

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.

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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 Tønning,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.

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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-

ing the patient a cerebral biopsy and not altering substantially the prognosis in the case of a tumour, as has previously been suggested by van der Velden *et al* in a similar case.³

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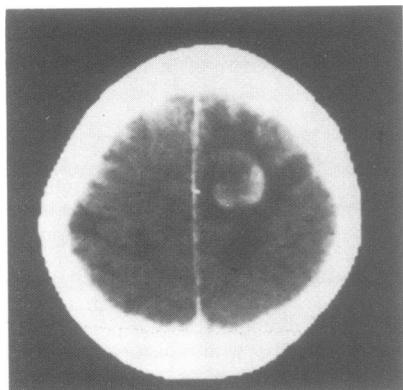


Fig 1A 1st CT scan showing an enhancing lesion in the right frontal area.

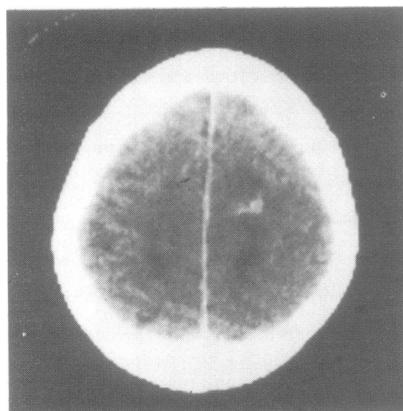


Fig 1B CT scan at 6 weeks showing marked regression of the lesion.

Sagar *et al* reply:

We were interested to hear of the case of multiple sclerosis described by Vanneste and Davies, although we have not seen the CT scan pictures. This case, and the one recently reported by Abbott *et al*¹ are similar to the eight cases reported and reviewed by us in showing not only mass effect on their CT scans but also the "cerebral" form of the disease (in these two cases, aphasia and/or seizures).

We agree that increased recognition of these features of multiple sclerosis should reduce the need for diagnostic biopsy particularly in cases with a compatible past history. Attempts to improve the clinical state by treatment with dexamethasone may be reasonable in a very sick patient in whom the differential diagnosis is limited but, as Vanneste and Davies remark, its efficacy in the disease is questionable. In general, it is clearly important to base the investigation and management on the precise clinical and radiological features of each case since these scan appearances may be due to lesions other than glioma or multiple sclerosis and are occasionally caused by infective lesions.^{2,3}

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A case of receptive amusia with prominent timbre perception defect

Sir: Mazzucchi *et al*¹ describe a patient with a right temporal lesion and found that the patient had deficits in recognising the timbre of music and other sounds. These deficits are remarkably similar to disturbances in prosody which also occur with right hemisphere lesions. As Weintrub *et al*¹ (Arch Neurol 1981;**38**:742-4) pointed out prosody "refers to the distribution of stress and melodic contour in speech.

Modulation of prosody can thus be used to impart affective tone, introduce subtle shades of meaning, and vary emphasis in spoken language". In patients with disturbances in prosody secondary to right hemisphere lesions, such deficits as inability to distinguish stress patterns in compound words and inability to discriminate intonations and phonetic stress in sentences were found. It would thus appear that the timbre perception defect described by Mazzucchi *et al* was a particular form of dysprosody.

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- ¹ Mazzucchi A, Marhini C, Budai R, Parma M. A case of receptive amusia with prominent timbre perception defect. *J Neurol Neurosurg Psychiatry* 1982;**45**:644-7.

Mazzucchi replies:

We thank Dr Procopis for the comments on our paper *A case of receptive amusia with prominent timbre defect*. We would however underline that prosody and timbre correspond unquestionably to different definitions: *prosody*, as reported by Dr Procopis, refers to "distribution of stress and melodic contour in speech", in other terms, prosody is related to a sequence of different sounds in which their *intensity* and *rhythm* vary; *timbre* depends on the harmonic content of the sound, that is it is the peculiar quality of overtones that allows recognition of an instrument or voice as being different from another. Therefore, in *semeiological and descriptive terms*, prosody and timbre must be considered as two distinct concepts and both corresponding disorders may be present in the same patient. If you proceed from the semeiological level to the level of the underlying neural mechanisms, this distinction could be more doubtful. In any case, until now no data exist on the possible relationship between prosody and timbre, just as it is not clear, generally, whether there is a unitary mechanism of sound and musical cognition or, rather, a combination of abilities which are independent of each other.

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