427

Mapping speech: More analysis, less synthesis, please

Michael Studdert-Kennedy

Queens College, City University of New York, Flushing, N.Y. 11367; and Haskins Laboratories, New Haven, Conn. 06510

Stimulation mapping would be of little interest if its achievement were merely to assign brain loci to categories of linguistic or psychological description. Our understanding of complex, intermodal functions, such as naming or reading, would be blocked rather than advanced if we were to conclude, as Ojemann speculates, that each is a macrocolumn or module, an impenetrable, vitreous chip in the great mosaic of language. The promise of stimulation mapping is rather that it may reveal, by some pattern of association and dissociation, the simpler mechanisms from which a function emerges and, ultimately, its underlying neural circuitry. To fulfill this promise, stimulation studies should not adopt uncritically the familiar, nonanalytic, modality-based tests of aphasia assessment. Rather, these tests or others must be given a functional analysis in terms of clearcut psycholinguistic hypotheses. Naming, for example, is not a unitary function: Naming errors may reflect perceptual, semantic, or phonological deficits (Goodglass 1980), and the source of naming errors may often be inferred from their form (e.g., Katz 1982). Similarly, deficits in oral reading are open to increasingly sophisticated analysis in terms of phonological segmentation, lexical access, and phonetic execution (e.g., Liberman, Liberman, Mattingly & Shankweiler 1980). Unfortunately, little in the target article suggests that systematic analysis of this kind was attempted.

Ojemann did, however, test one "lower" function that might plausibly be expected to enter into a pattern of relations with several others, namely, orofacial mimicry. If the ability to produce simple movements of the mouth is impaired, one would not be surprised if the ability to speak, in naming or reading, or even to recall a word (if short-term storage engages a motor representation) were also impaired. In fact, very often these relations were observed (though not with perfect consistency). Yet interpretation of even this modest pattern of associations is hazardous.

Before turning to this, consider how we might interpret the most controversial link with impaired mimicry: impaired phoneme identification. The key, at least to the frontal lobe sites, is hinted at in the sparsely reported findings of Darwin, Taylor, and Milner (Ettlinger, Teuber & Milner 1975, p. 132), cited by Ojemann. These authors discovered that patients in whom the left facial regions had been excised for relief of epilepsy were impaired both in spelling from dictation and in the same phoneme identification task as Ojemann used. Since these patients could understand and talk normally, Darwin and his colleagues concluded that their difficulty was confined to tasks stressing the phonetic structure of speech sounds. This conclusion implies that the identification of phonemes in nonsense words may be essentially a nonlinguistic task (inasmuch as it bypasses the lexical and syntactic processes of normal speech perception), a task very close, in fact, to mimicry. Ojemann stresses that stimulation during the phoneme identification test occurred only during presentation of the stimulus. However, since the patients simply had to reproduce what they heard, their task reduced to finding, *during presentation*, the motor pattern specified by the stimulus. Moreover, if, as Ojemann suggests, motor sequence programs were stored in the temporo-parietal region, this account would also handle the temporo-parietal links between phoneme identification (accomplished by execution of a two-syllable nonsense word) and three-gesture mimicry.

We may note, incidentally, that all phoneme identification tasks, which call for metalinguistic judgments, may reveal more about structural correspondences between audition and articulation than about normal processes of speech perception. Accordingly, even if a motor-reference theory of speech perception were still viable, Ojemann's findings would have little bearing on it. In fact, the link between perception and production is probably deeper and less tortuous than the old motor theory proposed. As Ojemann himself hints, the link is clearest in language acquisition, when the child learns to speak by discovering the articulatory dynamics specified by the speech he or she hears.

I come next to the pattern of associations and dissociations between functions, rather confusingly charted for a seven-subject series in Ojemann's Figures 3 and 4. Apparently, the circles of Figure 3 correspond to the large circles of Figure 4. There are 52 large circles (25 frontal, 9 parietal, 18 temporal), representing sites where all five language functions were tested. In addition, Figure 4 displays (by my count) 27 small circles (4 frontal, 10 parietal, 13 temporal), representing sites where all functions except orofacial mimicry were tested: This brings us to a total of 79 sites. Of these, 15 seem to have yielded no result, leaving us with 64 effective sites (24 frontal, 18 parietal, 22 temporal), an average of about 9 per patient. By arduous tabulation, we can discover the links between functions impaired at each site – though not, unfortunately, how these links were distributed across subjects.

Among my findings was the fact, touched on by Ojemann in his discussion of discrete localization, that every function (except, interestingly, mimicry) is disturbed alone on at least one site, a dissociation that effectively demonstrates the absence of causal relations among the functions in at least those patients who display it. But what is most remarkable is that every function (except single-gesture mimicry, confined to the frontal lobe) is impaired in every area. Thus, short-term memory (STM) is impaired at 16/24 frontal, 11/18 parietal, 7/22 temporal sites; reading and/or naming are impaired at 13/24 frontal, 9/18 parietal, 11/22 temporal sites; three-gesture mimicry is impaired at 4/23 frontal, 5/8 parietal, 5/13 temporal sites; phoneme identification is impaired at 9/24 frontal, 5/18 parietal, and 6/22 temporal sites. How are we to square this distribution with Ojemann's model, assigning retrieval to the frontal, storage to the temporo-parietal lobes? Moreover, Ojemann reports that STM was tested with stimulation at the time of input, storage, or retrieval, but these distinctions are not preserved in the report of the data. Were all frontal STM deficits confined to retrieval, all temporo-parietal deficits to storage?

The problem worsens as soon as we leave these statistical patterns and consider individual subjects – a reasonable move if we are interested in mechanism. Ojemann acknowledges a high degree of individual variability, but assures us that "the interrelationships described in the model can be readily identified in

individual patients, such as the one illustrated in Figure 2." Why, then, were we not given a comparable figure (or at least a table) laying out the pattern of relations for each subject? Yet, even if we had the individual data, we would have to be cautious in interpretation. If two functions are dissociated, we can be confident that there is no necessary connection between them. However, even if they are regularly associated, we cannot infer a necessary connection. This is so because electrodes are large relative to nervous tissue, so that we cannot be sure that the association is not due to blocking of distinct, though closely neighboring, functions. These limitations are inherent in the stimulation-mapping technique in its present state of refinement.

On the other hand, as Ojemann argues, the fact that certain associations do recur over wide areas of peri-Sylvian cortex is encouragement enough for continued research. Ojemann is to be honored for rediscovering a valuable technique, the most precise that we have to analyze the neural circuitry of functioning human cortex, and for leading the way toward important new discoveries.

ACKNOWLEDGMENTS

My thanks to Virginia Mann for advice. Preparation of this comment was supported in part by NICHD Grant No. HD 01994 to Haskins Laboratories.