BRAIN DAMAGE AND PHONOLOGICAL ORGANIZATION

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Phonological structure is often studied independently from the other components of language and, until recently, it has been studied in greater detail than these other components for obvious reasons: its accessibility to instrumental measurement and the presumed limitation or finiteness of its scope and content. The result is that we have a very rich set of hypotheses concerning the elements, the structural interrelationships and the processes of the sound system of a number of different languages, as well as a preliminary guess as to what aspects of phonology might be universal to all languages. Since it is not necessarily the case that a linguistic model of phonology is an appropriate characterization of how the human brain organizes the sound system of a language, it seems reasonable to inquire into the extent and success of applying these hypotheses to aphasia, a domain of data not ordinarlly considered by the linguist and phonetician. We would expect a neurologically adequate model of the phonological component to be able to predict the phonetic effects of brain damage on observed speech production and recognition, and to correlate these effects consistently with the underlying brain mechanisms.

Beginning with the initial insight of Broca that the faculty of articulated language is mediated by the foot of the third frontal convolution in the left hemisphere, there has been a steady accretion of knowledge about the language areas of the human brain. However, in the past few years there have been a number of studies which can provide a fairly coherent neuro-linguistic overview of the phonological component, at least with respect to production. Speech perception studies do not appear to conflict with this synthesis, but they will not be discussed in this paper.

Geschwind, Quadfasel and Segarra (1968) reported a case of carbon monoxide poisoning which in effect isolated the phonological component. There was extensive bilateral degeneration of most of the cortex, except in the classical speech areas: Broca's area (foot of the third frontal convolution), Wernicke's area (superior temporal convolution) and the Arcuate fasciculus (white matter fiber tract connecting Wernicke's and Broca's areas) (see Figure 1). This patient had evidently lost all language comprehension in the usual sense — she did not initiate conversations, did not reply to questions, nor demonstrate that she understood what was spoken to her.

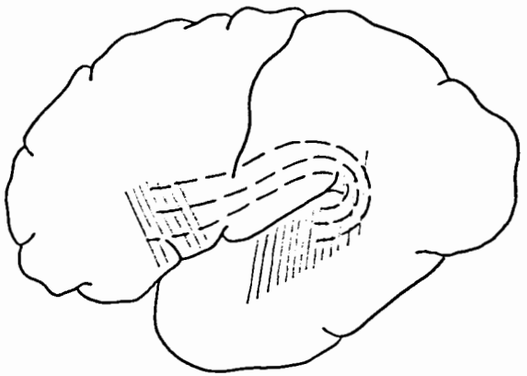

Fig. 1. Cortical structures of the phonological component Broca's area, arcuate fasciculus, Wernicke's area.

Nevertheless, she did retain a number of stereotyped expressions such as greetings, she could repeat things said to her, she would complete songs or proverbs after being given the first line and she demonstrated verbal learning by mastering new songs played on the radio which had not been composed prior to her illness. More importantly, her remaining verbal skills did not have a distorted or erroneous phonetic realization, i.e., there was no dysarthria, which is an inability to move in a normal way specific vocal tract muscles (cf. Espir and Rose 1970) and there was no apraxia of speech, which is an inability to execute or program the proper articulatory sequences in the language (cf. Johns and Darley 1970); in short, the phonology was intact. If the main features of the phonological component are a property of the brain areas outlined in the Geschwind, Quadfasel and Segarra case, then two clear conclusions follow: (a) brain damage in any part of this system should result in phonetic disintegrations that correlate with the phonology of the language in question and (b) brain damage in other parts of the language system should either have no phonetic consequences at all or should have phonetic consequences which are dependent upon semantic and syntactic aspects of the language. In other words, if an articulatory sequence is incorrectly realized in just those cases when a certain class of nouns are being spoken but the sequence is correctly realized in all other cases, the basis of the error is clearly syntactic or semantic and not phonological; we would expect that the classical speech areas are not damaged.

Blumstein (1970) investigated the linguistic nature of speech errors in three aphasic groups; each group was representative of brain damage in each of the three classical speech areas outlined above. Blumstein showed that the pattern of errors was systematic within groups, was similar across groups and reflected the phonological structure of English. Her data gives significant support for an abstract level of phonemic representation, for a distinctive feature analysis, for a psychological hierarchy of features or a marking convention, and for syntagmatic constraints in

the sound system probably at the syllable structure level. A detailed documentation of the phonological aspects of an aphasic patient whose brain damage was not in the classical speech area was done by Kehoe and Whitaker (1971) and Schnitzer (1971). The patient's lesion was located in the supramarginal gyrus (inferior parietal lobe) (see Figure 2). We showed that the phonetic errors could not be attributed to the

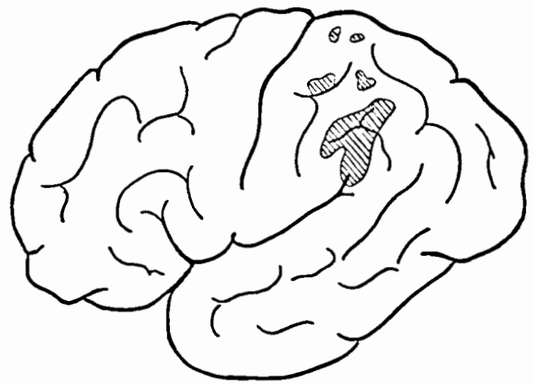

Fig. 2. Supramarginal Gyrus Lesion in case from Kehoe and Whitaker (1971).

length of the word nor to the presence of complex consonant clusters and that the errors were not systematic in terms of phonetic environment, which implies essentially that the patient did not have apraxia of speech or any general impairment of the phonological component as outlined by Blumstein. Instead, we showed that the errors could be systematically related to the derivational complexity of particular words, usually literary words of Latinate origins; e.g., adjectives derived from nouns ('bacteriological') or nominals re-derived from derived lexical sources ('nationalization'), were generally mispronounced by the patient even though nonsense words of equal phonetic complexity were pronounced properly. Given the lexical classification which predicted the occurrence of errors, Schnitzer (1971) then showed how a majority of the wrong phonetic realizations could be explained by postulating minor but plausible mistakes in the underlying dictionary representation. E.g., mistakes in the part of speech, in assigning the proper boundary to the derivational affix, in the proper vowel quality and omissions and substitutions of underlying syllables, were all shown to predict the incorrect phonetic shape just in case one assumed that the phonological component was intact.

The research reported above can be viewed as supporting a neuro-linguistic approach to phonological organization in which linguistic, psychological and neurological insights complement each other. One of the more important problems is that of extending a model based on brain damage to account for the normally functioning brain. We have taken some preliminary steps in this direction (Whitaker and McAdam 1971, McAdam and Whitaker 1971) in our studies of normal subjects' electro-

encephalograms (EEG) associated with language production. Greater EEG activity is localized over Broca's area prior to speaking than in other motor facial areas of the cortex; conversely, the EEG activity for vocal tract gestures such as spitting or coughing is evenly distributed in both hemispheres. In some pilot work not yet experimentally verified we found evidence that increased EEG activity is localized in Broca's area during the production of nonsense words 'polufratz', 'peenotine', etc., and multiple syllable repetitions 'papapa', 'tatata', etc., which leads us to hypothesize that Broca's area may control the vocal tract in part in syllable-sized chunks. We have evidence both from the EEG studies and the aphasia studies which suggests that very little semantic or syntactic encoding takes place in Broca's area; hopefully, future research can make more explicit the particular contribution of the three cortical structures of the phonological component.

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DISCUSSION

PILCH (Freiburg im Breisgau)

1. How did you make your patient repeat utterances, as she was (by your report) unable to respond to speech?

2. What is the realistic basis of your diagram (if any)?

WHITAKER

According to Geschwind, Quadfasel and Segarra, their patient responded spontaneously to various utterances. E.g., on occasion the patient was told "Ask me no questions," to which she replied "I'll tell you no lies". This is referred to as a COMPLETION PHENOMENON and is by no means rare in aphasia. What the mechanism for this spontaneity is, remains open to speculation.

It was perhaps not completely clear in the brief presentation above that the basis of the diagram was the post-mortem neuropathology of two patients, the one of Geschwind *et al.* and the one of Kehoe and Whitaker. In the cases reported by Blumstein, there was a complete neurological diagnosis on each patient, done by the neurological service at the Boston VA Hospital. With respect to the normal subjects studied by McAdam and Whitaker, the localization data is perhaps a bit less pinpointed since EEG signals picked up by an electrode can originate from an extent of cortex larger than the electrode itself. While these cases may not be completely convincing, one should remember that they are completely consistent with countless other case histories reported in the literature since 1861.

MACNEILAGE (Austin, Tex.)

Was there any subcortical involvement in Geschwind's patient, and if not, could you include the pulvinar in your model?

WHITAKER

There was subcortical involvement in the patient of Geschwind, Quadfasel and Segarra. They described lesions in the basal ganglia, particularly the globus pallidus, some damage to the thalamic nuclei (although the postero-lateral ventral nuclei were well preserved) and a moderate loss of neurons in the pulvinar. There was a moderate loss of cells in the cerebellum but the brain stem was within normal limits. The anatomical dissection in this case was superbly done and a brief resume such as this hardly does credit to the detail presented in their paper. It is somewhat surprising that no dysarthria was present; presumably the extent of degeneration in the basal ganglia was not sufficient to cause this speech disorder. With respect to the pulvinar, yes, it would definitely be incorporated in any adequate model of the language system in the brain. Although it is still unclear exactly what role it may have, a number of studies based upon electrical stimulation and neurosurgically produced lesions have shown that the left pulvinar (in left hemisphere dominant persons) is involved in the system for retrieval from the lexicon. Stimulation and lesions generally cause an anomia comparable to that caused by lesions to the posterior temporal and inferior parietal regions. However, the long-range follow-up of such cases seems to indicate a clearing of this aphasia deficit; this is not generally seen with cortical lesions. Extending this further at this time would, I think, be even more speculative than this paper has been.

BOND (Columbus, Ohio)
In regard to the second patient you discussed, would you please elaborate: what sort of errors lead you to believe that the errors were in lexical representation?

WHITAKER
Phonetic errors of a peripheral nature (apraxia of speech and/or dysarthria) have certain predictable characteristics. In the case of apraxia, specific consonant clusters are more subject to error than others, the initial segments of words are more likely to be mispronounced than the final, longer words are more difficult than shorter, and the semantic/syntactic features of the word have little if any affect. In the case of dysarthria, specific muscles are affected and hence the phonetic error is predictable on that basis alone. The patient we studied made errors which typically related a derived form to its base form; i.e., most difficulty was experienced on the derivational affixes rather than on the stem morpheme. Nonsense words of equal phonetic complexity (number of syllables and consonant clusters) were pronounced without error. This led us to consider the hypothesis that the lexical representation of these words (the derived or "literary" words in English) might be affected while the realization rules for specifying the final phonetic shape might be intact. This hypothesis was strongly supported by Schnitzer [1971] who demonstrated that minor errors in the lexical representation would in fact predict the actual output of the patient. Some of the errors which Schnitzer identified were noun vs. verb category error, incorrect choice of derivational affix, incorrect choice of the feature [tense/lax] in the vowel of the base (lexical) form, and the like.

DARWIN
Did the patient with damage to the supra-marginal gyrus have any further brain damage? What was the origin of her pathology?

WHITAKER
As indicated in the diagram, there were a few very small lesions in the superior parietal lobe of the left hemisphere with the same etiology as the supra-marginal gyrus lesion. These lesions were due to the pressure on the cortical surface generated by a very massive subdural hematoma, caused by a ruptured artery. In addition, there was a very old superficial lesion in the temporal lobe, approximately one-fourth the size of the supra-marginal gyrus lesion, which affected only the first two or three layers of neurons in the cortex; both the neurologist and the neuro-pathologist agreed that this lesion was unlikely to have caused any of the patient's recent problems; it was several years old.