Matters arising
Alexia and agraphia in Wernicke's aphasia

Sir; In the J Neurol Neurosurg Psychiatry, Dr Kirshner et al presented three patients who had “otherwise typical Wernicke’s aphasia”, in whom reading appeared to be disproportionately impaired relative to auditory comprehension. The authors claim that these cases represent exceptions to the more commonly seen patients with Wernicke’s aphasia, in whom auditory and reading comprehension are impaired to an approximately equal degree. We take exception, however, to the diagnostic classification of the patients discussed in this paper, and would argue that none of the three patients in fact represent examples of Wernicke’s aphasia.

Although there are no generally agreed criteria for classification of aphasic patients, one of the most commonly used systems is that of the Boston Diagnostic Aphasia Examination (BDAE). According to Goodglass and Kaplan, the critical features of Wernicke’s aphasia are markedly impaired auditory comprehension that “is evident even at the one-word level” and “fluent articulated paraphasic speech”. The BDAE range of auditory comprehension performance compatible with the diagnosis of Wernicke’s aphasia is given as a z-score of -2.0 to -0.5. Although there was not sufficient information to calculate actual z-scores for the three patients reported, based on the test results that were given, it is highly unlikely that any of the three patients would have had a mean z-score within that range. Case 1 demonstrated normal responses to multistep commands and complex ideational material (the two most difficult BDAE subtests of auditory comprehension) already at 4 days after onset. The second case showed severely impaired comprehension during the first day or so, but over the next few days her “auditory comprehension and following of commands recovered substantially, except for right-left errors”, and “resting six weeks after onset revealed almost normal auditory comprehension” (p721). The third patient, when tested at bedside 6 days after onset, “followed 8/8 spoken commands of increasing complexity and answered appropriately to yes/no and nonsense questions” (p721). The patients also were given the auditory version of the Token Test between 16 days and 4 weeks post-onset, and none of them obtained scores of less than 90% correct, suggesting that the auditory comprehension deficit had resolved almost completely by at least the fourth week. Six patients, with the BDAE diagnosis of Wernicke’s aphasia recently seen in our centre, had a mean Token Test (auditory presentation) score of 31.2% correct (SD = 24.8%) at four weeks post-onset. A group of four patients with the BDAE diagnosis of conduction aphasia, in comparison had a mean score of 77.8% correct (SD = 14.1%). None of the three patients reported by Kirshner et al therefore was impaired sufficiently on tests of auditory comprehension to be described as a typical case of Wernicke’s aphasia.

The authors consider the alternative explanation that their patients in fact had a conduction aphasia and alexia with agraphia, but dismiss this possibility because the patients were not “typical” of that syndrome either. They conclude that “the important point, however, is the linkage of alexia with agraphia to the fluent aphasia”. (p723). If that were the main point of the report, we would agree that this combination does occur, but we would simultaneously stress that such a combination would not be very surprising. Both fluent alexias and alexia with agraphia have been reported to occur in association with posterior lesions, and thus, the combination of these deficits might be expected. However, the authors place considerable emphasis, both in the text and in the title, on the classification of these patients as having Wernicke’s aphasia. They maintain that their findings are evidence that a selective reading deficit is not uncommon in Wernicke’s aphasia, in opposition to the frequent assertion in the literature that Wernicke’s aphasia is a supramodal disturbance in which reading and auditory comprehension typically are impaired about equally. A second problem with the data presented is that the dissociation of auditory and reading comprehension was not a very persistent one for at least two of these fluent patients. A comparison of the auditory and visual mode of presentation of the Token Test revealed only a 5% discrepancy at 16 days after onset for Case 3, and only a 12% discrepancy at 4 weeks after onset for Case 1. Only in Case 2 did a marked dissociation persist.

Although the cases reported are of importance in that they document a rapid amelioration of comprehension deficits in the very acute stage of recovery from aphasia, the patients reported, in our opinion, do not represent examples of Wernicke’s aphasia. Nor is there evidence that the disproportionate impairment of reading comprehension persisted beyond approximately 4 weeks after onset for two of these three patients. The issue of modality biases in aphasic syndromes is of great theoretical interest; however, since these patients did not have Wernicke’s aphasia, they do not represent an exception to the widely held opinion that reading and auditory comprehension are commonly impaired to a similar degree in Wernicke’s aphasia.

References

Kirshner & Webb reply:

Drs Selnes and Niccum take issue with the diagnosis of Wernicke’s aphasia in our three cases with alexia out of proportion to auditory comprehension deficit. As they themselves state, the diagnosis of Wernicke’s aphasia is not agreed upon generally by any statistical definitions. Aphasiologists have danced on the head of this terminological pin for over a century. Dr Goodglass, an originator of the Boston Diagnostic Aphasia Examination (BDAE), recently stated at a symposium on aphasia classification that the BDAE was not meant to be used as a set of rigid statistical criteria for classification of aphasic cases, but rather as a means of gathering standardised information. Wernicke’s aphasia is a clinical diagnosis, based on the central features of fluent, paraphasic speech and impaired auditory comprehension. These central features were clearly present in all three of our patients at an early stage in their deficits, and the clinical neurologists and speech pathologists involved in their care had no difficulty in recognising them as such. We agree that their deficit profiles and scores on the BDAE evolved in a direction not considered typical of Wernicke’s aphasia; it was just this atypicality which prompted us to report their case histories. We anticipated the argument that these cases might simply represent examples of a separate syndrome, such as pure alexia with agraphia. We therefore described the full evolution