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

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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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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

deficits in Alzheimer type dementia,⁴ we suggest a relationship between the functional integrity of the cholinergic and olfactory systems.⁵ The report of Simpson *et al.*¹ may provide the biochemical basis for our behavioural findings.

Previous animal work has suggested an important role for acetylcholine transmission in the olfactory system,⁵⁻⁷ although studies demonstrating a direct link between acetyl choline and olfaction are limited in number. One report has documented the lowering of olfactory threshold following local nasal application of acetyl choline and acetyl-betacholine.⁸

We are currently investigating the effects of pharmacologic manipulations of central cholinergic activity on several aspects of olfactory performance.

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St Clair *et al* reply

We have suggested that in Alzheimer-type dementia there is disease of the olfactory pathway and that this may produce olfactory deficits.¹ The impairment of olfactory quality discrimination in senile dementia reported by Serby *et al.* (above) and Waldton² could be related to degeneration of the temporal lobe,³ since surgical resection of the temporal lobe produces loss of odour discrimination.⁴ Olfactory threshold, which was not affected by the latter procedure, is a better measure of the functional integrity of the primary olfactory afferents.⁴ We have therefore tested olfactory threshold in addition to odour discrimination⁴ in 13 patients with Alzheimer-type dementia (78 ± 10 yr, mean $\pm$ SD) and 24 age-matched controls (78 ± 11 yr). Only those patients with dementia who were able to discriminate pitch, colours and roughness were tested. Our preliminary results show that both the normal aged and elderly demented patients had difficulty with olfactory quality discrimination and, in accord with the results of Serby *et al.*, the demented patients had more difficulty than the aged controls. Olfactory threshold in these demented patients was not markedly increased compared with age-matched controls. Threshold was, however, grossly elevated in the patients with senile dementia and in aged controls compared with younger controls, and residual function may have been due to trigeminal stimulation.⁵ We conclude that olfactory threshold does not discriminate well between populations of elderly subjects. We are currently investigating younger patients with Alzheimer-type dementia to determine if olfactory threshold in these patients is greater than in age-matched controls.

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