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NEUROBEHAVIORAL GRAND ROUNDS—INTRODUCTION

Comments on a case of pure word deafness

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Perception of speech is effortless under most circumstances; this may explain why the complexity of this cognitive process is so often unappreciated. While most other sounds in the environment are acoustically very distinctive, the 30 or 40 speech sounds people make are discriminable only by rapid analysis of rather subtle acoustic cues. Given the importance of language, it seems plausible not only that our auditory system evolved a degree of specialization for handling such analyses, but also that this specialization should assimilate substantial neural resources. Recent work in comparative physiology and human brain imaging suggests, in fact, that much of the superior temporal gyrus in humans is devoted to this perceptual feat.

Damage to this system is the topic aptly addressed by Stefanatos, Gershkoff, and Madigan in this inaugural Neurobehavioral Grand Rounds feature of *JINS*. Although relatively rare, the syndrome of pure word deafness has been acknowledged as a clinical fact for over a century. Despite numerous case reports, both the functional and anatomical bases of the syndrome remain in dispute. The functional dispute focuses on whether the deficit affects speech-specific processes or more general, nonlinguistic auditory processes. Stefanatos et al. provide important new evidence on this matter by demonstrating a defect involving analysis of the spectrotemporal form of rapid acoustic events. Their patient was severely impaired at discriminating complex tones ("isolated formants") moving rapidly in different directions in the frequency domain. This rapid analysis of spectrotemporal form is critical for discriminating certain types of speech sounds, particularly consonants, and less critical for recognition of (steady-state) vowels. Further tests confirmed that the patient performed much more poorly with consonants than with vowels.

Anatomical discussions of pure word deafness often center on whether a unilateral lesion can cause the syndrome. The vast majority of patients have had bilateral damage, which also tends to cause more severe deficits and more obvious impairment with nonspeech environmental sounds. The case documented by Stefanatos et al. provides clear confirmation that a unilateral lesion in the superior temporal gyrus, if sufficiently extensive, can cause lasting pure word deafness. As the authors point out in their concise but thorough review, the available case material suggests important differences between the unilateral and bilateral cases, with unilateral lesions more likely to affect processes that are particularly relevant to speech perception, and the bilateral lesions producing more general auditory impairments. This issue aside, virtually nothing is known concerning how placement of the lesion within each hemisphere affects the pattern of resulting deficits. Resolving these questions will require careful behavioral and anatomical studies. Unfortunately this has not been the norm for case studies of pure word deafness, which typically emphasize behavioral performance patterns or anatomical data, but rarely both at once. The case of Stefanatos et al. is an important exception to this general rule.

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