Matters arising

Figure Three examples of the patient's writing production, arranged in chronological order from top (at the beginning of the symptom), centre (about six months after), to bottom (about one and a half years after). Black marks outline right and left page margins.

of paper in about one and a half years. He could write for hours without getting tired. At the beginning of this symptom he could spontaneously write meaningful sentences, with a neat handwriting. His handwriting, which was inattentive to the left, became progressively careless and increasingly unreadable over time (fig.), until his writing finally stopped late in 1987, about one and half years after the beginning of the symptom.

Brain CT scan at the time of admission showed moderate cortical and subcortical atrophy without anterior-posterior or right-left differential involvement, ruling out Pick's disease.2 An EEG showed mainly subcortical changes, which is typically seen in Alzheimer's Disease.3

Polyglucosan bodies are not an unusual finding in temporal lobe epilepsy

A recent short report excited my interest. The report described the massive occurrence of polyglucosan bodies (PBs) in a surgically-resected temporal lobe.1 However, I am afraid that I have to disagree with the authors' contention that the occurrence of PBs is "very unusual in neuropathological examinations of epileptogenic foci."

Of the 40 temporal lobes resected for temporal lobe epilepsy which I recently reviewed, 15 contained corpora amylacea (PBs) in numbers far in excess of what would be expected for the patient's age and the anatomical site. In half of these the PBs were associated with mesial temporal (Ammon's horn) sclerosis and in the remainder they apparently constituted the sole abnormality.2 Over half of the Birmingham cases with excess PBs also had a history of febrile convulsions in childhood, although not all with such a history manifested mesial temporal sclerosis. Although they claim that "unusually large numbers of PBs were the only significant abnormality", the fact that there was "severe neuronal loss in the pyramidal layer of the Ammon's horn" indicates that the case of Loiseau et al. also demonstrated mesial temporal sclerosis.

The association with febrile convulsions and mesial temporal sclerosis suggests that the PBs are a result of either the epileptic activity, or the original process which damaged the temporal lobe, rather than "the origin of an epileptic focus."

The authors do, however, correctly highlight the sparsity of previous references to PBs in epileptic temporal lobes—they are not mentioned in Brutton's detailed study of 249 cases,3 and only one of the 81 specimens studied by Jackson et al. was reported as having unusually large numbers of white matter corpora amylacea.4 It is highly unlikely that the sparsity of reports reflects the rarity of epileptic temporal lobes containing excessive numbers of PBs. It is much more probable that the PBs have previously been overlooked by pathologists, either because their attention has been directed to more obvious pathology within the temporal lobe, or because the numbers of PBs have not been appreciated as excessive in relation to the patients' young ages (34 in Loiseau's case and a mean age of 24.6 for the Liverpool cases), or because they have been poorly stained. (PBs stain intensely with Ehrlich's haematoxylin, which is routinely used in Liverpool, but weakly with Harris' and Meyer's haematoxylin.)

If the interesting report by Loiseau et al. stimulates pathologists to scrutinise more carefully resected temporal lobes, then it will have served a very useful purpose.

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