

experimental factor. The initial consonant was varied along six steps between /g/ and /k/ and the following context was either /Ift/ or /Is/. Thus, there were six levels of bottom-up phonological information s_i and two contexts c_j . A free parameter is necessary for each level of bottom-up information but it is reasonable to assume that the contextual support given by /Is/ is one minus the lexical support given by /Ift/ so that only one value of c_j needs to be estimated. Thus, seven free parameters are used to predict the 12 independent points.

The lines in Fig. 3 also give the predictions of the FLMP. As can be seen in the figure, the model generally provides a good description of the results of this study. The root mean squared deviation (RMSD) between predicted and obtained is 0.017 on the average across all 12 independent fits. For the 10 subjects showing appropriate context effects, the RMSD ranges from 0.003 to 0.045 with a median of 0.007. Thus, for each of these individuals, the model captures the observed interaction between phonological information and lexical context: the effect of context was greater to the extent that the phonological information was ambiguous. This yields a pattern of curves in the shape of an American football, which is a trademark of the FLMP.

The model tests have established that perceivers integrate top-down and bottom-up information in language processing, as described by the FLMP. This result means that sensory information and context are integrated in the same manner as several sources of bottom-up information. These results pose problems for autonomous models of language processing.

See also: Audition and Hearing; Speech Production, Neural Basis of; Speech Production, Psychology of; Speech Recognition and Production by Machines

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D. W. Massaro

Speech Production, Neural Basis of

1. Brain Regions Involved in Speech Production

Over a century ago, the French neurologist Paul Broca demonstrated that speech mechanisms could be localized in the human brain. He did this by interviewing a patient with a severe speech production disorder with output limited to the recurring utterance, 'tan.' Upon the patient's death, Broca examined the brain and concluded that the patient's inability to speak was due to a lesion in the inferior part of the frontal lobe (Broca 1861b). A second patient also had severely reduced speech and was subsequently found to have a similar cortical lesion (Broca 1861a). Since the time of Broca, scientists have found that lesions to Broca's area alone are not enough to produce lasting speech deficits (e.g., Alexander et al., 1989; Dronkers et al. 2000; Mohr 1976). Many have attempted to diagram

and map additional areas that might also subserve speech and language functions. This chapter will focus on evidence related to brain mechanisms of speech production. Specifically, evidence will be reviewed from lesion studies, electrocortical stimulation, and functional neuroimaging, all of which are helping to elucidate the neural basis of the planning and execution of articulatory movements.

The involvement of these neural structures can best be understood in the framework of a working model that follows the utterance from its conception to its ultimate articulation. Numerous studies have confirmed that the temporal lobe is important for the translation of concepts into linguistic representations. Once the utterance has been linguistically formulated, it must be transferred to the speech mechanisms that will carry out its production. Articulation itself requires planning, initiation, modification, and execution. In addition to Broca's area, it has become clear that many areas are involved in these aspects of speech production. New research has suggested a role for the anterior insula in articulatory planning, the supplementary motor area for the initiation of sequential speech movements, the basal ganglia and the cerebellum in the modification of pitch, loudness and rate, and the primary motor face cortex and pre-motor cortex in the execution of articulatory movements. Each of these areas will be discussed below in terms of the lesion studies that have helped to identify them and the functional neuroimaging research that has corroborated these findings. This chapter will begin by discussing the structures that are believed to convey linguistic information to frontal lobe speech mechanisms from posterior language cortex and end with a description of those mechanisms that execute the articulatory plan.

Superior longitudinal and arcuate fasciculi. The arcuate fasciculus is a bundle of fibers that connects the temporal and frontal lobes. As it travels through the parietal lobe, it becomes part of the superior longitudinal fasciculus and incorporates parietal fibers as well. The arcuate fasciculus was identified by Norman Geschwind as the tract that could potentially connect Wernicke's language area to Broca's speech area as described in Wernicke's original model (see Geschwind 1970; Wernicke 1874). The Wernicke-Geschwind model predicts that disruption of this tract results in isolated repetition deficits. However, recently a more comprehensive disorder has been reported. Dronkers and colleagues found that lesions that sever the arcuate/superior longitudinal fasciculus result in a severe production deficit with a complete loss of propositional speech (see Dronkers et al. 2000). Only automatized utterances remain. These patients have lost more than just repetition skills—they are unable to transfer any information from temporal lobe language areas to anterior speech mechanisms.

Broca's two original patients were also noted to have this same production disorder, which Broca

termed 'aphemia' (Broca 1861a; 1861b). Dronkers et al. (submitted) recently had the opportunity to scan the postmortem brains of Broca's two original patients. They found that deep lesions also existed in these brains, in addition to the lesions Broca noted in the inferior frontal lobe. These deep lesions involved the superior longitudinal fasciculus in both cases, suggesting that this lesion also played a role in these historic patients' production deficits.

Insular cortex. The insula is a region of neocortex hidden beneath the intersection of the frontal, parietal, and temporal lobes. In the past, the insula has been implicated only vaguely in speech production (e.g., Ojemann and Whitaker 1978; Tognola and Vignolo 1980). More recently, Dronkers (1996) linked lesions in a specific region of the insula to an inability to plan and coordinate the appropriate movements necessary for articulation. She found that all patients studied with this disorder, known as 'apraxia of speech,' had lesions in the superior tip of the precentral gyrus of the insula while those without speech apraxia did not. Dronkers concluded that this small portion of the insula is critical for coordinating articulatory movements.

In keeping with this conclusion, recent neuroimaging studies have reported activation in the insula with tasks such as articulation of single words, word reading, picture naming, and word generation (see Indefrey and Levelt 2000). Interestingly, the insula appears to be activated only when tasks involve articulation of non-repeated and phonologically complex words (e.g., Wise et al. 1999), but not when only automatic or simple articulatory patterns are produced (e.g. Murphy et al. 1997).

Supplementary motor cortex. The supplementary motor area is located in the superior frontal gyrus and extends medially between the hemispheres. Penfield and others described the participation of the supplementary motor area in speech by documenting the effects of electrocortical stimulation on this region in patients undergoing surgical removal of epileptic tissue (see Penfield and Roberts 1959). Stimulation to the supplementary motor area either caused patients to make involuntary vocalizations or interrupted their ability to speak.

Infarctions of the supplementary motor area often result in a transcortical motor aphasia (See Rapcsak and Rubens 1994 for a review). Spontaneous speech is initially dysfluent, but comprehension, repetition, and reading remain relatively intact. Similarly, complete excision of the supplementary motor area causes a transient aphasia with deficits in articulation that resolve quickly (Penfield and Roberts 1959).

A review of the lesion literature suggests that the supplementary motor area is involved in the initiation of sequential, voluntary movements, including those for speech (e.g., Ziegler et al. 1997). This also seems to be true for the right supplementary motor area, as demonstrated by several case studies as well as

functional imaging data (e.g., Indefrey and Levelt 2000; Laplane et al. 1977). Other functional neuroimaging tasks that elicit supplementary motor cortex activation include the control of breathing for speech and vocalization (Murphy et al. 1997) and automatic speech (e.g., reciting months of the year; Ackermann et al. 1998).

Basal ganglia. The basal ganglia are subcortical nuclei comprised of the caudate, putamen, and globus pallidus. These structures interact with the cortex and with a number of other subcortical structures in a series of feedback loops that help to maintain motor activity (see Love and Webb 1996). Lesions to the basal ganglia can result in several types of movement disorders including Parkinson's and Huntington's disease, which each have their own speech characteristics (Duffy 1995). Patients with Parkinson's disease (which results from a lack of dopaminergic innervation of the caudate/putamen) show hypokinetic speech with decreased intensity and little modulation of pitch or loudness. In contrast, patients with Huntington's disease (which results from a loss of GABAergic, inhibitory neurons in the caudate) exhibit hyperkinetic speech with erratic control of pitch and loudness. These data suggest a role for the basal ganglia in the modulation of articulation and phonation.

Similar disorders can be seen in patients with focal lesions in the basal ganglia. For example, Pickett et al. (1998) reported a case study of a patient with bilateral lesions in the putamen and caudate. Her speech was initially characterized as dysarthric and was very difficult to understand, with poor articulatory and phonatory control. Similarly, Fabbro and colleagues reported at least two cases in which lesions in the basal ganglia resulted in hypophonia and a reduction of speech initiation and output (Fabbro et al. 1996).

In functional neuroimaging studies, caudate activation has been noted in only a small number of speech production studies (see Indefrey and Levelt 2000), but this may be due to the fact that it is not generally treated as a region of interest in imaging studies of speech.

Cerebellum. The cerebellum has traditionally been thought to be involved in fine coordination of motor acts including speech. Evidence for this comes from patients with focal cerebellar lesions. Ataxic dysarthria following cerebellar injury is a motor speech disorder that appears to be due to a discoordination in muscular control of the speech apparatus, as opposed to loss of initiation or execution (Duffy 1995). Mutism has also been associated with midline lesions in the cerebellum, in both children and adults (Coplin et al. 1997). However, this mutism is often transient and can resolve into ataxic dysarthric speech.

A broader theory of cerebellar function suggests that the cerebellum acts as a timing mechanism. That is, the cerebellum's role in motor coordination (for speech and other coordinated motor acts) is based on

a more general mechanism that allows for the precise temporal control of such movements (Ivry and Keele 1989). Consistent with this hypothesis, Gandour and Dardarananda (1984) and others have shown that cerebellar patients have abnormal voice onset time distributions, indicating a timing disorder.

In the late 1990s, the cerebellum was implicated in speech production as a store for verbal short-term memory. Silveri et al. (1998) studied a patient following right cerebellar hemispherectomy and found that the patient had poor verbal short-term memory due to an impaired phonological output buffer. Functional neuroimaging data have also suggested that the cerebellum may play a role in verbal short-term memory (Ivry and Fiez 2000).

The role of the cerebellum in speech is probably multi-faceted, based on the functional heterogeneity of the cerebellum in motor control. Functional neuroimaging studies may help to tease out the functions of the various subregions of the cerebellum and their roles in speech and language.

Broca's Area. Traditionally, the literature has emphasized Broca's area as a major structure for speech production. However, the function of Broca's area has been re-evaluated. Data from stroke patients and from neurosurgical excision have revealed that removal/injury to Broca's area results in a transient mutism that resolves in 3–6 weeks (Dronkers et al. 2000; Penfield and Roberts 1959). Also, focal lesions to Broca's area do not result in a persisting Broca's aphasia, and Broca's aphasia may result from lesions outside of Broca's area (Dronkers et al. 2000).

With the increasing acknowledgment of the contribution of other brain areas to speech production (discussed in this chapter), Broca's area is no longer considered to be as crucial to speech as once thought. Though clearly involved in articulation, Broca's area functions within a network of brain regions that support speech production. Indeed, neuroimaging studies have reported networks of activation that include Broca's area in tasks involving phonological encoding and articulation, such as picture naming, word generation, and reading (see Indefrey and Levelt 2000).

Motor face and pre-motor cortex. The muscles of the vocal mechanism are innervated by cranial nerves V, VII, IX, X, XI, and XII, which receive impulses from the cortex via the corticobulbar tract. The motor face area of primary motor cortex, as well as pre-motor cortex, are believed to be the source of these projections (Duffy 1995). Lesions to these areas result in a pure motor speech disorder with slow, effortful speech and impaired articulation but intact language (Alexander et al. 1989). Such lesions are presumed to disrupt the corticobulbar fibers that activate the cranial nerves necessary for speech.

Penfield and colleagues originally showed that a number of regions in primary motor and pre-motor cortex were involved in speech output, as electro-

cortical stimulation of these sites resulted in speech arrest (see Penfield and Roberts 1959). Similarly, Ojemann and Mateer (1979) showed that, in four cases, speech arrest followed stimulation of inferior motor and premotor regions in the left frontal cortex.

Activation of primary motor cortex has been reported in fMRI studies of speech production. Wildgruber et al. (1996) found that tongue movements elicited comparable right and left activation in the inferior region of the motor strip. Monotone recitation of the months of the year activated more left than right motor cortex, while singing activated more right than left motor cortex. Ackermann et al. (1998) also found left motor cortex activation for silent repetition of months of the year.

2. Conclusions

Much has been learned about the neural processes involved in speech based on studies that have examined the specific deficits associated with lesions in particular brain regions. While language mechanisms cluster in posterior neocortical areas, speech production is generally supported by anterior cortical regions. Broca's area, motor face area, supplementary motor area, and the insula all play important roles in aspects of speech production, as evidenced by the effects of lesions in these areas. The superior longitudinal fasciculus provides the mechanism by which language information is transferred to these anterior speech regions. The basal ganglia and cerebellum also assist in speech production through modulation, coordination, and timing of speech movements. Functional neuroimaging studies have corroborated these findings and have allowed the visualization of the networks involved in speech production on-line.

See also: Language Development, Neural Basis of; Speech Perception; Speech Production, Psychology of; Syntactic Aspects of Language, Neural Basis of

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N. F. Dronkers and J. V. Baldo

Speech Production, Psychology of

Speech production refers to the cognitive processes engaged in going from mind to mouth (Bock 1995), that is, the processes transforming a nonlinguistic conceptual structure representing a communicative intention into a linguistically well-formed utterance. Within cognitive psychology, research concerning speech production has taken various forms such as: research concerning the communicative aspect of speaking; research concerning the phonetics of the produced speech; and research concerning the details of the cognitive processing machinery that translates conceptual structures into well-formed linguistic utterances. We focus on the latter.

Native adult speakers produce on average two to three words per second. These words are retrieved from a lexicon of approximately 30,000 (productively used) words. This is no small feat: producing connected speech not only entails retrieving words from memory, but further entails combining this information into well-formed sentences. Considering the complexity of all the encoding processes involved, it is impressive that we produce speech at such a fast rate while at the same time remaining highly accurate in our production. Bock (1991) estimated that slips of the tongue occur in speech approximately every 1,000 words despite the ample opportunities for errors. In the following sections the cognitive processes subserving this ability will be discussed.

1. Methodological Issues

The prototypical empirical approach in cognitive psychology consists of systematically varying some properties of a stimulus (input), and to measure a corresponding behavior. Systematic relations between

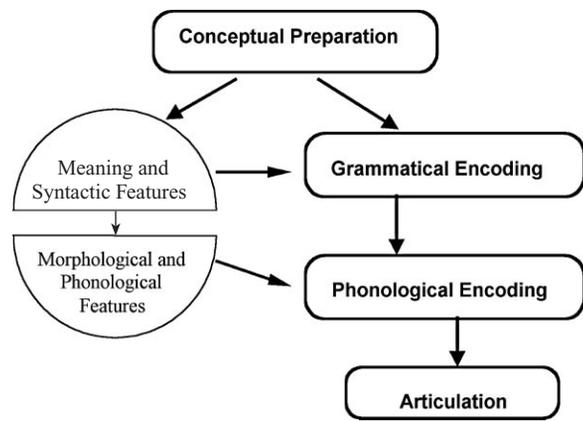


Figure 1

Levels of processing in speech production. Left part refers to stored representations, right part refers to processes integrating these representations into well-formed linguistic utterances

input and behavior are then used to infer the cognitive processes mediating between them. For example, researchers interested in language comprehension may systematically vary properties of a linguistic input such as the syntactic complexity of sentences or the frequency of words, and measure a corresponding behavior, such as reading times. Differences in the latter are taken as an indication of differences in the processes engaged in building an interpretation of a sentence (the output). Thus, the input is directly observable and completely accessible to systematic manipulation, while the output and the cognitive processes leading to it are inferred from the behavior.

The situation is different for research in speech production. The input (the conceptual structure to be expressed), is not directly observable and not readily accessible for experimental manipulation. By contrast, the output (i.e., the spoken utterance), is directly observable. Given this situation, research on speech production has started by focusing on properties of the behavior. Most prominent in this respect are analysis of speech errors, and of hesitations and pauses as they occur in spontaneous speech. These analyses motivated the general theoretical framework for speech production (Fig. 1) which is described in detail below. More recently researchers have started to make speech production accessible to standard experimental approaches from cognitive psychology (see Bock 1996).

2. Levels and Processes

Figure 1 provides an outline of the levels of processing involved in speech production. These levels are shared by most current psycholinguistic models (e.g., Dell 1986, Garrett 1988, Levelt 1989). *Conceptual prep-*