Panel: Unusual Aphasias: Their Implications for a Definition of and the Neurological Basis for Aphasia: Introduction

Robert T. Wertz Veterans Administration Medical Center, Martinez, California

The comfortable notion that aphasia erupts following damage to the left hemisphere has been challenged by reports of subcortical aphasia, slowly progressive aphasia, and crossed aphasia. These disrupt the traditional definition of aphasia and muddle most conceptions of the neurological bases of aphasia. This session includes individual presentations on subcortical aphasia, crossed aphasia, and slowly progressive aphasia followed by a discussion of their implications for a definition of aphasia and the neurological substrates for language.

Subcortical Aphasia. Aphasia resulting from subcortical lesions has crept into the literature. Some have even subdivided it into anterior and posterior subcortical aphasia. Most reports include the presence of dysarthria coexisting with some, not all, subcortical aphasias. The presenter, a behavioral neurologist, has reviewed the reported cases and added some of his own. In all, there is evidence of a subcortical lesion or lesions. However, in most, there is also evidence of coexisting cerebral pathology. Thus, are the reported cases subcortical aphasia, or are they cases of aphasia with coexisting cortical and subcortical lesions? The purpose of the presentation is to suggest that if we are really serious about the existence of subcortical aphasia, we should try to prove it by utilizing the tools at our disposal -- CT, PETT, MRI, and detailed speech and language analysis. Until we produce studies of aphasia subsequent to well-defined subcortical lesions with no coexisting pathology elsewhere, we may want to be careful in promoting the presence of subcortical aphasia.

Crossed Aphasia. The notion of crossed aphasia is not new. Early definitions have been refined to reserve crossed aphasia for those righthanded persons who become aphasic following a right hemisphere lesion. Reports of incidence range from less than one percent to two percent. Thus, though rare, crossed aphasia challenges the concept that language resides exclusively in the left hemisphere. Further, acknowledging the existence of aphasia and crossed aphasia excludes another small group of patients; the left-handed who are aphasic following a right hemisphere lesion. The presenter, a neuropsychologist and neurolinguist, will review the reported cases of crossed aphasia and present a comparison of crossed and uncrossed aphasic patients from her own clinic. When right-handed aphasic patients with left hemisphere lesions were matched on the basis of intrahemispheric location and size of lesion with right-handed aphasic patients with right hemisphere lesions, crossed aphasia, and a left-handed aphasic patient with a right hemisphere lesion, differences emerged. Generally, there were few differences between crossed and uncrossed aphasic patients in auditory comprehension and reading. However, crossed aphasic patients displayed better oral-expressive language and writing than their uncrossed aphasic cohorts. In addition, some of the crossed aphasic patients displayed symptoms (e.g., disrupted visual-spatial abilities) seen in right hemisphere brain damaged nonaphasic patients. Speculation about what crossed aphasia contributes to our understanding of language organization in the brain is a part of the purpose of this presentation.

Slowly Progressive Aphasia. The notion that aphasia erupts has been buffeted by reports of slowly progressive aphasia. Thus, the rapidity of onset which used to differentiate aphasia from dementia may not be as solid a sign as once believed. The presenter, a speech-language pathologist, will review the reported cases and add some from his own clinic. Comparison of cases of aphasia subsequent to a rapid onset with those that appear to be slowly progressive should refute or confirm whether the latter are really aphasic or demented. Slowly progressive cases who display symptoms of both aphasia and dementia but whose language is disproportionally disrupted will be given special attention. Further, the literature is confined to slowly progressive patients whose aphasia is fluent. However the presenter has documented a possible case of slowly progressive aphasia that was nonfluent. The ramifications of slowly progressive aphasia are many. For example, what is the slowly progressive patient's future? Does the aphasia progress and then subside, or does it continue to progress? What are the implications for management? Considering whether what has been called slowly progressive aphasia is really aphasia and, if it is, what we can do about it constitute this presentation's purpose.

Discussant. The existence of subcortical aphasia, crossed aphasia, and slowly progressive aphasia suggest a need to reexamine or, at least, worry about the way we have been defining aphasia. If these aphasias exist, our current concepts of the neurosubstrates of language need reconsideration. Perhaps there is a need to focus on vascularity rather than cortex as a basis for understanding and classifying what we call aphasia. Certainly, the reality of subcortical, crossed, and slowly progressive aphasias has implications for management. All of those things that we do less than well with aphasia as we currently understand it -- appraisal, diagnosis, prognosis, focusing treatment -- are vitiated by aphasia resulting from subcortical or right hemisphere lesions or aphasia whose onset creeps. The discussant, a speech-language pathologist will mince, dice, slice, and blend the material in the three previous presentations. Possible conclusions are that we have been and continue to be right as rain, or we should quit acting like all aphasic people got that way following a stroke involving the left hemisphere middle cerebral artery. One might suspect he will lean toward the latter.