Assessment of cerebral atrophy: discrepancy between pneumoencephalography and computed tomography

SIR,—The association between radiological cerebral atrophy and dementia is well established, but it is also clear that dementia may occur without atrophy and atrophy without dementia.¹ Thus, when clinically uncertain it may be unwise to rely upon radiological findings, especially since two long-term follow-up studies have shown that the diagnosis of presenile dementia may subsequently be refuted.^{2,3} Both these studies antedated the introduction of computed tomography (CT) and atrophy was demonstrated by pneumoencephalography (PEG). The following case questions the validity of pneumoencephalographic atrophy.

A 49-year-old male clerk was referred for psychiatric opinion because of severe weight loss of 25 kg in six months for which no physical cause could be found. There was no significant previous personal or family history of mental illness. One to two years previously, following difficulties at work, he had visited his doctor with various physical complaints. On being told that his blood cholesterol was slightly elevated he began to diet and lose weight. On admission he was cachectic (weight 54.5 kg) and looked at least twenty years older than his age. He admitted low spirits, but was alert and cognitively intact. There were no psychotic phen-

omena. He took food and drink only with strenuous coaxing. Later there were doubtful and transient indications of cognitive impairment. Extensive laboratory investigation, including CT scan, failed to reveal any abnormality. Five weeks after admission he developed an ataxic gait, upper limb cerebellar signs and a left extensor plantar reflex. PEG was performed under general anaesthesia to exclude an infiltrating lesion in the hypothalamic area which might not have been seen on CT. Soon afterwards his neurological signs receded. His weight remains low and the diagnosis uncertain.

Pneumoencephalography showed moderate, especially frontal, cortical atrophy. The left lateral ventricle was enlarged, septum-caudate line (SCL)=21 mm, maximum width of the body of the lateral ventricle (MWLV)=24 mm. CT scan showed no surface atrophy, except slightly at the right insula. Asymmetry of the lateral ventricles was confirmed but both were of normal size (figure). Because of the neuroradiological discrepancy CT was repeated. The second scan did not differ from the first.

Although not greatly enlarged, the dimensions of the left lateral ventricle on PEG exceeded those found by Mann⁴ to differentiate demented from non-demented patients in a 5–10 year follow-up study (SCL=18 mm, MWLV=20 mm). No demented patient in that study had smaller dimensions, but measures greater than these did not

necessarily indicate dementia. We consider that the PEG findings in our patient may perhaps be due to relative dehydration allied to high oxygenation, consequent lowering of the arterial pCO_2 and cerebral vasoconstriction. This patient, whose fluid intake had already been low, was starved for about 14 hours before PEG. His general state of health caused some concern to the anaesthetist and he was well ventilated during the procedure. A fall in the arterial pCO_2 has a potent vasoconstrictor effect on cerebral arterioles,⁵ and could have contributed to shrinkage of the brain in this case. The use of PEG in the assessment of clinical dementia has greatly diminished with the advent of CT, but the capital cost of scanners is high and the facility is therefore limited to large centres. This case underlines the need for caution in the interpretation of pneumoencephalographic atrophy, and emphasises the advantages of computed tomography.

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Figure

Letters

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