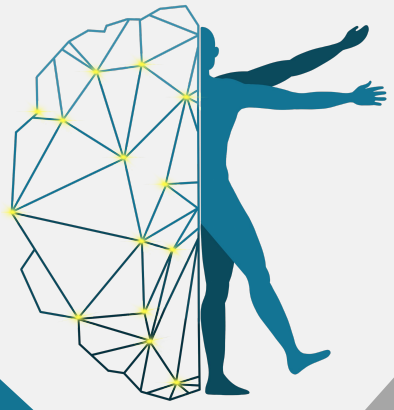
Physiology Team 439

MED439
KING SAUD UNIVERSITY



Prof.Meo's Final revision

Abdulaziz Alsuhaim

DISCLAIMER!

This file is completely a personal effort, it is not by any mean a primary source to study and prepare for the exam, study your slides or the team work first then check this file, some points I want to clarify:

- I suggest opening this file hand by hand while reading the lectures and focusing on the important things mentioned by doctor to have a full picture and imagination.

- Any slide number mentioned in this file it would be according to Prof.Meo's slides

Good luck

Cerebellum

R

- Slide 5 **VERY IMPORTANT** "please remember this slide" (Rule of 3)

- Slide 8 **IMPORTANT** "please remember these names"

- Slide 9 "just know the peduncles and where the got their inputs from"

- Slides 10-12 he went through them quickly "just read them".

- Slides 13-14 are **VERY IMPORTANT** Slides.
- He skipped slide 15.
- He just read slides 16-17.
- Slide 18 cerebellum lobes functions are **IMPORTANT**
- He said about slides 18-20 "Please keep these slides in your mind, **VERY IMPORTANT** slides"

- He just went through slide 21 quickly.
- Slide 22 "Clinical features & tests related to cerebellum are **IMPORTANT**"

- Dysdiadochokinesia & tremors are **VERY IMPORTANT**

- Slide 23 (Finger nose test), he just went through it quickly.

- Slides 24-26 were skipped. "I just explain these things"

Cerebellum

R

- Slide 27 “If motor and sensory systems are intact, an abnormal asymmetric heel to shin test is highly suggestive of an ipsilateral cerebellar lesion”
- slide 28 He just read the graph very quickly.

Autoregulation of Cerebral blood flow

R

- Slide 3 was skipped
- Slides 4-5 he just read them
- Slide 6 is **VERY IMPORTANT** slide
- Slide 7 “know the red highlighted factors”
- Slide 8 he just read it
- Slide 9 is **VERY IMPORTANT** slide
- Slide 10 is **VERY IMPORTANT** (know the values)
- Slide 11“Make sure to understand it”
- Slide 12 “know the myogenic theory”
- Slide 13 is **VERY IMPORTANT** (know the range of regulation 60-140)
- Slide 14 he read it quickly
- Slide 15 is **VERY IMPORTANT** (CO_2 is very IMPORTANT)
- Slides 16-20 he just read them
- Slide 21 “please know the oxygen limits”
- Slide 22 he just read it
- Slide 23 he just read it and said “ANS and Neurochemical control has minor role”
- Slides 24-25 he just read them
- Slides 26-29 were skipped
- Girls Dr : **the Values are important**

Taste and smell

R

- Slides 2-4 he just read them.
- Slide 5 “Saliva is very important in taste process”
- Slide 6 he just read it quickly
- Slide 7 “Please remember this slide”
- Slide 8 is **VERY IMPORTANT** (know the receptors)
- Slides 9-13 are **VERY IMPORTANT** slides
- Slide 14 “same previous slides”
- Slide 15 he read it and explained each pathophysiology briefly.
- Slides 17-18 were skipped
- Slide 19 he just read it
- Slides 20-23 were skipped
- Slide 24 is **VERY IMPORTANT**
- Slide 25 “same mechanism mentioned on slide number 24”
- Slides 26-28 were skipped
- Slide 29 he read it quickly without saying any hints, but I think it is important.
- Slide 30 he just read each pathophysiology quickly without explanation.

Speech

R

- Slide 3 (know the type of communications)
- Slide 4 he went through it quickly
- Slide 5 he just read the definitions
- Slide 6-13 he just read them without any hints
- Slide 14 "**please remember this slide**"
- Slide 15-21 he just read them
- Slide 22 is **VERY IMPORTANT** slide
- Nuclei X and XII receive corticobulbar pathway from both ipsilateral and contralateral hemispheres (**Bilateral innervation**).
- Slide 23-24 he just read them and said please remember slide number 24.
- Slide 25 is **VERY IMPORTANT** slide
- Slides 26-28 were skipped
- Slide 29 he just read it "Stuttering is more common in children"
- Slide 30 he just read it
- Slide 31 was skipped
- "**please remember slides 22, and 25 they are very IMPORTANT slides please do not miss them**"

- Girls Dr: **Function + Lesion are important**
- don't worry about the difference between girls and boys slides ذاكروا سلايداتي

Cerebellum

★ functions are IMPORTANT

R

Cerebellar Divisions

- The anterior & posterior lobes on each side constitute **2 large cerebellar hemispheres**, which are separated by a narrow band called the **vermis.***
- **Guyton:** The flocculonodular lobe is the oldest of all portions of the cerebellum; it developed along with (and functions with) the vestibular system in controlling body equilibrium.

male slide only

Anatomically	Physiological	Functional
Anterior lobe	Paleocerebellum	Spinocerebellum
Posterior lobe	Neocerebellum	Cerebrocerebellum
Flocculonodular lobe	Archicerebellum	Vestibulocerebellum

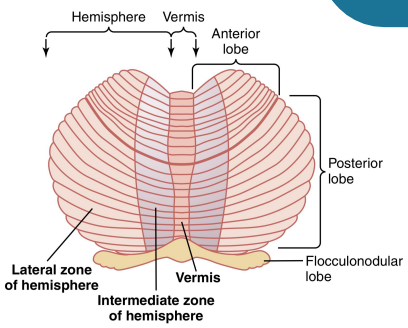
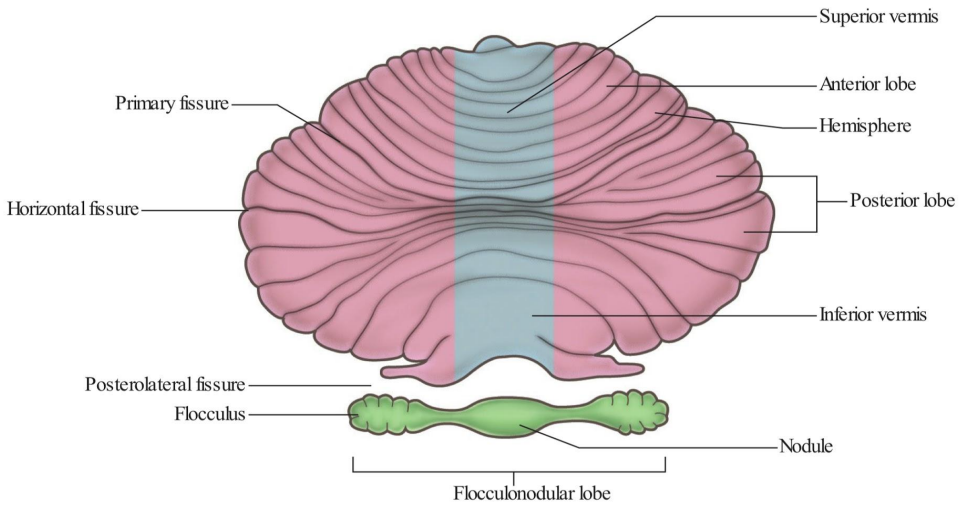


Figure 56-2 Functional parts of the cerebellum as seen from the posteroinferior view, with the inferiormost portion of the cerebellum rolled outward to flatten the surface.



Functional Divisions: Extra : Explanation for images in slides

- **Spinocerebellum**: Regulation of **muscle tone, coordination of skilled voluntary movement**. Receives proprioceptive input and copy of the motor plan from the motor cortex and sends impulse to medial and lateral descending system for motor execution. **Guyton:** This consists of most of the vermis of the posterior and anterior cerebellum plus the adjacent intermediate zones on both sides of the vermis.

- **Cerebrocerebellum**, **planning and initiation of voluntary activity.** Interacts with motor and premotor cortices. **Guyton:** This consists of the large lateral zones of the cerebellar hemispheres, lateral to the intermediate zones.

- **Vestibulocerebellum**: **Maintenance of balance, control of eye movement.** Connects with vestibular nuclei. **Guyton:** This consists principally of the small flocculonodular cerebellar lobes that lie under the posterior cerebellum and adjacent portions of the vermis.

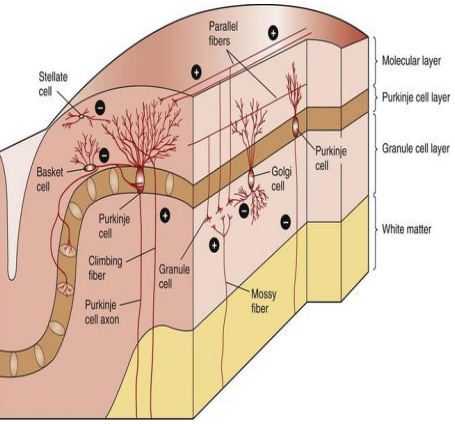
VERY IMPORTANT SLIDE

Structure and Connections of the cerebellum

R

Cells	Fibers	Nuclei
Cortex nuclei 1. Purkinje cell. 2. Granule cell. 3. Basket cell. 4. Golgi cell. 5. Stellate cell.	1. Climbing fiber. 2. Mossy fiber. 3. Parallel fiber.	1. Inferior olivary nucleus. 2. Deep cerebellar nuclei.

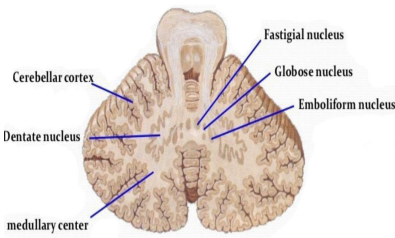
- The cortex is deeply infolded, giving a large surface area, and it contains five different cell types.*
- The cerebellum has an external cerebellar cortex (gray matter) separated by inner white matter =medulla from the deep cerebellar nuclei as follows:



cerebellar cortex		
Layer*	**Cells**	**Action**
Molecular Layer	Stellate cells (inhibitory Interneuron)	Release Taurine*, GABA (**Inhibition**)
	Basket cells (inhibitory Interneuron)	Release GABA (**Inhibition**)
Purkinje Cell Layer	Purkinje cells (**output cells**) inhibit the deep nuclear cells(DNCs). (its axons take feedback from cortex to deep nuclei, then deep nuclei give the efferents)	
Granular Layer	Golgi cells (inhibitory Interneuron)	
	Granular cells has $GABA_A$ receptors	Release Glutamate (**Excitation**)

Cerebellar nuclei (Deep nuclei in white matter):

- **D**entate Nucleus in cerebrocerebellum
- Interpositus nucleus in spinocerebellum
  - **E**mboliform Nucleus
  - **G**lobose Nucleus
- **F**astigial Nucleus in the spinocerebellum and vestibulocerebellum



From lateral to medial
"**D**on't **E**at **G**reasy **F**ood "

- All afferent fibers (have specific target cell) relay first at the deep nuclei and the cerebellar cortex, then the latter discharges to the deep nuclei, from which the efferent fibers originate and leave the CB.*

Cerebellum

R

- The CB **receives both sensory** and **motor information** through a rich afferent nerve supply.
- This arises from : 1-Other areas of the brain 2-Peripheral receptors. and **enters the CB via the 3 cerebellar peduncles.**

IMPORTANT Types of Afferent Fibers

Climbing fibers	Mossy fibers
From the Inferior olivary nucleus	From all other afferents that enter the cerebellum + some fibers coming from the inferior olivary nucleus (So they are greater than climbing fibers)
It learns the cerebellum to **perform new patterns of movements precisely** movement skills لتعلم اي	**Help the precise execution** (التنفيذ الدقيق) **of the voluntary movements** (concerning their initiation, duration and termination), which occurs by controlling the turn on and turn off output signals from the cerebellum to the muscles.
They synapses with only one Purkinje cell.	They synapses with Granule cells, which in turn affect many Purkinje cells.
They both give of afferent fibers to to deep cerebellar nuclei, which are excitatory.	

VERY IMPORTANT The Rule of 3*

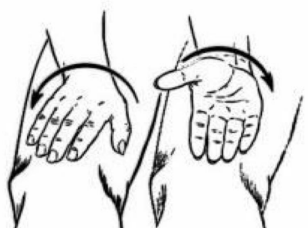
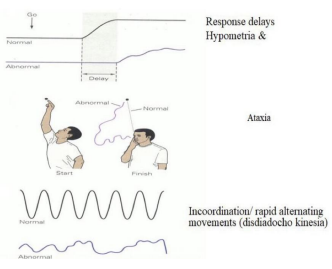
3 lobes:	Floculonodular Lobe	Anterior lobe	Posterior lobe
3 Cortical Layers:	Molecular layer	Purkinje cell layer	Granular layer
3 purkinje's cells afferent paths:	Mossy fibers	Climbing fibers	Aminergic fibers
3 pairs of deep nuclei:	Fastigial	Interposed (globose & emboliform)	Dentate
3 pairs of peduncles:	Superior (pri.output)	Middle (pri.Input)	Inferior (pri.Input)
3 functional division:	Vestibulocerebellum	Spinocerebellum	Cerebrocerebellum

Cerebellum

Abnormalities associated with cerebellar disease*

Disorder	Description
Ataxia	Reeling, wide-based gait
Decomposition of movement	Inability to correctly sequence fine, coordinated acts
Dysarthria	Inability to articulate words correctly, with slurring and inappropriate phrasing
Dysdiadochokinesia	Inability to perform rapid alternating movements
Dysmetria	Inability to control range of movement
Hypotonia	Decreased muscle tone
Nystagmus	Involuntary, rapid oscillation of the eyeballs in a horizontal, vertical, or rotary direction, with the fast component maximal toward the side of the cerebellar lesion
Scanning speech	Slow enunciation with a tendency to hesitate at the beginning of a word or syllable
Tremors	Rhythmic, alternating, oscillatory movement of a limb as it approaches a target (intention tremor) or of proximal musculature when fixed posture or weight bearing is attempted (postural tremor)

TESTS RELATED TO CEREBELLUM*

Test	Description	Image
Finger nose test	While the examiner holds his finger at arm's length from the patient. Patient touches her nose and then touches the examiner's finger. After several sequences, the patient is asked to repeat the exercise with her closed eyes. **A patient with a cerebellar disorder tends to miss the target.**	 Finger-to-nose test. Patient cannot direct finger accurately with eyes closed
Heel to shin test	The heel to shin test is a measure of coordination and may be abnormal if there is loss of motor strength, proprioception or a cerebellar lesion. If motor and sensory systems are intact, an abnormal, asymmetric heel to shin test is highly suggestive of an **ipsilateral cerebellar lesion.**	
Dysdiadochokinesis	Dysdiadochokinesis: Inability to perform rapidly alternating movements. Is called dysdiadochokinesia. It is usually caused by **multiple sclerosis in adults** and **cerebellar tumors in children**. Patients with other movement disorders (e.g. Parkinson's disease) may have abnormal rapid alternating movement testing secondary to akinesia or rigidity, thus creating a false impression of dysdiadochokinesia.	
Cerebellar signs	- Response delays Hypometria. - Ataxia. - Incoordination/ rapid alternating movements (disdiadocho kinesia).	 Response delays Hypometria & Ataxia Incoordination/ rapid alternating movements (disdiadocho kinesia)

Autoregulation of Cerebral blood flow

1 Cerebral Circulation

R

Brain receive its blood supply from four main arteries:

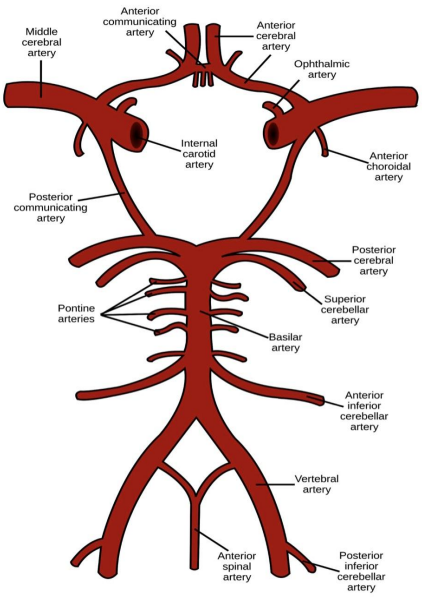
1-two internal carotid arteries.

2-two vertebral arteries. → The **vertebral arteries** unite to form Basilar artery

↓

The Basilar artery and the carotids form the **circle of Willis.**

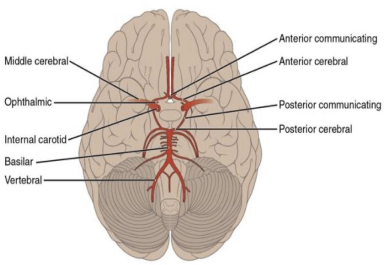
1 and 2 form the **Circle of Willis**: A group of arteries near the base of the brain. → The circle of Willis consists of six large vessels

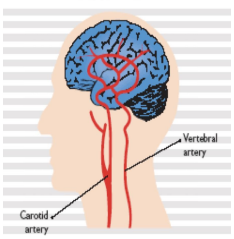


Female slides only

1. Anterior cerebral artery (left and right)
2. Anterior communicating artery
3. Internal carotid artery (left and right)
4. Posterior cerebral artery (left and right)
5. Posterior communicating artery (left and right)
6. Basilar artery

- The Middle Cerebral Arteries (MCA), supplying the brain, are not considered part of the circle.
- The MCA is by far the largest cerebral artery and is the vessel most commonly affected by cerebrovascular accident.
- The clinical consequences of vascular disease in the cerebral circulation is depend upon which vessels or combinations of vessels are involved.
- Substances injected into one carotid artery distributed completely to the cerebral hemisphere on that side. Normally no crossing over occurs because of equal pressure on both sides.





IMPORTANT Slide

2

Cerebral Blood Flow

R

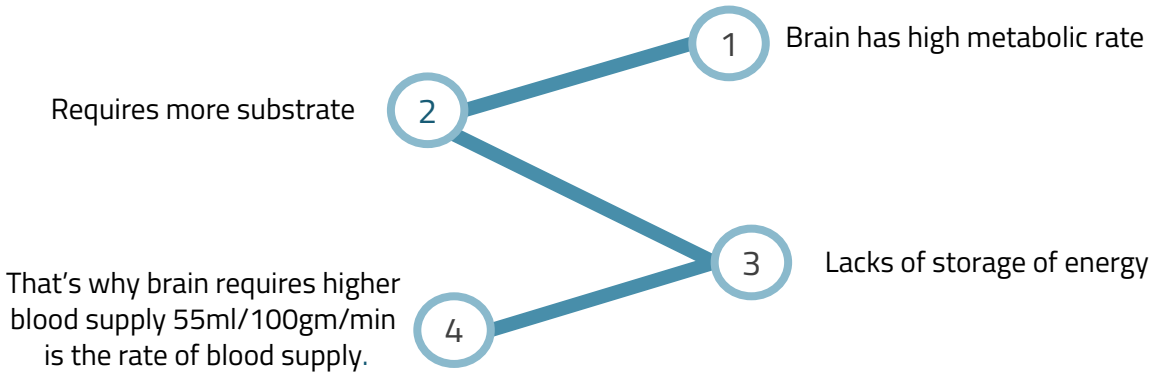
- CBF is tightly regulated to meet the brain's metabolic demands*
- Brain: **1350 gm**; **2%** of Total Body Weight*
- Normal blood flow through the brain of the adult person averages = ***50 to 65 ml /100 grams of brain tissue / minute.***
- For entire brain: **750 to 900 ml/min** = 15 % of the resting cardiac output.
- It is important to maintain CBF within narrow limits because:-*
  - ❖ Too much blood can raise intracranial pressure (ICP) which can compress and damage delicate brain tissue.
  - ❖ Too little blood flow causes ischemia
- **Ischemia** results if blood flow to the brain is below:- **18 to 20 ml** / 100 gm brain tissue / minute*
- **Tissue death** (Necrosis) occurs if flow drops below **8 to 10 ml** / 100 gm brain tissue / minute.*

*Values are **important**

Physiological considerations:

*Male slides only

Brain accounts for 2% of body weight yet requires 20% of resting oxygen consumption .
O2 requirement of brain is 3-3.5 ml/ 100 gm /min.
And in children it goes higher up to 5ml/100gm/min.



factor	factor
CHEMICAL	MYOGENIC
CMR	Autoregulation / MAP
anesthetics	
temperature	BLOOD VISCOSITY
PaCO2	
PaO2	NEUROGENIC

Factors Affecting Regulation of CBF

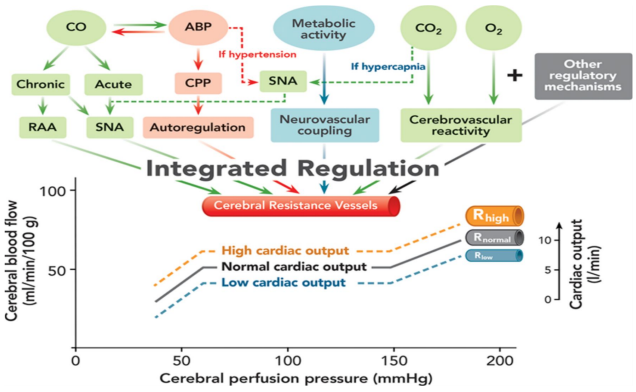
- A Myogenic
- B Neurogenic
- C Metabolic

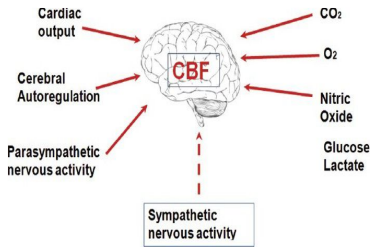
Note that: Nitric oxide & adenosine are autoregulation mediators.

VERY IMPORTANT slide

3 Regulation of CBF

R



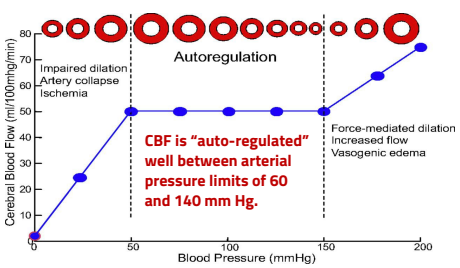


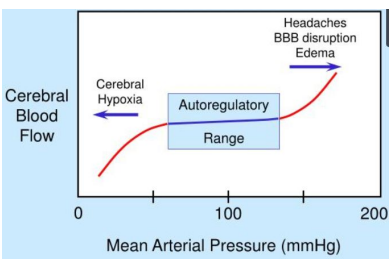
*Cardiac output (CO); Sympathetic nervous activity (SNA); Renin–angiotensin–aldosterone (RAA) system; Arterial blood pressure (ABP); **Cerebral perfusion pressure (CPP)**; Carbon dioxide (CO2) and oxygen (O2); (R) Cerebral resistance vessels at high (R high), normal (R norm), and low (R low)*

A Myogenic AutoRegulation

- **-Arterioles dilate or constrict** in response to changes in BP and ICP to maintain a constant CBF.*
  -Vascular smooth muscle within cerebral arterioles **contract to stretch response**, regulating pressure changes. Autoregulation of CBF completely **BP-dependent.***
- **Myogenic theory:** The vascular smooth muscles are highly responsive to changes in pressure, a process called myogenic activity, that contributes to auto-regulation of cerebral blood flow.*
- **Cerebral blood flow (CBF) is well extremely** "**auto-regulated**" between arterial pressure limits ***of 60 and 140 mm Hg.***
- The brain maintains proper **Cerebral Perfusion Pressure** (CPP) through the process of autoregulation.*
- ***Mean arterial pressure*** can be decreased acutely to as low as 60 mm Hg or increased to as high as 140 mm Hg **without significant change in cerebral blood flow.***
- - The response to **lower pressure is arteriolar dilation** in the brain, while when blood **pressure rises they constrict.***
- - At their most constricted condition, blood vessels create a pressure of 150 mmHg, and at their most dilated the pressure is about 60 mmHg.*
  Thus, changes in the body's overall blood pressure do not **normally** alter cerebral perfusion pressure (CPP) drastically.*
- When pressures are outside the range of **60 to 150 mmHg (to 180 mmHg)** , the blood vessels' ability to autoregulate pressure through dilation and constriction is lost, and cerebral perfusion is determined by blood pressure alone without autoregulation.
- Thus, **hypotension can result in severe cerebral ischemia** (=***If arterial pressure falls*** below 60 mmHg, cerebral blood flow become severely decreased => ischemia) & **hypertension can result in stroke.**

Guyton: Cerebral Blood Flow Autoregulation Protects the Brain From Fluctuations in Arterial Pressure Changes. During normal daily activities, arterial pressure can fluctuate widely, rising to high levels during states of excitement or strenuous activity and falling to low levels during sleep. However, cerebral blood flow is "autoregulated" extremely well between arterial pressure limits of 60 and 140 mm Hg. That is, mean arterial pressure can be decreased acutely to as low as 60 mm Hg or increased to as high as 140 mm Hg without significant change in cerebral blood flow.





Autoregulation of Cerebral blood flow

1 - Carbon dioxide (CO2) & 2 - Hydrogen ions (H+)

Acidity & Carbonic Acid

- **Carbon dioxide increase cerebral blood flow** by combining first with water in the body fluids to form carbonic acid, with subsequent dissociation of this acid to form **hydrogen ions.***
  (**CO2** diffuse through Blood-Brain-Barrier (BBB) into the CSF to form **H+** via (Carbonic acid) which then causes the **vasodilation**.)
- When activity in a given region of the brain is heightened, the increase in **CO2 and H+ concentrations causes cerebral vasodilatation**, and deliver more blood to the area to meet the increased demand.*
- Blood vessels dilate in response to low pH (acidity), Arterioles dilate in response to potent chemicals that are by-products of metabolism such as **lactic acid**, **carbon dioxide**, **pyruvic acid** and **H+ .**
- tissue metabolism → acidic substance (ex; CO2, H+) → **increase CBF .**

Acidosis:

- Increase in cerebral blood flow, due to **vasodilation.**
- **Depress neurotransmitter.**

Alkalosis:

- increase neurotransmitter.
- No effect on cerebral blood flow.

1 - CO2 is a potent vasodilator

Increased CO2 (Hypercapnia)

- **Hypercapnia** is a condition of abnormally elevated carbon dioxide (CO_2) levels in the blood.
- Increased CO2 / decreased BP → **Vasodilation.***
- As the arterial tension of CO2 (PCO2) **rises**, **CBV** and **CBF** increases.*
- Excess carbon dioxide can dilate blood vessels up to 3.5 times their normal size.*
- 70 % increase in arterial PCO2 approximately doubles the cerebral blood flow.*

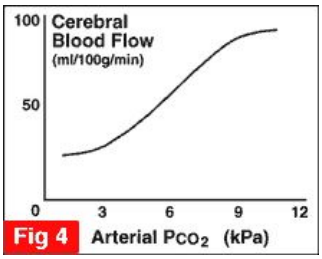
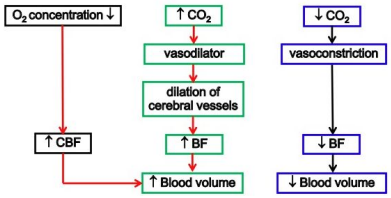


Fig 4

Decreased CO2 (Hypocapnia)

- Decreased CO2 / increased BP → **Vasoconstriction**.*
- As the arterial tension of CO2 (PCO2) **falls**, **CBV** and **CBF** Decreases, When it is decreased vasoconstriction is induced.*
- **During hyperventilation → decreased CBF → cerebral hypoxia.**

CBF: CO2 and O2



Taste and Smell

Taste and smell

R

Distribution

- there are 5 established taste buds
- Distribution of taste buds on the tongue is not uniform
- There are no taste buds at the mid dorsum of the tongue

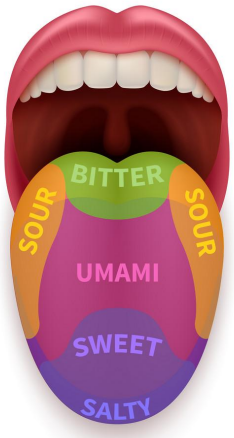
Sweet tongue tip

Sour tongue margins

Bitter back of tongue

Salt widely distributed on the edges.

Umami "Beef taste" of steak, widely distributed all over the tongue.



Taste Sensation

When the taste buds are stimulated they produce nerve impulse to specific brain area through:

- Anterior 2/3 of the tongue → VII Facial
- Posterior 1/3 of the tongue → IX Glossopharyngeal
- Receptors on the palate, pharynx, epiglottis → X Vagus

Taste Mechanism

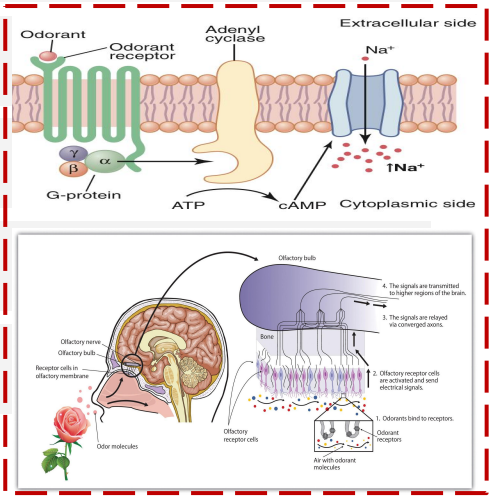
1	Molecules dissolve in the saliva without it? less taste
2	Attached to receptors on cillia of gustatory cells. Combination between molecules and receptors are week (since taste can be easily abolished by washing mouth with water)
3	receptors potential
4	action potential

IMPORTANT slide

R

Olfactory Mechanism

1. Molecules dissolve in mucus layer
2. combine with receptors on cilia (Odorant + receptor protein)
3. stimulate adenylate cyclase (G protein → adenylate cyclase)
4. increase intracellular cAMP (ATP → cAMP)
5. opening of Na channels → Na influx
6. receptors potential (depolarization)
7. AP in olfactory pathway



Olfactory Pathway

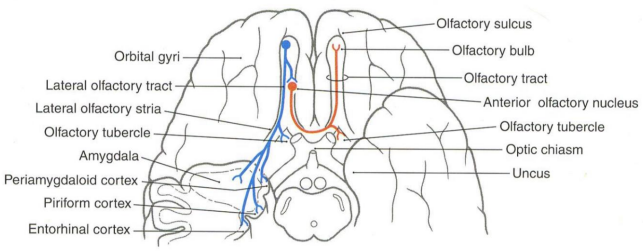
As in the boys slides

Olfactory Receptors → Olfactory nerve → Olfactory bulb → Olfactory Tract

Click to see the original pic

From the Olfactory tract

Lateral olfactory area



Thalamus → Orbitofrontal cortex (newer system) for conscious perception of smell

Prepyriform cortex,Pyriform cortex , Amygdala →Hippocampus limbic system (less old factory system) For odor memory

Medial olfactory area

Septal nuclei → Hypothalamus limbic system (primitive parts,very old) for motivational and emotional aspects of smell

Speech

- Girls Dr: **Function + Lesion are important**
- don’t worry about the difference between girls and boys slides ذاكروا سلايداتي

The whole page is on Male slides only

Speech

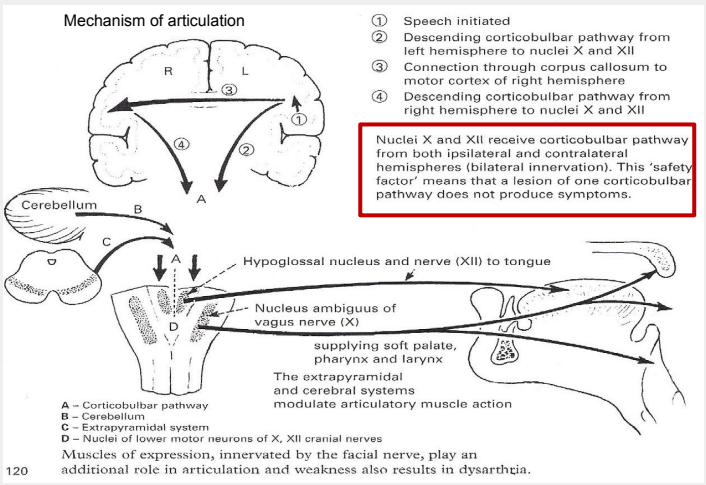
R

03 ARTICULATION:

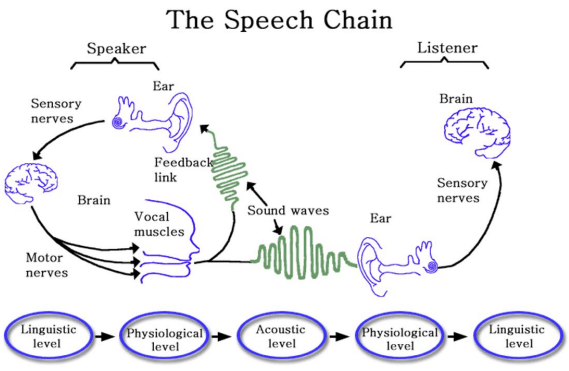
- **Muscular movements** of the mouth, tongue, larynx, vocal cords.
- Contribution by structures to shape airflow.
- A variety of speech sounds can be produced in terms of another way of airstream change – Articulation.
- Articulation is done mainly at **vocal cord.**
- An specific part of the vocal apparatus involved in the production of a speech sound.
- **Active articulators:** Lips, tongue, lower jaw, velum (structures of the mouth).
- Responsible for the intonations, timing, and rapid changes in intensities of the sequential sounds.

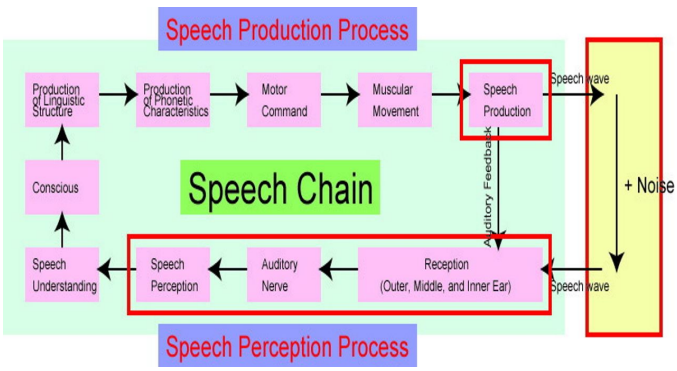
Male Dr. : Very important picture specially this Part (red square).

- Phonation: عملية إنتاج الأصوات
- Articulation: عملية ترتيب هذه الأصوات لإنتاج الكلمات



More pictures explaining the Speech production processes:





Speech

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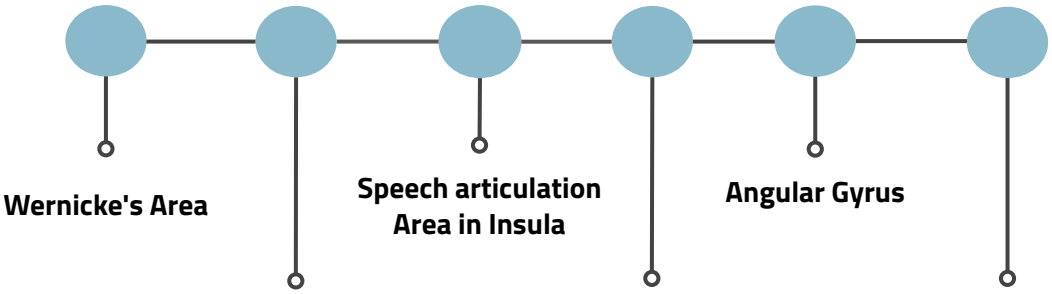
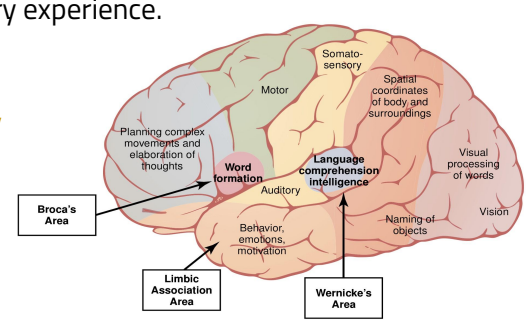


1 Wernicke's area:*

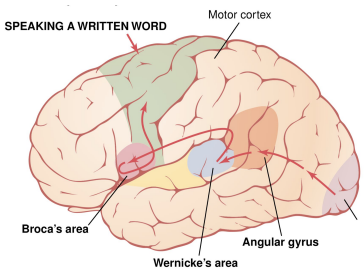
- At the posterior end of the **superior temporal gyrus.**
- Closely associated with 1 & 2 auditory areas.
- Responsible about **comprehension of auditory & visual information**, then projects it to Broca's area via arcuate fasciculus.
- Interpretations of sensory experience.
- Formation of thought in response to sensory experience.
- Choice of words to express thoughts.

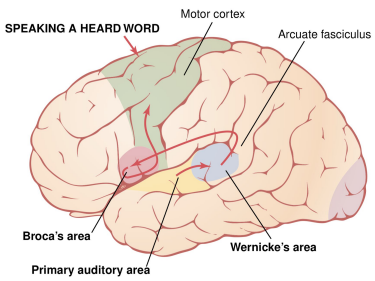
Language comprehension, intelligence,
cognitive function , analysis ,choice of the way we response,



2 Broca's area:

- A special region in the frontal cortex, called Broca's area, provides the neural circuitry for **word formation**.*
- This area, is located partly in the posterior lateral prefrontal cortex and partly in the premotor area (At the lower end of premotor area*).*
- It is here that **plans and motor patterns for expressing individual words** or even short phrases are initiated and executed.*
- This area also works in close association with Wernicke's language comprehension center in the temporal association cortex.*
- In adult who learn second language during adulthood. The MRI shows portion of Broca's area concerned with it is adjacent to but separate from area concerned with the native language. But in children who learn second language early in life there is only single area involved for both languages. *





Speech

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Lesions of Different Areas in The Brain: This table is in males slide only + Important

Area	Lesion Features
Auditory association areas	Word deafness
Visual association areas	Word blindness called dyslexia
Wernicke's Aphasia	Unable to interpret the thought
Broca's Area Causes	Motor Aphasia
Global Aphasia	Unable to interpret the thought Motor Aphasia

Lesion area	Type of aphasia	Girls Dr said its IMPORTANT
Wernicke's Area	**Sensory or Wernicke's aphasia (fluent):** Broca's area receive unprocessed disorganized information from wernicke's area ❖ Lesion of wernikes area +/- arcuate fasucul.* ❖ Impaired comprehension / understanding.* ❖ Loss of intellectual function.* ❖ Failure to interprets meaning of written or spoken words.* ❖ Meaningless words & excessive talk (in severe cases).* ❖ FLUENT: Meaningless words with loss of comprehension/ understanding.*	
Broca's Area	**Motor or Broca's aphasia (non fluent):** Projection to motor cortex is not working ❖ Patient will understand spoken & written words but find it difficult to speak or to write.* ❖ NON FLUENT, Understanding normal but Voice production defective.* ❖ Poorly articulated speech, slow with great effort & abnormal rhythm.* ❖ In some cases speech may be limited to 2-3 words.*	
Arcuate Fasciculus*	**Conductive aphasia (fluent):** ❖ Lesion of nerve fibres of arcuate fasiculus. ❖ Patient understand speech of others but can not repeat it. ❖ Meaningless speech. ❖ Normal comprehension but the Transmission from wernicke's area to broca's area is disrupted.	
Angular Gyrus*	**Anomic aphasia** ❖ Lesion of angular gyrus, thus Broca's & Wernicke's are intact.* ❖ Unable to name the objects.* ❖ Speech & auditory comprehension is normal but visual comprehension is abnormal, due to visual information is not processed & not transmitted to wernicke's area.* ❖ **Dyslexia** (word blindness) interruption in the flow of visual experience into Wernicke's area from visual area.* Depending on the extent of lesion in the angular gyrus ،major lesion or minor a specific letter or number will be affected مثلاً ما راح يقدر يشوف حرف الألف فتكون مشوشة أو مقلوبة أو معوجة يحسبو عنده صعوبات تعلم ولكن هو ما يشوف الحرف They do well after special classes and training, **finds it hard to differentiate between d,b and p,q**	

The whole page is EXTRA

Summary of Speech Disorders

Dysphonia

Abnormal sound production due to problem in vocal cord.

Dysarthria

Abnormality in articulation (motor dysfunction) Due to neurological conditions involving motor function.
e.g. :

1. Slurred Speech:
Language is intact, Paralysis, slowing or in coordination of muscles of articulation or local discomfort causes various different patterns of dysarthria.

2. Stuttering:
Talking with involuntary repetition of sounds, especially initial consonants.

Aphasia

loss of or defective language from damage to the speech center within the left hemisphere.

1. Wernicke's aphasia (fluent):

Impaired comprehension + Meaningless words. (Sensory)

2. Broca's aphasia (non fluent):

understand spoken & written words but find it **difficult to speak** or to write (motor).

3. Conductive aphasia (fluent):

Normal comprehension but the Transmission from wernicke's area to broca's area is disrupted.

4. Anomic aphasia:

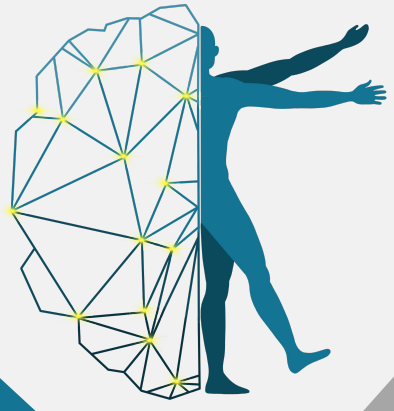
visual comprehension is abnormal, due to visual information is not processed & not transmitted to wernicke's area.

5. Insula damage: Progressive non-fluent aphasia:

deterioration of normal language function.

6. Global Aphasia:

the **combination** of the expressive problems of **Broca's** aphasia and the loss of comprehension of **Wernicke's**.



Thank you