

جامعة نيويورك أبوظبي



PSYCH-UH 2218: Language Science

Class 12: Language Disorders

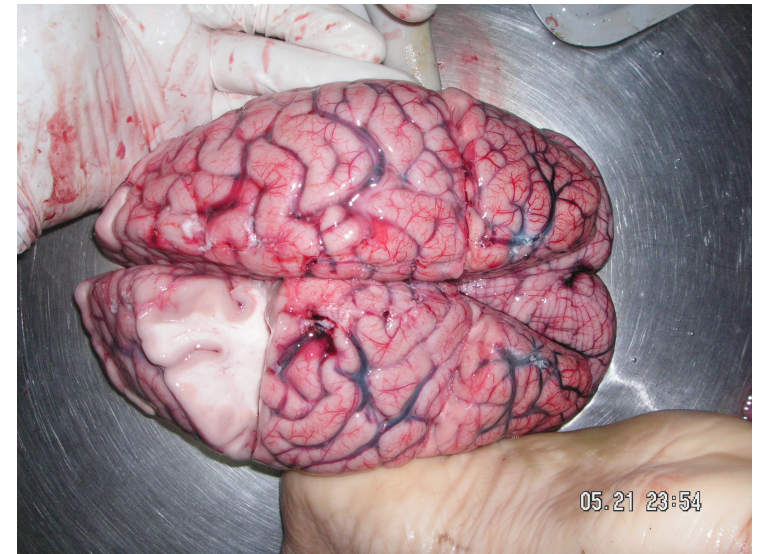
Prof. Jon Sprouse  
Psychology

# Two goals for today

1. Descriptive goal: Explore the symptoms associated with cognitive language disorders (aphasias and semantic dementia)
2. Theoretical goal: Begin to understand the functional organization of cortical areas that support language

First impressions of the brain are that it is just one undifferentiated lump, not unlike other organs.

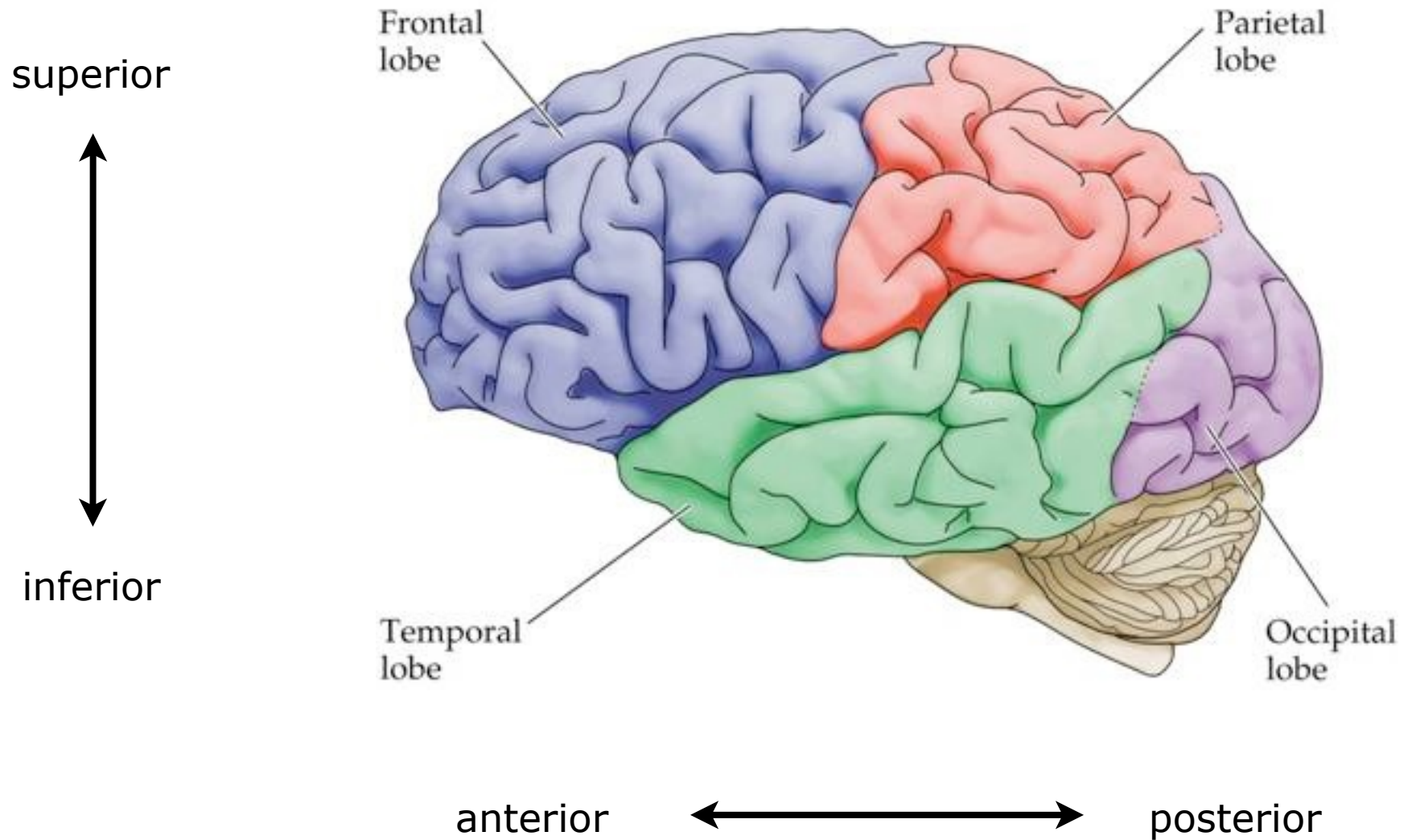
There are some basic differences: the grey outer layer (grey matter) and the white inner layer (white matter), and a few smaller structures, but it still seems mostly like a relatively unstructured mass.



This led to a major debate in 1800s and into the 1900s: Is the brain just one relatively uniform organ, or **do different parts of the brain perform different cognitive functions?**

Some brain basics to help us talk  
about things

# Gross Anatomy: Lobes

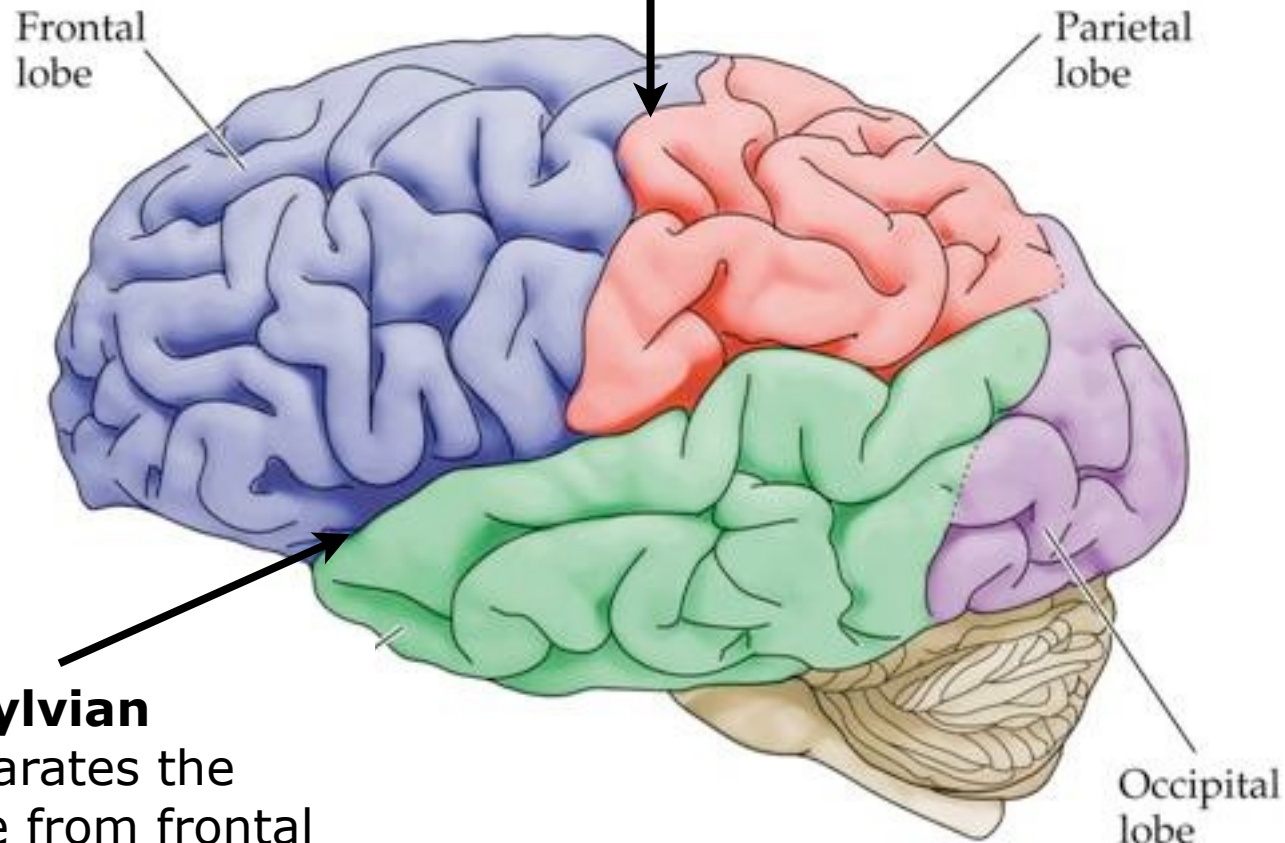


# Gross Anatomy: Gyri and Sulci

**Gyrus:** a ridge of the cerebral cortex. Plural: gyri. Also called a convolution.

**Sulcus:** a furrow (valley) of the cerebral cortex. Plural: sulci. Also called a fissure, especially for the major sulci.

**Central Sulcus:** separates the frontal and parietal lobes



**Lateral or Sylvian Fissure:** separates the temporal lobe from frontal and parietal lobes

# Naming Gyri and Sulci

We can use relative directions and lobe names to name gyri and sulci:

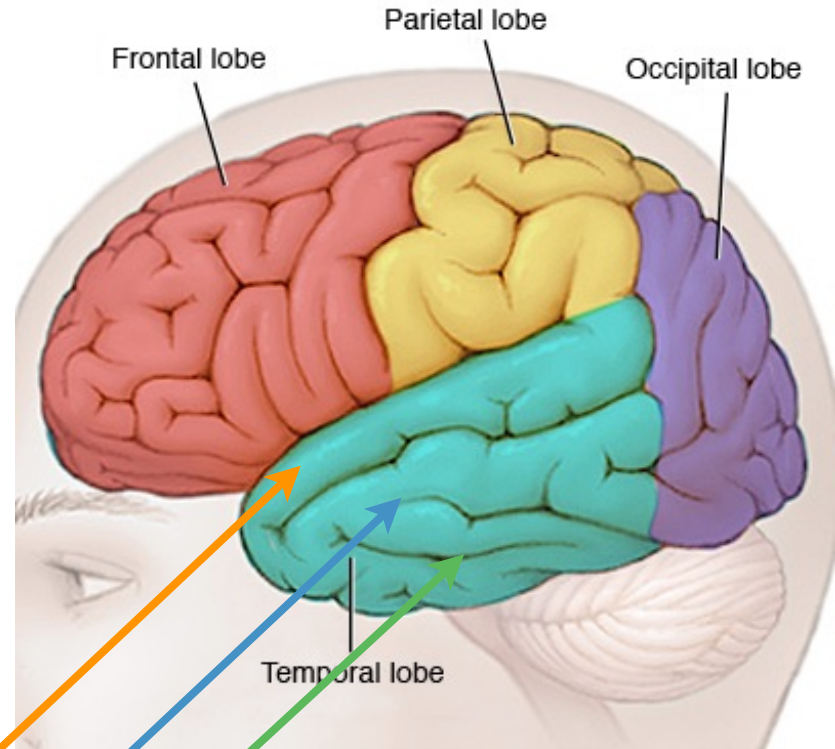
## **Directions**

superior  
medial  
inferior

anterior  
posterior

## **Lobes**

frontal  
temporal  
parietal  
occipital



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Superior Temporal Gyrus

Medial Temporal Gyrus

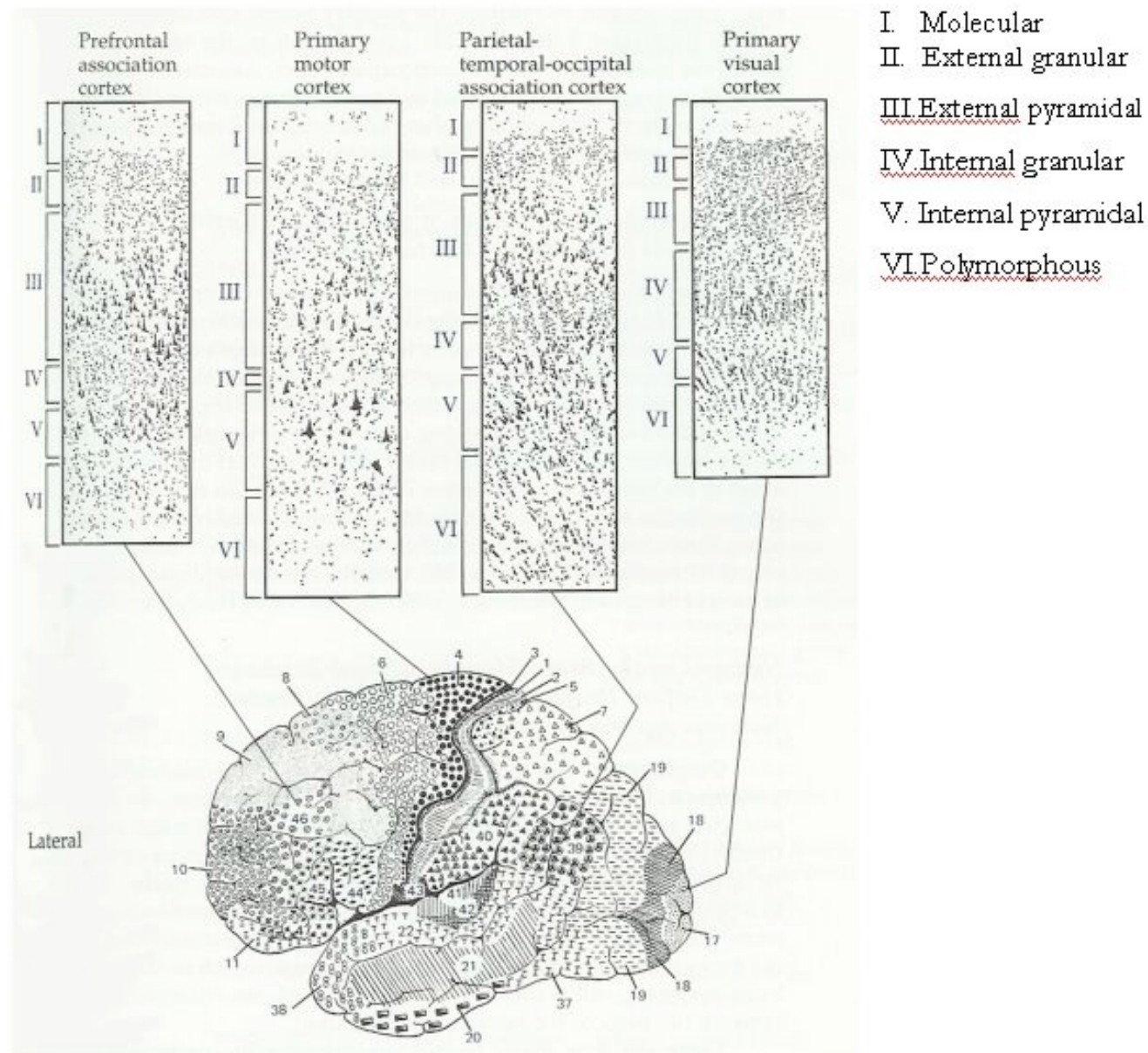
Inferior Temporal Gyrus



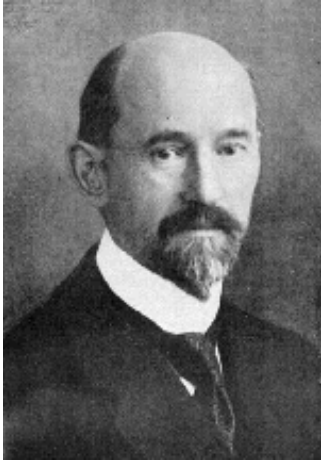
# Cytoarchitectonics and Brodmann areas

**Cytoarchitectonics** refers to the arrangement of neurons in layers.

It turns out that different areas of the brain display different cytoarchitectonics (different arrangements of neurons in layers).



# Cytoarchitectonics and Brodmann areas

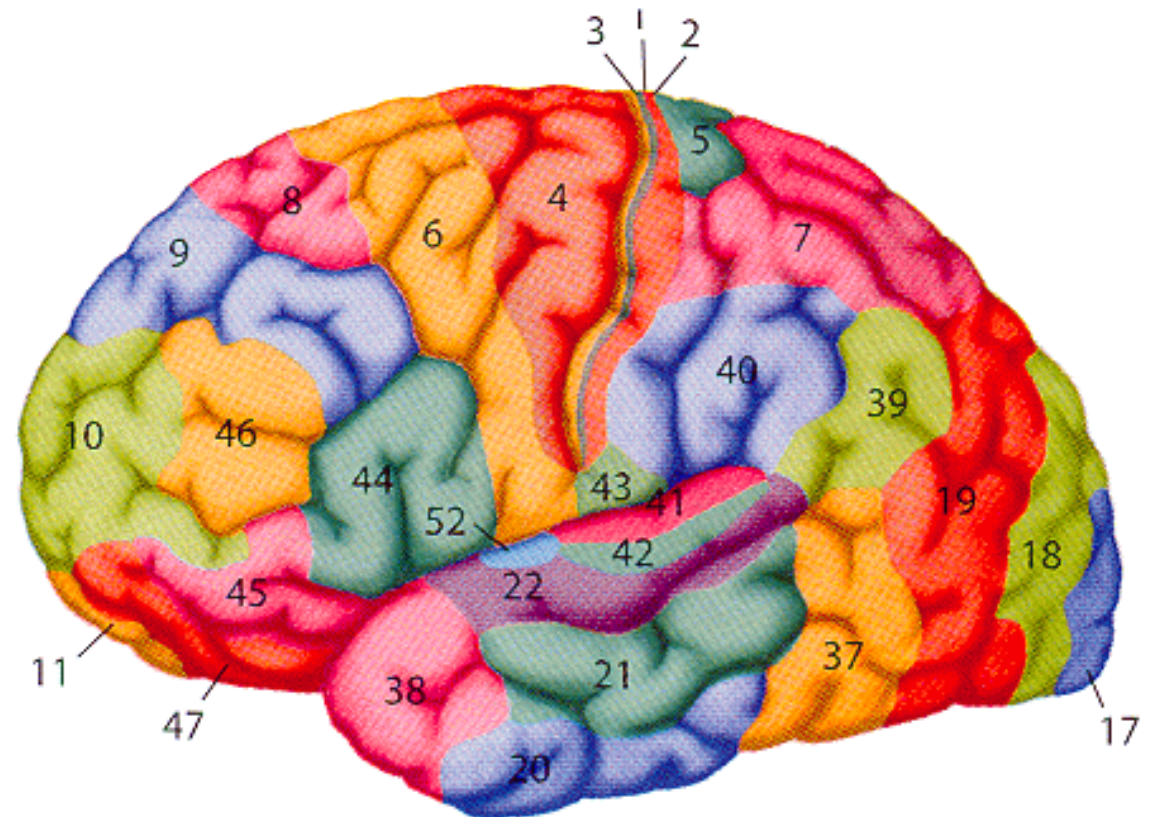


German neurologist **Korbinian Brodmann** spent years slicing and staining different areas of human (and monkey) brains.

In 1909 he published a map of his results, dividing the brain into areas that share the same cytoarchitectonics.

Although his results have been refined by more recent studies, we still use the term **Brodman areas** to refer to areas of the brain identified based on cytoarchitectonics.

The hypothesis is that areas with **different forms** perform **different functions**.





The strongest evidence for the functional organization of the brain comes from cases of brain injury or disease

# Phineas Gage (1823-1860)



Phineas Gage was a 24 year old foreman of railroad construction crew in Vermont in the US in 1848.

He was overseeing the detonation of some rock formations that were in the way of track construction, when an accidental explosion sent his tamping iron **straight through his head** (literally 25 feet in the air).

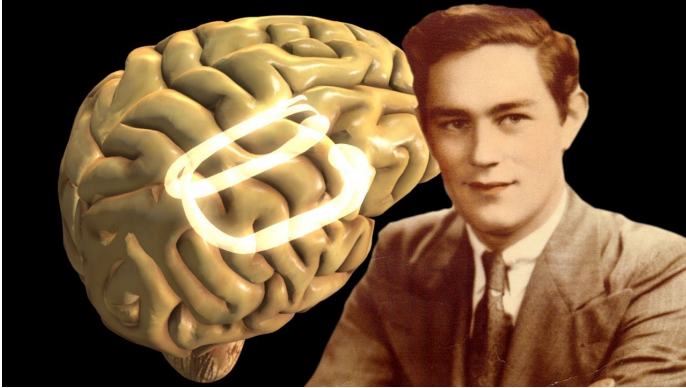
What made this incident famous is that he **survived for another 12 years**, and lived a relatively normal life.

The iron damaged the left frontal portion of his brain.

Gage suffered a fairly substantial personality shift, turning from a mature, responsible foreman into an impulsive, possibly rude and aggressive man.

Gage was still able to perform relatively complex tasks (speaking, living, driving a coach on a mountain pass!).

# Henry Molaison (HM; 1926-2008)



Born (and lived) in Connecticut in the US.

A bicycle accident at age 7 led to an injury that caused seizures. The seizures became life threatening as he got older.

In 1953, at age 27, a neurosurgeon at Hartford Hospital removed parts of the medial temporal lobe in both hemispheres, ending the seizures. But also removing both hippocampi (singular: hippocampus).

This caused **anterograde amnesia**. He remembered everything from before the surgery, but **he could not form new long-term episodic memories**. (HM is the inspiration for the movie trope - like "50 First Dates")

HM volunteered to work with researchers for the rest of his life. He taught us quite a bit about the biological bases of memory. He taught us that long-term episodic memory is different from short-term memory (which was not affected) and procedural memory for skills (which was also not affected).

# Vision: Prosopagnosia

**Prosopagnosia** is the **inability to recognize faces**. It can either be the result of injury to the brain or a congenital issue.

## **Symptoms**

1. Failure to recognize a close friend or family member.
2. Focusing on hairstyles or clothes instead of faces.
3. Confusing characters in movies and on TV shows.
4. Failure to recognize yourself in older or other people's photographs.
5. Difficulty recognizing people out of the typical context (e.g., a coworker on the street) or after a haircut or wardrobe change.



The **fusiform face area** appears to be dedicated to face recognition. There are active questions about whether it only does faces or whether it can support any sort of expert visual discrimination.

# Imagination: Aphantasia

**Aphantasia** is the inability to imagine sensory experiences, such as visualizing a scene, hearing music in the mind, or imagining a specific smell.

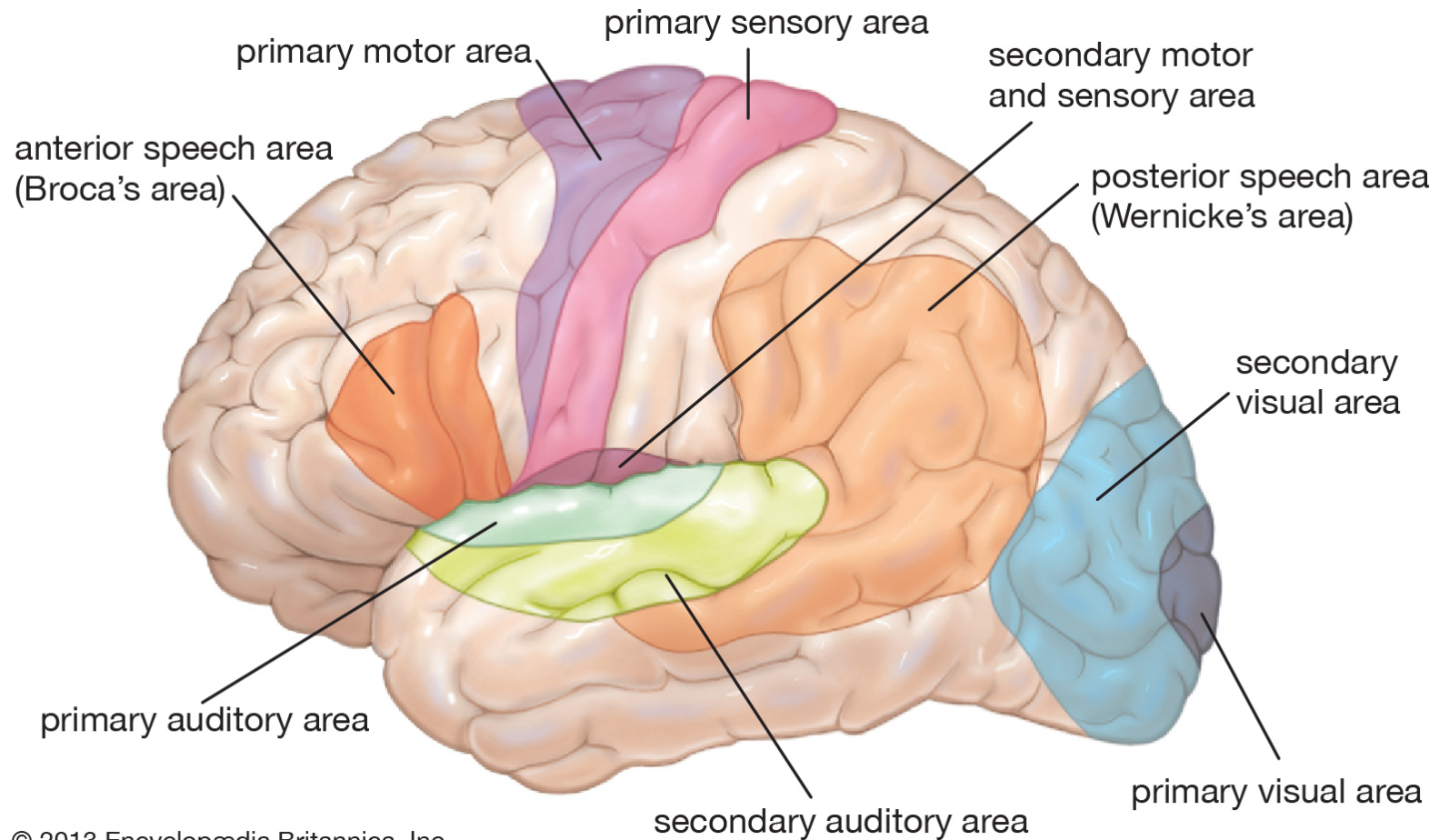
Aphantasia has not been extensively studied. It was first named in 2015 (though it may have been noticed earlier), and didn't gain widespread attention in the media until one of the co-creators of Firefox (Blake Ross) wrote an essay about how he suffers from aphantasia and never realized that he was different from the rest of the world.

## **A quick aphantasia test:**

1. Imagine an image of a friend that you see frequently. How clearly can you see their face, head, and body?
2. Imagine an image of a rising sun. How clearly do you see the colors of the sun? The colors of the clouds in the sky?
3. Imagine a lightning storm erupting in the sky. How well can you see the clouds and lightning?
4. Imagine a rainbow appearing after the storm? How vivid is the picture of the rainbow in the sky?



# Sensory and Perceptual cortices



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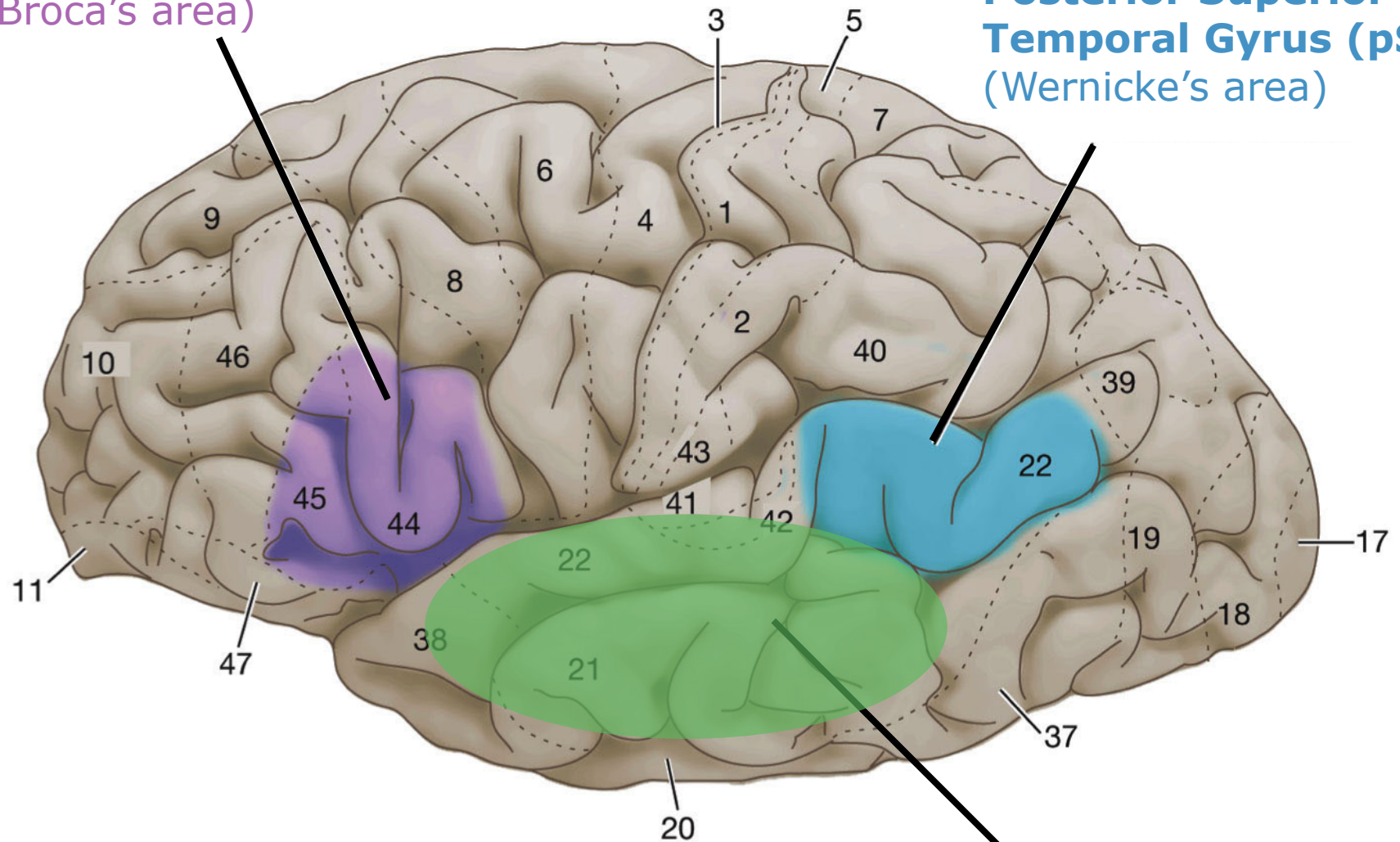
Sensation and perception (like hearing and vision and touch) offer some of the strongest evidence for functional specialization in the brain. We have identified areas of the brain that seem to give rise to them.

But what about language?

# There is no single "language area"

**Left Inferior Frontal Gyrus (LIFG)**  
(Broca's area)

**Posterior Superior Temporal Gyrus (pSTG)**  
(Wernicke's area)

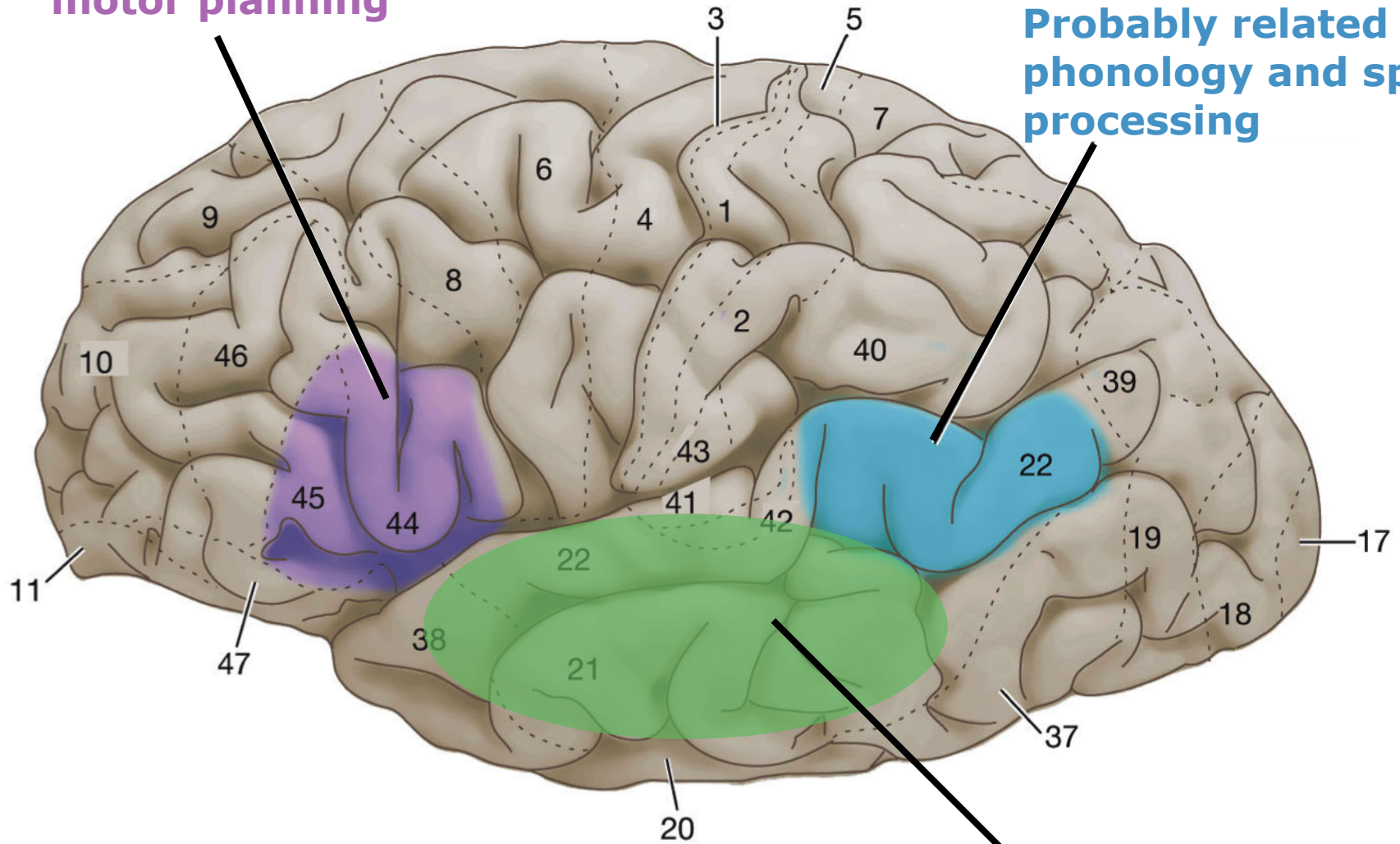


**Temporal Lobe**  
(lots of portions of it)

# There is no single "language area"

Probably related to syntax and motor planning

Probably related to phonology and speech processing



Probably related to Morphology/Lexical Access

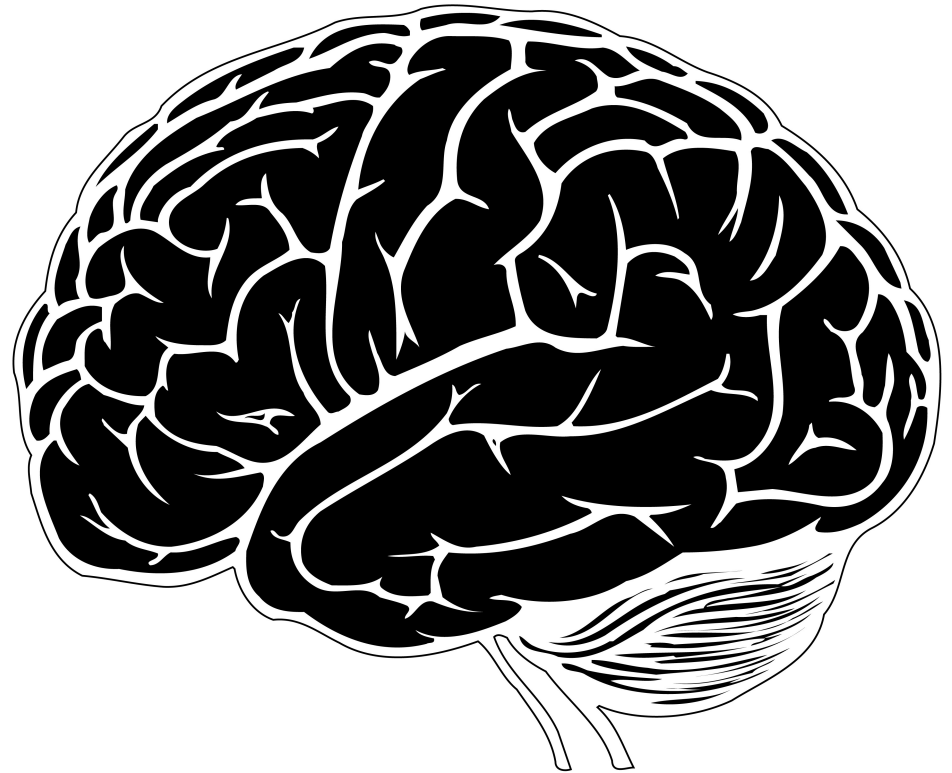


# Lots of disorders affect language, but only **aphasia** is specifically about language!

**Dysarthria** is muscle weakness of the vocal tract. It is caused by damage to the nervous system (including the motor cortex) from stroke, Parkinson's, ALS, etc.

**Apraxia** is paralysis or other malfunction of the vocal tract. It is caused by damage to the motor cortex that disrupts the motor signals to the vocal tract (typically stroke).

**Dementia** is a general term for impairments in mental ability. Dementias can affect language ability if they affect the motor cortex (speech), or **memory centers relevant to words/concepts**.

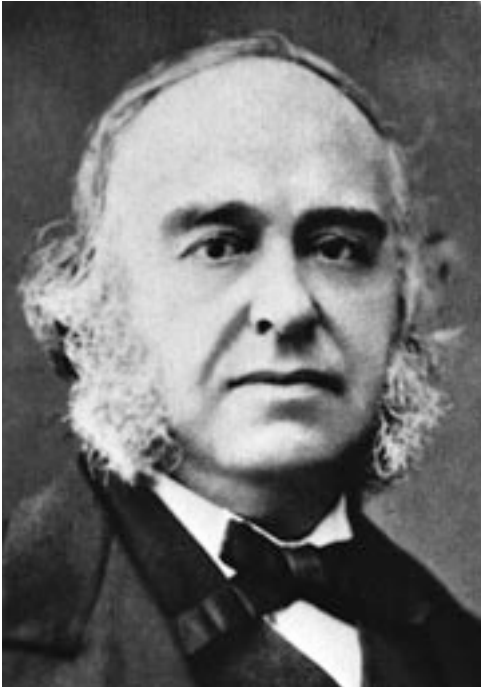


**Aphasia** is a language impairment that arises from damage to an area of the brain that is relevant for language processing beyond the motor cortex or memory systems (typically stroke). As such, aphasias are the most relevant disorder for the study of language in the brain (we won't look at dysarthria or apraxia in this class, and only one dementia).



The classical model of aphasia: Broca,  
Wernicke, and conduction aphasia

# The first aphasia: Broca's Aphasia



Pierre Paul Broca

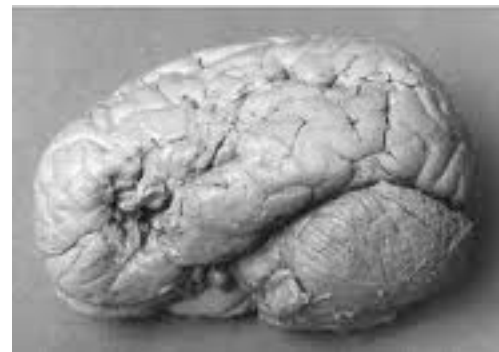
Pierre Paul Broca was a French physician in the 19th century who found himself embroiled in early debates about whether brain function is localized or not.

He encountered two patients that led him to believe that brain function is localized.

The first, Leborgne, was a patient who could only produce one word "Tan" (you will often find him referred to as "Tan" in the literature.)

The second, Lelong, could only produce five words: yes, no, three, always, and 'Lelo' (in French).

At autopsy, Broca found that both patients had lesions in approximately the same location of LiFG. An area we now call **Broca's area**.



Leborgne's brain



Lelong's brain

# Carl McIntyre - Aphasia the movie



Carl suffered a stroke that left him with Broca's aphasia. He is an actor, so he created a movie about it. He also travels around the US giving talks about it.

# Symptoms of Broca's aphasia

Damage to LiFG, sometimes called Broca's area, is classically associated with a disorder known as **Broca's Aphasia**.

Symptoms of Broca's Aphasia include:

1. Patients with Broca's aphasia have **difficulty producing fluent speech**. This was classically interpreted as an issue with "motor" aspects of speech.
2. Their speech often **lacks grammatical function words** like auxiliary verbs and prepositions.
3. Patients with Broca's aphasia have **difficulty comprehending sentences with complex syntax**, especially when the order is not subject-verb-object:

relative clauses:     The reporter that the senator called \_\_\_ wrote the story.

Strokes primarily affect older people, but it can happen to the young. It is worth learning the symptoms of a stroke so you can recognize it quickly and find medical attention for someone:

[www.youtube.com/  
watch?v=q5XHH1XfAbM](http://www.youtube.com/watch?v=q5XHH1XfAbM)

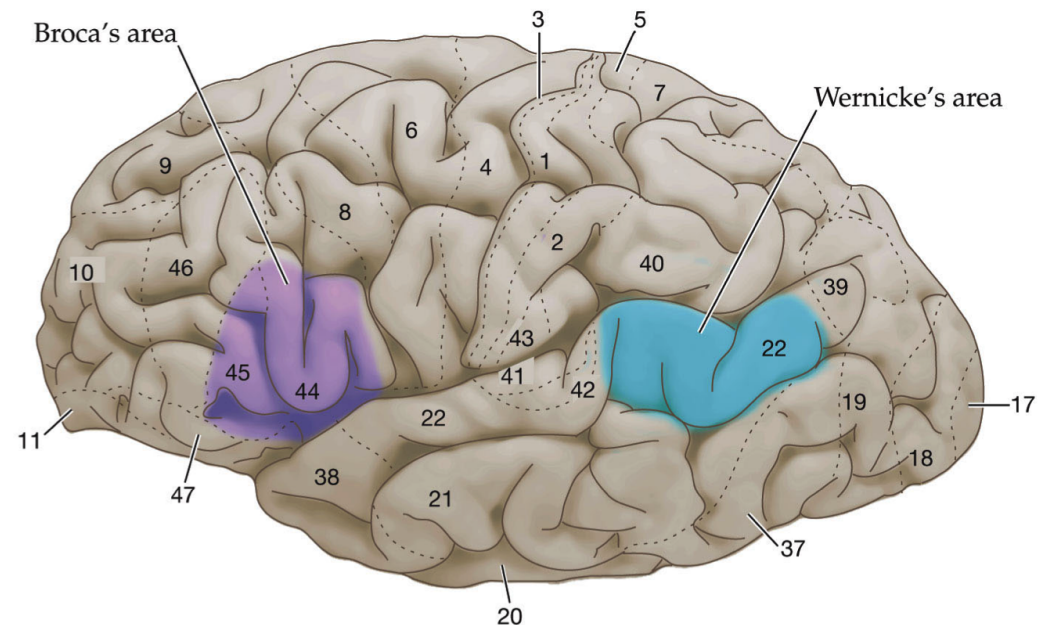
# The second aphasia: Wernicke's aphasia



Carl Wernicke was a German physician in the 19th century.

He was keenly interested in Broca's work on aphasia, and set out to perform his own studies on patients suffering from language disorders.

He found that not every language disorder resulted from a lesion to **Broca's area**. In particular, he found several patients with damage to the **posterior superior temporal gyrus (pSTG)**. This region is now known as **Wernicke's area**.





# Symptoms of Wernicke's aphasia

Wernicke's aphasia was first identified by Carl Wernicke in the late 1800s (~1874):

[https://www.youtube.com/watch?v=3oef68YabD0&ab\\_channel=tactustherapy](https://www.youtube.com/watch?v=3oef68YabD0&ab_channel=tactustherapy)

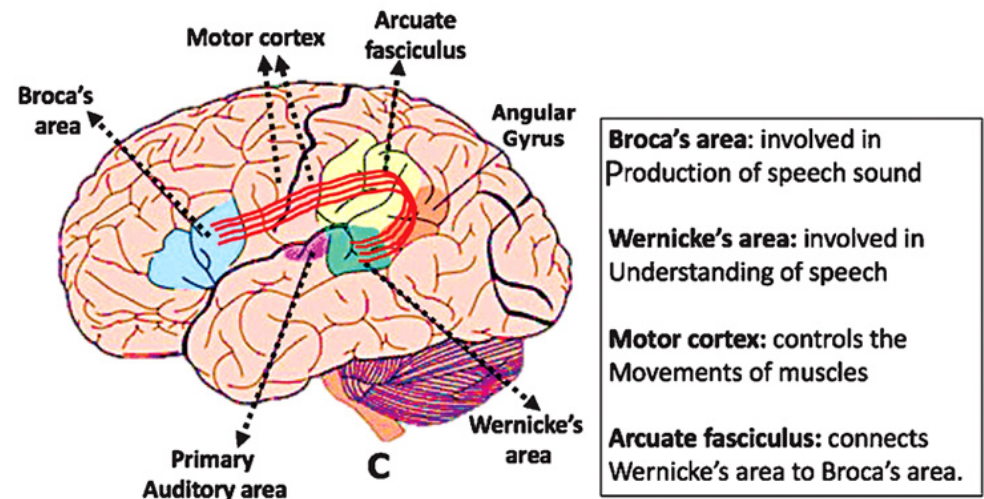
Wernicke's aphasia is sometimes called **fluent aphasia** because patients appear to produce fully grammatical sentences. However, the productions tend not to be composed of real words (gibberish). Even when real words are used, they tend not to form sentences that make any sense (suggesting that they aren't the words that are intended).

It is also sometimes called **receptive aphasia** because patients suffer from catastrophic comprehension difficulties. They simply cannot understand what people are saying to them.

This suggests an impairment of **speech processing**, which would then affect both comprehension and production of speech.

# The “classical model” of aphasia

The classical model of aphasia is that Broca’s area is related to motor planning for speech (hence the production problems), while Wernicke’s area is related to auditory processing including the phonology of words (hence the comprehension difficulties, and the non-sensical speech).



Parts of the Brain that Control Speech

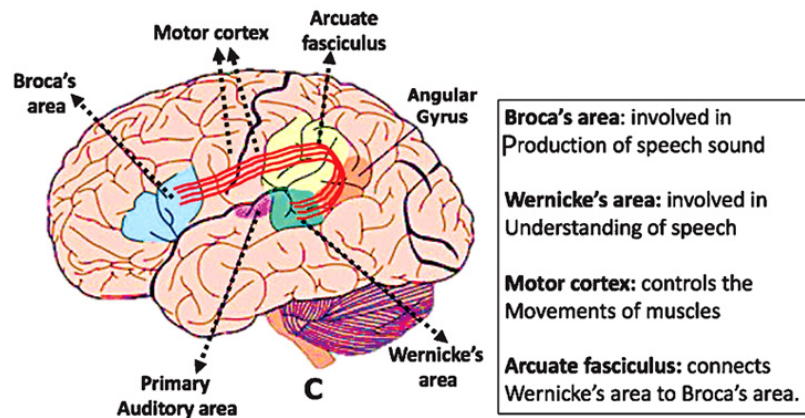
The idea is that these two areas must work in concert for most speech tasks.

These two areas are connected by a bundle of axons (white matter) called the **arcuate fasciculus**, which seems to fit with the idea that Broca’s area and Wernicke’s area must work together for speech. You will often find diagrams like the one above to show this “classical model”.

The classical model is very good for patient care. It captures the general symptoms of the two most common aphasias, and it fits generally with lesion sites of strokes. But **very few neuroscientists think it is accurate.**

# Conduction Aphasia - evidence for, and ultimately against, the classical model

Conduction aphasia is characterized as relatively intact speech comprehension, but difficulty with repeating what is heard, and some errors in speech production. Here is a video showing some errors in speech production:



**Parts of the Brain that Control Speech**

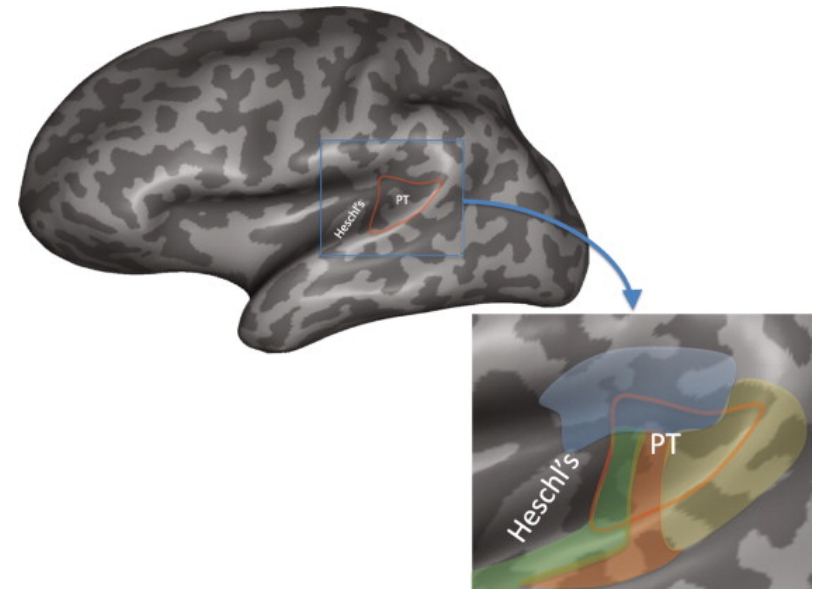


[https://www.youtube.com/watch?v=G94TvTvjeeU&ab\\_channel=InstantNeuro](https://www.youtube.com/watch?v=G94TvTvjeeU&ab_channel=InstantNeuro)

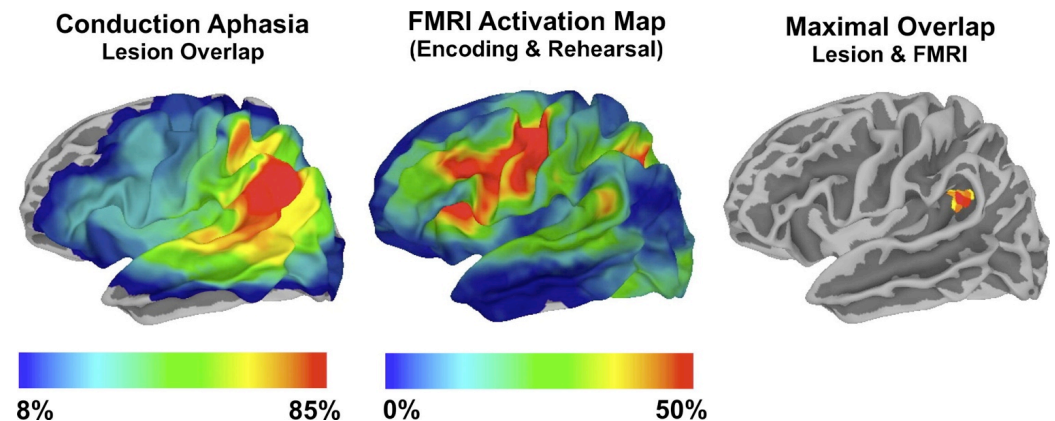
The idea is that conduction aphasia is caused by a lesion impacting the arcuate fasciculus, such that comprehension (from auditory cortex to Wernicke's area) is spared, but planning/production (from Wernicke's to Broca's) is damaged.

# The problem is that conduction aphasia tends to arise from damage to SPT/Wernicke's area

There have been a number of studies in the last two decades showing that conduction aphasia tends to be driven by damage to the Superior Planum Temporale (SPT), an area next to the auditory cortex and part of Wernicke's area, rather than damage to the arcuate fasciculus.



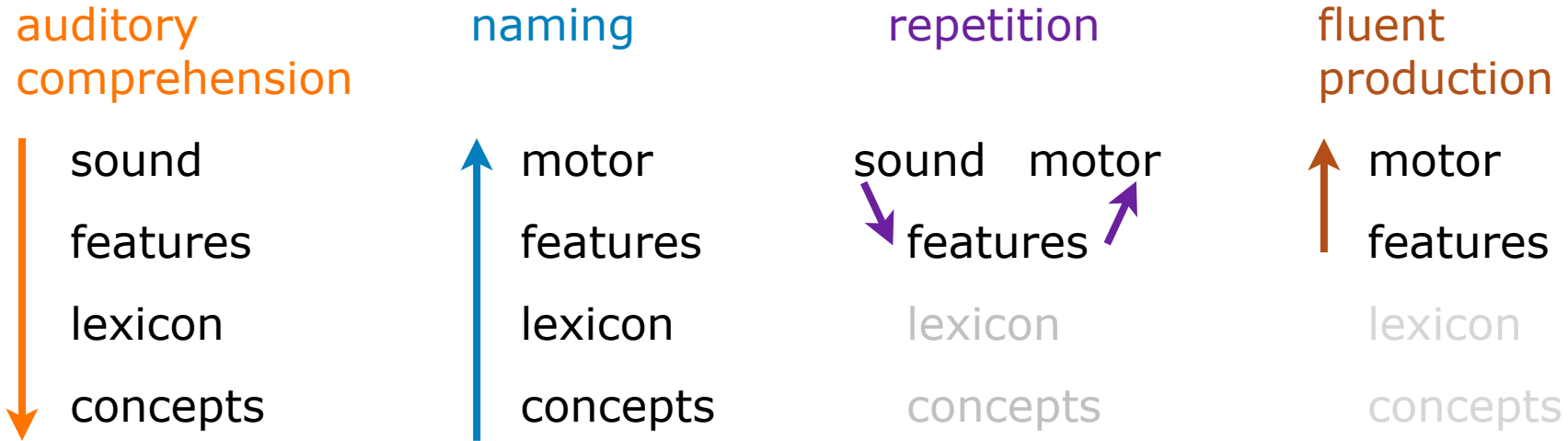
For example, Buchsbaum et al. 2011 compared lesion overlap for 14 patients with conduction aphasia with activation overlap in participants without aphasia during repetition tasks. They found maximal overlap for the lesions at SPT, and overlap between the two groups at SPT.



Going beyond the classical model -  
more aphasia types, and a more  
articulated functional theory

# Four common tasks for aphasia studies

First, aphasias are defined based on symptoms, and diagnosed with various tests.



**Auditory comprehension:** patients must perform some action.

**Naming:** patients are shown images of objects, and must name them.

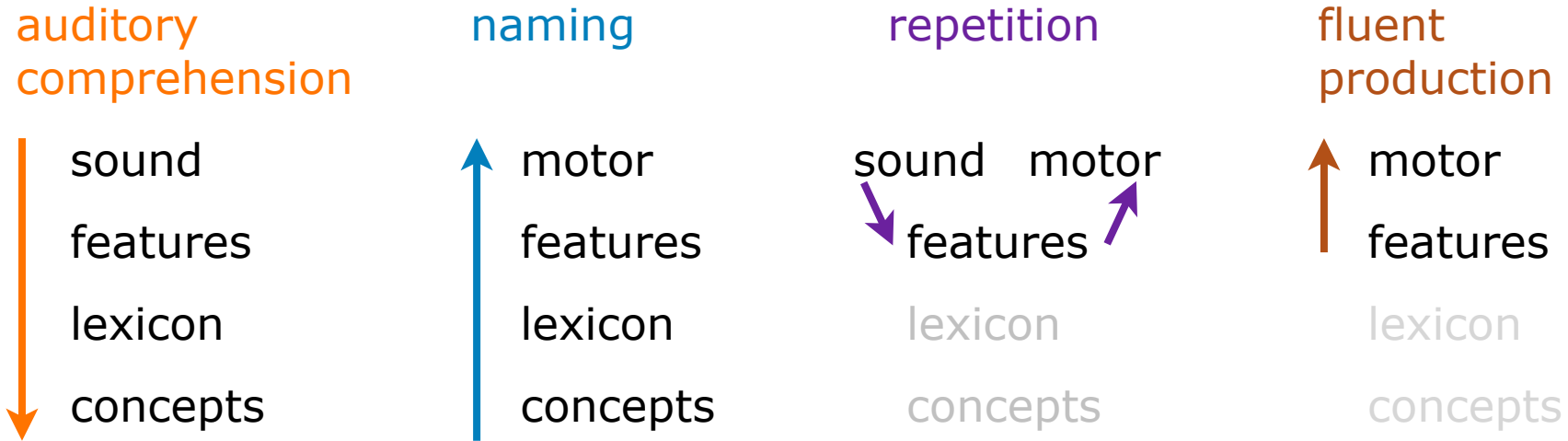
**Repetition:** patients listen to a word or sentence, and must repeat it.

**Fluent production:** doctors simply listen to the patient's spontaneous speech.



# Aphasias are defined by symptoms, not lesions

We can classify aphasias based on patients' performance in these tasks.



aphasia	auditory	naming	repetition	fluent
Broca's	✓	✓	✓	✗
Wernicke's	✗	✗	✗	✓
Conduction	✓	%	✗	✓
TSA	✗	%	✓	✓
Anomia	✓	✗	✓	✓
Semantic Dem.	✓/✗	✗	✓	✓

<b>aphasia</b>	<b>auditory</b>	<b>naming</b>	<b>repetition</b>	<b>fluent</b>
Broca's	✓	✓	✓	✗
Wernicke's	✗	✗	✗	✓
Conduction	✓	%	✗	✓
TSA	✗	%	✓	✓
Anomia	✓	✗	✓	✓
(Semantic Dem.)	✓/✗	✗	✓	✓

**Transcortical Sensory Aphasia** is a rare aphasia that leads to catastrophic loss of auditory comprehension and fluent nonsensical speech, but preserves repetition and some amount of naming.

[www.youtube.com/watch?v=bpeZ4xm62DM](http://www.youtube.com/watch?v=bpeZ4xm62DM)

**Anomia** is a common aphasia that only impacts object naming, and only for selected objects. The word anomia means "no names".

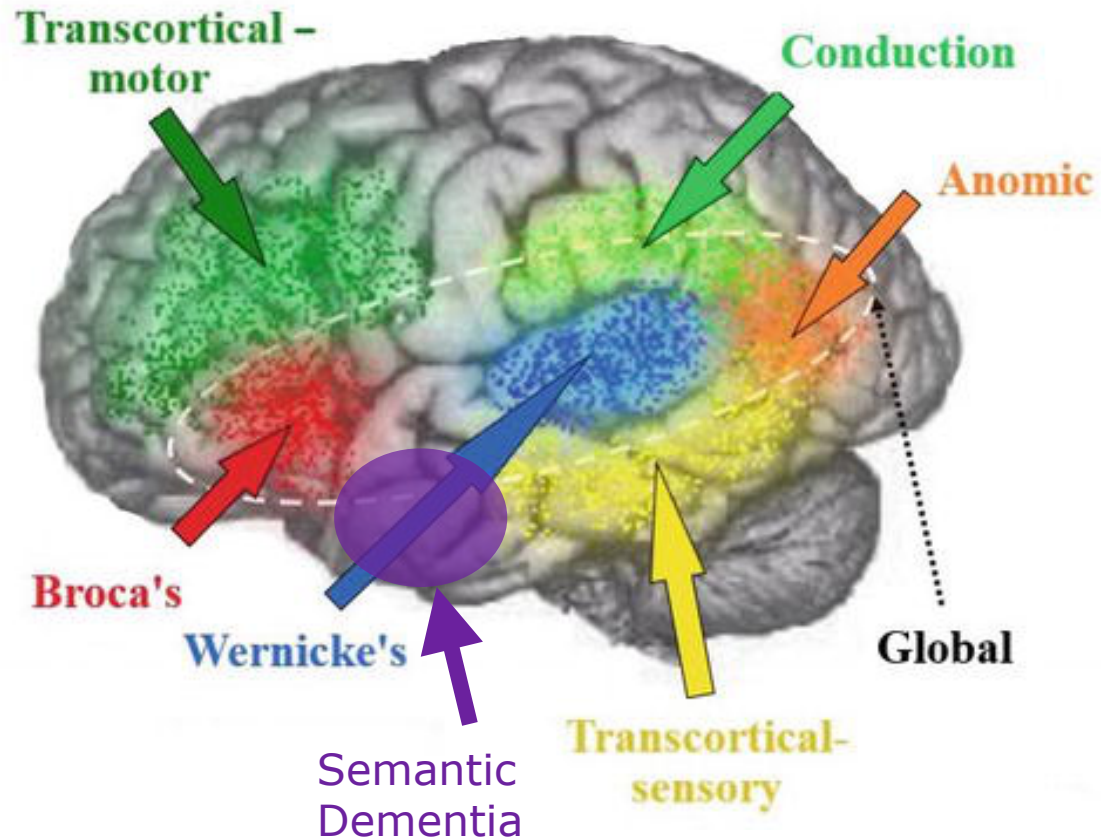
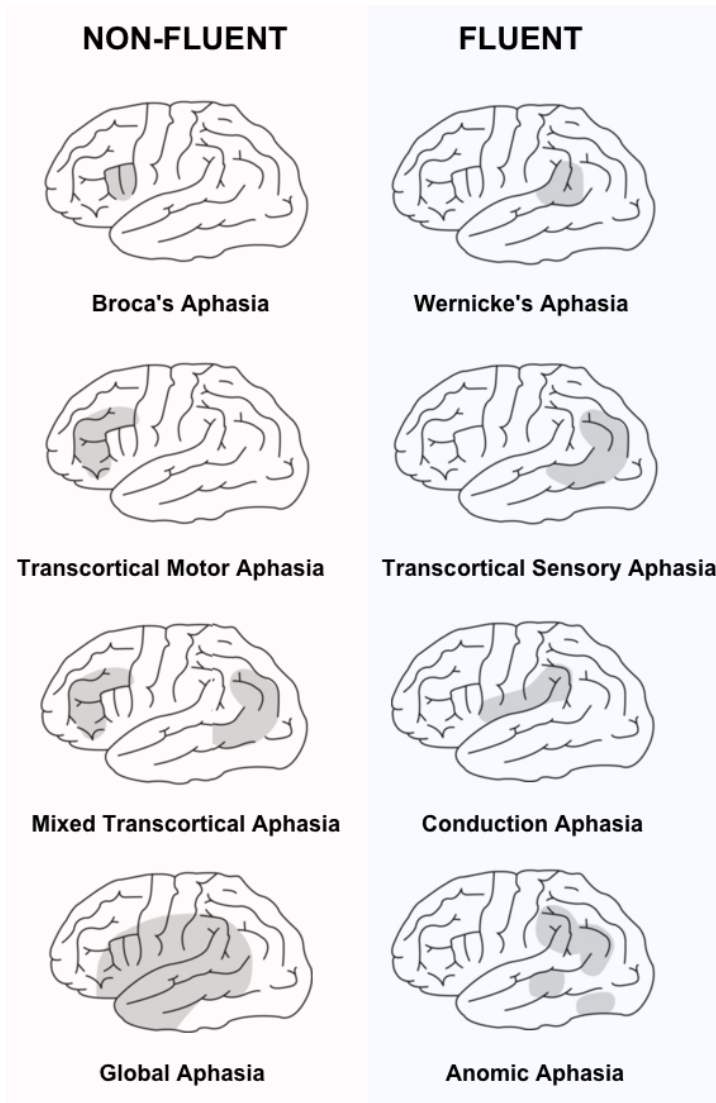
[www.youtube.com/watch?v=LWAUmsgk8eg](http://www.youtube.com/watch?v=LWAUmsgk8eg)

**Semantic Dementia** is not an aphasia. It is a rare progressive dementia that, in the early stages, leads to the loss of certain concepts. The exact cause of SD is not clear, but has been tied to some viral infections in the brain.

[www.youtube.com/watch?v=fkKrsbwQvrE](http://www.youtube.com/watch?v=fkKrsbwQvrE)

# Lesion locations in aphasia

Lesion studies are not necessarily part of the diagnosis criteria for doctors. But you will find diagrams like this that link specific aphasia types to lesion sites:



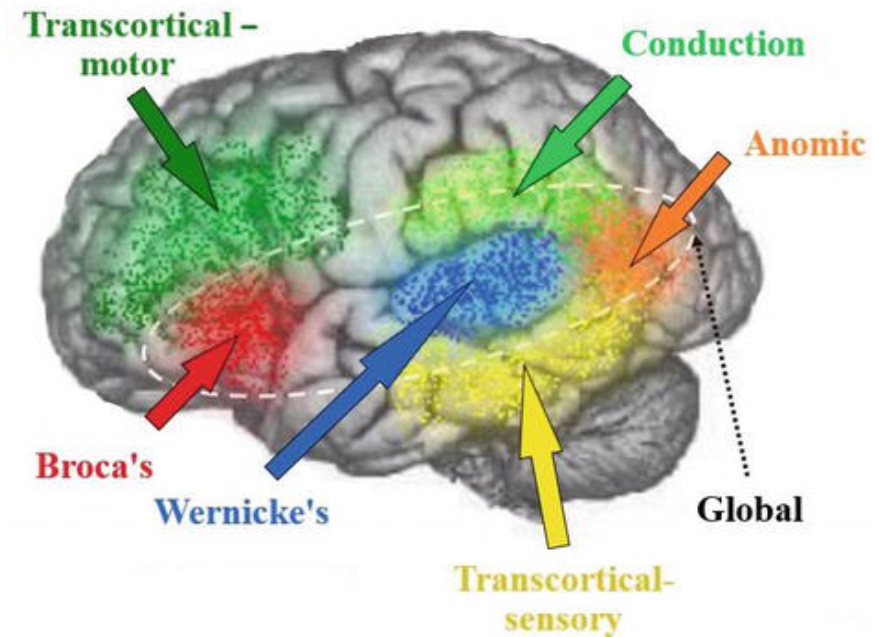
# Aphasia as evidence for neuroscience

As neuroscientists, we would like to use information from aphasia studies to build a theory of the cortical organization language. But the specific locations of lesions that cause different aphasias is an active area of investigation.

There are multiple challenges. The first is that strokes tend to impact fairly large sections of the brain, likely spanning multiple areas, each with several (dozens!) of functions. The deficits we see are never as clear as a textbook presentation.

The second is that there could be a decent amount of variability between patients in their functional organization to begin with. We do not have information about the organization before the stroke.

The third is that there is some amount of reorganization in the brain after a stroke. This can mask functional deficits that might be expected from the lesion itself.



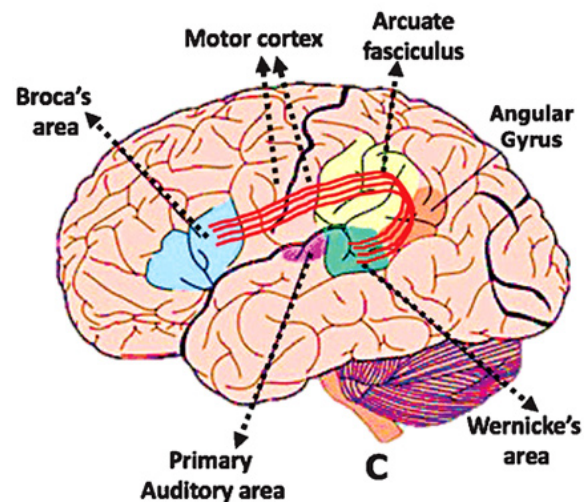




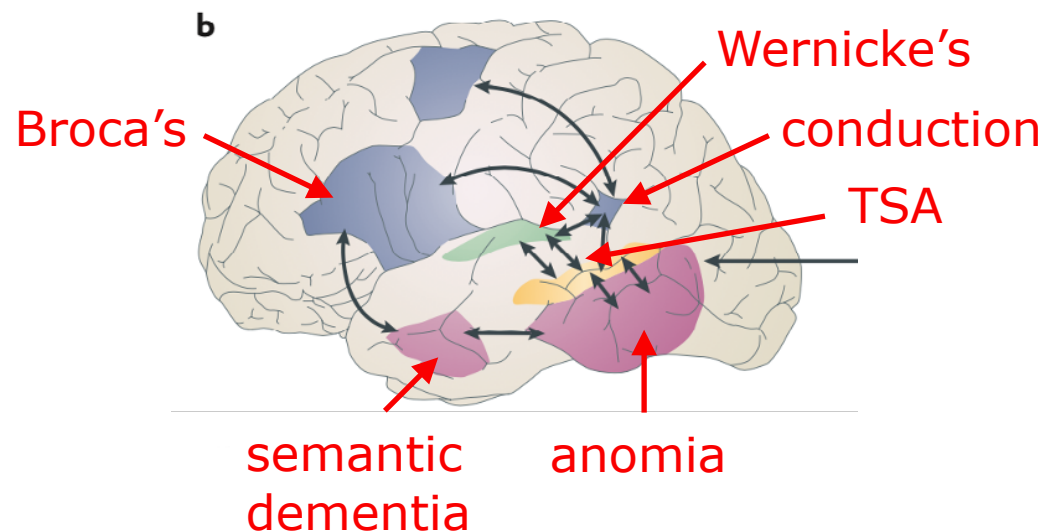
# Comparing models

You can see that the dual stream model is an elaboration of the classical model, incorporating new evidence from aphasia (and fMRI) studies:

## Classical model



## Dual stream model



But there is a ton of work left to do further identifying the actual cognitive functions in language, the localization of functions (e.g., lesion studies), and the temporal relationships among the functions and areas.