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.¹ 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).² 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 "fluently 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 left-right errors", and "retesting 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 aphasias". (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 aphasias 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.

OLA A SELNES

NANCY NICCUM

Hennepin Medical Center,
701 Park Avenue So,
Minneapolis,
Minnesota 55415, USA

References

- ¹ Kirshner HS, Webb WG. Alexia and agraphia in Wernicke's aphasia. *J Neurol Neurosurg Psychiatry*, 1982;45:719-24.
- ² Goodglass H, Kaplan E. *The Assessment of Aphasia and Related Disorders*. Philadelphia: Lea and Febiger, 1972:59-61.

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.¹ As they themselves state, the diagnosis of Wernicke's aphasia is not agreed upon generally by any statistical definition. 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),² 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.³ 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

Matters arising

from typical Wernicke's aphasia to the later picture of disproportionate but not "pure" alexia and agraphia. The persistent aphasic deficits of paraphasic speech and mildly impaired auditory comprehension still showed the residual fluent aphasia.

As stated in our paper, we sought to stress not the issue of nomenclature of aphasia classification, but rather the point that reading and auditory comprehension deficits do not always go hand in hand in fluent aphasia. Our earlier, companion paper,⁴ which described cases of Wernicke's aphasia with poor auditory comprehension but intact reading, also exemplifies the dissociation of modalities in some cases of fluent aphasia. The numerous other studies cited in that paper provide additional evidence for this dissociation. We believe that the exploration of modality differences in aphasic syndromes is not only of theoretical interest but also of importance in guiding strategies of speech and language rehabilitation, through the use of spared modalities.

HOWARD S KIRSHNER

WANDA G WEBB

Dept. of Neurology,
Vanderbil University,
Nashville, Tennessee 37232, USA

References

- Kirshner HS, Webb WG. Alexia and agraphia in Wernicke's aphasia. *J Neurol Neurosurg Psychiatry* 1982;**45**:719-24.
- Goodglass H, Kaplan E. *The Assessment of Aphasia and Related Disturbances*. Philadelphia: Lea and Febiger, 1972.
- Goodglass H. Symposium on Classification of Aphasia at the Academy of Aphasia, Mohonk, New York, October, 1982.
- Kirshner HS, Webb WG, Duncan GW. Word Deafness in Wernicke's Aphasia. *J Neurol Neurosurg Psychiatry* 1981;**44**:197-210.

Facial palsy and diabetes mellitus

Sir: The question of the association and possible aetiological relationship between Bell's palsy and diabetes mellitus, something of a hardy perennial, has been raised once more in the article by P Pecket and A Schattner.¹ They reported finding chemical or overt diabetes in 39% of a series of 126 patients with Bell's palsy. Other reports reflect a wide divergence of opinion regarding the incidence of diabetes in this condition, ranging from 6% abnormal glucose tolerance tests² to an astonishing 66%.³ It seems likely that such gross discrepancies can only be explained by either differences in methodology or patient

selection, or both. In Korczyn's series³ capillary blood was used for glucose estimation, a method shown by Mosenthal⁴ to give values at 2 hours after glucose ingestion, some 40 mg/100 ml higher than those determined by examining venous blood. In our own series of 60 patients⁵ we were able to demonstrate the crucial importance of the patient's age. In the age range of 10-19 years all tests were normal and 20-49 year-olds yielded only three abnormal results. However, in the age group 50-69 years, 85.7% were abnormal, and over the age of 70 years all patients had reduced tolerance. Venous blood was examined in all cases. This importance of the age factor was confirmed by Adour and associates⁶ with rates ranging from 3.8% (age 30-39) to 38.5% (age over 60 years). Nonetheless these impaired tolerance rates were considerably less in non-paralytic individuals of the same age (1.3% and 7.0% respectively), and the authors concluded that diabetes is more common among patients with Bell's palsy than among those who had never had this disease. However, Adour in his most recent publication on the subject⁷ states his belief that it is almost universally accepted that Bell's palsy is a viral inflammatory demyelinating disease, manifested by patchy areas of longitudinal involvement extending from the brain stem to the periphery. Although warnings against attempts at precise topographical localisation by clinical testing are given by Tonning,⁸ the finding by Pecket and Schattner¹ of taste impairment in 83% of their non-diabetic patients and in only 14% of the diabetics is striking, and may indeed point to two different pathological and aetiological processes. It would be interesting to know, in addition to the question of the type of blood used in their study (capillary or venous), how their various findings are related to the age of their patients, before any conclusions can be derived regarding the role, primary or secondary, of diabetes mellitus in the aetiology of Bell's palsy.

J BRAHAM

J TURGMAN

I SAROVA-PINHAS

Y GOLDHAMMER

Department of Neurology,
Sheba Medical Centre, Tel-Hashomer,
Sackler School of Medicine,
Tel Aviv University, Israel

References

- Pecket P, Schattner A. Concurrent Bell's palsy

and diabetes mellitus: a diabetic mononeuropathy? *J Neurol Neurosurg Psychiatry* 1982;**45**:652-5.

- Aminoff MJ, Miller AL. The prevalence of diabetes mellitus in patients with Bell's palsy. *Acta Neurol Scand* 1972;**48**:381-5.

- Korczyn AD. Bell's palsy and diabetes mellitus. *Lancet* 1971;**i**:108-9.

- Mosenthal HO. Interpretation of glucose tolerance tests. *Med Clin North Am* 1947;**31**:299-312.

- Turgman J, Sarova-Pinhas I, Goldhammer Y, Braham J. Prevalence of glucose intolerance in patients with Bell's palsy. *Israel J Med Sci* 1973;**9**:8;1054-56.

- Adour KK, Wingerd MA, Doty HE. Prevalence of concurrent diabetes mellitus in idiopathic facial paralysis (Bell's palsy) *Diabetes* 1975;**24**:449-51.

- Adour KK. Diagnosis and management of facial paralysis. *New England J Med* 1982;**307**:348-51.

- Tonning FM. The reliability of level-diagnostic examinations in acute peripheral facial palsy. *Acta Otolaryngol.* 1977;**84**:414-5.

CT scans suggestive of tumour in multiple sclerosis

Sir: The paper of Sagar, Warlow, Sheldon and Esiri¹ describing unusual CT scan appearances suggesting cerebral tumours in three cases of multiple sclerosis is interesting. We would like to report a similar case and comment on the diagnostic and therapeutic management.

A 57-year-old woman with an 11 year history of multiple sclerosis presented in December 1979 with a history of a grand mal seizure followed by a progressive left hemiplegia for three weeks. New clinical findings consisted of a hypotonic hemiparesis most pronounced in the leg. A CT scan showed a right frontal subcortical enhancing lesion with oedema, suggesting a metastasis (fig 1A). Search for a primary tumour was negative and dexamethasone at a dose of 9 mg a day was followed by rapid clinical improvement. Follow-up CT scans at three and six weeks (fig 1B) disclosed progressive disappearance of both oedema and the lesion. The recovery of the left hemiparesis persisted for three years.

Although the therapeutic effect of corticosteroids in multiple sclerosis remains questionable² we feel that in the case of a large cerebral lesion with oedema, therapy with dexamethasone may provide some benefit by reducing the oedema and thus local compression and neurological deficit. In addition repeat scans in the following weeks will further differentiate between a tumour and multiple sclerosis plaque, spar-