Introduction to Aphasia Classifications (and their limitations)

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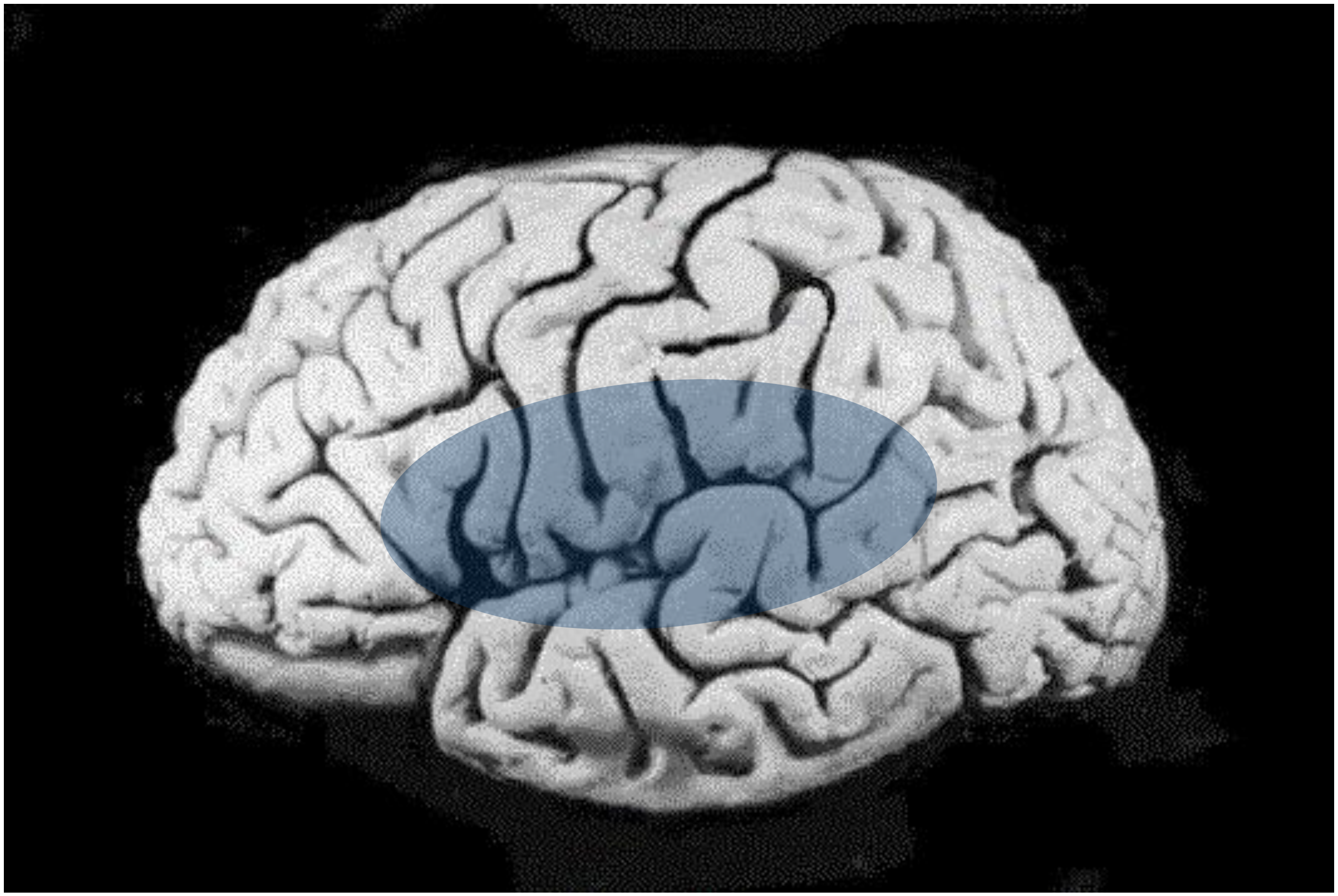
Personal background:

- Aphasia researcher and certified speech-language pathologist.
- **Training:**
  - BA and PhD focused on psycholinguistics and experimental psych
  - Clinical master's in SLP.
- **Clinical Experience:**
  - Aphasia and cog rehab experience at Mass General and VA Pittsburgh.
- **Teaching:**
  - Aphasia, cognitive rehab, and counseling content at Pitt CSD grad programs.
- **Research agenda:**
  - Adaptive computer-based aphasia treatments.
  - Integrating counseling and language treatments to improve communication participation and psychosocial outcomes.
  - Working with aphasia community and game designers to create therapeutic aphasia games

Outline:

1. Intro to Aphasia
2. Classical aphasia categories as a guide to core processes in aphasia
3. Current approaches to brain-behavior relationships in aphasia
4. Neurobiological predictors of aphasia treatment response.

Aphasia is a language disorder caused by brain damage, usually to the left hemisphere.



Aphasia is a language disorder caused by brain damage, usually to the left hemisphere.

Talking

Listening Comprehension

Writing

Reading

Aphasia Incidence, Prevalence, and Impact:

- Incidence: ≈ 250,000 new cases per year (National Aphasia Organization).
- Prevalence: >2,500,000 people in North America (Simmons-Mackie, 2018)

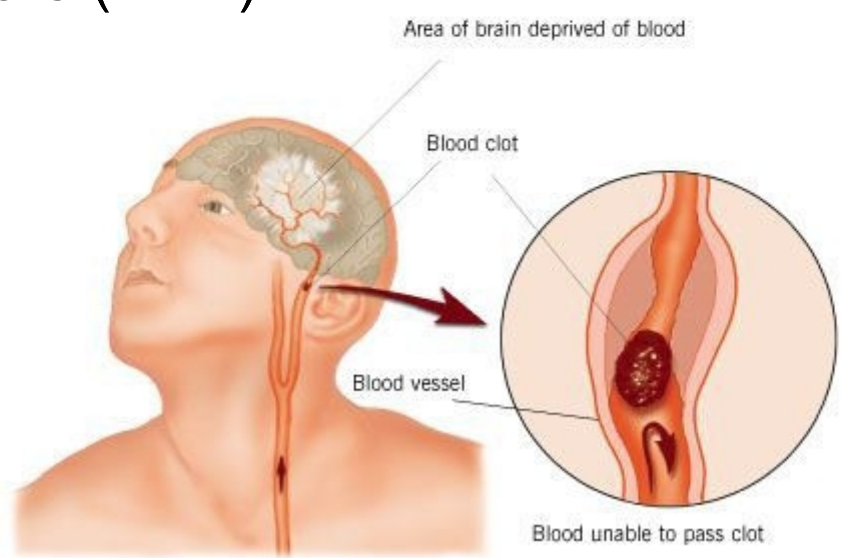
- **More common than Parkinson's disease, ALS, and cerebral palsy, but with low public awareness.**
  - 2020 National Aphasia Association survey: of 1,001 US respondents ages 25 and older, **only 7% of respondents** had heard of aphasia and could identify it as a language disorder.

- Negative effects on quality of life can be **greater** than those of cancer, Alzheimer's, or Amyotrophic Lateral Sclerosis. (Lam & Wodchis, 2010)

- Aphasia leads to high levels of **social isolation, depression, anxiety, psychological distress**. (Simmons-Mackie, 2018)
  - Roughly twice the rates of stroke survivors without aphasia

Etiology:

- Stroke ≈ 85%
  - 25-40% of stroke survivors have persisting aphasia (Watila & Balarbe, 2015)
- Other ≈ 15%
  - Traumatic Brain Injury (TBI)
  - Brain tumor
  - Infection
  - Progressive neurological disorders (PPA)



Definitions of Aphasia

aphasia.org

- *"Aphasia is an acquired communication disorder that impairs a person's ability to process language, but does not affect intelligence.*
- *Aphasia impairs the ability to speak and understand others, and most people with aphasia experience difficulty reading and writing.*
- *The diagnosis of aphasia does NOT imply a person has a mental illness or impairment in intelligence."*

Definitions of Aphasia

Wertz (1985; adapted from Darley, 1969, 1982)

- "… impairment, due to brain damage, of the capacity to interpret and formulate language symbols, a multimodal loss or reduction in decoding conventional meaningful linguistic elements (morphemes and larger syntactic units); disproportionate to impairment of the intellectual functions; not attributable to dementia, sensory loss, or motor dysfunction, manifested in reduced ability of vocabulary, reduced efficiency in applying syntactic rules, reduced auditory retention span, and impaired efficiency in input and output channel selection." (p.2)

Aphasia is *Not*…

<u>Speech & language impairments related to</u>:

- Delirium or psychiatric disorder
- Coma
- Sensory loss
- Motor dysfunction (e.g. dysarthria or apraxia of speech).
- Dementia (e.g., AD, PD, Lewy body dementia)

In aphasia, language impairments are *prominent* compared to any other cognitive deficits that may be present (see Bek, Varley, et al., 2010)

Aphasia Definition Implications

These definitions imply:

- Aphasia does not necessarily involve a *loss* of anything (in particular, intelligence or personhood).
- Linguistic *representations* are still present (distributed across partially-damaged cortex).
- *Rules* that are used to assemble these representations into coherent units of language also still present.
- The problem is one of an impairment of “accessing” the rules and representations or “activating/inhibiting” information in properly timed and regulated ways to build representations.
- Language does not have to be “relearned,” but efficient access, activation, and inhibition of representations and rules must be *restored*. **This is a goal of restorative language therapy**.

Common Aphasic Deficits and Characteristics:

Associated Linguistic Deficits Include:

- Anomia (semantic, phonological, or mixed paraphasias).
  → difficulty w/ word finding (tip of tongue on steroids)
- Auditory comprehension deficits.
- Agrammatism (ommission of function words, difficulty with verbs/ verb morphology).
- Dysgraphia, dyslexia.

  trouble w/ writing

  trouble w/ reading

Common Aphasic Deficits and Characteristics:

Associated Linguistic/Performance Characteristics Include:

- Variable performance.
- Slowed processing times.
- Reliance on situational context to understand/ convey meaning.
- Reduced sentence length/ complexity, perseverative speech.
- Reduced verbal STM and WM span.
- Reduced gestures and co-verbal behaviors.

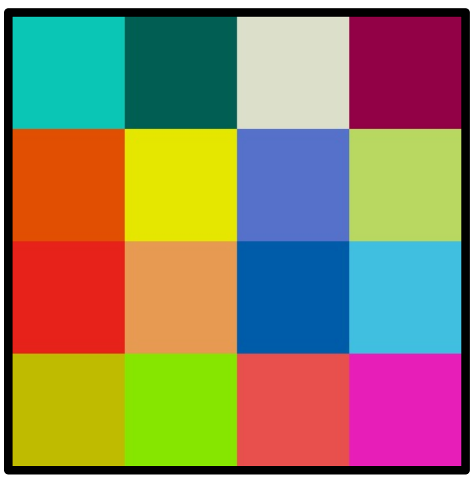
Aphasia Syndromes:

- Over 30 different aphasia classification systems have been proposed to date (McNeil & Copland, 2011).
- Nearly all have proposed anatomic/lesion correlate classification categories.

- Most common systems in use today are:
    1. The Centers and Pathways (Wernicke/Lichtheim Model) aka "the Boston Classification System."
    2. Fluent/Nonfluent Classification.
    3. <u>No Classification</u> – focus characterizing language profile.

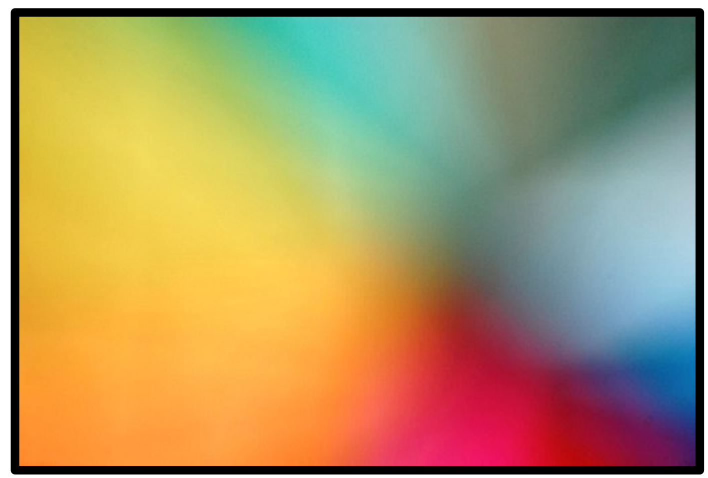
Why study aphasia classification?

- Provides important historical context.
- Still in common use clinically based on common aphasia assessments (e.g., Western Aphasia Battery).
- Learning framework: providing examples of different aphasia classifications provide a structured way to work through and contrast different key features of aphasia.
- Tied to vascular syndromes and useful for neurologists especially in early in acute recovery phase (Hillis, 2007).



similar extremes

Vs.



Discrete boxes of constructs

Real World

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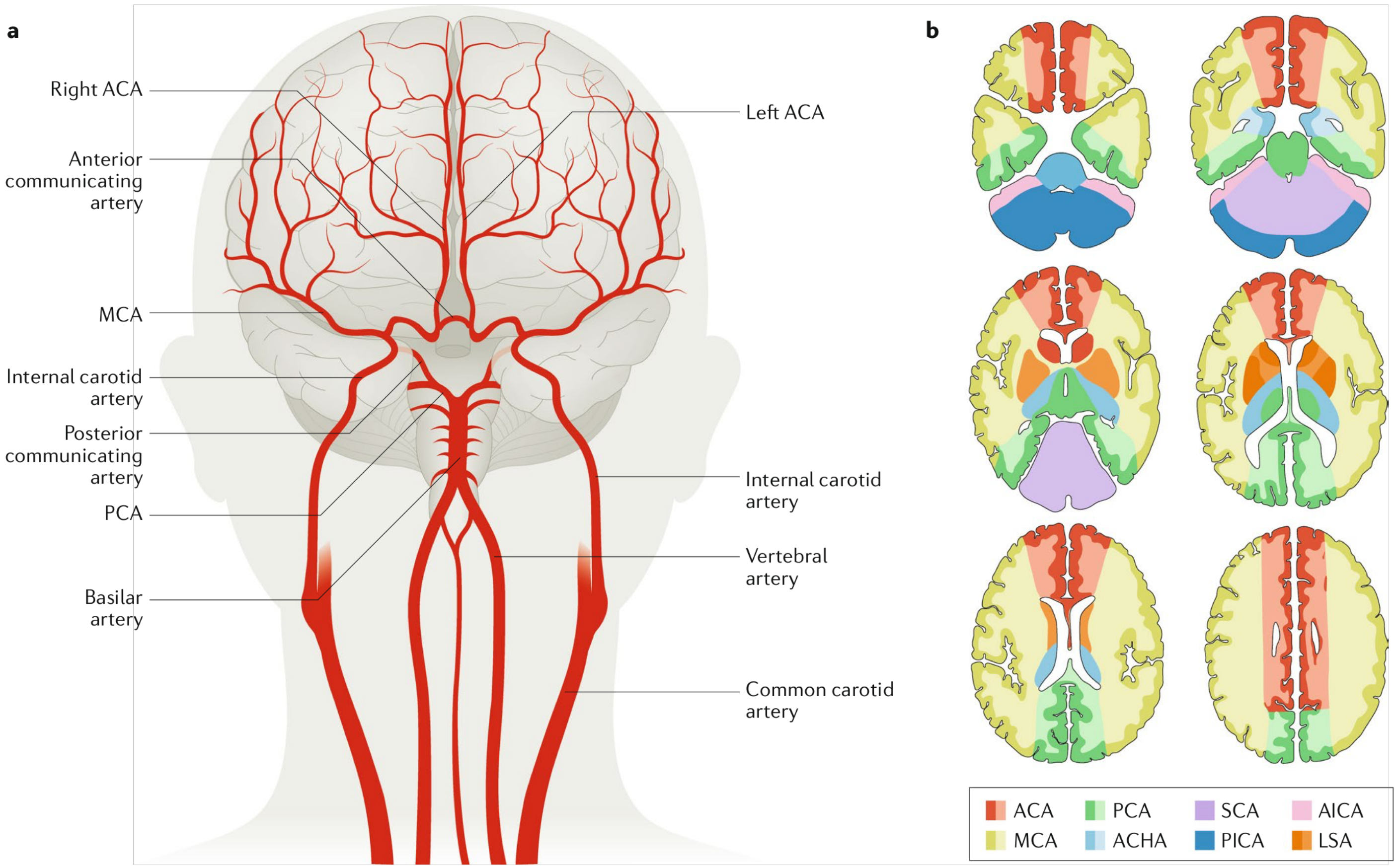


Fig. 3 | **Cerebral vasculature.** The major arteries of the brain (part **a**) and their vascular territories (part **b**). Although simplified here for illustrative purposes, an ischaemic stroke in one of these vessels could cause tissue damage in the regions highlighted. ACA, anterior cerebral artery; ACHA, anterior choroidal artery; AICA, anterior inferior cerebellar artery; LSA, lenticulostriate artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; PICA, posterior inferior cerebellar artery; SCA, superior cerebellar artery. Part **b** adapted from REF.[197], Springer Nature Limited.

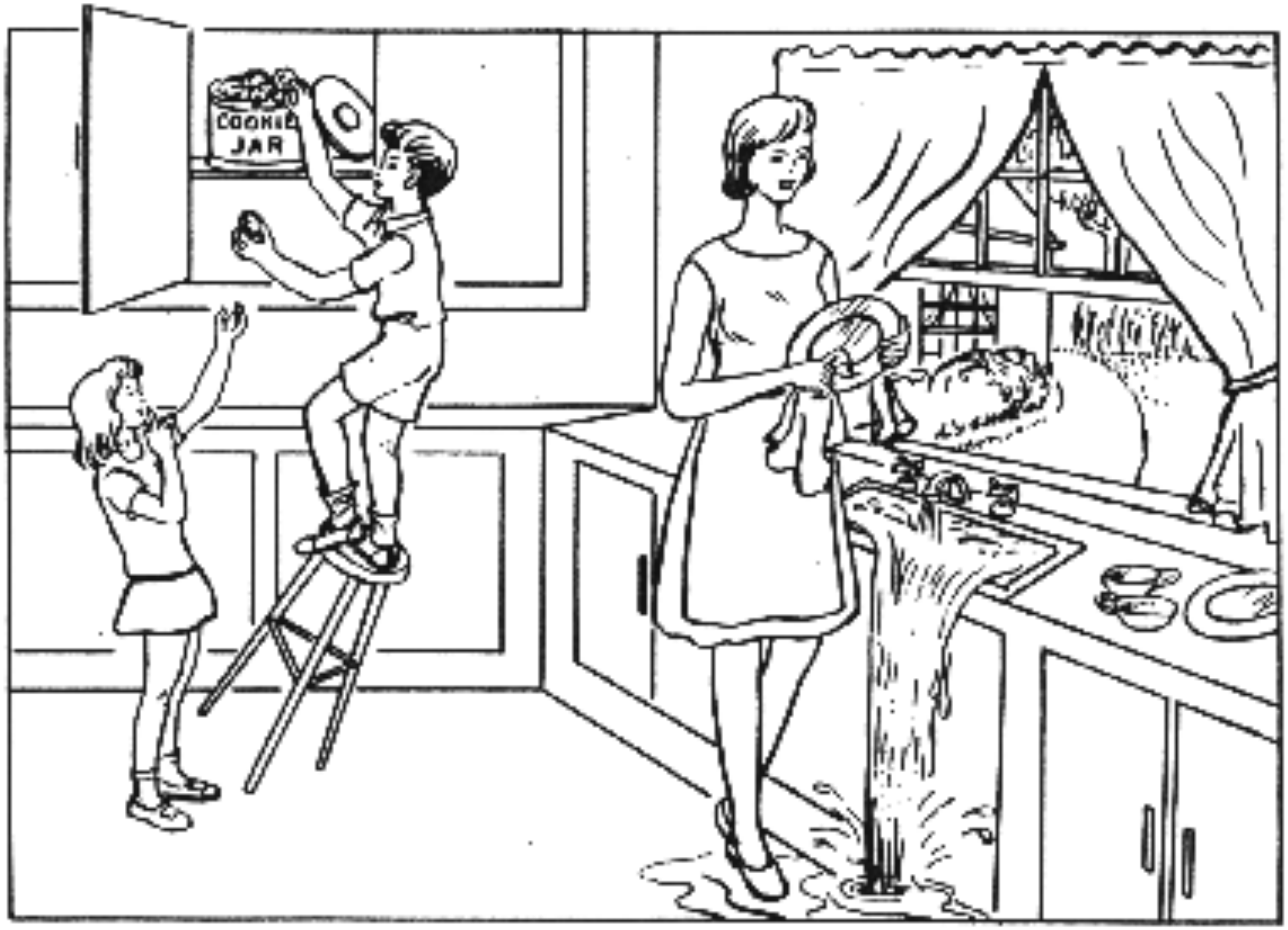
Campbell et al. (2019)

Wernicke-Lichtheim (Boston, Neo-classical, Centers-and-Pathways) Classification System

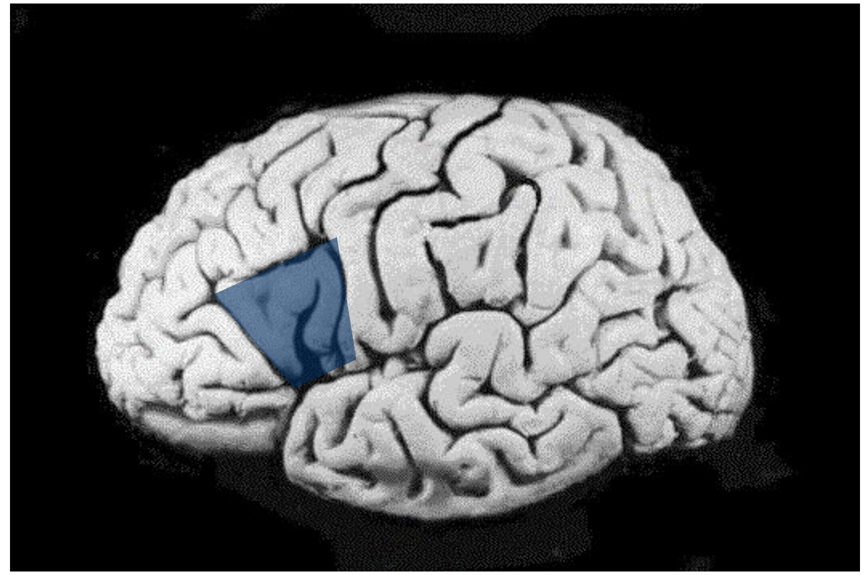
	FLUENCY	AUDITORY COMPREHENSION	REPETITION	APHASIA TYPE
Aphasia	Fluent	Good	Good	Anomic
			Poor	Conduction
		Poor	Good	Transcortical Sensory
			Poor	Wernicke's
	Nonfluent	Good	Good	Transcortical Motor
			Poor	Broca's
		Poor	Good	Mixed Transcortical
			Poor	Global

Cookie Theft
Broca's

nonfluent
good auditory comprehension
poor repetition



Broca's aphasia



- •Nonfluent output, good comprehension, poor repetition.
- •Effortful speech.
- •Distorted articulation.
- •Short phase length.
- •Agrammatic Production.
  - – Omission of function words (e.g., articles, auxilary verbs, prepositions).
  - – Omission of bound morphemes (e.g., -ed, -ing, -s).
  - – High noun/verb ratio.
- •Agrammatic Comprehension.
  - – Relatively poor comprehension of sentences with non-canonical ordering of subject, verb, and object (like passives).

- •Often hemiparetic.

- •*Classic <u>postulated</u> lesion site:*
  - •Posterior 2/3 of the inferior frontal gyrus; pars triangularis and pars opercularis; BA 44, 45.

Broca's aphasia

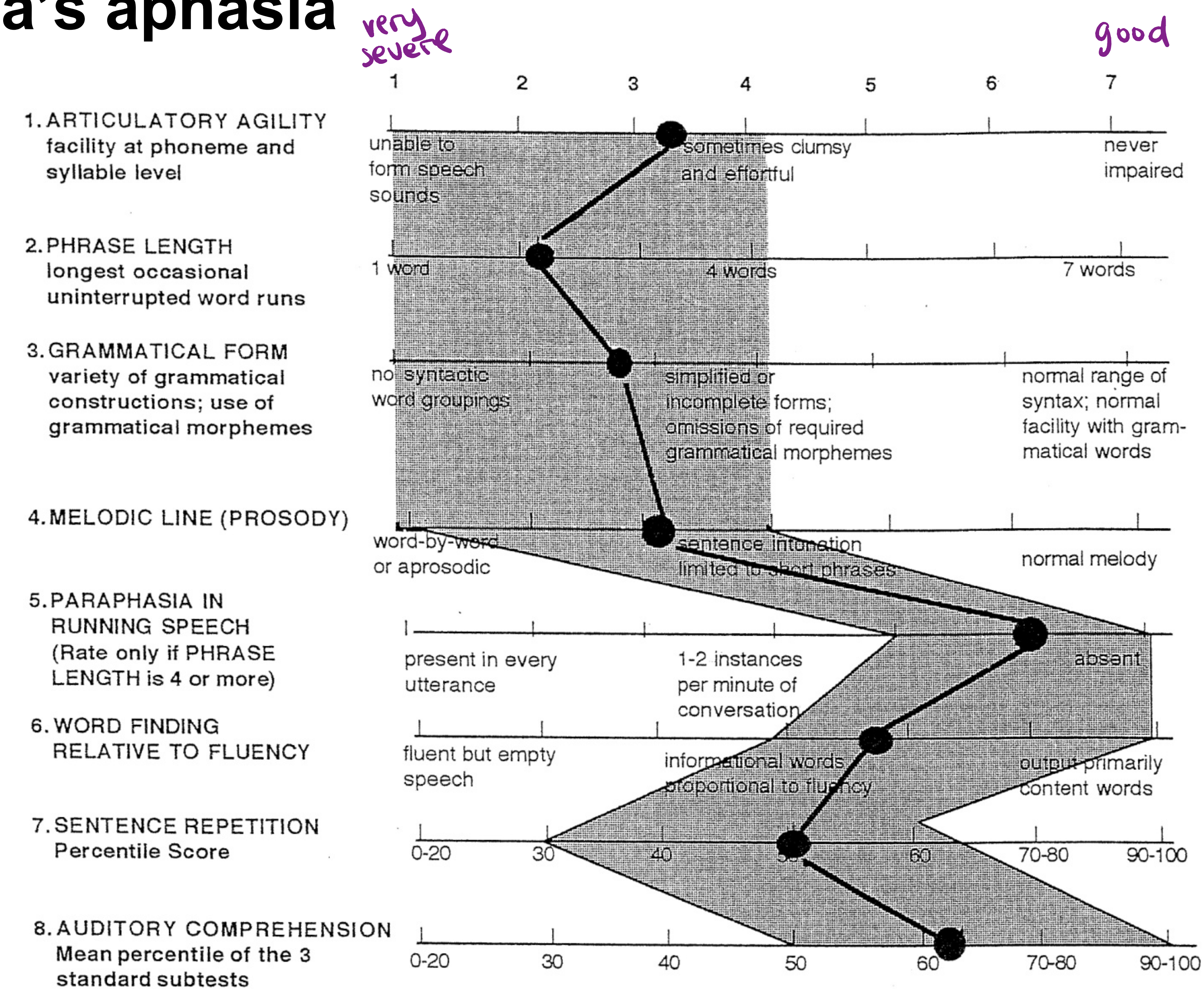
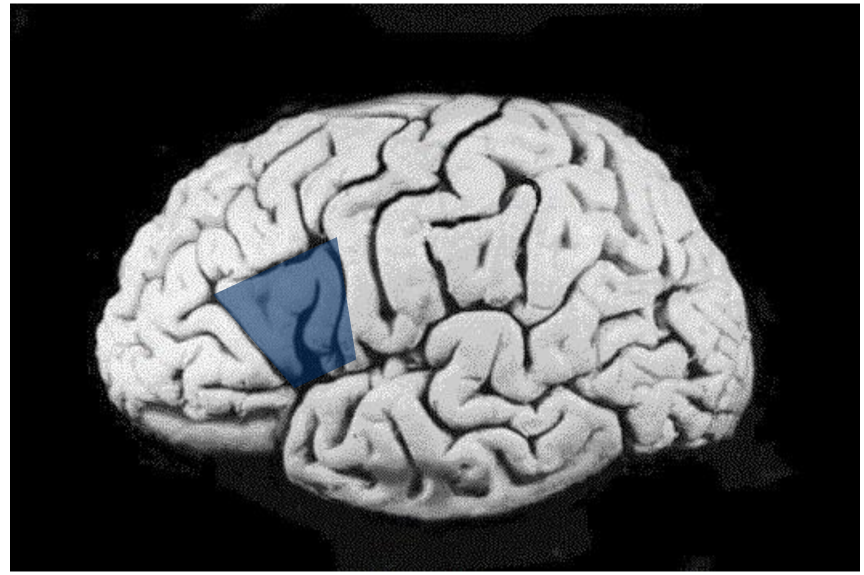


FIGURE 6-4. Broca's Aphasia: Range of Possible Ratings. Rating Scale Profile of Speech Characteristics.

Broca's aphasia



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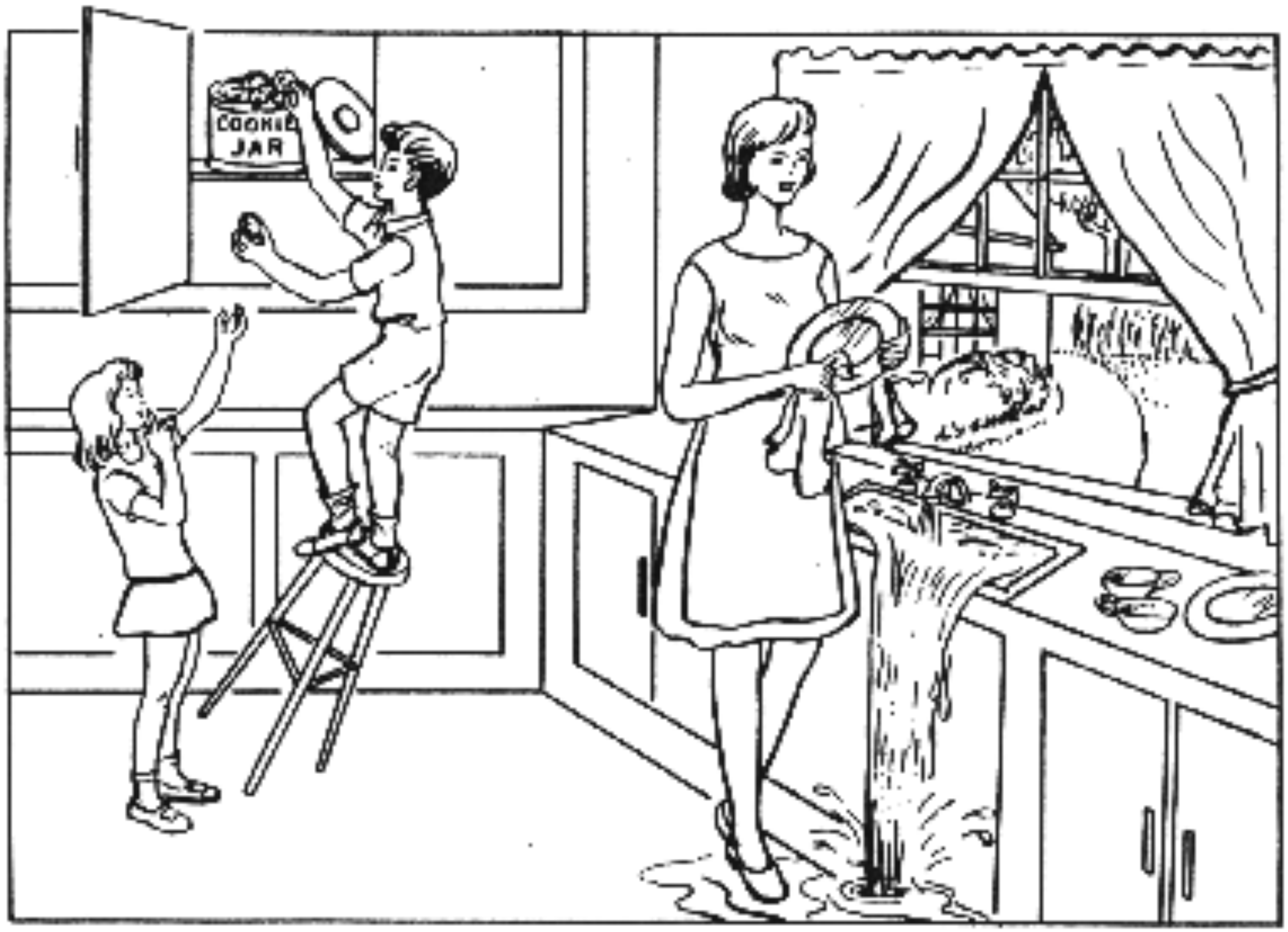
	Nonfluent	Good	Good	Transcortical Motor
			Poor	Broca's
		Poor	Good	Mixed Transcortical (Isolation)
			Poor	Global

Cookie Theft
Wernicke's

Fluent
Poor Auditory comprehension
Poor repetition



Wernicke's aphasia

•Fluent output, poor comprehension, poor repetition.

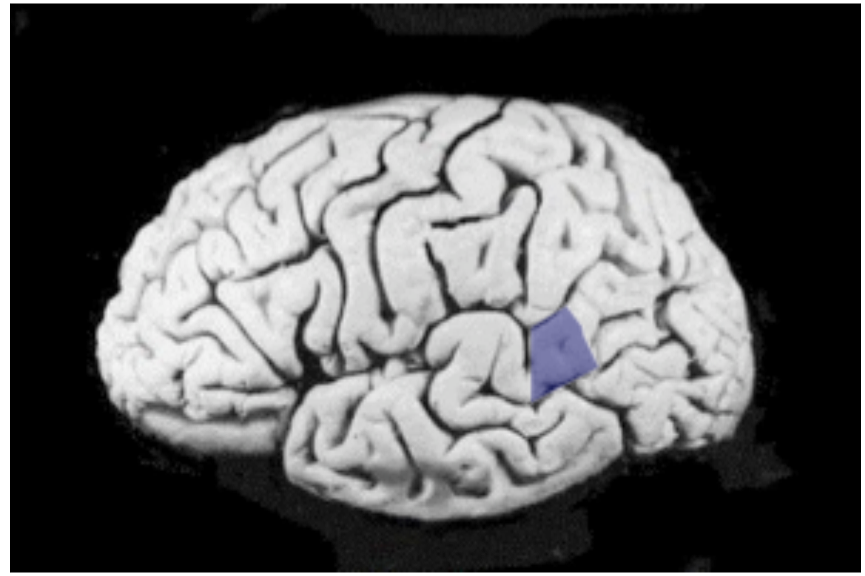
•Well-articulated speech, without distortions.

•Normal prosody.

•Long, syntactically varied utterances.

•Poor self-monitoring; striking lack of awareness of many of their errors.

•Demonstrable lexical-semantic and often conceptual-semantic impairments.



•Motor deficits uncommon

•*Classic <u>postulated</u> lesion site:*

  •Posterior part of the superior temporal gyrus; BA 22

Wernicke's aphasia

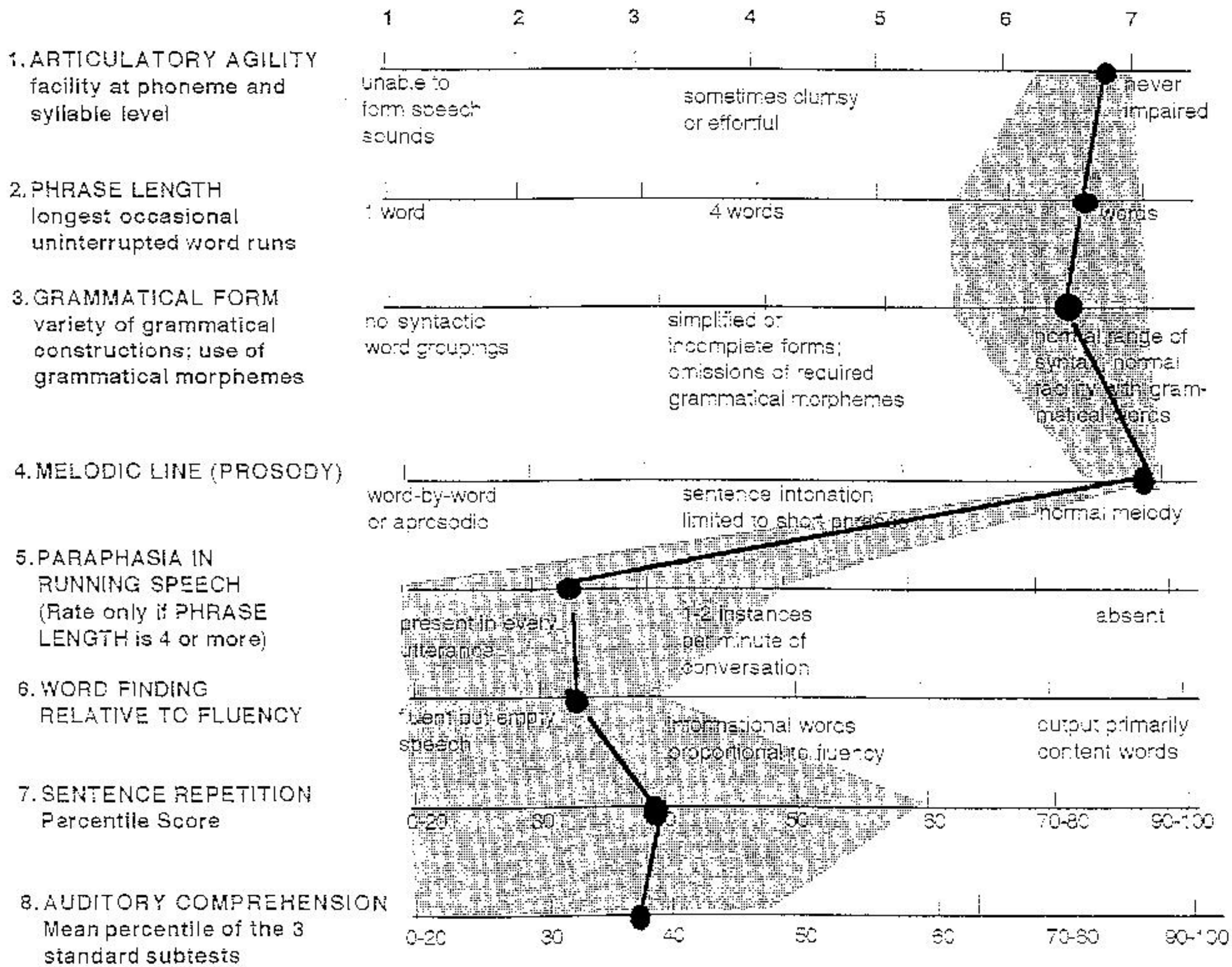


FIGURE 6-7. Wernicke's Aphasia: Range of Possible Ratings. Rating Scale Profile of Speech Characteristics.

Wernicke's aphasia

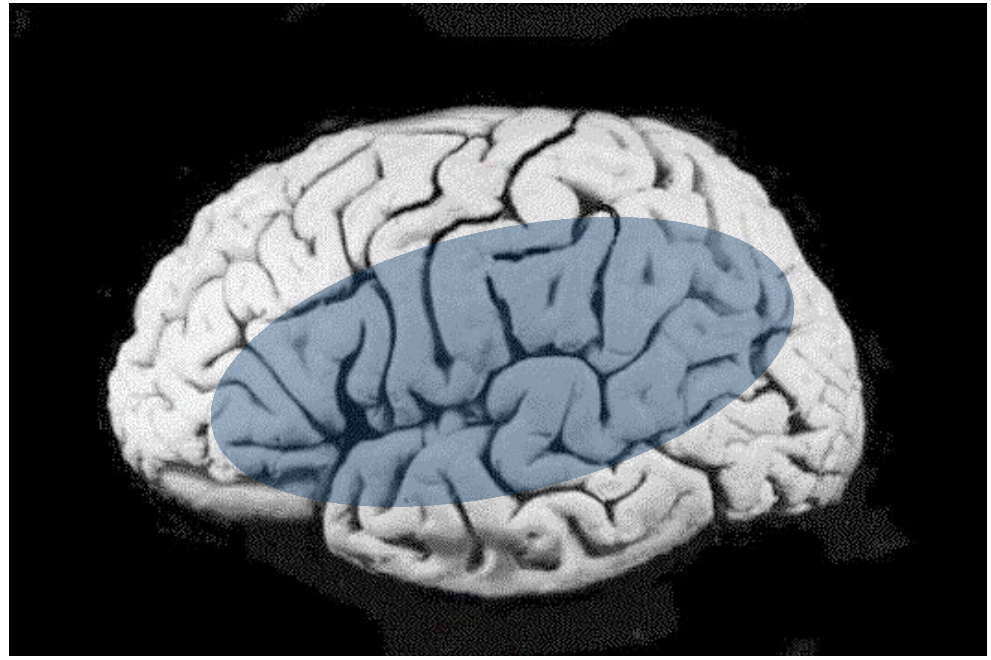
- Fluent output, poor comprehension, poor repetition.
- Well-articulated speech, without distortions.
- Normal prosody.
- Long, syntactically varied utterances.
- Poor self-monitoring; striking lack of awareness of many of their errors.
- Demonstrable lexical-semantic and often conceptual-semantic impairments.

empty content

doesn't make sense

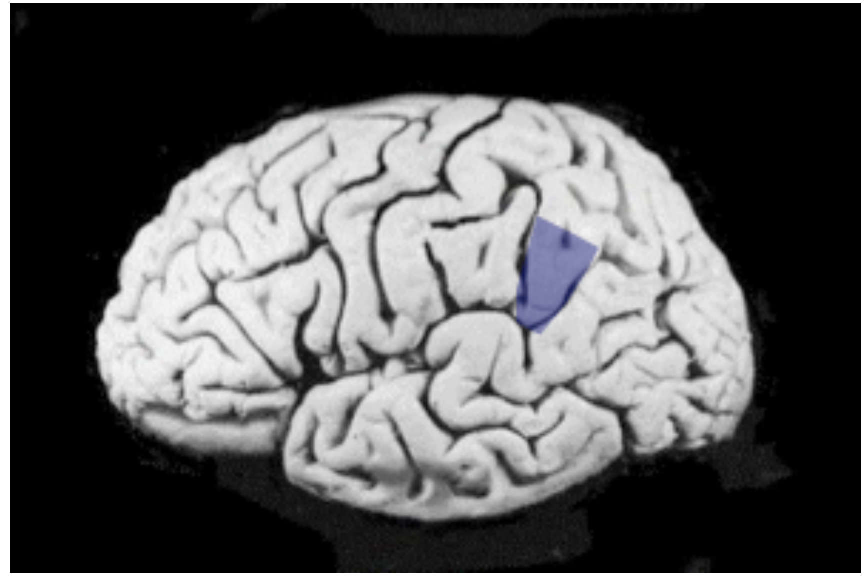
Global aphasia

- Nonfluent output, poor auditory comprehension, poor repetition.
- May have varied, often neologistic output or recurrent stereotypy.
    - e.g., “I know,” “tan”
- Usually hemiparetic or hemiplegic.
- Lesion site.
    - Large, perisylvian.



Nonfluent
Poor Auditory comprehension
Poor repetition

Conduction aphasia

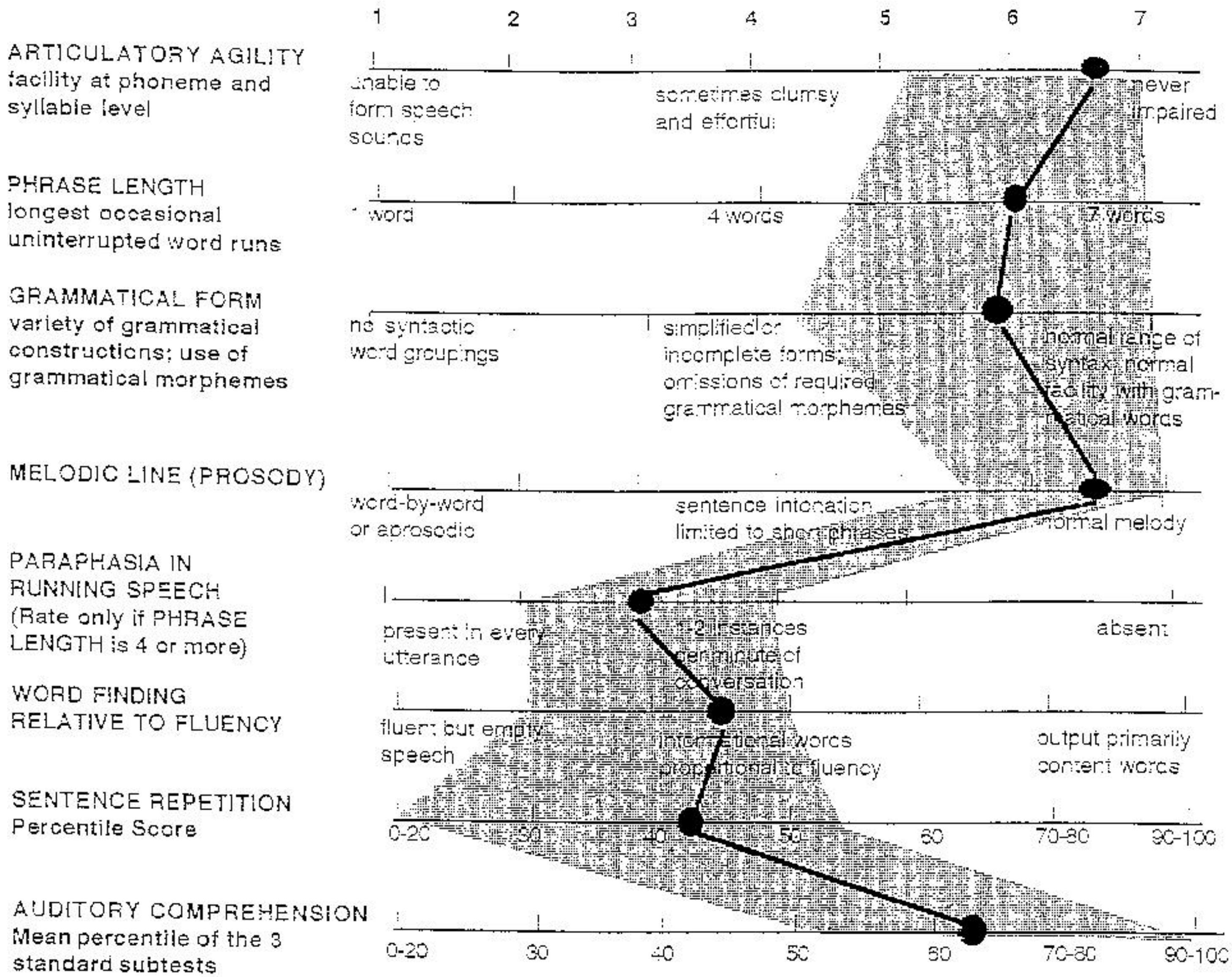


- •Fluent output, good comprehension, poor repetition.
- •Able to accurately paraphrase sentences that they cannot correctly repeat.
- •Frequent phonemic or literal paraphasias.
- •Conduite d'approche: multiple production attempts that progressively more closely approximate the target.
- •More fluent than Broca's, less fluent than Wernicke's.

- •Motor deficits uncommon.
- •*Classic postulated lesion site:*
  - •Arcuate fasiculus and/or supramarginal gyrus; BA 40.

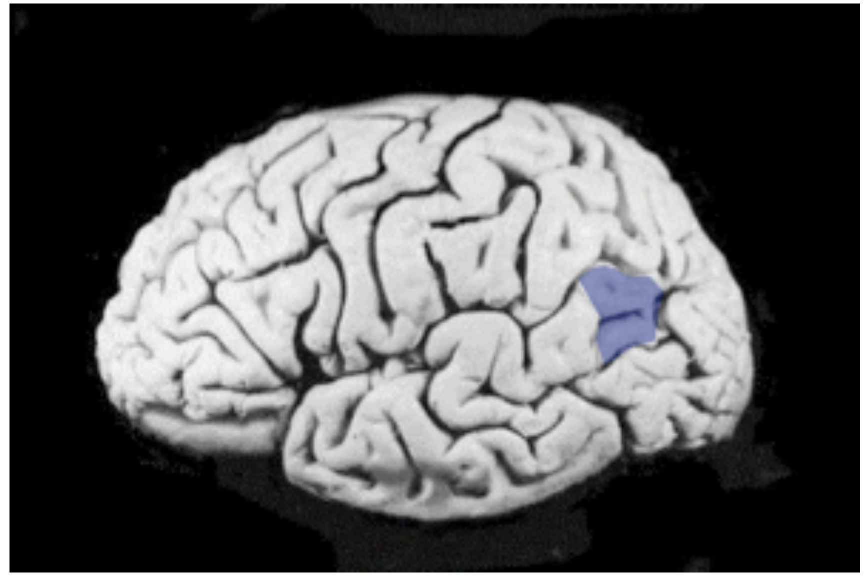
Fluent
Good Auditory comprehension
Poor repetition

Conduction aphasia



GURE 6-10. Conduction Aphasia: Range of Possible Ratings. Rating Scale Profile of Speech Characteristics.

Anomic aphasia



- Fluent output, good comprehension, good repetition.
- Circumlocution and semantic paraphasias may be the predominant error types.
- May be the initial classification in mild cases, or may be the end result of recovery from other types.
- Motor deficits uncommon.
- *Classic* ***postulated*** *lesion site:*
  - Angular gyrus, BA 39.
  - Temporal lobe lesions have also been associated with category-specific naming deficits.
    - Temporal pole: proper names.
    - Anterior ITG and MTG: animals.
    - Temporo-occipital junction: tools.

Fluent
Good Auditory comprehension
Good repetition

Anomia:
difficulty w/ word retrieval
hallmark of aphasia

Anomic aphasia

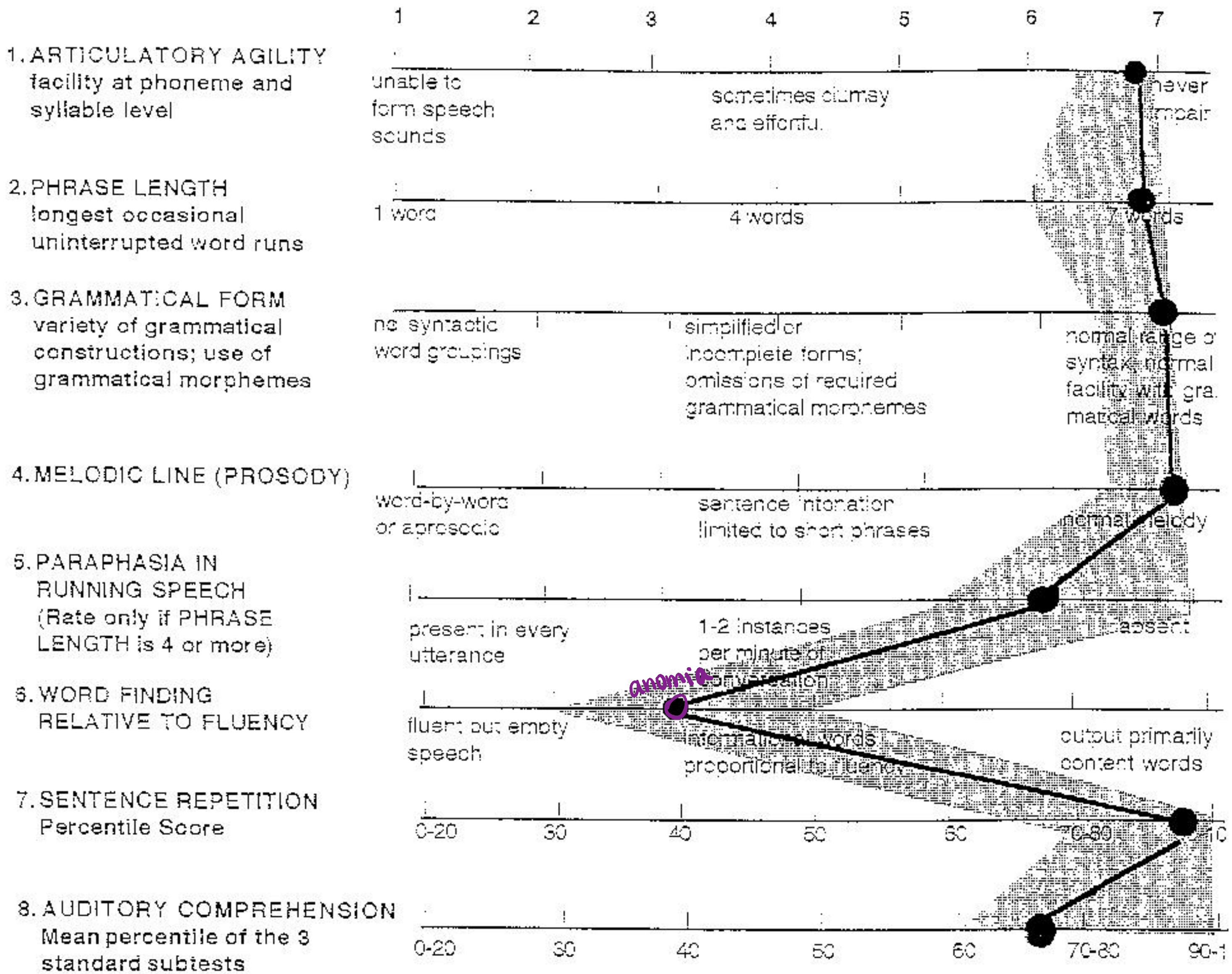
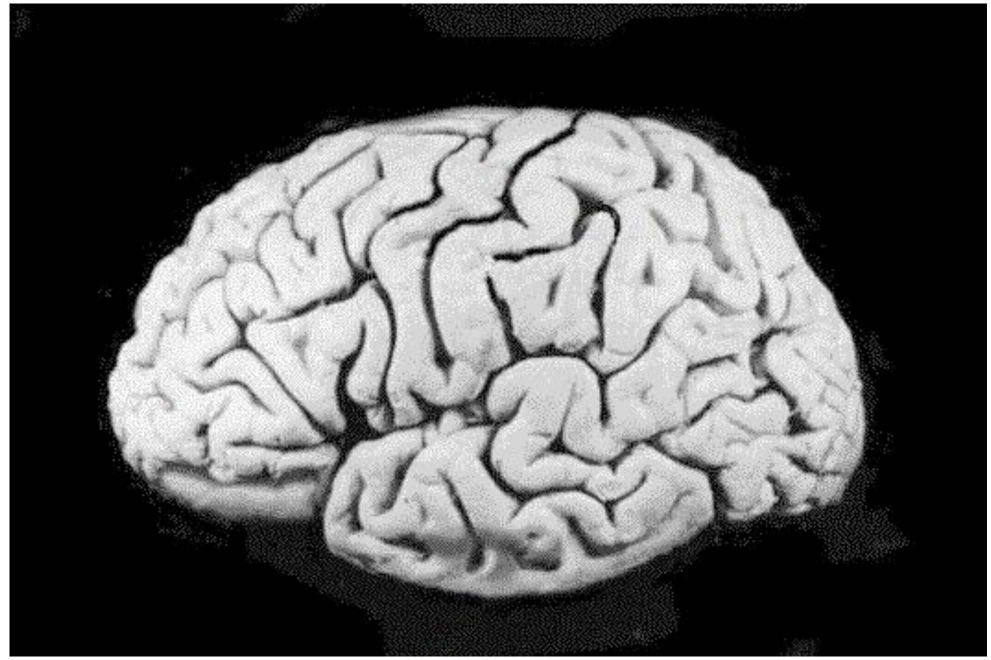


FIGURE 6-13. Anomic Aphasia: Range of Possible Ratings. Rating Scale Profile of Speech Characteristics.

Transcortical aphasias



- Transcortical motor: nonfluent output, good comprehension, good repetition.
  - May have generally akinetic or bradykinetic presentation.
  - LE > UE hemiparesis.
  - *Classic postulated lesion site:*
    - watershed region between MCA and ACA; SMA (BA 6); anterior & superior to Broca's area.

- Transcortical sensory: fluent output, poor comprehension, good repetition.
  - May be echolalic.
  - Lesion site: watershed region between MCA and PCA; temporo-occipital junction.

The Wernicke-Lichtheim classification system is based on outdated assumptions.

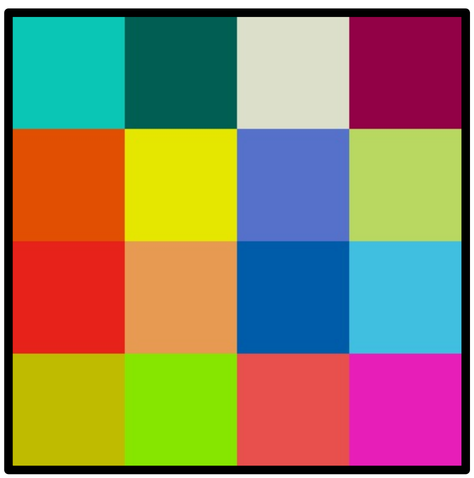
Outdated assumptions include:

- Language representations are stored in discrete "centers."
- Centers are connected to one another by unique and discrete pathways.
- Lesions in specific centers or pathways create specific, differentiable types of aphasia.
- The behavioral dimensions of fluency, comprehension, and repetition are unitary, localizable, and bimodally distributed.

The Wernicke-Lichtheim classification system is based on outdated assumptions.

Unfortunately, little predictive value in terms of:

- Domains *not* included in classification.
- Language treatment planning: need severity and full linguistic profile.
- Recovery prognosis.
- Only provides general information about lesion location.
- **Even classic cases have been found to be more complicated from a lesion-symptom perspective…**



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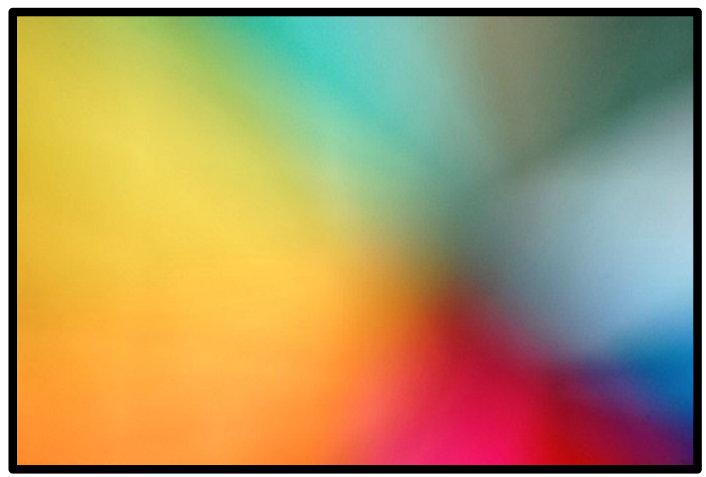
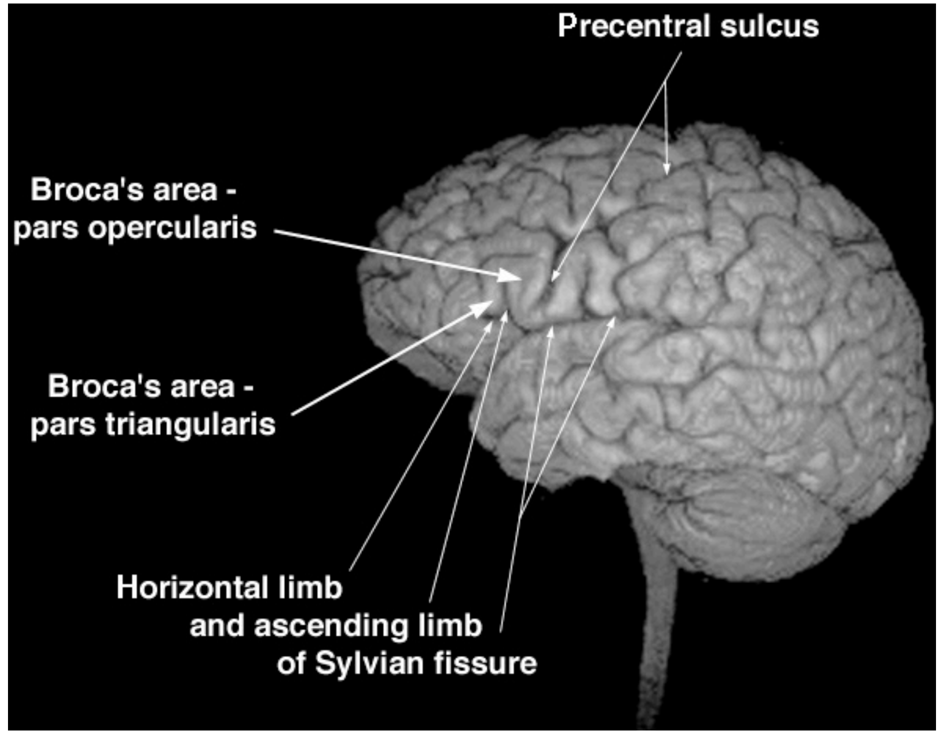


Dronkers et al., 2007, *Brain*

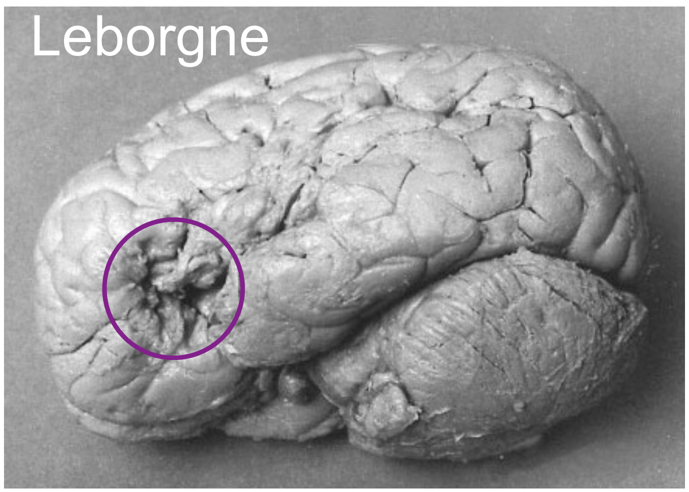
- Paul Broca's famous patients, **Leborgne** and **Lelong**, had lesions involving the left inferior frontal gyrus (IFG) and behavioral deficits consistent with Broca's aphasia.

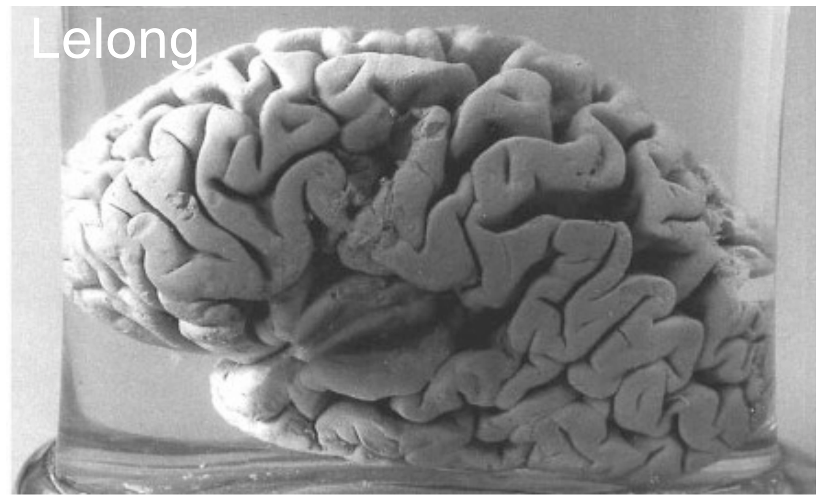
- Dronker et al (2007) used high-resolution MRI to examine the brains of Leborgne and Lelong to more completely characterize the extent of their lesions.



Dronkers et al., 2007, *Brain*

- *Gross anatomy*



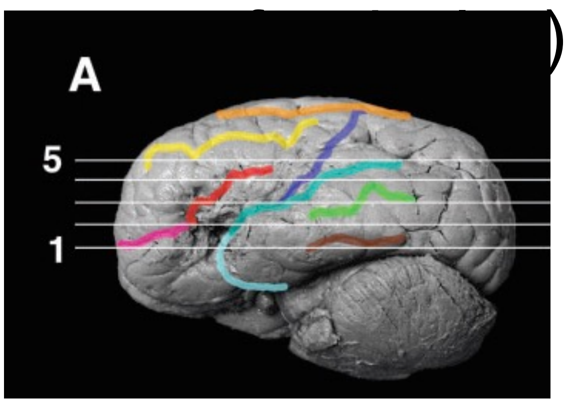


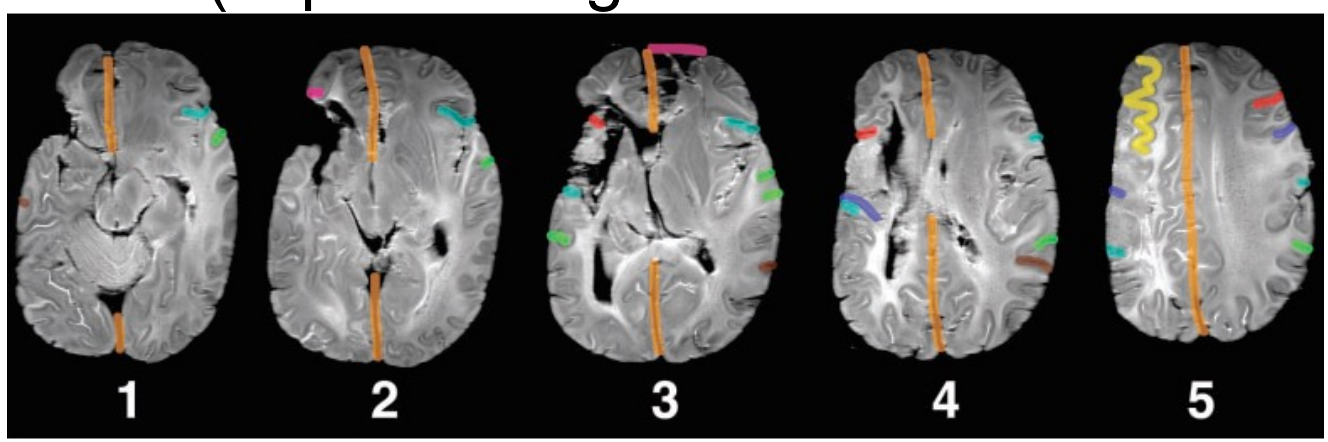
- Clearly visible lesion, left IFG.
- Extension into tissue superior and posterior to IFG.
- ***Suggested additional cortical/ subcortical involvement.***

- IFG involvement appears limited to posterior portion (pars opercularis), with sparing of anterior portions.

Leborgne: MRI findings

- Lesion involved:
  - **Much more** than lateral IFG lesion…
  - LIFG
  - Deep inferior parietal lobe
  - Anterior temporal lobe
  - Insula
  - Subcortical structures (basal ganglia)
  - White matter tracts (superior longitudinal)

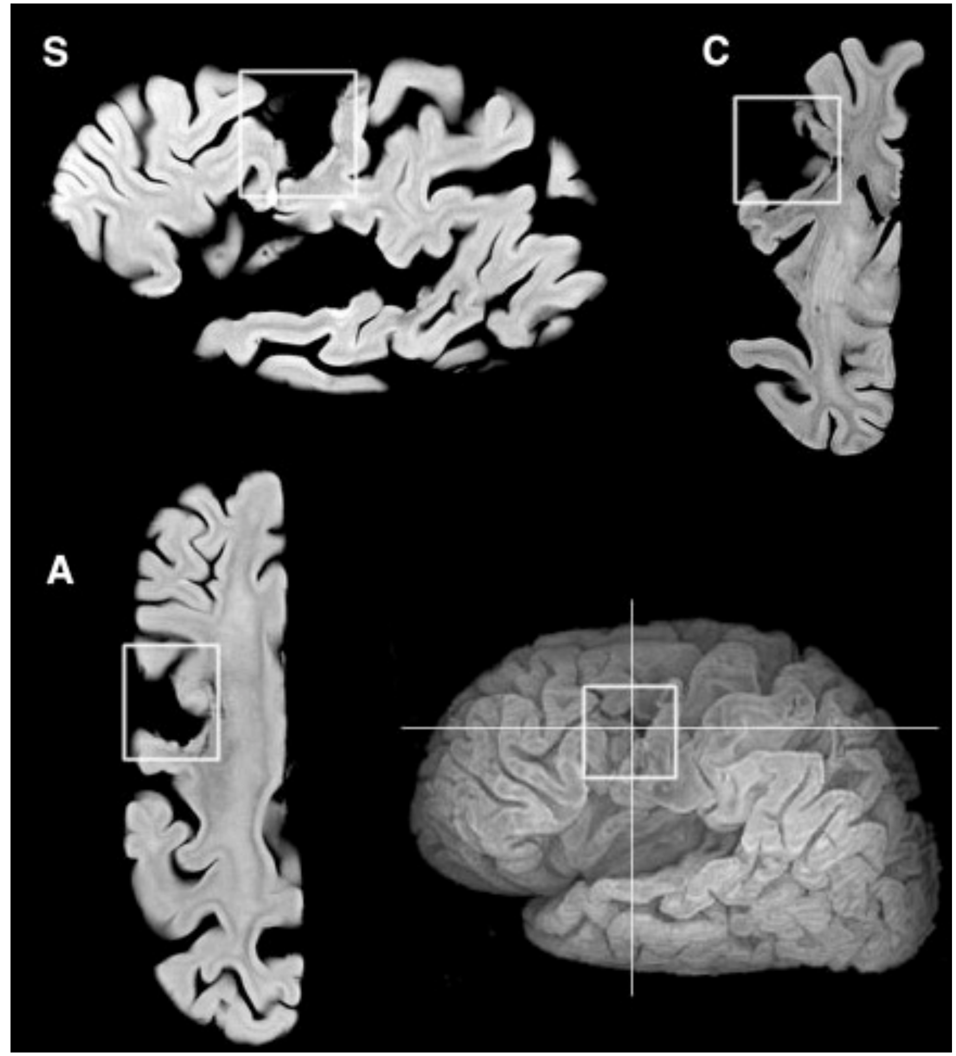




colors delineate various structures as points of reference (eg, orange = interhemispheric fissure; aqua = sylvian fissure; red = inferior frontal sulcus);

Lelong: MRI findings

- Lesion involved:
  - Posterior Broca's area (i.e., posterior pars opercularis
  - Superior longitudinal fasciculus
  - Other deep structures spared.



- *Note: Lelong's RH was not preserved with his LH.*

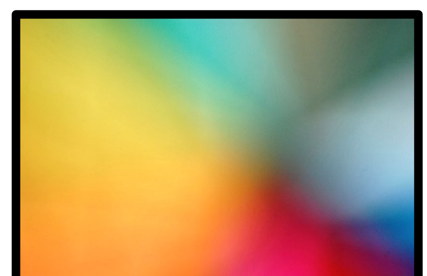
Dronkers et al., 2007, *Brain*

- Both patients' lesions involved deep structures that Broca could not discern.
- He chose to preserve the brains instead of dissecting them. (thank you!)
- Both patients had involvement of the superior longitudinal fasciculus (large fiber tract that connects anterior and posterior cortical structures).

- **Findings suggest that the "Broca's aphasia" observed even in original cases was not solely due to damage to Broca's area**.

More trouble for classic classifications

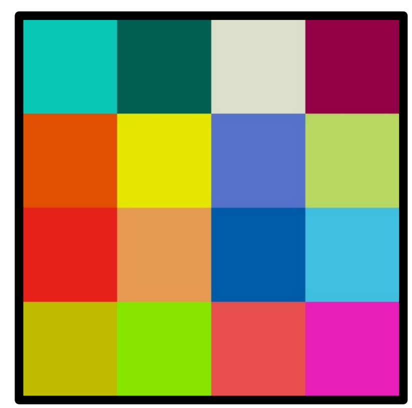
- Lesion-syndrome correlations tenuous at best...
- Willmes & Poeck (1993)
  - Acute/subacute and chronic patients with single contiguous LH lesion
  - Broca’s Aphasia
    - P (Broca’s | anterior) = 17/48 = 35%
    - P (anterior | Broca’s) = 17/29 = 59%
  - Wernicke’s Aphasia
    - P(Wernicke’s | posterior) = 23/48 = 48%
    - P(posterior | Wernicke’s) = 23/26 = 90%



Motor speech may be more localizable...

- Apraxia of speech is relatively localizable to Broca's area (Hillis et al. 2004)
  - P (AOS | Broca's lesion/hypoperfusion) = 26/30 = 87%
  - P (Broca's lesion/hypoperfusion | AOS) = 26/31 = 84%
  - P (AOS | insular lesion/hypoperfusion) = 12/29 = 41%
  - P (insular lesion/hypoperfusion | AOS) = 12/31 = 39%

Apraxia of speech (AOS):
motor planning deficit



As you move ↑ in language system,
things get ↑ fuzzy

Moving beyond classic classifications

- What are more *current* approaches to making sense of brain-behavior relationships in aphasia?
- Examples:
  - Lacey et al. (2017)
  - Hope et al. (2018)

Lacey et al., 2017, *Neurorehabilitation and Neural Repair*

- Goal of modern aphasia assessment is not syndrome classification but to **ID specific language deficits.**
- Important to establish a clear mapping between:
  - Language tests
  - Underlying cognitive-linguistic processes
  - Lesion location

Lacey et al., 2017, *Neurorehabilitation and Neural Repair*

left-hemisphere

- Participants: N = 38 chronic LH stroke survivors of ranging aphasia severity.
- Approach:
  - Principle component analysis (PCA) to ID core language and cognitive processes affected by LH stroke on a wide battery of tests.
  - Applied multivariate lesion symptom mapping to determine which brain areas are associated with these core processes.

Lacey et al., 2017, *Neurorehabilitation and Neural Repair*

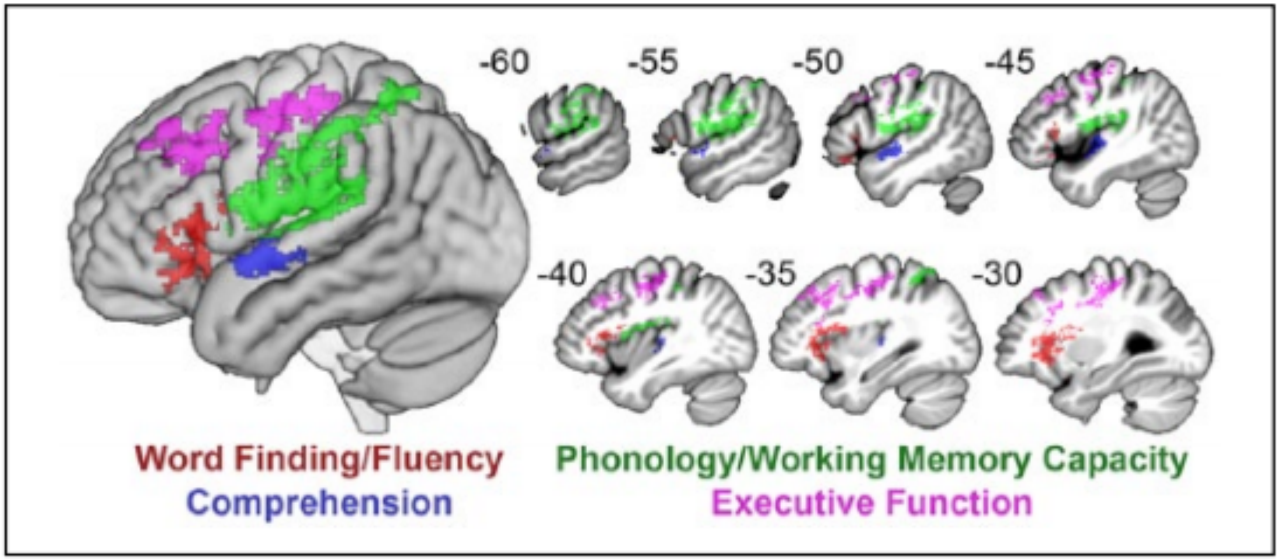
- PCA Identified 4 factors explaining 81% of test score variance:
  1. Word finding/fluency
  2. Comprehension
  3. Phonology/working memory capacity
  4. Executive function

	Factor 1 **Word Finding/Fluency**	Factor 2 **Comprehension**	Factor 3 **Phonology/Working Memory Capacity**	Factor 4 **Executive Function**
Variance accounted for by Factor	28%	21%	18%	14%
	Factor Loadings			
Philadelphia Naming Test (PNT)	0.842	0.247	0.249	0.251
WAB Object Naming	0.84	0.287	0.275	0.246
Reading Real Words	0.791	0.3	0.414	0.1
Category Fluency (Total 3 Categories)	0.74	0.249	0.256	0.413
WAB Spontaneous Speech Fluency	0.695	0.381	0.357	0.201
WAB Spontaneous Speech Content	0.678	0.406	0.316	0.299
WAB Responsive Speech	0.73	0.477	0.324	0.083
Letter Fluency (Total 4 Letters)	0.56	0.186	0.394	0.435
WAB Sentence Completion	0.606	0.488	0.434	0.005
PNT - Written Naming	0.618	0.118	0.129	0.566
BDAE Semantic probe	0.251	0.831	0.14	0.25
WAB Sequential Commands	0.226	0.725	0.39	0.228
WAB Yes/No Questions	0.179	0.737	0.415	-0.234
WAB Word Recognition	0.423	0.738	0.295	0.182
Auditory word-to-picture matching	0.439	0.708	-0.111	0.366
BDAE Complex Ideational	0.438	0.609	0.403	0.286
ABA Increasing Word Length	0.278	0.066	0.815	-0.034
Pseudoword Repetition (1-5 Syll.)	0.452	0.293	0.686	0.149
Digit Span Forward	0.275	0.33	0.668	0.452
BDAE Embedded Sentences	0.358	0.421	0.511	0.414
WAB Repetition	0.54	0.39	0.612	0.203
Reading Pseudowords	0.486	0.292	0.518	0.423
CLQT Executive - Composite score	0.161	0.078	0.139	0.839
Pyramids and Palm Trees	0.218	0.569	-0.153	0.643
Digit Span Backwards	0.344	0.172	0.565	0.621

which factors explain as much of the variance as possible?

Lacey et al., 2017, *Neurorehabilitation and Neural Repair*

- Lesion-symptom mapping analyses indicated that impairments in different domains (factors) were associated with lesions in distinct areas.



1. Word finding/fluency → inferior frontal gyrus (IFG), anterior insula, and dorsal parietal white matter
2. Comprehension → superior temporal cortex anterior to Heschl's gyrus
3. Phonology/working memory → ventral motor and somatosensory cortex, supramarginal gyrus, and posterior planum temporale
4. Executive function → middle frontal gyrus (dorsolateral prefrontal cortex), dorsal Rolandic cortex, and posterior white matter in the superior longitudinal fasciculus

Lacey et al., 2017, *Neurorehabilitation and Neural Repair*

- Lesion-behavior relationships:
  - IFG lesions were associated with impaired word finding/fluency. Consistent with studies showing that functional activation in IFG is associated with verbal fluency in controls.

- Results support modern models of speech-language processing by demonstrating that these functions extend beyond Broca's and Wernicke's areas.

Hope et al., 2018, *Neuroimage: Clinical*

- https://www.ucl.ac.uk/ploras/
- Predicting standardized test results from clinical imaging (T1-weighted MRI).
- Large study (by aphasia research standards) – **818 stroke survivors!**
- Methods:
  - Computed lesion load (i.e., proportion of damaged tissue) in 116 gray matter regions from the Automatic Anatomical Labelling atlas.
  - Computed white matter disconnection via the Network Modification Toolbox.
  - Machine learning-based regression approach.
    - Compared models predicting performance on 28 language scores from the Comprehensive Aphasia Test (CAT) based on lesion and WM data.

gold-standard

- **Provides general benchmark for current best brain-behavior predictions we can hope to make based on real-world clinical data.**

Hope et al. (2018).

- Models assessed by correlation between empirical vs. predicted language scores for each model.

TASK			R: Predicted vs. Empirical						
	Med. (range)	N (all/impaired)	L	C(r)	C(f)	LC(r)	LC(f)	LsC(r)	LsC(f)
Fluency	68 (38)	812/255	0.72	0.73	0.73	0.73	0.73	0.72	0.70
Comprehension of spoken words	65 (40)	814/158	0.50	0.51	0.50	0.51	0.50	0.51	0.50
Comprehension of spoken sentences	63 (44)	813/370	0.66	0.67	0.66	0.67	0.66	0.67	0.65
Comprehension of spoken paragraphs	60 (26)	805/116	0.44	0.43	0.39	0.43	0.40	0.44	0.45
Comprehension of spoken language	63 (48)	805/283	0.66	0.67	0.66	0.67	0.67	0.66	0.65

Med = median score; N = number of patients contributing to the model; L = model included lesion load; C = model included WM connectivity (i.e., disconnection)

How well did lesion data predict performance?

Hope et al. (2018).

- Evaluated correlations between CAT performance and lesion-based prediction models.
- Overall, r^2 ranged from ≈19%-55% variance explained for subtest performance.
- Lesion + WM models not superior to models without WM (high lesion/WM collinearity).

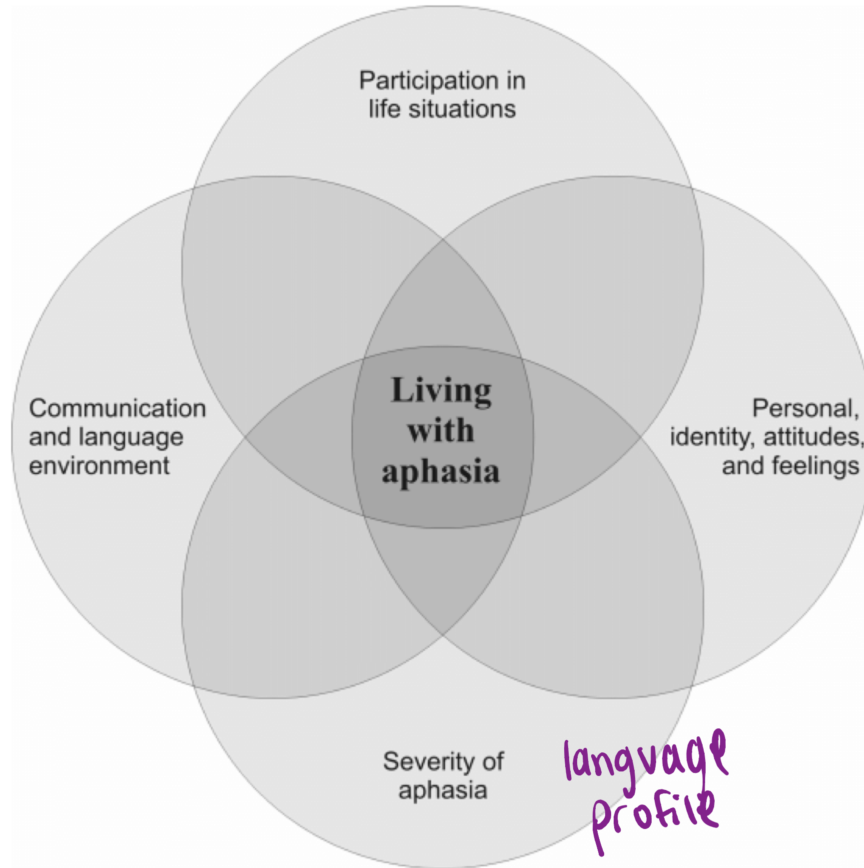
- *Take-aways:*
  - Can currently predict ≈55% of patient variance in picture naming based on clinical MRI.
  - Hopeful progress, but not definitive.
  - Developments continue!

Correlations: empirical vs. predicted language performance	*r*
Fluency	0.72
Comprehension of spoken words	0.50
Comprehension of spoken sentences	0.66
Comprehension of spoken paragraphs	0.44
Comprehension of spoken language	0.66
Comprehension of written words	0.54
Comprehension of written sentences	0.67
Comprehension of writing	0.67
Repeating words	0.63
Repeating complex words	0.64
Repeating non-words	0.57
Repeating digit strings	0.70
Repeating sentences	0.76
Repeating (all)	0.73
Object naming	0.71
Action naming	0.68
Naming (all)	0.74
Spoken picture description	0.72
Reading words	0.68
Reading complex words	0.69
Reading function words	0.60
Reading non-words	0.70
Reading	0.72
Writing (copying)	0.45
Written picture naming	0.58
Writing to dictation	0.68
Writing	0.67
Written picture description	0.71

How do Speech-language pathologists help people with Aphasia?

Behavioral interventions:

1. Restorative impairment-focused treatment
2. Compensatory treatment (e.g., strategy training)
3. Counseling
4. Education (understanding aphasia, advocacy, available resources)



Kagan et al., 2008

Aphasia Treatment and Brain Structure/Function

- Treatment can be a paradigm for testing causal mechanisms:
  - patterns of convergent/divergent improvements in response to well-specified treatments can shape and falsify theory (Nickels, Rapp, Kohnen, 2015).

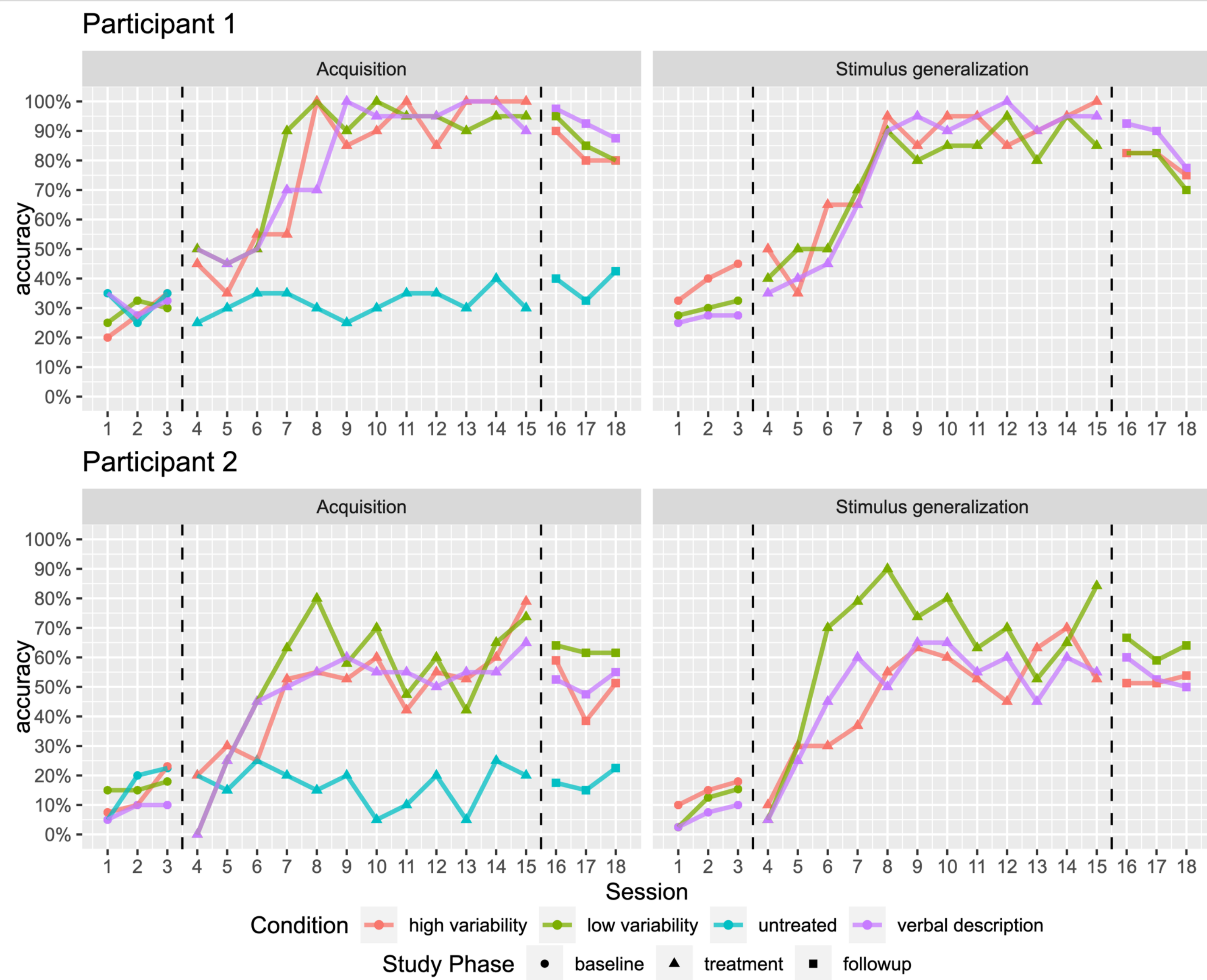
Behavioral example: Evans et al. (under review) Stimuli variability in computer-based flashcard tx

- Stimuli variability facilitates retention in developmental learning (Aguilar et al., 2018).
- However, anomia treatments often train and probe a single picture…

Results:

- Robust stimulus generalization was observed *across* conditions.
- Evidence of generalizable improvement in lexical access.
- Consistent with theories of post-stroke anomia vs. PPA/ semantic dementia (Jefferies & Lambon Ralph, 2006).

Results



Aphasia Treatment and Brain Structure/Function

- Current restorative treatment research mainly focused on how pre-tx structure/function may predict tx outcomes.
  - Very current example: Wiley et al (2021)

    Hula et al (2018)

Hot off the preprint press!

Prediction of post-stroke aphasia treatment outcomes is significantly improved by inclusion of local resting-state fMRI measures

Robert W. Wiley[1,7]*, James Higgins[2], David Caplan[3], Swathi Kiran[4], Todd Parrish[2], Cynthia K. Thomspon[5,6], and Brenda Rapp[7]

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ACADEMY OF APHASIA

2021 Annual Meeting

Academy of Aphasia 59th Annual Meeting
October 24-26, 2021
Online, US Eastern Daylight Time

Wiley et al. (2021 preprint)

- Local resting-state fMRI is easy to collect, can delineate healthy/lesioned tissue and index domain-specific language deficits (e.g., Demarco & Turkeltaub, 2020)
- Wiley et al. examined predictive value of resting-state fMRI on aphasia treatment outcomes, above and beyond behavioral and demographic predictors.

Wiley et al. (2021 preprint)

Methods:

- Large multi-center clinical trial. 64 PWA following a single left-hemisphere stroke treated for deficits in naming (n = 28), spelling (n = 22), or syntax (n = 14).
- Pre-tx fMRI data used to calculate local resting state measure: fractional Amplitude of Low Frequency Fluctuations (fALFF; Zou et al., 2008).

Wiley et al. (2021 preprint)

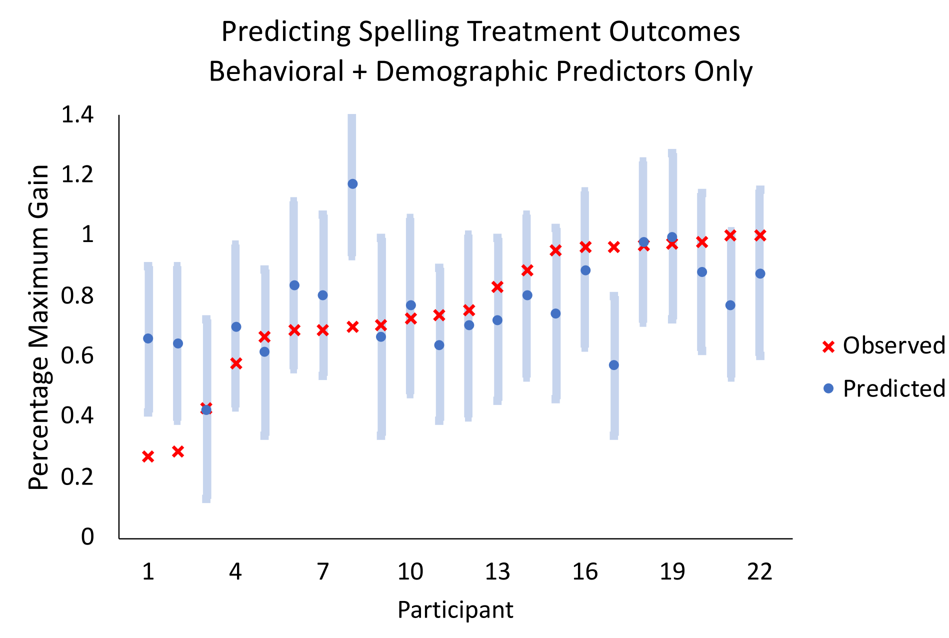
Methods:

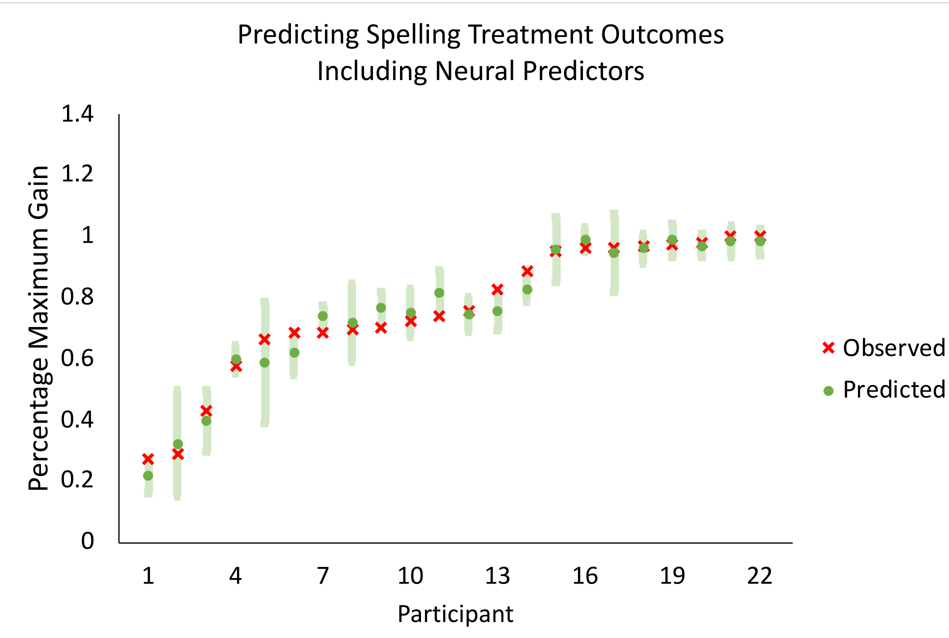
- Response to treatment predicted using best reduced set of demographic and behavioral measures. Assessed via cross-validation.
- Process repeated including rs-fMRI (fALFF) and lesion volume.
- Best set of neural measures selected via elastic net regression (Zou & Hastie, 2005).
- *Behavioral only* vs. *Behavioral + neuro* models compared via Monte Carlo analysis.

Wiley et al. (2021 preprint)

Results:

- Inclusion of local rs-fMRI significantly improved precision **and** accuracy of tx response predictions.
- Mean absolute error for behavioral-only prediction models: 11-17%
- Mean absolute error for behavioral + neuro prediction models: 1-3%
- **Evidence for emerging benefits of resting state fMRI for treatment prognosis!**





Summary for today:

- **Aphasia is a language disorder caused by brain damage, usually to the left hemisphere, which can affect spoken language production, auditory comprehension, reading, or writing.**
- Aphasia classification has important historical context and continues to offer educational and some (limited) clinical utility.
- However, current advances focusing on data-driven approaches to improving our understanding of brain-behavior relationships without relying on classic aphasia syndromes.
- As a result, modern analytical approaches are just starting to improve treatment prognosis.

PWA from VA PIRATE:

- Veterans with aphasia interviewed while attending the VA Pittsburgh Program for Intensive Residential Aphasia Treatment and Education (PIRATE)
- http://vrfpittsburgh.org/pirate/

Thank you!

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