NAMING AND LANGUAGE PRODUCTION

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ABSTRACT

Naming and sentence production are complex tasks, each requiring a number of cognitive processes and representations, which can be selectively impaired by focal brain damage, such as stroke, or by neurodegenerative disease. The types of errors made by the patient and the pattern of performance across tasks can provide clues regarding the location of the lesion and sometimes the most likely pathology. Understanding the nature of the deficit can help the physician provide guidance on how to facilitate communication.

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INTRODUCTION

Impaired word retrieval or naming is a ubiquitous problem in all neurologic diseases that affect language. It is also a cognitive function that declines with normal aging. Word retrieval is only one aspect of language production that can be affected by age or neurologic disease, however. Other aspects that will be considered in this article are grammatical sentence production and speech articulation. Organization of discourse will be only briefly touched on, because the complexity of this aspect of language would require greater space to be adequately covered.

First, the cognitive processes underlying naming (including articulation of the name) will be described. The focus will be on naming visual stimuli, but one can also name objects from their sound (eg, a flute or a dog), tactile exploration, smell, or a description. All of these tasks require the same cognitive processes, other than the earliest levels of perception and recognition (which will only be described for vision). Comparison of naming performance across these modalities of input is often useful in identifying the patient's impairment, as will be illustrated in some of the cases. Evidence for proposing the role of each of the cognitive processes underlying naming comes from consideration of the demands of the tasks and from neurologically impaired patients whose pattern of performance across language tasks can be explained by assuming selective damage to that cognitive function. Then, the neurologic diseases and sites of lesions in the brain that lead to impairments of each of these components of naming will be discussed. The section on sentence production will have a similar structure, moving from the cognitive processes underlying sentence production to associated diseases. The article will end with

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KEY POINT

- The task of producing the name of a pictured item requires many distinct cognitive processes and recruits a complex network of brain regions.

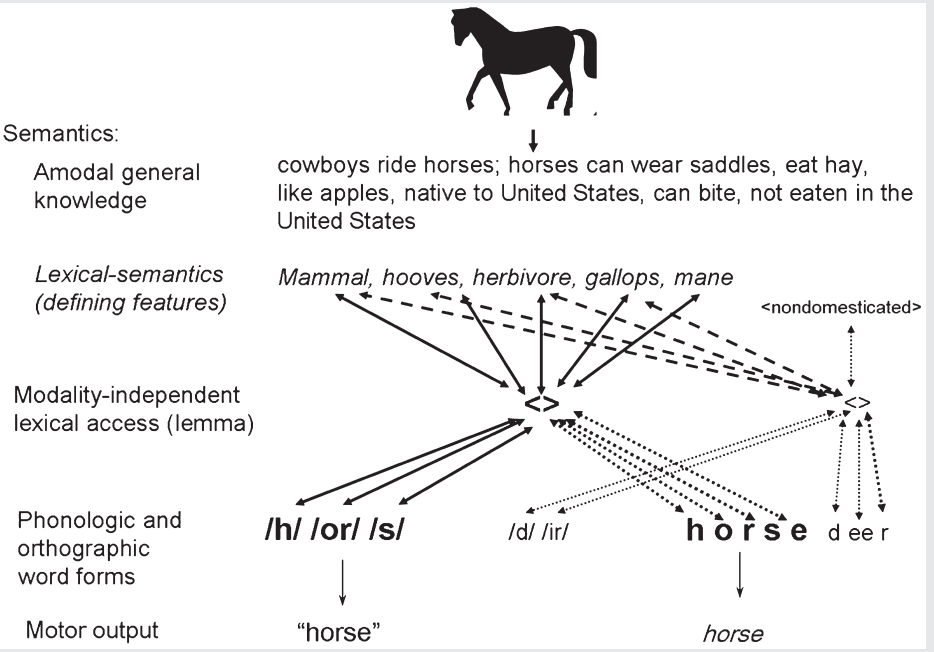

FIGURE 2-1 Schematic representation of the cognitive processes underlying naming.

practical recommendations for evaluation and management of naming and sentence production deficits.

COGNITIVE PROCESSES UNDERLYING NAMING

The task of producing the name of a pictured item, such as a horse, would seem to be very simple. However, this task requires many distinct cognitive processes (schematically represented in **Figure 2-1**) and recruits a complex network of brain regions (as reflected in functional imaging studies of naming (**Figure 2-2**). The importance of each cognitive process will be introduced by describing patients who have relatively selective impairment in the process or level of representation.

Impaired Visual Recognition

In order to name a pictured horse one must first recognize the horse as a familiar entity. This in itself requires computation of several levels of visual representation[1] that culminate in a three-dimensional image (independent of the orientation, size, or location of the picture) that can be matched to stored information about familiar items (sometimes called structural descriptions). Impairments of this level of processing are known as *apperceptive visual agnosia*. A patient with apperceptive visual agnosia would be able to see the picture and trace it, but would not recognize it or be able to match it to a picture of the

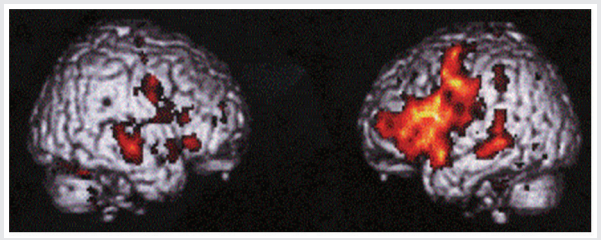

FIGURE 2-2 fMRI scan showing areas of the brain activated during oral naming relative to rest in 10 neurologically healthy control subjects. *Left:* right hemisphere, *Right:* left hemisphere.

Adapted from Prabhakaran V, Raman SP, Grunwald MR, et al. Neural substrates of word generation during stroke recovery: the influence of cortical hypoperfusion. Behav Neurol 2007;18(1):45–52. Copyright © 2007, with permission from IOS Press.

Case 2-1

A 70-year-old man first had trouble reading 3 years ago and had several car accidents before he was told by a neurologist not to drive. He initially had right homonymous hemianopia, but had progressive difficulty with visual perception in both visual fields. He could not recognize faces of even familiar people but recognized them by voice. He could avoid bumping into things but needed to touch or hear objects to recognize them. He could not name any pictures correctly but often named them as something visually similar (eg, he called a picture of a pear a light bulb). He began to develop mild verbal memory deficits. His spoken language was normal in conversation, but he could not read. He orally spelled words well, but his writing was illegible. Serial MRI scans showed progressive bilateral occipitotemporal atrophy. He was diagnosed with the ventral form of posterior cortical atrophy, which is usually caused by Alzheimer disease pathology. (The dorsal form of posterior cortical atrophy is characterized by Balint syndrome, with impaired visually guided reaching, simultagnosia, and optic apraxia, and can be due to either Alzheimer disease or corticobasal degeneration pathology with atrophy in the occipitoparietal cortex bilaterally.)

Comment. Like many patients with posterior cortical atrophy, his initial symptoms were motor vehicle accidents and trouble reading. He developed alexia without agraphia and optic aphasia (impaired access to semantics from vision, as described later in this chapter) and proposagnosia (impaired face recognition, all consequences of bilateral temporooccipital damage or left temporooccipital damage combined with damage to the white matter tracts that connect the hemispheres. Eventually he developed apperceptive agnosia.

KEY POINTS

- A patient with apperceptive visual agnosia would be able to see the picture of a horse and trace it but would not recognize it or be able to match it to a picture of the same horse from a different viewpoint.
- Patients with impaired conceptual knowledge often use objects inappropriately, particularly less familiar objects.

same horse from a different viewpoint (**Case 2-1**).

Impaired Semantics (Conceptual Semantics and Lexical Semantics)

Once an item, such as horse, is recognized as a familiar entity, it is essential to access its meaning. Meaning or semantics includes at least two levels of knowledge: (1) conceptual knowledge or information shared by a culture about the use and associations of the item (eg, that cowboys ride horses, that horses wear saddles, that they are not typically eaten in the United States), and (2) lexical semantics—the defining features of all things that share the name (eg, what makes a horse a horse and what makes it distinct from related items like a cow or a deer, including physical features such as a mane and more abstract features such as ''can be domesticated''). Patients with impaired conceptual knowledge, such as the woman in **Case 2-2**, often use objects, particularly less familiar objects, inappropriately.[2,3]

Impaired Lexical Semantics and Impaired Access to Lexical Semantics From Vision

Cases 2-3 and **2-4** describe individuals who are impaired in accessing only the subset of semantic features known as lexical semantics or lexical semantic representations, which allow a person to know what makes a horse a horse and what distinguishes it from related items such as a deer or a cow. Patients with impaired lexical semantics (but intact conceptual knowledge) do not use items inappropriately, but incorrectly label them or match them to their names. For example, a patient

KEY POINT

- A patient with impaired lexical semantics (or impaired ability to access lexical semantics from vision, a problem known as associative visual agnosia or optic aphasia) might point to a cow when asked to point to a horse. The patient, however, would neither try to saddle a cow nor consider milking a horse.

Case 2-2

A 66-year-old woman had progressive difficulty communicating for the past 2 years. She initially had impaired word retrieval only, but then had fluent, well-articulated, but meaningless, speech. She could not follow directions or point to named objects. When asked to point to the ceiling, she said, ''Ceiling? What is a ceiling?'' She had been living independently until her family became concerned when she served boiled pizza for dinner. She also had tried to eat soup with a knife. She could read out loud fairly well but made regularization errors in reading irregular words (eg, read *bear* as ''beer''), and did not understand what she was reading (a pattern of reading known as surface dyslexia). Her MRI showed bilateral anterior and inferior temporal atrophy, worse on the left. She was diagnosed with the semantic variant of primary progressive aphasia (PPA) (also called semantic dementia).

Comment. This woman shows typical defining features of the semantic variant of PPA, including impaired word meaning in the face of spared speech fluency and grammar. It is typical for these patients to ask the meaning of nouns such as *ceiling*. They often show the pattern of surface dyslexia and surface dysgraphia (reading and spelling by phonics). The semantic variant of PPA is usually associated with atrophy of the anterior and inferolateral temporal cortex, more marked on the left.

with impaired lexical semantics, like the patient in **Case 2-3** (or impaired ability to access lexical semantics from vision; a problem known as *associative visual agnosia* or *optic aphasia*, as in **Case 2-4**), might point to a cow when asked to point to a horse. However, neither of them would try to saddle a cow or consider milking a horse. In each case, the person might access a subset of features (eg, hooved animal) that are equally compatible with semantic-related words (horse, deer, cow), and so they make semantic paraphasias—incorrect words that are semantically related to the target.

Case 2-3

A 66-year-old accountant had sudden onset of jargon speech and could not understand what was being said to him. He was found to have Wernicke aphasia due to a large left temporoparietal stroke caused by new-onset atrial fibrillation. After a few days, he began to say some intelligible words but often made semantic paraphasias in naming pictures (eg, named a bicycle as car). He made the same sorts of errors in conversation and naming objects from tactile exploration. When asked to point to named objects, he pointed to semantically related objects as often as to the correct object. However, he was independent in activities of daily living and did not use objects inappropriately. He called a knife a spoon but did not try to eat soup with the knife. He also had surface dyslexia (**Case 2-2**).

Comment. This patient has impaired lexical semantics (so he misnames items and points incorrectly to named items), but has spared conceptual semantics (so he uses items appropriately). He does not understand printed words, but can sound them out, resulting in surface dyslexia.

Case 2-4

A 72-year-old woman developed sudden inability to read or see things in her right visual field 1 week ago. She had fluent, well-articulated, and grammatical speech and did not make semantic paraphasias in conversation. She followed directions well, except when asked to point to named pictures or objects. In these tasks, she pointed to semantically related objects. She also made semantic paraphasias in naming pictures or objects from vision but named the same objects correctly after exploring them with her hands. She named them accurately if they were described to her (eg, she named a pictured bird as ''dog'' but said ''bird'' when asked what animal can fly). She could not read accurately but tried to read by slowly reading letter by letter. However, she made some errors in identifying the letters. When words were spelled aloud to her, she named them accurately. For example, she read chair as, ''C-n-a-l-r. . . canaler?,'' but when it was spelled to her, she said, ''chair''). She wrote accurately to dictation. She was diagnosed with optic aphasia and alexia without agraphia, secondary to a stroke in the left posterior cerebral artery (PCA) distribution, with infarct involving the left occipital cortex and splenium of the corpus callosum.

Comment. This patient has classic pure alexia or letter-by-letter reading as well as optic aphasia. Since the 1880s these two clinical syndromes have been described as disconnection syndromes with the same basic account. In most cases, typically a lesion in the left occipital cortex prevents visual information from reaching left hemisphere language cortex directly, and a lesion in the splenium of the corpus callosum prevents visual information from the right occipital cortex from reaching the left hemisphere language areas via the corpus callosum. Therefore, visual information cannot be named or read aloud. The patient, however, can name items from tactile input or letters that are read aloud to her (and often resorts to tracing each letter with her finger to read). Sometimes visual information can slowly cross to the left hemisphere through nonsplenial white matter paths, but inefficiently and inaccurately, resulting in errors naming visual stimuli.

Impaired Access to Modality-Independent Lexical Representations (Lemmas)

The meaning of the item, or lexical semantic representation, is used to select a lexical representation or *lemma* that is independent of output modality (oral versus written). Impairments at this level of processing are manifest as *anomia* or impaired word retrieval. This deficit is well-known to all of us (increasingly with age) when we have a word ''on the tip of the tongue.'' We can neither write the word nor say it, although we may retrieve some partial information, such as the first letter or sound, the approximate length, and so on. This partial information often activates phonologically similar words for output, such that the person makes a phonemic paraphasia (eg, calling a horse a horn) or activates semantically related words for output, such that the patient makes a semantic paraphasia (eg, calling a horse a cow). Sometimes the partial phonologic information and partial semantic information combine to result in mixed errors, such as calling a shirt a skirt (**Case 2-5**).

Impaired Access to Modality-Specific Lexical Representations

The lemma is used to select a modality-specific lexical representation—the *phonologic representation* (spoken word

KEY POINTS

- Anomia, or impaired word retrieval, is well-known to all of us when we have a word on the tip of the tongue. This partial information often activates phonologically similar words for output, such that the person makes a phonemic paraphasia or activates semantically related words for output, thus making a semantic paraphasia.
- Some patients can write names even when they cannot retrieve the pronunciation of the names (despite intact motor speech). Other patients show the opposite—the ability to say the name but inability to retrieve the spelling of the same name.

Case 2-5

A 62-year-old woman developed sudden difficulty retrieving words in spontaneous speech. The first day of her symptoms, she spoke fairly fluently, except for hesitations for word retrieval and circumlocutions (eg, ''It's the thing you use to cut paper...knife... no, I can't think of it''). She followed directions and repeated sentences well but was very poor in saying or writing the names of objects on examination. MRI with dynamic contrast perfusion-weighted imaging showed hypoperfusion in the left posterior middle/inferior temporal cortex, with minimal infarct. She had severe stenosis of the inferior branch of the left middle cerebral artery (MCA). She also had low blood pressure (mean arterial pressure of 80 mm Hg). She was given boluses of saline and midodrine to increase her blood pressure to normal (mean arterial pressure of 100 mm Hg). A repeat MRI showed reperfusion of the previously hypoperfused area, and repeat testing showed resolution of her anomic aphasia (**Figure 2-3**).

Comment. This patient has pure anomic aphasia resulting from acute ischemia in an area of cortex posterior and inferior to Wernicke area. Stroke limited to this area or to the left angular gyrus or other parts of the parietal cortex can cause pure anomia. Anomic aphasia can also be the residual deficit after recovery from nearly any aphasia subtype.

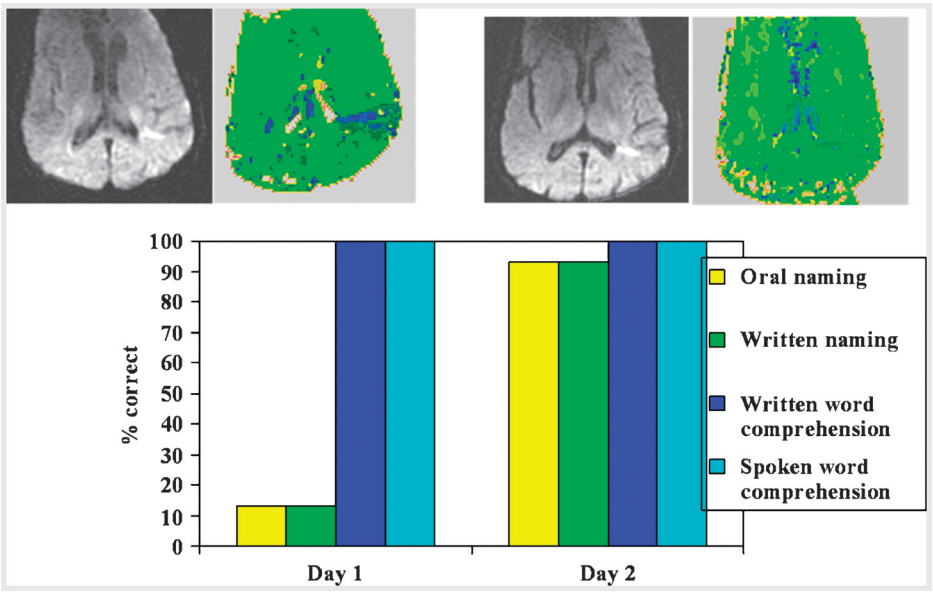

FIGURE 2-3 MRI (diffusion-weighted image showing area of acute infarct [*left*], dynamic contrast perfusion weighted image showing area of hypoperfusion [*right*]) scans at day 1 (*left*) and day 2 after reperfusion therapy (*right*) in a patient who had severe anomia at day 1 and recovered to normal naming at day 2 after reperfusion of the posterior, inferior temporal cortex.

form or learned pronunciation of the name) or the *orthographic representation* (written word form or learned spelling of the name). Patients with selective impairment in accessing one or the other have been described (**Case 2-6**).[4–8] Some patients can write names even when they cannot retrieve the pronunciation of the names (despite intact motor speech). Other patients show the opposite—the ability to say the name but inability to retrieve the spelling of the same name.

Once a spoken word form or phonologic representation has been accessed, it is must be spoken aloud. There are two aspects to this process. One requires maintaining the phonologic representation (the correct sequence of speech sounds that comprise the pronunciation) while the sounds are produced, and the

Case 2-6

A 74-year-old woman had progressive difficulty speaking for 10 years. She was independent in activities of daily living, including driving. Her speech articulation was effortful, with distorted speech sounds, and limited to nouns or short phrases with nouns. She rarely produced a complete sentence. Her spoken output was generally intelligible. She made sound substitution errors in naming and spontaneous speech but self-corrected them. Her repetition was similar to her spontaneous speech. She followed simple directions and had good single-word comprehension but had trouble understanding syntactically complex sentences (eg, passive voice). In oral-naming tasks, she named nouns (objects) much more accurately than verbs (actions) but made some errors on both. However, she wrote the names of both nouns and verbs accurately. Her MRI showed atrophy in the left posterior inferior frontal cortex and insula. She was diagnosed with the agrammatic/nonfluent variant of PPA. She had impaired access to phonologic or spoken word forms, in addition to some motor speech impairment (apraxia of speech).

Comment. This patient had the main features of the nonfluent/agrammatic variant of PPA, including effortful, halting, distorted speech and agrammatic sentence production. She also had impaired comprehension of syntactically complex sentences, which is common in this variant. Additionally, she had impaired access to phonologic word forms, but spared access to written word forms, so writing was much more accurate than speaking even for words that she could articulate well.

second is motor output—articulation. Failure to activate or maintain activation of the complete phonologic representation will result in phonemic paraphasias such as substitution, insertion, and transpositions of phonemes (speech sounds) resulting in a different word (eg, horn for horse) or nonwords (eg, *porse* for horse). Articulation of the word requires motor planning or programming of the complex movements of the lips, tongue, palate, vocal folds, and respiratory muscles, followed by implementation of these movements. Impairment of motor planning or programming of speech articulation is sometimes known as *apraxia of speech* and can also be manifest by errors of insertion, deletion, transposition, substitution of speech sounds, or distortions of speech sounds in the absence of impaired strength, range, or rate of any of the speech muscles. Importantly, patients with apraxia of speech are very aware of their errors and try to correct them, while those who make phonemic paraphasias are generally unaware of their errors. Apraxia of speech is often characterized by various off-target productions of the word when attempting to say the same word multiple times and is more apparent in production of polysyllabic words, which require more complex motor planning. Even when motor planning is intact, the word might be articulated incorrectly because of dysarthria, a motor speech impairment caused by impaired strength, range, rate, or timing of movements of the lips, tongue, palate, or vocal folds. Dysarthria can be distinguished from apraxia of speech by its consistency across words (eg, the same speech sound will typically be distorted in both short and long words consistently across trials in dysarthria, but is much more likely to be inconsistently misarticulated in long words compared to short words in apraxia of speech). Dysarthria is also associated with weakness or reduced range/rate of movement of the muscles involved in speech.

KEY POINTS

- Patients with apraxia of speech are aware of their errors and try to correct them, while those who make phonemic paraphasias are generally unaware of their errors. Apraxia of speech is characterized by various off-target productions of the word, when attempting to say the same word multiple times, and is more apparent in polysyllabic words.
- Dysarthria can be distinguished from apraxia of speech by its consistency across words and is associated with weakness or reduced range/rate of movement of the muscles involved in speech.

KEY POINTS

- Impaired visual recognition, or apperceptive visual agnosia, is typically caused by bilateral temporal or fusiform lesions.
- Associative visual agnosia, or optic aphasia, with impaired access to semantics from vision, is typically caused by stroke in the distribution of the posterior cerebral artery. Both the right occipital lobe (including striate cortex) and the splenium of the corpus callosum are damaged.
- Impaired access to conceptual semantics requires damage to both hemispheres, with the anterior and inferior temporal lobes probably being the most critical, perhaps as an essential node or hub in a complex network that links different types of information about items stored in separate parts of the brain.

Diseases and Sites of Lesions Associated With Impairment of Each Cognitive Process Underlying Naming

Impaired visual recognition, or apperceptive visual agnosia, is typically caused by bilateral temporal or fusiform lesions. Such lesions can occur as result of head trauma, herpes encephalitis, top-of-the-basilar stroke, or neurodegenerative disease (particularly a rare form of Alzheimer disease that selectively involves these areas known as the ventral form of posterior cortical atrophy.[9–12]

Associative visual agnosia, or optic aphasia, with impaired access to semantics from vision, is typically caused by stroke in the distribution of the PCA. Both the left occipital lobe (including striate cortex) and the splenium of the corpus callosum are damaged. Patients will have right homonymous hemianopia, such that all visual information is initially processed in the right hemisphere. However, the visual information cannot be transferred to the left hemisphere language cortex to be named. The patients' visual information can only access the limited conceptual information in the right hemisphere, which would allow them to produce gestures appropriate to the picture and match it to pictures of the same item from different views.[8,13–15]

Impaired access to conceptual semantics requires damage to both hemispheres, with the anterior and inferior temporal lobes probably being the most critical, perhaps as an essential node or hub in a complex network that links different types of information about items stored in separate parts of the brain.[16] Information about color, smell, and shape might be stored in parietal lobes, while information about how it is manipulated or held is stored in the frontal lobes, and how it moves is stored in area MT (also called area V5), the visual motion area of the temporal lobe. These aspects of the conceptual and lexical semantic representations might be integrating in the anterior temporal lobes bilaterally. Damage to only one side does not disrupt the meaning of objects or their use, perhaps because this critical function is duplicated in the two hemispheres because of its evolutionary importance. The most common disease affecting the bilateral anterior temporal lobes (usually left greater than right) is the semantic variant of PPA (formerly called semantic dementia).[17–19] Herpes encephalitis also can affect this area bilaterally. Patients with the semantic variant of PPA and herpes encephalitis show impaired use of objects, particularly less familiar ones.

Lexical semantics refers to a subset of the semantic representation that defines the word.[20] Evidence from stroke suggests that the posterior, superior temporal gyrus or Wernicke area is essential for accessing this subset of semantic information that represents the meaning of the word.[21–24] Acute infarcts or hypoperfusion in this area causes impaired word comprehension, and restoration of perfusion to Wernicke area can result in recovery of word comprehension.[20,25–27]

Impaired access to the lemma, or modality-independent lexical representation, is known as *anomia*. Anomia, manifest as inability to retrieve either the spoken or the written name, can be the residual deficit in individuals who have recovered from any type of aphasia caused by stroke (eg, Broca aphasia, caused by infarcts in the territory of the superior division of the left MCA, or Wernicke aphasia, caused by infarcts in the territory of the inferior division of the left MCA). Anomia can also result from isolated infarcts or hypoperfusion in the left angular gyrus,[28] left thalamus,[29] or left posterior inferior/middle temporal gyrus.[20,30] Restoring blood flow to the last area can result in recovery of oral and written naming.[31] Anomia is often the first sign of PPA, the

most disabling deficit in the logopenic variant of PPA,[32] and a common deficit in Alzheimer disease, where it likely results from dysfunction in the posterior temporal and or inferior parietal cortex.

Modality-specific naming deficits, caused by impaired access to phonologic or orthographic lexical representations (ie, spoken or written word forms) are uncommon but can be observed in the agrammatic/nonfluent variant of PPA,[7] in which atrophy in the left posterior inferior frontal cortex and insula is present,[19] or after stroke in the left posterior inferior frontal cortex.[6,33] Apraxia of speech occurs with damage in the same areas,[34,35] caused by the same diseases.

Dysarthria, which is not a language deficit but rather a motor deficit, can result from any lesion to the subcortical structures (eg, internal capsule, basal ganglia), motor cortex, cerebellum, or brainstem. When it is caused by supratentorial lesions, it is typically mild, unless bilateral damage is present.

COGNITIVE PROCESSES UNDERLYING SENTENCE PRODUCTION AND ASSOCIATED LESIONS

In a model of sentence production described by Garrett (**Figure 2-4**), production of a sentence starts with a concept to be conveyed (the message level).[36] Then, a particular syntactic structure and particular modality-independent content words (lemmas) are selected at the functional level. Next, at the positional level of representation, a sentence planning

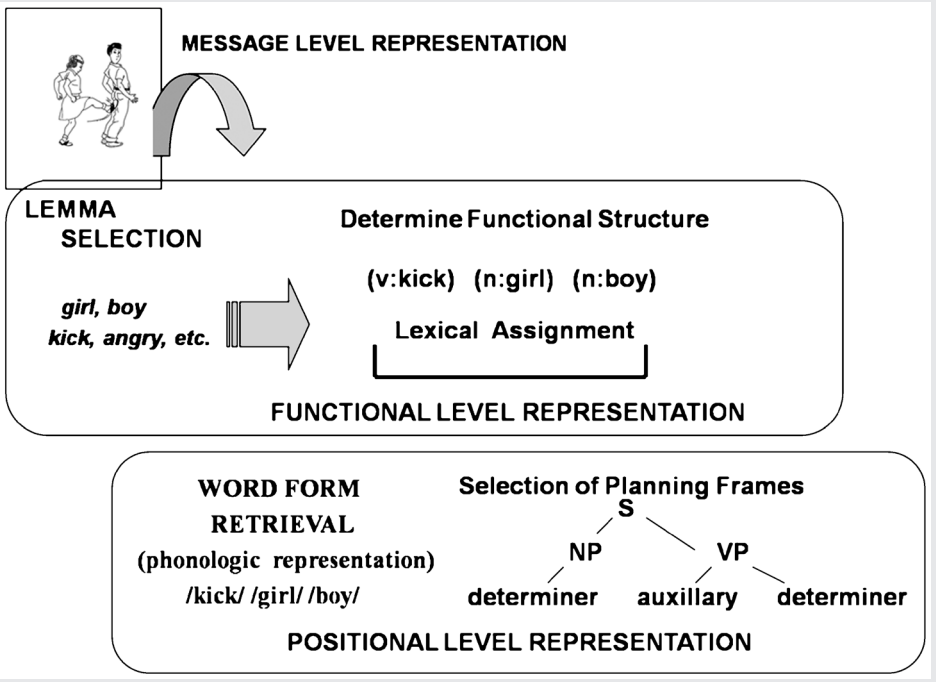

FIGURE 2-4 A schematic representation of the levels of representation underlying sentence production.

v = verb; n = noun; S = sentence; NP = noun phrase; VP = verb phrase.

Data from Mitchum CC, Berndt RS. Verb retrieval and sentence construction: effects of targeted intervention. In: Humphreys GW, Riddoch JM, eds. Cognitive neuropsychology and cognitive rehabilitation. East Sussex, UK: Lawrence Erlbaum Associates, 1994:317–348.

KEY POINTS

- Evidence from stroke suggests that the posterior, superior temporal gyrus, or Wernicke area is an essential area for accessing the subset of semantic information that represents the meaning of the word.
- Anomia, manifest as inability to retrieve either the spoken or the written name, can be the residual deficit in individuals who have recovered from any type of aphasia caused by stroke.
- Dysarthria, which is not a language deficit but rather a motor deficit, can result from any lesion to the subcortical structures (eg, internal capsule, basal ganglia), motor cortex, cerebellum, or brainstem. When it is caused by supratentorial lesions, it is typically mild, unless bilateral damage is present.

KEY POINTS

- Impairment at the message level of sentence production is manifest by jargon speech, which may be strings of words that do not make sense together, or neologisms (nonsense wordlike utterances) or a combination of both.
- Impairment at the functional level results in agrammatic speech with incorrect or missing verbs or other content words.
- Impairment of the positional level also results in agrammatic spoken output, but the correct content words have been selected in this case. However, the word order may be incorrect, or the sentence may be missing determiners, auxiliary verbs, or word endings.

frame is created that specifies the word order, the grammatical morphemes (eg, suffixes and prefixes, determiners like *the*, auxiliary verbs). Specific word forms or phonologic lexical representations are then selected to fill the ''slots'' in the sentence planning frame.

Impairment at the Message Level of Sentence Production

Impairment at the message level of sentence production is manifest by jargon speech, which may be strings of words that do not make sense together, or neologisms (nonsense wordlike utterances), or a combination of both (**Case 2-7**). Disruption at this level is often seen in the semantic variant of PPA (affecting left greater than right anterior and inferior temporal cortex [**Tables 2-1** and **2-2**]), Wernicke aphasia (affecting the left superior temporal gyrus [**Tables 2-1** and **2-2**]), and transcortical sensory aphasia (affecting left posterior temporal/parietal or temporal/occipital cortex). Although stroke is the most common cause of the latter two vascular syndromes, tumors, abscesses, or other lesions in the same regions can result in the same clinical syndromes.

Impairment of the Functional Level of Sentence Production

Impairment at the functional level results in agrammatic speech with incorrect or missing verbs or other content words (eg, ''The girl hit the boy'' might be produced as, ''The boy was hit the girl''). It is often associated with anomia and working memory deficits, as shown in **Case 2-8**. It can be caused by left frontal or left inferior temporoparietal lesions or atrophy (often involving supramarginal gyrus) as in severe logopenic variant PPA and severe conduction aphasia (**Table 2-1**).

Impairment at the Positional Level of Sentence Production

Impairment of the positional level also results in agrammatic spoken output, but the correct content words have been selected in this case. However, the word order may be incorrect, or the sentence may be missing determiners, auxiliary verbs, or word endings (eg, ''The boy was kicked by the girl'' may be produced as, ''Girl boy kick.''). Deficits at these two levels often co-occur, as both are typically seen with damage to the left posterior, inferior frontal cortex (including Broca area), as seen in the agrammatic/

Case 2-7

A 78-year-old woman awoke from bypass surgery with some deficits in understanding and producing language. A CT scan showed a posterior watershed stroke, an infarct in the area between the left MCA and left PCA in the posterior parietal and inferior temporal cortex. When she tried to describe her occupation as a nurse, she said, ''I the things that you know we all wish we did with the other ones.'' However, she added gesture and intonation that convey some meaning. She made semantic errors in naming and word comprehension, but her sentence repetition was nearly perfect. A speech-language pathologist told her family that she had transcortical sensory aphasia.

Comment. Transcortical sensory aphasia is characterized by jargon speech, impaired comprehension, but relatively spared sentence repetition. It is typically a result of lesions surrounding Wernicke area, but often posterior and inferior to Wernikce area. It can be seen in Alzheimer disease and other dementias.

TABLE 2-1 Aphasia Syndromes and Associated Sites of Damage and Etiologies—Vascular Syndromes

Aphasia Syndrome	Spontaneous Speech	Comprehension	Repetition	Site of Damage or Atrophy	Most Common Etiology
Broca aphasia	Nonfluent, agrammatic, articulatory errors	Relatively intact except for syntactically complex sentences	Nonfluent, agrammatic, articulatory errors	Left posterior, inferior frontal cortex and insula	Stroke involving left superior division MCA
Wernicke aphasia	Fluent jargon	Impaired for words and sentences	Fluent jargon	Left posterior, superior temporal cortex	Stroke involving the left inferior division MCA
Global aphasia	None, or one or two perseverative utterances (eg, no, no, no)	Impaired for words and sentences	Profoundly impaired	Usually large area involving left frontal, temporal, and/or parietal cortex	Stroke involving the entire left MCA distribution
Conduction aphasia	Fluent with some phonemic paraphasias (eg, sleep→ ''skeet, →skeep'')	Relatively intact except for syntactically complex sentences	Disproportionately impaired	Controversial; usually left supramarginal gyrus +/− arcuate fasciculus	Stroke involving branch of the left inferior division MCA
Transcortical motor aphasia	Nonfluent, agrammatic, articulatory errors	Relatively intact except for syntactically complex sentences	Relatively spared (more accurate than spontaneous speech)	Watershed area between left MCA and ACA territories, or left medial frontal cortex	Left ICA stroke or left ACA stroke
Transcortical sensory aphasia	Fluent jargon	Impaired for words and sentences	Relatively spared (more accurate than spontaneous speech)	Watershed area between left MCA and PCA territories or left thalamus	Left ICA stroke or stroke involving branch of left PCA to thalamus
Mixed transcortical aphasia	None, or one or two perseverative utterances (eg, no, no, no)	Impaired for words and sentences	Relatively spared (more accurate than spontaneous speech)	Watershed area between left MCA and ACA and between left MCA and PCA territories	Left ICA stroke or dementia (eg, Alzheimer disease)
Optic aphasia	Normal, but makes errors in naming visual stimuli	Normal, but makes errors in pointing to visual stimuli	Normal	Left occipital cortex and splenium	Left PCA stroke

MCA = middle cerebral artery, ACA = anterior cerebral artery, ICA = internal carotid artery, PCA = posterior cerebral artery.

TABLE 2-2 Primary Progressive Aphasia Syndromes and Associated Sites of Damage and Etiologies

Primary Progressive Aphasia Syndromes	Spontaneous Speech	Comprehension	Repetition	Site of Damage or Atrophy	Most Common Etiology
Nonfluent/ agrammatic variant PPA	Nonfluent, agrammatic, speech sound errors	Relatively intact, except for syntactically complex sentences	Nonfluent, agrammatic, speech sound errors	Left posterior, inferior frontal cortex and insula	Tauopathy (eg, corticobasal degeneration, frontotemporal lobar degeneration)
Logopenic/ phonologic variant PPA	Fluent with some phonemic paraphasias (eg, sleep→ ''skeet, →skeep'')	Relatively intact, except for syntactically complex sentences	Disproportionately impaired	Left inferior parietal, superior temporal cortex	Alzheimer disease
Semantic variant PPA	Fluent jargon	Impaired for words and sentences	Fluent jargon	Left anterior and inferior temporal cortex	Ubiquitinopathy (eg, TDP-43)[37]

PPA = primary progressive aphasia.

nonfluent form of PPA, and superior division MCA strokes (**Case 2-9**).

Discourse

Normal communication requires conveying ideas through putting together sentences in a way that makes the idea clear to the listener and augmenting the words and sentences with prosody (changes in vocal pitch, loudness, and duration, to add stress and intonation to convey emotion or meaning), gesture,

Case 2-8

A 68-year-old man had progressive impairment in speech production, characterized by marked anomia, for the past 3 years. He had trouble naming pictures or sounds and thinking of words in conversation. His speech production was slow and hesitant, with long pauses for word retrieval. He understood conversation well. He had marked trouble repeating sentences or writing down phone numbers that were spoken to him. In describing his work as a car mechanic he said, ''I, well, do the cars when they are . . .you know. When your car is. . .it won't go. . . and I, you know, do it. . .and then it can.'' When asked to repeat the sentence, ''The mechanic fixes foreign cars,'' he said, ''The man, like me, does the cars. . .from other. . .other, you know, places. . .big places.'' He was diagnosed with logopenic/phonologic variant PPA.

Comment. This patient is more nonfluent than many patients with logopenic variant PPA because he is so severely anomic. But he has islands of fluently articulated speech with no motor speech problems in oral reading, making nonfluent/agrammatic variant PPA less likely. It is his disproportionate impairment in repetition of sentences that is the key feature of logopenic variant PPA in this case, along with spared motor speech and spared word comprehension. Patients are sometimes described as having a primary progressive conduction aphasia, because of their poor repetition and phonologic errors.

Case 2-9

A 65-year-old man had sudden-onset difficulty with speech production, with relatively good comprehension. An MRI showed a left posterior frontal stroke. He had mild dysarthria and mild apraxia of speech, but his main problem was in formulating sentences. When asked to describe a picture of a woman washing dishes with the sink flowing over and children behind her stealing cookies from a cookie jar, he said, ''Woman wash disk, no, dish. Water over to foor...floor...and oh no, two kid...cookie... steal...gonna fall, the stool...chaos!'' When asked to repeat, ''It's a sunny day in Maine,'' he said, ''Maine...sunny day.'' His Broca aphasia was caused by occlusion of the superior branch of the left MCA. His oral reading of the same sentence was similar to his repetition.

Comment. The key feature of Broca aphasia in this case is that the patient was agrammatic in all sentence production tasks—spontaneous speech, repetition, and oral reading. He had a motor speech impairment and relatively spared comprehension.

and facial expression. These aspects of communication are more often disrupted by right hemisphere lesions than left hemisphere lesions. Patients with right hemisphere MCA territory stroke or other large lesions of the right hemisphere may have trouble putting together a concise and meaningful story, and often have flat tone of voice even in trying to convey emotional information (**Case 2-10**). Focal dementias involving the right hemisphere (eg, behavioral variant frontotemporal degeneration) or dementias that affect both hemispheres can cause similar problems.

EVALUATION OF LANGUAGE PRODUCTION

The neurologist's bedside evaluation should include short tests of oral and written picture naming, naming from another modality (eg, from a verbal description), and reading aloud. Repeating words with increasing number of syllables (eg, but, butter, butterfly) is also important to distinguish between motor speech impairments. Sentence

Case 2-10

A 72-year-old woman was found by her husband to have left-sided weakness and seemed confused. She was unaware of left-sided weakness initially but later knew that she had left hemiplegia caused by a large right MCA stroke. However, when asked why she was in the hospital she said, ''I was watching baseball on TV. I like to watch baseball and my husband usually watches it with me. It was a good game and the mayor was there. We like the mayor, we voted for him twice. He does a good job.'' When redirected to explain why she was in the hospital, she continued, ''Well, I was watching TV, and then I fell. I fell onto the carpet. It is a new carpet, with swirls in it, and is 2 inches thick. It is a lovely color, and we got it on sale.''

Comment. This response illustrates the difficulty patients with right hemisphere stroke have with getting to the point when speaking. They often address the question without ever answering it. They have trouble sorting out relevant from irrelevant information to integrate components into a whole and meaningful picture.

KEY POINTS

- Patients with right hemisphere middle cerebral artery territory stroke or other large lesions of the right hemisphere may have trouble putting together a concise and meaningful story, and often have flat tone of voice even in trying to convey emotional information.
- The neurologist's bedside evaluation should include oral and written picture naming; naming from another modality; and reading aloud, repeating words with increasing number of syllables to distinguish between motor speech impairments, sentence production, word comprehension, and comprehension and repetition of sentences with various grammatical complexities.

KEY POINTS

- Improvement of language production is common after stroke and other focal, stable lesions. The mainstay of management is speech and language therapy.
- Naming and sentence production are complex tasks, each requiring a number of relatively distinct cognitive processes or representations, which can be selectively disrupted by focal brain damage, most often caused by stroke or neurodegenerative disease (such as PPA).

production should be evaluated through description of complex pictures or responses to open-ended questions (eg, "Why are you in the hospital?"). Oral reading or even scrambled words (anagrams) to formulate into sentences (to tease apart agrammatism from severe anomia as the cause of inability to formulate complete sentences) can also be helpful. Assessment of word comprehension and both comprehension and repetition of sentences with various grammatical complexities is essential to distinguish among the various types of aphasia.

Treatment of Naming and Language Production

Improvement of language production is common after stroke and other focal, stable lesions. The mainstay of management is speech and language therapy. The therapist will identify the patients' priorities in communication and then develop ways to facilitate communication to allow them to reach their goals. Often, therapy involves guided or cued practice in sentence production, naming, or other affected skills. Sometimes the focus is on using intact modalities of communication (gesture, drawing, etc) to compensate for speech and language deficits. Sometimes anomic patients benefit from creating a pocket-sized word notebook, with pages or sections containing names of family members, names of friends, places they like to go, foods they like to eat, and so on. When they are unable to think of a word or name, they can often find it in their notebook. Therapies that involve medications (stimulants, cholinesterase inhibitors, or dopaminergic medications), transcranial magnetic stimulation, or other modalities to augment behavioral therapies are also under investigation.

SUMMARY

Naming and sentence production are complex tasks. Each requires a number of relatively distinct cognitive processes or representations, which can be selectively disrupted by focal brain damage, most often caused by stroke or neurodegenerative disease (such as PPA). Careful analysis of the types of errors and patterns of performance across tasks can provide clues as to the localization and even the most likely pathology in some cases. For example, bilateral but asymmetric anterior and inferior temporal damage is most likely caused by herpes encephalitis or the semantic variant of PPA, the latter usually associated with a ubiquitinopathy.[38,39] These two disorders are easily distinguished by the time course (very rapid onset in the case of herpes encephalitis, and more gradual, progressive course in PPA) and associated symptoms (eg, fever in encephalitis). Understanding the underlying cognitive impairment is also useful in facilitating communication with the affected individual.

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