Matters arising

Behavioural manifestations of third ventricular colloid cysts

Sir: I was quite pleased to read the article on behavioural manifestations of third ventricular colloid cysts by Lobosky, Vangilder and Damasio.¹ It is with this in mind that I would like to present the following case. A 67-year-old female has a 1 year history of intermittent periods of confusion, manifested by forgetfulness and inability to do her normal activity. For example, she was driving as a passenger with her husband, and it was time for them to get out of the car. She found herself unable to go through the mechanical action of getting out of the car and actually had to be carried out. Once she was out of the car, she had no difficulty walking into the house. In relation to her skills of daily living, she is able to continue to cook, dress and bathe herself, and go to the store and shop.

Mental status examination revealed that the patient was disoriented to time and place; she thought that she was in her home. She could not remember her own birthdate. When asked to attend to four objects given to test her recent memory, she could not maintain attention long enough to maintain these words in immediate memory; thus making it impossible to check for recent memory deficit. When asked to perform simple commands, she was able to close her eyes or open her mouth. When asked to show her right or left hand, she was confused and unable to do this consistently and accurately. When asked to put her right index finger on her left ear, she responded with, "Do I have to get up to touch my ear?" She seemed a bit confused at that time, and when asked to point to just her ear, she did this accurately. There was no difficulty with naming simple objects such as a watch, a pair of glasses, or a tie. There was no clear evidence of dysarthria or aphasia. There was an inability of the patient to perform complex motor activities. When asked to go from the sitting position to the lying down position, she could not do this. It was clear that this was not on the basis of impaired strength or coordination.

Neurological examinations revealed cranial nerves 2-12 intact. The muscles were of normal tone, bulk and strength. Sensation could not be tested well, owing to poor cooperation, but was intact to pinprick and proprioception. Reflexes were 2+ and symmetric in the upper extremities, 2+ at

the knees, 1+ at the ankles, and the toes were downgoing bilaterally. Finger to nose and heel to shin tests were normal. The gait was normal as were heel walk, toe walk, tandem walk.

CT scan revealed a colloid cyst of the third ventricle without hydrocephalus. EEG revealed diffuse slowing with frontal intermittent rhythmic delta activity of high voltage. Blood studies revealed normal thyroid, liver function, B-12 and folate, and VDRL. Neurosurgical consultation was obtained with three different neurosurgeons, all of whom declined to pursue an operative course.

This case calls to attention behavioural manifestations associated with colloid cyst of the third ventricle without hydrocephalus. In general, it is the impression of my neurosurgical colleagues in the United States that in the absence of hydrocephalus, removal of a colloid cyst is not mandatory.

It is my hope that reports of cases such as this will raise the awareness of behavioural manifestations in colloid cysts of third ventricle without hydrocephalus.

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Reference

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Positive bromide partition test in the absence of tuberculous meningitis

Sir: I was interested to read the paper by Weinberg and Coppack on the bromide partition test.

They may be interested to know that in 1963 we showed that the blood CSF barrier to bromide is also broken down in cases of diffuse meningeal tumour.²

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Neuropsychological evaluation of mild head injury

Sir: Over the last decade, evidence for neuropsychological impairment following mild to moderate closed head injury¹ has been growing.²⁻⁴ Recently, Gentilini *et al*⁵ concluded that any structural damage following mild head injury (PTA < 20 min) did not leave persisting neuropsychological deficits one month post-injury.

This conclusion must be criticised. It is based on a single multivariate analysis on a battery of six neuropsychological tests purporting to measure intelligence, memory and attention. Reasons for selection of the tests used are not given. Previous investigations have shown that mild-moderate head injury causes specific cognitive deficits. Taking the overall result from a battery of tests would obscure these.

Considering each test in turn, deficits in forward digit span are rarely found even in severe head injury (PTA 1-7 days),¹⁰ and although lower performance IQ (Raven's Matrices) is found in more severe cases, there is no evidence to suggest that this should be the case following mild injury. Other tests include verbal recall, verbal recognition and an obscure "working memory test", whose validity must be questioned. Previous findings might however predict deficits in the "Selective Attention Test" if this is indeed a test of attention and separate analysis of this test did in fact reveal poorer performance in the mild head injury group.

Gentilini *et al* argue that it is not acceptable to analyse the tests in the battery separately because of the increased probability of type one errors, and ignore this finding. However, it is striking that published studies have consistently found deficits in attention or the rate of information processing as shown by Gronwall and colleagues using the Paced Auditory Serial Attention Test (PASAT),²⁻⁴ and hence there is an *a priori* reason to analyse attention tests separately.

In conclusion, because selection of tests in the battery used is inappropriate and because there is an *a priori* reason to consider differences in the expected direction on the "Selective Attention Test", it would seem that the findings of Gentilini *et al* can justifiably be reinterpreted as supporting

the view that cognitive deficits exist even after one month post-injury in mild head injury cases, as reported by Gronwall and Wrightson.²

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tive performances might in some way be disturbed after mild head injury. It would therefore be interesting to address the issue of this specific cognitive impairment by a new carefully controlled study

¹ Rimel RW, Giordani B, Barth JT, Boll TJ, Jane JA. Disability caused by minor head injury. *Neurosurgery* 1981;9:221-8.

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Nichelli et al reply

To our belief, prior to this study, consistent *a priori* evidence was not available that impairment after closed head injury was confined to tests evaluating attention. In fact, mild neuropsychological impairment has been reported in the vast majority of Halstead-Reitan Neuropsychological Test Procedures, including tests of higher level cognitive functioning, new problem solving skills, attention, concentration and memory.¹

Therefore one might expect that tapping different abilities should increase and not obscure differences between experimental and control groups. This is why a simple multivariate analysis was preferred. Nevertheless our study provides some support for those studies claiming that atten-

Ambulatory EEG monitoring

Sir: While Dr Cull¹ has shown some advantage in ambulatory EEG over routine recordings, it is slight, especially when the routine EEG is normal: in such patients additional abnormalities were only detected in three out of 35 (9%) compared with four out of 11 (36%) where the routine EEG was judged equivocal. All the excess abnormalities were in patients with weekly, or more frequent attacks. His data therefore suggest that ambulatory EEG is probably only worth doing if the routine EEG is equivocal or attacks happen once a week or more frequently: such an approach would save EEG departments much unproductive work and reduce the risk of the technique falling into disrepute for a low yield.

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Reference

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Cull replies:

Dr Dunstan has made two interesting points. As clearly stated in my paper the overall yield from ambulatory EEG monitoring compared with routine EEG recording is relatively slight (34% compared with 26% detection of abnormalities) but in patients with clinical attacks occurring once a week or more frequently ambulatory EEG recording detected abnormalities in 53% of patients compared with 34% routine EEG records. This was brought out in the paper but Dr Dunstan has made the interesting additional point that where the routine EEG was equivocal, ambulatory EEG recording

detected abnormalities in 36% of patients. Putting these two pieces of information together we can say that from this study the data suggest that ambulatory EEG recording is unlikely to provide useful diagnostic information if the routine EEG is normal and clinical attacks are occurring less frequently than once a week. I agree that selection of patients for ambulatory EEG recording is desirable and have adopted this policy in my own department.

Olfaction in dementia

Sir: We noted with interest the recent report by Simpson and associates.¹ These investigators documented a significant reduction in choline acetyltransferase (CAT) levels in the olfactory tubercles of subjects with Alzheimer type dementia, Down's syndrome and Huntington's disease. The authors state that they are currently investigating deficits in the sense of smell in patients with Alzheimer type dementia. We have previously reported² significant olfactory recognition deficits in this type of dementia and in Parkinsonian dementia. We assessed the capacity of demented patients and non-demented volunteers to identify odours in the following fashion. The olfactory test procedure consisted of a ten pair olfactory identification task in which subjects were presented with two masked vials per trial containing common substances. The subject was then told the identity of one odour and asked to choose the vial containing that substance. Each pair was presented twice quasirandomly. An analogous tactile recognition task served as a control. Subjects scoring below 17 of 20 on the tactile task were excluded from further testing. Degree of dementia was rated with the Global Deterioration Scale.³

Eleven subjects with Alzheimer type dementia had a mean olfactory recognition score of 12.09 ± 2.55 (SD) out of a possible 20. The Parkinsonian subjects ($n = 5$) performed as poorly (mean score: 12.40 ± 4.4). In contrast, a group of demented alcoholics ($n = 12$) scored an average of 18.59 ± 1.70 , and old controls aged 60 to 82 yr ($n = 20$) scored 18.05 ± 1.29 on this task. An analysis of covariance was performed with age and Global Deterioration Scale as covariates, confirming that diagnosis *per se* was a source of significant olfactory performance difference [$F(6, 82) = 16.00, p < 0.001$].

Given the well documented cholinergic