controls, no plaques or tangles were seen. CAT was measured in the olfactory tubercle, dissected to exclude the anterior perforated substance. The activity of CAT in olfactory tubercle from the cases of Alzheimer-type dementia (mean 56 ± 40 SD nmol/h per mg protein) was considerably less (p < 0.001, Student’s t test) than in the controls (153 ± 32) – see Fig. Similarly low levels of CAT were observed in Down’s syndrome (29 ± 22) and in Huntington’s disease (34 ± 13). CAT activity in tubercles from the five female Alzheimer type dementia cases (81 ± 26), but there was no significant difference between enzyme activities in five female (142 ± 17) and ten male (158 ± 38) controls. In control olfactory tubercles, CAT activity was weakly correlated (r = -0.54, p < 0.05) with post-mortem interval but not significantly related to age.

The olfactory tubercle and ventral striatum contain neurons which have very similar afferent and efferent connections. In Huntington’s disease there is a loss of neurons and CAT activity from the striatum. The reduced CAT observed in the tubercle in this disease may therefore be related to degeneration of striatal-like neurons or their connections in the tubercle. In Alzheimer-type dementia and Down’s syndrome, reduced CAT of the order observed in the tubercle (63% and 81%, respectively) has been found in another area of the rhinencephalon, namely the amygdala, in which plaques and tangles are very numerous. These results, combined with those of Averback and Esiri and Wilcock, suggest that olfactory regions of the brain are severely affected in Alzheimer-type dementia and in cases of Down’s syndrome with the neuropathological features of Alzheimer-type dementia. We are currently investigating whether Alzheimer-type dementia patients have a deficit in their sense of smell.

For the supply of necropsy brains, we thank clinicians and pathologists of Bangour Village, Bangour General, and Gogarburn Hospitals, West Lothian; Strathmartine and Ninewells Hospitals, Dundee; and the Royal Edinburgh, Royal Infirmary and Western General Hospitals, Edinburgh. We are indebted to Mr C Farquhar for technical assistance.

Fig CAT activity in olfactory tubercle from controls and cases of Alzheimer-type dementia (ATD), Down’s syndrome and Huntington’s disease.


Accepted 18 April 1984

High density lipoprotein cholesterol in transient global amnesia

Sir: Since the original report by Fisher and Adams, transient global amnesia has been considered to be a sudden loss of short-term memory characterised by transient inability to form new memories, repetitive queries, retrograde amnesia and absence of other neurological symptoms and signs. Although the aetiology is uncertain, most opinions favour a vascular or epileptic mechanism.

In recent years low levels of high density lipoproteins or low ratio of high density lipoprotein cholesterol to total cholesterol have been related to an increased risk of myocardial infarction and cerebrovascular disease. Recent studies have shown that high density lipoprotein cholesterol/total cholesterol ratios are related to severity of coronary artery disease more than high density lipoprotein cholesterol level above. Sirtori et al reported a decreased high density lipoprotein cholesterol level in men with transient ischaemic attacks, but there are no previous reports of this determination in transient global amnesia.

References
2 Esiri MM, Wilcock GK. The olfactory bulbs in
Table

<table>
<thead>
<tr>
<th>No. of subjects</th>
<th>Control subjects</th>
<th>Patients with transient ischaemic attack</th>
<th>Patients with transient global amnesia</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>23</td>
<td>23</td>
<td></td>
</tr>
<tr>
<td>Sex, M/F</td>
<td>54/11</td>
<td>17/6</td>
<td>10/13</td>
<td></td>
</tr>
<tr>
<td>Age, mean</td>
<td>57</td>
<td>58.6</td>
<td>60±8</td>
<td></td>
</tr>
<tr>
<td>High density lipoprotein/cholesterol, mg/dl</td>
<td>54 ± 16</td>
<td>56 ± 13</td>
<td>56 ± 15</td>
<td>NS</td>
</tr>
<tr>
<td>Total cholesterol, mg/dl</td>
<td>201 ± 53</td>
<td>248 ± 41</td>
<td>246 ± 49</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>High density lipoprotein/cholesterol ratio (×100)</td>
<td>27 ± 8</td>
<td>22.8 ± 6</td>
<td>23.2 ± 6</td>
<td>p = 0.012</td>
</tr>
<tr>
<td>Triglycerides mg/dl</td>
<td>120 ± 78</td>
<td>116 ± 41</td>
<td>116 ± 52</td>
<td>NS</td>
</tr>
</tbody>
</table>

Our study investigated serum high density lipoprotein cholesterol levels in transient global amnesia and transient ischaemic attack patients. High density lipoprotein and total cholesterol levels were measured in fasting blood samples from 65 healthy subjects, 23 patients with a clinical diagnosis of transient ischaemic attacks and 23 patients with transient global amnesia. Transient ischaemic attack was defined as a sudden episode of focal cerebrovascular failure with complete resolution of all the symptoms within 24 hours, while transient global amnesia has already been defined above.

The high density lipoprotein cholesterol level was measured by an enzymatic method following heparin-manganese chloride precipitation. Student’s t test was used for statistical evaluation. Levels of serum high density lipoprotein cholesterol, total cholesterol, triglycerides, and high density lipoprotein cholesterol/total cholesterol ratio are given in the table.

The most striking features of our study was the increased total cholesterol levels and the decreased high density lipoprotein cholesterol/total cholesterol ratio in patients with transient global amnesia and transient ischaemic attacks as compared with the control group. No differences were found between patients with transient global amnesia and transient ischaemic attacks.

Our study suggests an inverse correlation between high density lipoprotein cholesterol/total cholesterol ratio levels and transient global amnesia. Although the possibility that high density lipoprotein may prevent the arterial deposition of atherogenic lipoproteins is not fully accepted, our study shows the presence of similar atherogenic risk factors in transient global amnesia patients and transient ischaemic attack patients, which supports the theory of a vascular mechanism for transient global amnesia.

### References


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High density lipoprotein cholesterol in transient global amnesia.

J Matias-Guiu, A Davalos, M Antem and A Codina

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