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, Vanderbilt University, Nashville, Tennessee 37232, USA

References

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 series4 capillary blood was used for glucose estimation, a method shown by Mosenthal5 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 patients6 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 Tonnig,8 the finding by Pecket and Schattner4 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 GOLDMAMMER
Department of Neurology, Sheba Medical Centre, Tel-Hashomer, Sackler School of Medicine, Tel Aviv University, Israel

References

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 frontotemporal 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-
Facial palsy and diabetes mellitus.

J Braham, J Turgman, I Sarova-Pinhas and Y Goldhammer

*J Neurol Neurosurg Psychiatry* 1983 46: 463
doi: 10.1136/jnnp.46.5.463

Updated information and services can be found at:
http://jnnp.bmj.com/content/46/5/463.1.citation

*Email alerting service*

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

*Notes*

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/