When Speech Goes Wrong: Evidence from Aphasia

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The sounds of speech are our primary means of communicating with the surrounding world. Speech sounds are critical for communication because they serve as the necessary link to the words, sentences, and meanings of the language system. And yet, speech sounds in both perception and production are vulnerable to errors. Whether fatigued, distracted, or just getting older, we all have experienced times when we have misperceived what was said, as in "let's look for the cape" misheard as "let's look for the cake" (Garnes and Bond, 1980, p. 232), or we have misspoke an intended message, as in "speech production" produced as "preach seduction" (Fromkin, 1980, p. 6). As annoying as these "slips of the ear" and "slips of the tongue" might be, they occur relatively rarely.

Unfortunately, this is not the case for those individuals who have sustained brain injury resulting in aphasia, where deficits in perceiving and producing speech are pervasive. Examining how speech breaks down in aphasia is crucial for understanding the nature of the impairment, and this ultimately informs the aphasia diagnosis and strategies for rehabilitation. Such analyses also afford a unique window into how speech is represented and processed in the brain and how it ultimately connects to language.

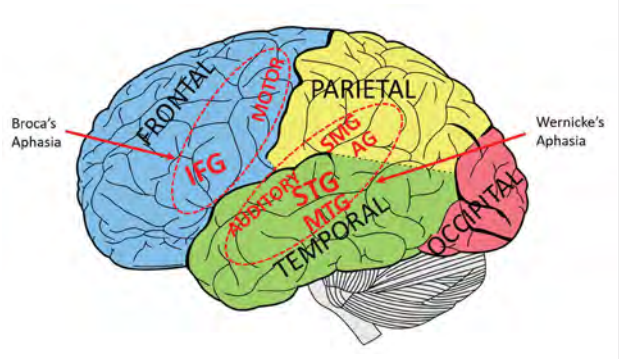
Two avenues of research are followed in this article. First is a look at how brain injury affects the production and perception of speech sounds. Then the acoustic properties giving rise to these sounds are examined. Focusing on the acoustics of speech allows for distinguishing "normal" variability in speech, often not perceived by listeners, from "pathological" variability.

A Brief Introduction to Aphasia

Aphasia can occur at any time across the life span. However, the focus is on those individuals who have sustained a brain injury as adults where language, speech, and the cognitive resources underlying their usage is normal and fully developed prior to the injury. By definition, aphasia is a language disorder affecting speaking and understanding as well as associated language skills such as reading and writing. The National Aphasia Association (see aphasia.org) reports that there are over 2,000,000 people who have aphasia in the United States, with about 180,000 new cases occurring annually. Stroke is the most common cause. Despite the fact that the brain has two structurally symmetrical hemispheres, it is the left hemisphere that is typically dominant for language and hence vulnerable to aphasia.

It is important to understand that there is not one aphasia. The type and severity of language impairment varies as a function of the locus and extent of the brain injury. It is beyond the scope of this article to review all of the

Figure 1. *The left cerebral cortex (**anterior to the left**) showing the four lobes of the human brain: frontal, temporal, parietal, and occipital. The neural areas (inferior frontal gyrus [IFG]; MOTOR; superior temporal gyrus [STG]; middle temporal gyrus [MTG]; AUDITORY; angular [AG] and supramarginal [SMG] gyri; **red**) are involved in language and speech processing. The **red dashed ovals** encircle the neural areas that, when damaged, typically result in Broca's and Wernicke's aphasia. The insula, an area implicated in Broca's aphasia, is not shown because it is located under the surface of the cortex.*



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aphasia syndromes and the details of their underlying neuropathology (see Goodglass, 1993; Damasio, 1998). Rather, I describe here Broca's and Wernicke's aphasia because most of the research exploring speech deficits in aphasia has focused on these two syndromes.

Figure 1 shows a lateral view of the left hemisphere of a human brain and identifies some (but not all) of the neural structures implicated in Broca's and Wernicke's aphasia (Damasio, 1998; Bates et al., 2003). Lesions in the frontal areas of the brain, including the inferior frontal gyrus, the insula, and often motor areas, typically produce Broca's aphasia. In contrast, lesions in the posterior areas, including the superior and middle temporal gyri and often extending into the parietal areas that include the supramarginal and angular gyri, produce Wernicke's aphasia. That said, there is considerable variability in the areas of brain injury that give rise to the classical aphasias, including Broca's and Wernicke's aphasia.

The language deficits in these two types of aphasia are qualitatively different (Goodglass, 1993). Individuals with Broca's aphasia have difficulty speaking: speech quality tends to be slow and labored and often includes sound errors and sound distortions. Additionally, a distinguishing characteristic of this disorder is agrammatism, which means that sentences tend to be grammatically simple and difficulties emerge in the use of grammatical words. For example, "the" may be omitted as well as grammatical endings such as plurals, "books" may be produced as "book." Comprehension, on the other hand, although not perfect, is relatively good.

In contrast, language production in Wernicke's aphasia is fluent and easily articulated. However, it is often empty of semantic content such as "I did it because it was so and I am." Speech errors occur as well as substitutions of words with those that may be semantically related, such as saying "mother" for "father." Additionally, those with Wernicke's aphasia have severe auditory language comprehension impairments.

That Broca's aphasia largely presents with a production impairment and Wernicke's aphasia with a comprehension impairment is not surprising given the neural architecture of the brain. Namely, motor and associated neural structures are located in the frontal lobe, and auditory and associated neural structures are localized in the temporal lobe (see **Figure 1**). However, despite good auditory comprehension, those with Broca's aphasia display speech perception deficits, and despite a fluent, well-articulated speech output, those with Wernicke's aphasia display speech production deficits. This provides the first indication that neither speech perception nor speech production is localized in one circumscribed area of the brain. Rather, the speech systems underlying production and perception are broadly represented in the left hemisphere.

Speech Production Deficits in Aphasia

Speech production errors commonly occur in aphasia regardless of the aphasia syndrome. Sounds may be substituted for each other, "pot" may be produced as "bot"; sounds may be produced in the wrong order, "dummy" is spoken as "muddy"; or they may be deleted, "train" becomes "tain," or added, "same" becomes "stame."

The most common error type for those with aphasia is sound substitution. Analyses of sound substitutions in Broca's and Wernicke's aphasia show both striking similarities and also differences (Lecours and Lhermitte, 1969; Blumstein, 1973; Haley et al., 2013). First, to the similarities. For individuals with either Broca's or Wernicke's aphasia, the occurrence of sound substitutions is variable, and it is not possible to predict if and when a substitution error will occur. Importantly, however, there is a pattern to these sound substitutions. When they occur, the substituted sound and target sound are "similar" to each other. What does this mean?

Speech sounds in language differ from one another in systematic ways. They can be broken down into a smaller set of dimensions or *features* that reflect how the sounds are articulated and their associated acoustic correlates (Jakobson et al., 1952). For example, as shown in **Figure 2**, consonant sounds like [b], [d], and [t] may be broken down into features that indicate how they are produced (their manner of articulation), where in the mouth they are produced (their place of articulation), and what the vocal cords are doing during the production of the sound (voicing). [b] is a consonant produced with a closure in the mouth, the closure is at the lips, and the vocal cords are vibrating during production. It is distinguished from [d] by one feature, place of articulation, and it is distinguished from [t] by two features, place of articulation and voicing; in the production of [d], the vocal cords are vibrating as the consonant is produced

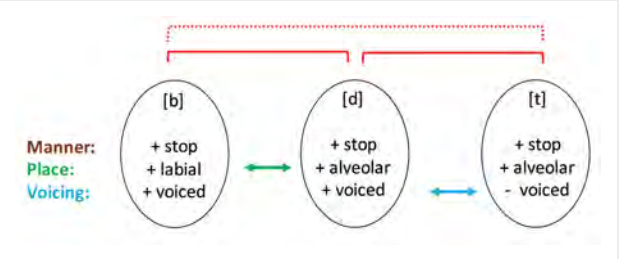


Figure 2. *The network associated with the consonants [b], [d], and [t] and the phonetic features defining them.* ***Red lines*** *signify their positions in in the network:* ***solid line****, closest sounds;* ***dotted line****, more distant sounds.*

(hence, it is called *voiced*), whereas in the production of [t], the vocal cords vibrate after the consonant is produced (hence it is called *voiceless*; see **Figure 2**). Thus, [b] is more "similar" to [d] than it is to [t] in terms of both its articulation and its acoustic correlates.

Critically, in current computational and neural models (McClelland and Elman, 1986; Mesgarani et al., 2014), speech sounds and their features are connected to each other in a network-like architecture, the strength of their connections varying depending on how close or similar the sounds are to each other in terms of their articulatory and acoustic features. Sounds that are more similar to each other are closer to each other and hence are more likely to influence each other.

The pattern of sound substitution errors in aphasia reflect this relationship. The more similar sounds are to one another, the more likely they are to trigger a substitution error. There are many more one-feature errors, such as [b] produced as [d], than errors of more than one feature, such as [b] produced as [t] (see **Figure 2**) in both Broca's and Wernicke's aphasia and irrespective of the lesion locus.

A number of conclusions can be drawn from the common pattern of sound substitution errors in Broca's and Wernicke's aphasia. First, the sounds of language are not lost but are vulnerable to errors. Second, that the same pattern emerges across syndromes and brain areas suggests that the speech production system is neurally distributed and not narrowly localized. Additionally, a common deficit underlies the production of sounds in Broca's and Wernicke's aphasia. Here, I propose that the brain injury introduces *noise* into the system, leaving intact the architecture (the connections) of the system and the speech sounds and their features. However, the differences are *blurred*, making sounds distinguished by a single feature more likely to be mistakenly selected. Interestingly, similar patterns occur in the production of sound substitutions in analyses of "slips of the tongue" (Fromkin, 1980), indicating that the occasional lapses in production that you and I have reflect "temporary" noise introduced into an otherwise normal production system.

One important difference does emerge between Broca's and Wernicke's aphasia. There are many more production errors in Broca's than in Wernicke's. These results are not surprising, given that the frontal areas are recruited for motor planning and articulatory implementation (see **Figure 1**).

On To Acoustics

One of the challenges in analyzing speech production errors is the difficulty of ensuring that examiners can accurately identify correct and incorrect productions. Indeed, how listeners perceive the speech of others is shaped and biased by their native language (Cutler, 2012), making it difficult, even with training, to accurately phonetically transcribe speech.

A case in point is the *foreign accent syndrome*. The characteristics of this syndrome are well-described by its name. After brain injury, a speaker sounds like she is now talking with a foreign accent (watch youtu.be/uLxhSu3UuU4). For example, someone who speaks English may now sound like she is a French speaker talking in English. There is often disagreement among listeners about what foreign accent the patient seems to have "acquired" (Farish et al., 2020). The question is whether the patient has truly acquired a foreign accent or whether the listener is perceiving the changes in the patient's speech incorrectly as a foreign accent.

Acoustic analyses can provide the answer by examining the different ways language articulates the sounds of its language. For example, a common feature used across languages of the world is voicing (Lisker and Abramson, 1964). Voicing relates to the timing of the vibration of the vocal cords (called *voice-onset time* [VOT]) as a sound is being produced (**Figure 3**, *left*). Both English and French use VOT to distinguish the voiced stop consonants [b d g] from the voiceless stop consonants [p t k] but not in the same way.

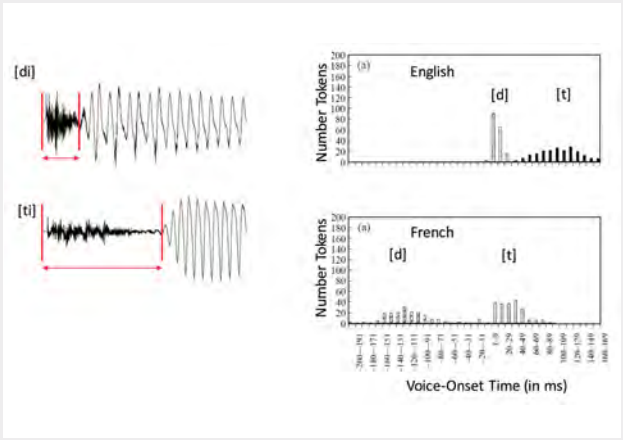


Figure 3. ***Left:*** *measurement of voice-onset time (VOT) for the syllables [di] and [ti]. VOT is the time (**red arrows**) between the release of the stop consonant closure (**left vertical line**) and the onset of voicing (**right vertical line**).* ***Right:*** *VOT distributions for [d] and [t] produced by English and French speakers. Note that English [d] and French [t] have similar distributions. Reproduced from Kessinger and Blumstein (1997).*

Figure 3, *right*, shows that in English, there is a short delay in the onset of vocal cord vibration relative to the release of the stop consonant in the production of [d] and a long delay in the onset of voicing relative to the release of the stop consonant in the production of [t]. In contrast, in French, [d] is produced with voicing beginning before the release of the stop consonant, whereas [t] is produced with a short delay in the onset of voicing. Thus, the two languages differ in how they produce voicing in stop consonants. Indeed, English [d] and French [t] are both produced with a short delay in voicing.

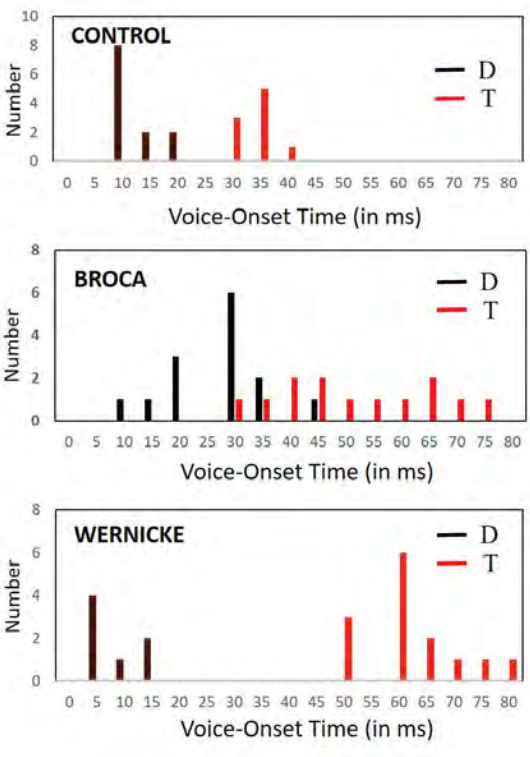
The question then is whether a foreign accent syndrome patient who speaks English, now with a French sounding foreign accent, produces the voicing difference between [p-b], [t-d], and [k-g] as a French speaker or as an English speaker. Analyses showed that the production of voicing was the same as an English speaker, *not* a French speaker (Blumstein et al., 1987). Further examination showed that the foreign accent was not a foreign accent at all. Rather, there were global changes to a number of parameters of English, none of which could be associated with the sounds of any other language. In other words, acoustic analysis revealed that the foreign accent was in the "ears" of the listener, not in the "voice" of the speaker.

Acoustic Analysis of Speech Production in Aphasia

As **Figure 3**, *right*, shows, speakers do not have sufficient articulatory control to produce exactly the same VOT value every time they produce a voiced or voiceless stop consonant. Instead, there is a range of values for each category. Importantly, there is no overlap in VOT between voiced and voiceless stops.

The story is different in aphasia (Blumstein et al., 1980; Itoh et al., 1982; Verhaegen et al., 2020). As **Figure 4** shows, those with Broca's aphasia show a deficit in producing VOT. They fail to maintain the distinction between voiced and voiceless stops. Instead, there is an overlap in VOT between voiced and voiceless sounds, suggesting that they have an articulatory problem in laryngeal (vocal fold) timing. This is not the case in Wernicke's aphasia where production of voicing is similar to the normal pattern.

Figure 4. *The VOT distribution of alveolar stop consonants produced by a control subject (**top**), a Broca's aphasic (**center**), and a Wernicke's aphasic (**bottom**). Number is the number of productions at a particular VOT value. **Black bars**, target voiced stops, [d] (D); **red bars**, target voiceless stops, [t] (T).*



These findings indicate that, like Wernicke's aphasics, Broca's aphasics display a deficit in the selection of sounds and their features. However, unlike Wernicke's aphasia, their deficit also affects articulatory implementation of those features, reflecting the role of the frontal areas of the brain in the motor planning and production of speech.

It is clear from these results and those from the foreign accent syndrome that a listener's ears cannot always identify the nature of speech production deficits. Such is also the case for sound substitution errors, such as producing [da] as [ta]. Indeed, there has been considerable debate in the literature about whether these errors reflect the mis-selection of a sound followed by its correct production or a *misperception* by the examiner that the produced sound was a substitution error (Buckingham and Yule, 1987). Here again, acoustic analysis can provide an answer.

Kurowski and Blumstein (2016) examined the acoustic characteristics of sound substitution errors between [s] and [z]. They compared the acoustics of [s]-[z] substitutions with correct productions of these sounds. The question was whether the perceived correct productions and the substitution errors were acoustically the same or different. Results showed that [s]-substitution errors were more [z]-like than correct productions of [s], and [z]-substitutions were more [s]-like.

These findings support the network-like architecture of the sounds of language described in **Speech Production Deficits in Aphasia**. Sounds are "connected" to one another in the network not only in their selection but also in their implementation. A selected sound partially activates and hence *coactivates* a sound similar to it in the network. As a consequence, a sound substitution contains a *trace* or acoustic residue of its coactivated neighbor.

The Perception of Speech

A first step in considering speech perception deficits is examining the extent to which those with aphasia can discriminate differences between speech sounds. An impairment in distinguishing sounds like [p] and [b] would not only indicate a deficit in perceiving the sounds of speech but could affect the discrimination of words like "pear" and "bear," potentially influencing language comprehension.

Similar to production, perception is influenced by the articulatory-acoustic feature distance between the sounds of speech. Sounds distinguished by one feature are harder to discriminate than sounds distinguished by more than one feature, whether discriminating nonsense syllables or words. This pattern emerges across languages, aphasia syndromes, and lesion site (Basso et al., 1977; Blumstein et al., 1977a), suggesting that speech perception recruits both anterior and posterior brain structures and that the properties of sound are organized, as in production, in a network-like architecture based on articulatory and acoustic similarity.

Although the pattern of errors is the same in discriminating nonsense syllables and real words, there are more errors discriminating nonsense syllables, even though the same sound contrast is compared (Blumstein et al., 1977a). For example, discriminating [pa] versus [ba] results in more errors than discriminating "peak" versus "beak." These results are not surprising. Words can be discriminated based on differences in their sounds as well as their meaning. Nonwords can only be discriminated based on their sounds.

One important difference has emerged between Broca's and Wernicke's aphasia. The number of speech discrimination errors was substantially greater in Wernicke's aphasia. Again, neuroanatomy provides an explanation. Primary auditory areas are located in the temporal lobe (see **Figure 1**). Here, the acoustic input is transformed into the acoustic features of speech (Turkeltaub and Coslett, 2010; Mesgarani et al., 2014). Hence, speech perception is more vulnerable with damage to the temporal areas than to the frontal areas.

The poor performance of patients with Wernicke's aphasia raises the possibility that their speech perception impairment underlies or contributes to the severity of their auditory comprehension deficits. This possibility was first noted by Luria and Hutton (1977) who proposed that the poor auditory comprehension in Wernicke's aphasia reflected a deficit in "phonemic hearing." In this case, misperception of similar sounds could result in perceiving the wrong words, leading to comprehension failures.

This hypothesis was tested by giving subjects an auditory word-picture matching task. Here, subjects point to the picture of an auditorily presented word from a group of pictures that includes a target word, such as "pear," a word perceptually similar to the target, "bear," and a word related in meaning to the target such as "apple." (Both pear and

apple are fruits). If a deficit in phonemic hearing underlies comprehension impairments, then performance on the matching task should predict auditory comprehension; the poorer the performance on the matching task, the poorer the performance on an auditory comprehension task.

Recent research with a large number of subjects, careful clinical assessment, lesion mapping, and well-constructed test materials has shown strong support for the "phonemic hearing" hypothesis (Robson et al., 2012a). Nonetheless, it is also the case that lesions resulting in Wernicke's aphasia may include the middle temporal gyrus (see **Figure 1**), an area implicated in semantic processing (Binder et al., 2009; Bonilha et al., 2017). Thus, it is possible that auditory comprehension deficits reflect both speech perception impairments and semantic impairments (Robson et al. 2012b). Regardless, it is clear that speech perception deficits have repercussions throughout the language system, affecting not only the perception of sounds but also access to words and their meanings (see also Blumstein, 2009; Dial and Martin, 2017).

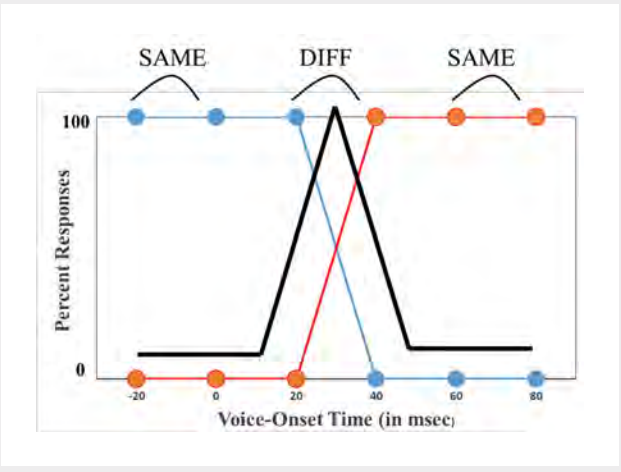
Of importance, the overall patterns of speech perception impairments mirror those shown in speech production. First, similar to speech production, deficits emerge in both Broca's and Wernicke's aphasia despite the fact that the lesions producing these syndromes differ, indicating that the speech perception system is neurally distributed, encompassing more neural areas than just the temporal lobe. Second, the pattern of errors suggests that the sounds of language are composed of features organized in a network-like structure based on their articulatory and acoustic similarities. Third, brain injury does not affect the architecture of speech but introduces noise into the system, rendering similar sounds more likely to be misperceived than sounds further apart in the network. Fourth, despite similar patterns of errors, the number of errors distinguishes Broca's and Wernicke's aphasia, reflecting the neural areas specialized for perception on one hand and production on the other.

Perception of the Acoustic Properties Underlying Speech

The perception of speech requires an auditory analysis of the speech input and a mapping of this input onto the acoustic features associated with the sounds of language. A question is whether speech perception deficits in aphasia reflect failures to perceive these acoustic features normally. For example, as discussed in ***On To Acoustics***, VOT is a temporal cue that distinguishes voiced, [b d g], from voiceless stop consonants, [p t k]. As in production, there is a range of VOT values associated with voiced stops and voiceless stops (see **Figures 3** and **4**). Construction of a synthetic speech continuum with equal VOT steps (in milliseconds) between stimuli shows that listeners separate the stimuli into two distinct categories (see **Figure 5**). Importantly, despite equal acoustic steps, listeners can only discriminate stimuli that cross the phonetic boundary between voiced and voiceless stops, and they fail to discriminate stimuli that lie within either the voiced or voiceless phonetic category (Liberman et al., 1967). Thus, speech is perceived *categorically*, with discrimination ability limited by category membership.

The perception of VOT is severely affected in aphasia regardless of clinical syndrome or lesion site (Carpenter and Rutherford, 1973; Basso et al., 1977). Discrimination performance is typically better than categorization (Blumstein et al., 1977b; Gow and Caplan, 1996). Importantly, discrimination functions are similar to those shown for non-brain-injured subjects, even for those aphasics who are unable to categorize the stimuli. This dissociation between categorization and

Figure 5. *Idealized representation of categorization and discrimination responses to a continuum of synthetic [da]-[ta] stimuli ranging in VOT from −20 to +80 ms in 20-ms steps. Subjects failed to discriminate stimuli within either the [d] (**blue line**) or [t] (**red line**) category, but could discriminate stimuli (**black line**) perceived as members of two different (DIFF; blue vs. red) categories, i.e. [d] and [t].*



discrimination performance again supports the view that the architecture of speech (the connections between sounds) is spared. The perception of the acoustic features of speech sounds is also spared in aphasia, but there is a deficit in identifying the category to which a sound belongs. Naming or identifying heard sounds is more difficult because it requires not only perceiving the acoustic cues but also classifying them into their appropriate sound.

Conclusion

As an experiment in nature, the study of aphasia has provided a unique window into the intersection of mind (speech) and brain (neurology). It not only has informed the scientific study of speech, but it has also placed this research in the context of a disorder that affects millions of people and their families.

A number of broad conclusions can be drawn from this review. In particular, both speech production and speech perception are neurally distributed, encompassing posterior and anterior brain structures, with the exception being those neural areas underlying speech output (motor areas) and speech input (auditory areas). Whether examining speech production or speech perception impairments, the architecture of the speech system is spared. That is, the sounds of language and the features associated with them as well as the network connecting them are intact. Brain injury introduces noise into the system, rendering production and perception processes less efficient and more prone to errors, which are nonetheless systematic.

This same pattern of results emerges even in the absence of brain injury. Single-feature errors are more common in production, as shown in analyses of slips of the tongue (Buckingham, 1992), and in perception, as shown by analyses of errors when listening to speech in noise (Miller and Nicely, 1955; Cutler et al., 2008). The similarity in the pattern of errors in speech production and perception in individuals with and without brain injury suggests that these two speech systems share a common representation for both sounds and their features. As **Figure 6** shows, in speech perception, the analysis of spectrotemporal (frequency-time) properties map onto sounds and features and, ultimately, to the words (lexicon) of a language. In speech production, the selection of a word maps to sounds and features and then to articulatory commands that implement the spectrotemporal properties of sounds.

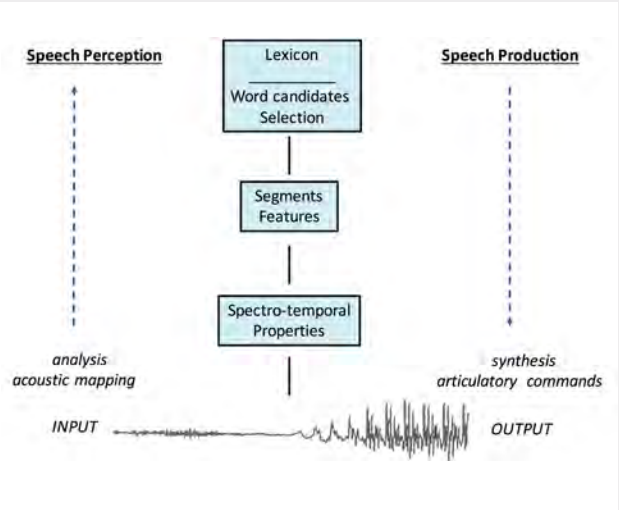


Figure 6. *A theoretical model of speech perception and speech production showing shared representations for the stages involved in speech perception and speech production.* ***Left up arrow****, direction of information flow in speech perception. The acoustic input that the listener hears is analyzed and converted to spectrotemporal properties of speech that are then mapped onto segments (sounds) and their associated features. These sounds access the lexicon (the mental dictionary) where a word candidate is selected from the set of words that sound similar to the candidate word.* ***Right down arrow****, direction of information flow in the stages involved in speech production. A word is selected from the words in the lexicon that are similar to it. The sounds of the word are represented in segments and their associated features, which are then put together (synthesized) and converted via articulatory commands to an acoustic output to be heard by the listener.*

Taken together, speech production and speech perception are integrated systems that are critical for language communication. They not only connect the speaker and listener to the outside world but also shape the internal structure and organization of the language system.

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