posterior cerebral artery and the internal carotid are linked on each side by the **posterior communicating artery**. This forms a complete circle (circulus arteriosus of Willis –figure 2-).

Each cerebral artery gives **central branches** and **cortical branches**. Most of the central branches enter to the brain through the anterior perforated substance (behind the olfactory tract).

1- The central branches are the **lateral striate** (from the middle cerebral artery) and the **medial striate** (from the anterior cerebral artery). They go to the basal ganglia (hence the name) and on their way they supply the internal capsule.

Blood supply of the internal capsule:

Anterior limb: **mainly from the middle cerebral** (the lateral striate branches). Also from the **anterior cerebral** through the medial striate or the recurrent branch (artery of Heubner).

Genu: mainly from the **middle cerebral** (the lateral striate branches). Also from direct branches from the **internal carotid.**

Posterior limb: mainly from the **middle cerebral** (the lateral striate branches). Also from the **anterior choroidal.**

Retrolentiform part: is supplied entirely by the **anterior choroidal** .

2- The cortical branches:

If we look at the lateral aspect of the motor area and the sensory area, **most of it is supplied by the middle cerebral** (most of it means the representative area of the face and the upper limb). The middle cerebral also supplies the **primary auditory cortex** (deep in the lateral fissure). The representative area of the lower limb is supplied by the anterior cerebral. The occipital and the lower temporal gyri are supplied by the posterior cerebral (figure 3).

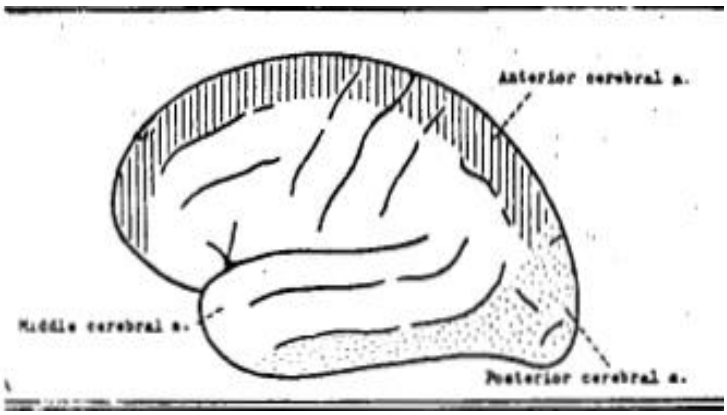


Figure 3

So the visual cortex is supplied exclusively by the posterior cerebral, **except** the macular area. The macula and fovea centralis are represented most posterior, and they are responsible for central vision. The macular area receives blood supply from **both** the posterior cerebral and the middle cerebral. If the posterior cerebral is occluded, the patient presents with hemianopia (lost vision in homonymous halves of his visual fields), yet he can still see in the central area of his visual field because the macula gets additional blood supply from the middle cerebral, so it remains safe. This is known as "**Macular sparing**".

The inferior surface (the orbital surface of the frontal, figure 4):

Medially it's supplied by the anterior cerebral.

Laterally it's supplied by the middle cerebral (including the temporal pole).

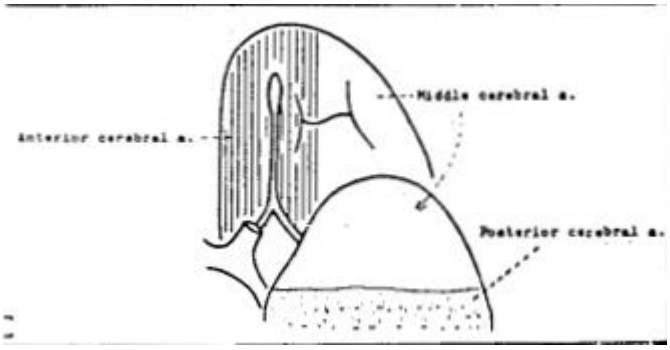


Figure 4

The paracentral lobule (Figure 5): the medial extension of area 4 and area 3,1,2. Responsible for motor and sensory control of the lower limbs and control over sphincters. It's supplied by the anterior cerebral artery mainly. Commonly injured due to meningioma of falx cerebri, the patient suffers from paraplegia and loss of continence.

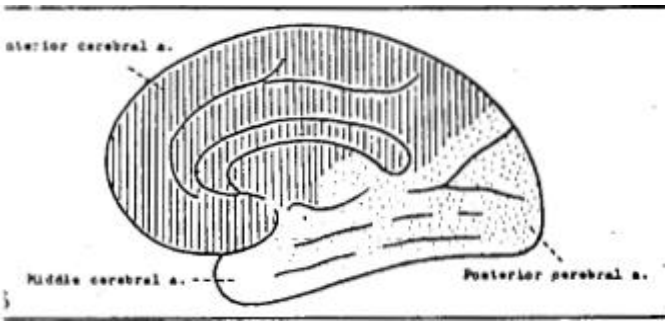


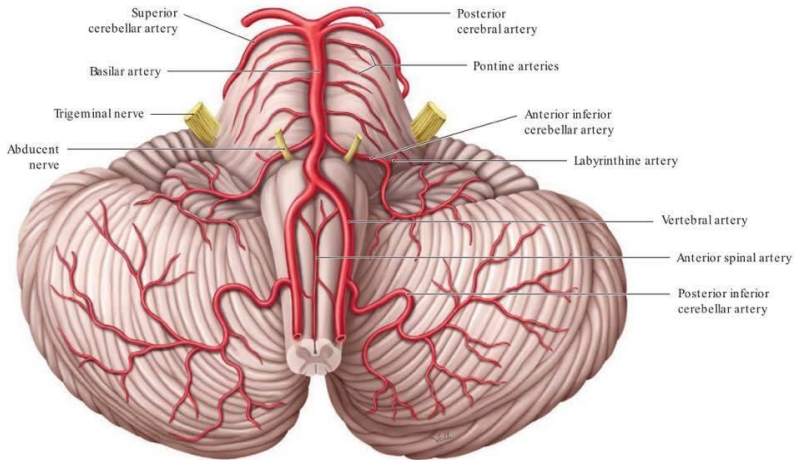
Figure 5

So in case of injury to the anterior cerebral artery, we don't only consider the blood supply to the corpus callosum and the callosal syndrome, but also we consider the paracentral lobule and paraplegia.

Blood supply to other organs:

- ➔ Blood supply of the spinal cord: After the vertebral artery enters through the foramen magnum, it gives two anterior spinal arteries that unite and form one anterior spinal artery, that supplies **most** of the spinal cord (the ventral horn, the pyramidal and extrapyramidal and the spinothalamic. It does NOT supply the dorsal column). The dorsal column is supplied by the posterior spinal artery.

- ➔ Blood supply of the cerebellum: The vertebral artery gives a very important branch called the **posterior inferior cerebellar artery (PICA)**. It ascends to supply the cerebellum, on its way it supplies the upper lateral part of the medulla. If an occlusion occurred in PICA or in the vertebral artery, the result is **<u>PICA syndrome</u>**.



The symptoms are:
-**Loss of pain, temperature and touch sensations from the ipsilateral side of the face and the contralateral side of the body** [Remember that the spinal trigeminal tract is not crossed in the medulla, but the spinothalamic tract that brings sensations from the body is crossed in the medulla].
-**Vertigo and vomiting** due to involvement of the vestibular nuclei.
-**Ataxia and tendency to fall toward the side of the lesion** due to involvement of the inferior cerebellar peduncle.
-**Nervous type of dysphagia** due to paralysis of pharyngeal muscles supplied by the nucleus ambiguous.
-**Horner's syndrome** due to involvement of the descending sympathetic.
***These symptoms can result from blocking PICA or the vertebral arteries.*

Other branches to the cerebellum (other than PICA):

-**Anterior inferior cerebellar artery** (AICA): branch from the basilar.
-**Superior cerebellar artery**: branch from the basilar.

So the cerebellum receives two branches from the basilar and one branch from the vertebral.

- ➔ Labyrinthine artery: branch from the basilar, exits through the internal acoustic meatus to the labyrinth (inner ear).
- ➔ The basilar artery also gives the paramedian and the circumferential arteries to supply the pons.
- ➔ Small branches from the posterior cerebral go to the midbrain.
- ➔ Blood supply of the thalamus: thalamoperforating arteries (to the anterior part of the thalamus), thalamogeniculate arteries (to the posterior part of the thalamus) and small branches from the posterior cerebral.
- ➔ **Thalamic syndrome**: abnormal sensation due to occlusion of one of the thalamic arteries. The patient feels light touch as if it was severe pain.

Overthinking will destroy your mood. Breathe and let go.

"وآخِرُ دَعوَاهُم أنِ الحمدُ للهِ رَبِّ العَالَمِين"