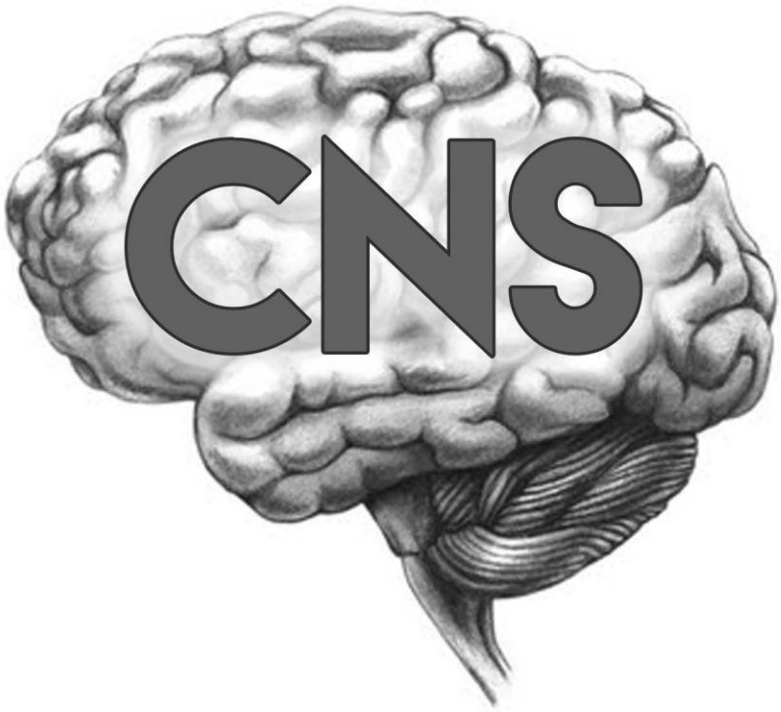

ANATOMY

☑ Sheet ○ Slide ○ Handout

Number

19

Subject

Aphasia

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Speech and language

Speech and language involve UNDERSTANDING of **spoken** and **printed** words and the ability to EXPRESS IDEAS in **speech** and **writing**.
So a normal individual must be able to **communicate** with others by language (talking) and by Reading.
So we can think of language and speech as a high cortical function. This implies that we have certain areas in our cortex that are specialized in speech.

The language and speech from cortical view

- In more than 95% of people, the Lt cerebral hemisphere contains the centers for language and comprehension (Dominant Hemisphere \ Talking Hemisphere).
  → Cortical areas of speech and language:
  1. **Broca's motor speech** area (area 44, 45) at the Lt inferior frontal gyrus.
  2. **Wernicke's area.**
     - While Broca's is considered motor speech area, Wernick's is considered sensory speech area.
     - Present at the parietal lobe. (posterior part of area 22).
  3. **Primary auditory area** (areas 41, 42).
  4. **Premotor and motor areas.**

Normal communication b/w people by spoken language needs at least the above-mentioned areas.

What about the Rt hemisphere?

- ✓ As said earlier the Lt hemisphere is considered the talking hemisphere, the Rt hemisphere cannot talk but it can recognize faces, analysis, solve

problems, Music. And many other functions of the non-dominant hemisphere.

It's important to bear in mind that *"both hemispheres comprehend"* (can understand spoken/written language) but only the Lt hemisphere can talk.
→A comprehensive example:
If someone handle u a key in your Lt hand, your Rt cortex will recognize that and a thought is produced (ahh! There's a key in the Lt hand) if u want to tell someone that u have a key, "the thought" must be transmitted from your silent-Rt hemisphere to your Lt-talky-hemisphere via the of corpus callusoum (CC).
Say the CC is cut → you won't be able to express that you are holding a key in your Lt hand. (Yet u recognize that u are holding a key).
After this introduction, we can understand what happens inside the cortex when we communicate by language.

Look at figure1

- ✓ To communicate well, you must be able to listen well then understand what we've listened, then we can talk back.
- ✓ When someone says "SALAM" the first cortical area involved in the response is the 1ry auditory area. By the cortex has "heard" the sound -but has not understand them yet.
- ✓ Then impulses are sent to the association auditory area -area 22-(specially Wernicke's area).
  نفهم ما نسمع (وما نرى في حالة القراءة)
- ✓ The Wernicke's area will send signals to Broca's area via the Arcuate bundle (long association tract).
- ✓ At Broca's area there are programs of speech stored→ these programs are sent to motor areas (area 4 + 6) from which the corticobulbar tract descends.
  The corticobulbar will supply cranial nerve motor nuclei → then muscles of speech can "move" and we talk.

  Side note: what are the muscles of speech?

  Muscles of palate, tongue, larynx and lips.

  So the corticobulbar will supply Nucleus ambiguous (for laryngeal muscles

involved in speech) and hypoglossal nucleus (tongue muscles) + facial motor (for lips) …etc
So we have to listen, understand and then talk → to communicate.

- To be able to understand what we read we need:

1. Primary visual area (to see words).
2. Association visual area -area 18 and 19- (to understand what u read) along with the
3. Lt angular gyrus, at the inferior parietal lobule, a VERY important cortical region that aid in comprehension of what we read.
   Note:
   we say Lt, as we consider the Lt Hemisphere as the dominant hemisphere in the majority of people. If this area is lesioned (say by a stroke) or a child has a congenital bad Lt angular gyrus → then this individual will face **reading difficulty.**
   هذه إحدى أسباب صعوبات التعلّم و ينبغي الانتباه هنا أن الطفل "مش غبي" إنما لا يمكنه التعلّم بالقراءة.
   What is important about this area, that it is important in converting words into their auditory equivalents. (**from Graphemes TO Phonemes**)
   لتحويل الكلمة من الصورة المرئية المكتوبة إلى الصورة المسموعة، عجيب!

From the **Lt angular gyrus** to the **Wernicke's area** in order to understand what we read.
(note in both cases Wernicke's received signals as auditory signals)
then from Wernicke to Broca … etc

By this we have understood what we hear and what we read and we can respond by talking or even by writing. (Details later)

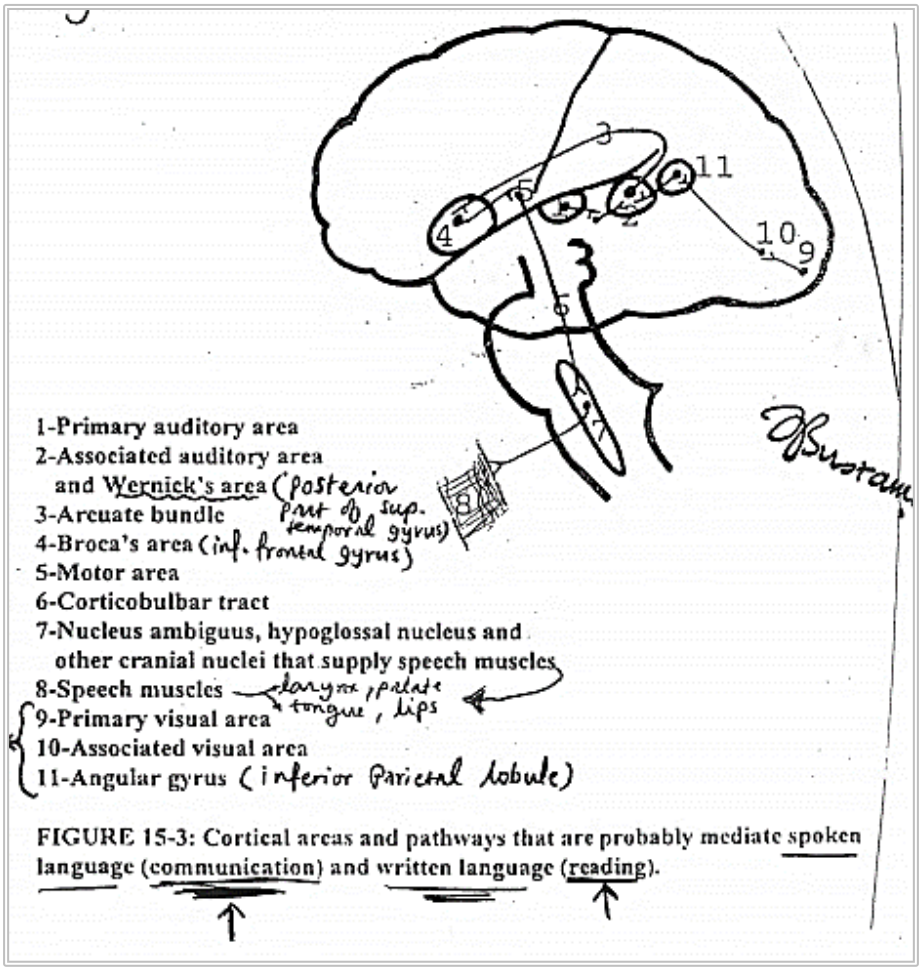

***Figure 1** : cortical areas of communication.*

Aphasia

- ✓ Definition:

  Abnormalities of language functions that are NOT due to defects of vision or hearing or to motor paralysis (of facial muscles), but they are rather caused by lesions in the categorical hemisphere.

- ✓ Types:
  - Motor
    1. Occur when motor-cortical regions are damaged, like Broca's area.
    2. AKA **non-fluent aphasia:** if they managed to talk they will just say simple words like (yes , No..) & generally their speech tend to be poor and slow.

- Sensory
  1. Occur when sensory-cortical regions are damaged, like Wernicke's area.
  2. AKA **fluent aphasia** , the patient tend to talk A LOT but in a non-purposeful manner - (أي حكي)
- At both types of aphasia, there is NO vision impairment or hearing impairment or motor paralysis .عضلات الكلام غير مشلولة

  Note: we have other types of aphasia, which are : nominal aphasia ,conducting aphasia and global aphasia(we will talk about them later at this sheet).

  Now we will talk with more details about each area (some info are repeated).

Wernicke's area – sensory speech area –الفهّامة

- ✓ AKA general interpretative area, it aids in GENERAL UNDERSTANDING of what we feel, see, and hear.
- ✓ It's the posterior part of area 22.(at the junction b/w temporal and parietal lobe)
- ✓ It receives input from wide areas, angular gyrus (which itself receives from visual association area), auditory association and receives from somatosensory areas.
  So Wernicke's input is auditor and visual signals.
- ✓ When all of these information are collected in Wernicke's → it can then generate thoughts.
  Then Wernicke's choose the proper words to express your thoughts.
- ✓ Wernicke's output can be verbal (spoken) or written.
  How u can respond by speaking :
  if Wernicke's send signals to Broca's area.
  How u can respond by writing :
  Wernicke's can send signals to the hand-skill-area at the premotor cortex → by this u are able to express your thoughts by writing.
  Note: this hand-skill-area is not far from Broca.

If Wernicke's is damaged :

- ✓ If Wernicke's is damaged the patient will show TOTAL FAILURE to comprehend spoken or written language. (despite that the vision and hearing sensation are intact).
  The patient will suffer from fluent aphasia (a lot of meaningless talk) and the patient is not aware of his problem (unlike motor aphasia patient who understand what he hear but cannot express himself by talking , such a patient might develop depression).
- ✓ Also if Wernicke's is damaged the patient won't be able to use the proper nouns in the proper place or in the proper context and things like these will manifest :
  1. **Circumlocution:** الدوران حول المعنى
     the patient will say Instead of 'I use a knife'>> 'I use the thing you cut with'.
  2. **Verbal paraphrasia** (the use of words of allied meaning):
     the patient will say Instead of 'I cut with a knife', 'I cut with a fork'.
  3. **Phonemic paraphrasia** (the use of made-up words having appropriate sounds).
     بخترع كلمات إلها نفس الصوت
     The patient say Instead of 'knife and fork', 'bife and dork'
     THESE PATEINTS ARE NOT SCHEZOPHRINIC ,THEIR TALKING CORTEX IS DAMAGED.

Broca's area and its lesion

- Occur At the frontal lobe.
- If Broca is damaged by a stroke then u must expect that area 4 and 6 are damaged too (as they are not far from each others).
  So if the patient have Rt monoplegia (or hemiplegia), he\she will suffer from motor aphasia, too.
- In Broca's damage, the type of aphasia is *non-fluent* \ *expressive* (slow+ poor) in which the patient understand what he hears and knows what he's going to say but he has difficulty in saying it.
  Such patient does not have speech muscles paralysis but he has lost

speaking programs which were stored at Broca's.

Would such a patient has a difficulty in writing?

If the hand-skill area at premotor cortex is affected then yes (remember that this area is not far from Broca's).

FLASH Q:

Abo-khaldon is suffering from Rt monoplegia along with aphasia, what type of aphesia u expect Abo-khaldon is suffering from? (Motor/ sensory)

-Ans: it's more properly motor aphasia, as motor areas that control the limbs and Broca's motor area of speech area adjacent.

Aphasia types

	Broca's	Wernick's
Articulation	Slurred	Normal
Speed	slow	Rapid
comprehension	Good	Poor
Awareness	YES (may develop depression)	NO (بهذرب ع راحته)

Angular gyrus and ALEXIA

Look at figure 2 and notice where the angular gyrus is

- ✓ It's area 39 at the parietal lobe.
- ✓ How to find this area grossly: follow the Lt Superior temporal sulcus backward.
- ✓ As said earlier Lt angular gyrus is concerned with converting visual input into auditory (from graphemes to phonemes), if this function wasn't there we wouldn't be able to understand what we read.
- ✓ Input of Lt angular gyrus: visual association (area 18+19) from ibsilateral and contralateral side and from the angular gyrus of the opposite side.

- ✓ Output of angular gyrus: to Wernicke's area.
- ✓ If the Lt angular gyrus is damaged → **ALEXIA** occur.

ALEXIA:

- ✓ Inability to understand what u read as the converter (i.e. the Lt angular gyrus) is damaged.
- ✓ u can examine alexia by asking the patient to read for u.
  If the patient is not educated (بعرفش يقرا لإنو ما دخل مدرسة أصلاً) can be tested for alexia by putting several objects in front of him (say key , cup , book..) and then sling him to pick particular object → he won't be able to do so. And he won't be able to name the object→ this type of aphasia is called **nominal aphasia** (aka anomic aphasia).
- ✓ Generally speaking, Alexia-s could be congenital or acquired:
  - Congenital/ developmental: the person could be born with defective Lt. angular gyrus → when such child enter the school they will discover that he cannot read nor understand what he reads تعلّم من صعوبة وسيعاني.
  - Acquired: the Lt angular gyrus could be damaged by a stroke or sth.

- ✓ **Hemi**-**alexia** : the patient will lose the ability of reading and understanding what he reads at the level of half his visual field. (the Dr just said this about hemi-alexia – I'm not sure whether its causative lesion is situated at the angular gyrus or not).

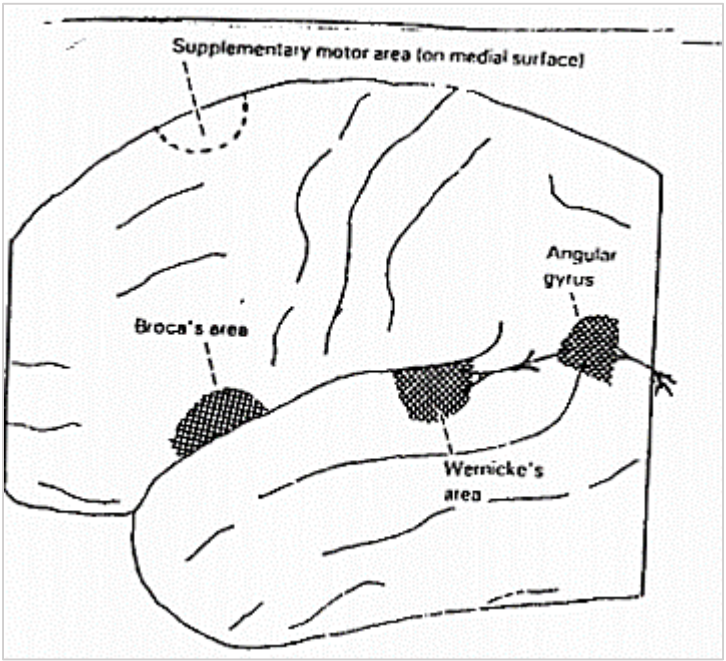

***Figure 2:** Angular gyrus with respective to Wernicke and Broca.*

Reading loud

Before we start talking about the exact steps, what is our TAREGT in this context, meaning: which cortical area is the "chief" of loud-reading ability?
THE **LT ANGUALR GYRUS** → so we have to collect all the inputs in this gyrus.

Ok now we can start
which areas we need to read loud: -figure3

1. Retina.
2. Optic nerve , after the optic chiasma it becomes
3. Optic tract , which ends at the level of LGB (thalamus).
   Note : we can say retino-geniculate tract instead of retina-optic nerve-optic tract.
4. From the thalamus the Optic radiation arise , AKA geniculo-calcarine tract , which transmit signals to :
5. The primary visual area and association visual area (connected together via association fibers).
   If we are at the Rt side of the cortex → signals must be transmitted to the Lt side-association visual areas→ in order to reach the Lt angular gyrus.
   So if we are talking about the Rt area 18, 19 → signals must be sent to →area 18, 19 at the Lt→ then to the Lt angular gyrus.
   Rem: signals are sent from Rt hemisphere to the Lt via the corpus callusoum (the splenium here).
6. At the Lt angular gyrus the transformation from visual to auditory info occur , then signals are sent to
7. Wernicke's area, for comprehension. And from there signals are sent to
8. Broca's area via arcuate tract. And from Broca to
9. The motor cortex, to move the speech muscles via the cortico-bulbar tract.

Applied neuro-anatomy:
If the Lt angular gyrus and the surrounding parietal association areas were lesioned, what will happen:

1. **Alexia.**
2. **Gerstmann syndrome** (aka Lt posterior parietal disease) , manifest as :

- Agraphia (inability to write) -which is acceptable as if u cannot read it is expected to not be able to write either.
- Acalculia (inability to do simple sums) – even if the patient were a great mathematician before his brain lesion, he won't be able to know the sum of 2+2… SADDLY!
- Difficulty in distinguishing right from left.
- Finger agnosia (inability to tell how many of the examiner's fingers are held up for inspection).

  All in all, these signs are difficult to explain but we know that they are just there.

Important note: sometimes the manifestations of this "angular gyrus syndrome "might be thought to be just dementia-signs → and the patient might be misdiagnosed as Alzheimer patient".

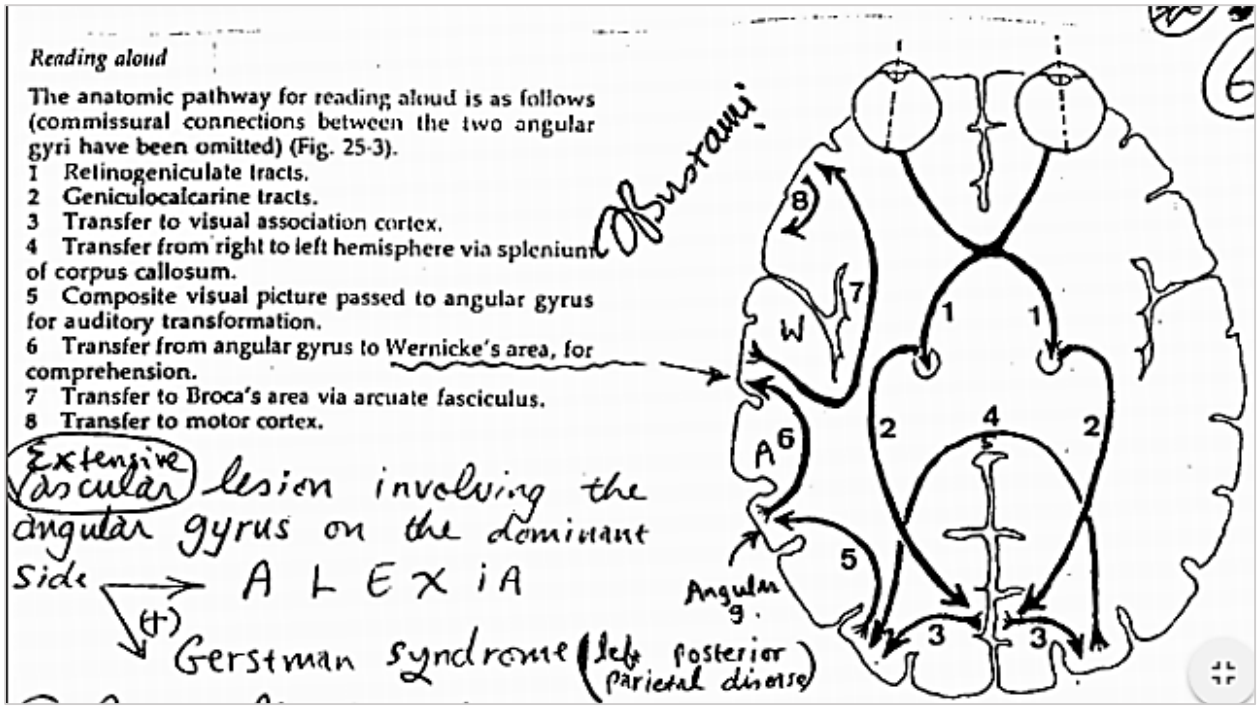

***Figure 3 :** Areas involved in reading loud.*

Conducting aphasia

- ✓ The lesioned area here is **area 40** (just in front of area 39 -which is the Lt angular gyrus).
  look at figure-4 .
- ✓ Area 40 = supramarginal gyrus.
- ✓ The lesion is not at the level of the cortex (i.e. the gray matter) it is rather at the underlying white matter.
- ✓ When this area is lesioned what actually happen that the tract b/w the Wernicke's area and Broca's area is lost which , i.e. the arcuate tract is cut (hence the name , conducting aphasia).
- ✓ The most important symptom of this aphasia:
  - Inability to repeat even simple phrases which spoken by the examiner.
    Explanation : when the patient hear the phrase that he should repeat , the input run all the way tell it reach Wernicke's, but the signals cannot reach Broca's as the tract is interrupted.
    → Some may mix Wernicke lesion with supramarginal lesion, but bear in mind that the patient won't understand u in Wernicke's lesion unlike the supramarginal one.
    (The Dr. said that naming objects is affected in this kind of aphasia)
- ✓ This kind of aphasia is accompanied with **facial apraxia.**
  - **Apraxia** : inability to perform a given movement of request , despite that the patient mentality and motor structure are intact (i.e. no paralysis).
    يعني المريض لا مشلول و عقله سليم لكنه لا يستطيع الامتثال للأوامر الحركية
  - Many types of apraxia exist, like limb apraxia (like when u ask the patient to handle a certain object, he won't) and facial apraxia.
  - In **facial apraxia:** it is thought that the causative factor of it is the interruption of long association fibers passing **from** the visual or/and auditory association areas **to** premotor area and lower of area 4.
    أي أننا نفقد الاتصال بين المناطق التي تجعلنا نفهم ما نرى أو ما نسمع والمناطق الحركيّة التنفيذيّة
    - If the visual connection alone are lost → the patient cannot **mimic** facial movement u ask him to do (like pursing the lips , squeezing the eyes) but the patient can respond to the same SPOKEN request.
      يعني هنا بالفحص إنت بتسأل المريض يقلد ما تفعله من حركات الوجه بس ما بقدر أما إذا أمرته شفوياً فسيفعل.

- If the auditory connections are lost, alone → the patient won't be able to do what u say but he is able to mimic u.
  بالاستماع لن يستطيع تنفيذ ما تطلبه منه ، و لكن بالإيماء سيفعل.

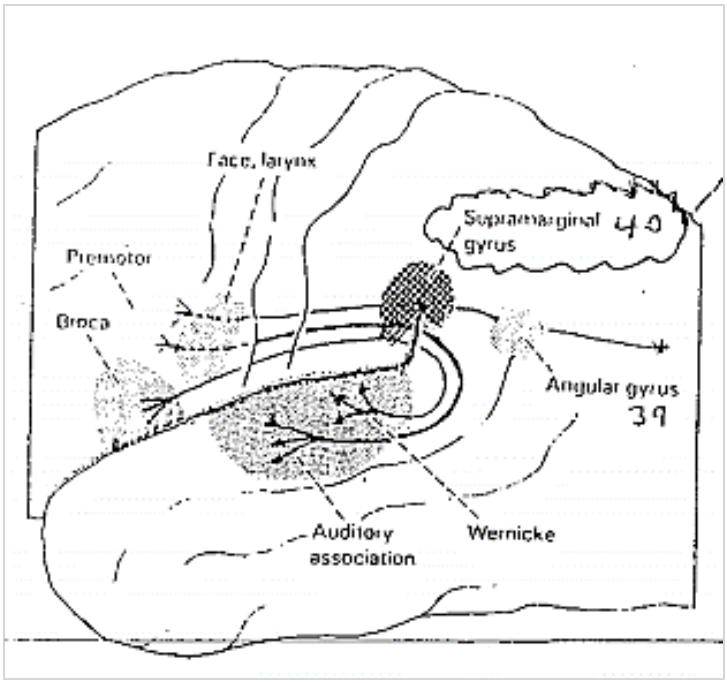

***Figure 4:** Supramarginal gyrus.*

Global aphasia

- ✓ It's a mixture of motor and sensory aphasia.
- ✓ It actually make sense , as:
  - Most brain lesions are vascular events and both Wernicke's and Broca's are supplied by the same artery (i.e. the middle cerebral artery) remember : anterior part of middle cerebral supplies Broca's , posterior part supplies Wernicke's.
  - Might be caused by an event causing narrowing of the carotid arteries (common /internal/ middle) mainly those at the Lt Side or just an embolus at the middle cerebral artery.
- ✓ Manifestations of global aphasia:
  - The patient cannot comprehend or talk. (both motor and sensory defects).

- Might be accompanied with hemiparesis as broca's is lesioned and it's not far from the motor areas (area 4+6) >> contralateral spastic paralysis.

✓ Prognosis: Bad, hard to manage and poor recovery.

- Important note: if the patient present to u with fluent aphasia (alone without paralysis) then most properly he has narrowing of the posterior part of his Lt middle cerebral artery , this patient should be referred to a specialist/surgeon to relive the narrowing (confirmed by angiogram) or remove the embolus (if it has arrived from the heart give the patient anticoagulants) , otherwise the same patient will present to u after a month with global aphasia→ so u must act fast so that the simple aphasia do not turn into global one and always remember that sudden fluent aphasia suggest embolus at the posterior part of the middle cerebral artery (so u just have to give the patient anticoagulants to prevent further embolism).

The significance of aphasia Diagnosis

Aphasia orient the physician: by knowing that a certain patient is suffering from aphasia (of any type) then by definition, we must know that his lesion is at the Lt Side -most properly-.
Further analysis: if the physician notice that the patient has non-fluent aphasia → then his Lt Frontal lobe is lesioned while if the patient has fluent aphasia then he has a more posterior lesion.

Imagine this clinical scenario : a patient present to u , complaining from weak hand girdling skills → u started to think of his upper limb muscles that are responsible for hand-girdling and u put other possibilities like : the patient could have neurological lesion → and this lesion could be UMN lesion or LMN lesion then the patient continue and give u another hint : I'M SUFFERING FROM APHASIA → then u are oriented that : AHH , this patient has an UMN lesion at his Lt frontal lobe.
APHASIA → BRAIN LESION.

Prognosis of different types of aphasias

- Global aphasia → poor prognosis.
- Anomic (nominal) and conduction aphasia → good prognosis + complete recovery occur frequently.
- Broca's and Wernicke's aphasia → intermediate prognosis (in between).
- The best prognosis of all aphasia is that which is caused by a trauma (better prognosis than stroke induced).

Examination of the aphasic patient

(Handout only / except the part of fluent aphasia)
to examine an aphasic patient:

1. Listen to his speech (is it fluent or not?)
2. orient ur-self :
   fluent aphasia →anterior lesion
   non-fluent → posterior lesion
3. Can the patient READ and WRITE?
   if yes the patient is not aphasic
4. Is there hemiparesis?
   If yes, → the lesion is anterior (frontal) involving motor areas.
5. In fluent aphasia check if the patient can comprehend / repeat/ name objects

Aphasia type>>	*Wernicke's*	*Conducting*	*Anomic*
Repeat ability	Cannot	cannot	Can
Comprehending words	Cannot	Do comprehend	Can
Naming objects	poorly	Poorly	Trouble with naming objects

Note : the naming of objects ability is of unknown etiology.من الصعب تفسيرها

Alexia or dyslexia again

- ✓ As said earlier it is the inability to comprehend written language (reading disability).

This definition is valid for educated people ,for non-educated people it is defined as the describe what u see and to name objects

- ✓ Can be acquired (in stroke patients) or developmental (inability to learn to read normally from childhood bcz of Lt angular gyrus defect).
- ✓ Acquired alexia could be :

1. **Pure alexia** (without graphic), AKA *pure word blindness*. Look at Figure5 this patient cannot understand what he reads but can write, surprisingly such a patient cannot read what he wrote himself.
   the lesion here is at two places :
   - The Lt Visual cortex (area 17 at the Lt), rem. That this area receives visual info from the Rt side. And the conducting of Rt visual field info stop at this area is as it is lesioned.
   - The splenium of the corpus callosum. (This actually make sense as by this lesion we've lost the connection with the Rt hemisphere → the Rt-sided info won't reach the Lt angular → and info won't be converted nor comprehended).

     So we can notice that the conducting of both the Rt and Lt visual info is interrupted :
     **The Rt visual field CUT→at the level of the Lt area 17 and
     **The Lt visual field CUT →at the level of the splenium of corpus callosum.
     So we can say that this kind of alexia is complete alexia.

     note : sometimes the patient could have pure alexia without splenic lesion >> but the Lt visual field will stay interrupted as there will be a deep lesion in the occipitotemporal junction which will isolate the visual cotices from the Lt angular gyrus>
2. **Alexia with agraphia** (AKA *parietal alexia*)
   cannot comprehend what he read and cannot write. The lesion here at the Lt angular gyrus (hence the name parietal alexia).

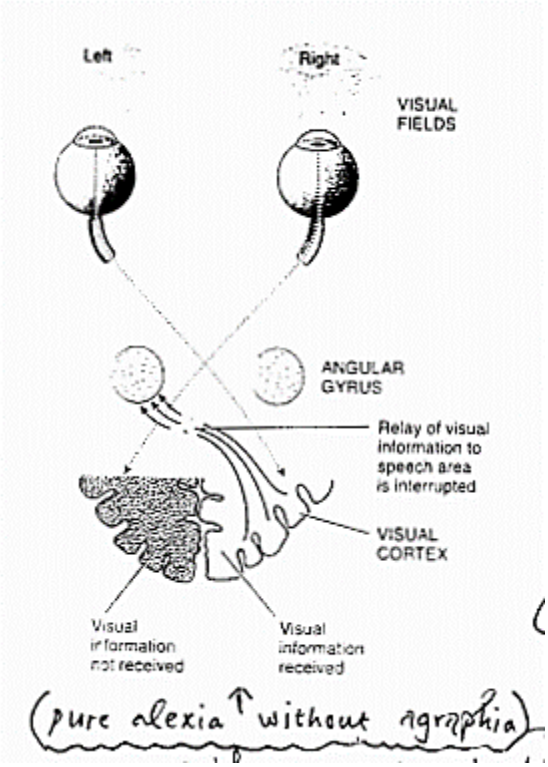

***Figure 5**: Pure alexia without agraphia.*

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