Cognitive dysfunction after concussion - Authors did not to comment on the single truly significant result

Citation for published version:
Strachan, MWJ, Frier, BM & Deary, IJ 1998, 'Cognitive dysfunction after concussion - Authors did not to comment on the single truly significant result' BMJ, vol 316, no. 7134, pp. 865-865.

Link:
Link to publication record in Edinburgh Research Explorer

Document Version:
Publisher's PDF, also known as Version of record

Published In:
BMJ

Publisher Rights Statement:

General rights
Copyright for the publications made accessible via the Edinburgh Research Explorer is retained by the author(s) and / or other copyright owners and it is a condition of accessing these publications that users recognise and abide by the legal requirements associated with these rights.

Take down policy
The University of Edinburgh has made every reasonable effort to ensure that Edinburgh Research Explorer content complies with UK legislation. If you believe that the public display of this file breaches copyright please contact openaccess@ed.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.
saries which are directly supervised by—often comparatively junior—programme staff can assume better control and contribute to programme activities in many ways.

The situation in private practice is even more confused. Patients may select only part of the treatment regimen because of the expense involved and may default from treatment after a few weeks, once they start to feel better. Attempts to trace such patients who drop out are rarely undertaken. Cooperation with the control programme—which can help with training, open access to sputum microscopy services, free supplies of drugs, and accompanying monitoring—is essential if there is to be a unified strategy on how to treat the disease.

The national programme incorporating “DOTS” (directly observed treatment, short course) needs to be flexible. In Indonesia, a nominated observer (usually a relative, but it could be a neighbour or influential fellow villager) is briefed carefully and entrusted to be responsible for seeing every home dose taken. This observer can be as effective as a worker based at a health centre. Many patients have their disease diagnosed and documented, receive advice and encouragement, and are started on treatment at a health centre, with their nominated observer in attendance. These patients do not have to go to the district or provincial hospital. We are beginning to see satisfactory cure and completion rates from the rural area. The cities, however, are quite another problem.

Richard de Soldenhoff
Regional medical officer
Sulawesi (Support Programme from the Government of Indonesia), Ujung Pandang, Sulawesi (29 November.)

The other main finding, that the risk of concussion was greater in subjects with premorbid cognitive dysfunction, is readily understandable. It is disconcerting, however, that a similar degree of cognitive dysfunction was not apparent in subjects tested after head injury. The authors suggested that this may have been due to differences in the mean age of men tested before and after head injury (20.6 years and 18.0 years, respectively). There was indeed a greater frequency of dysfunctional scores in men injured at age 18 than in those injured at age ≥ 19. These data, however, were derived from the combined test results of men examined both before and after head injury. The authors’ argument would be strengthened if a similar age difference were to be shown individually in both groups.

Mark W J Strachan
Clinical research fellow
Brian M Frier
Consultant physician
Department of Diabetes, Royal Infirmary of Edinburgh, Edinburgh EH13 9YW

Cognitive dysfunction after concussion

Authors did not to comment on the single truly significant result

Editor—Our finding of an increased rate of cognitive dysfunction among subjects tested within one week of sustaining concussion was unsurprising given the numerous studies pointing to the same conclusion.1 The marginal lack of significance of the binomial test (P = 0.06) is due to a lack of statistical power when only eight subjects are studied. That the lower limit of the 95% confidence interval for the risk ratio should nevertheless lie above 1.0 (1.23) is certainly anomalous, but such discrepancies can arise given the different calculations involved.

Interpretation of significant cognitive dysfunction over 200 days after concussion needed to be deferred until the results for those injured after being tested were examined. It then seemed that there was an increased rate of cognitive dysfunction among subjects whether they were tested before or after sustaining concussion. This pointed to cognitive dysfunction being a risk factor for concussion. That the risk factor had manifested itself more strongly in those subjects who were injured after being tested could have been due to their being relatively older at injury than those subjects injured before being tested (four fifths of whom were injured more than six months before testing). We found a lower rate of cognitive dysfunction among those injured at age ≤ 18 than those injured at age ≥ 19. Strachan et al suggest that this argument would be strengthened if both groups were subdivided according to whether they sustained concussion before or after being tested. The table shows the relevant data.

The table provides only partial support for our argument in that the age effect appears only among those injured after testing. There is, however, substantial confounding between age at injury and whether testing took place before or after the injury. Furthermore, dichotomising age involves a reduction of information. In a stepwise logistic regression we found age at injury to be significantly related to the test score (dysfunctional/normal) (P = 0.017), and thereafter there was no significant contribution of injury before or after testing (P = 0.45). In default of alternative hypotheses, we therefore continue to believe that the poorer performance in cognitive tests of those young men who were tested before they sustained concussion may well be explained by factors related to their relatively greater age at injury.

Thomas W Teasdale
Associate professor
Psychological Laboratory, University of Copenhagen, 2500 Copenhagen S, Denmark

Aase Engberg
Senior resident
Department of Neurology, Odense University Hospital, 5000 Odense C, Denmark

Determined prognosis after acute myocardial infarction in the thrombolytic era

Rescue angioplasty after failed thrombolysis may put patients at risk

Editor—Beller brings to readers’ attention the fact that routine invasive procedures after acute myocardial infarction offer no significant benefit over that offered by the routine practice of risk stratification with non-invasive methods.1 We are concerned, however, with the blanket statement that high risk patients should have early angioplasty or rescue angioplasty after failed thrombolysis. This technique should be used with caution.

A meta-analysis by Ellis et al indicated a mortality of 10.0% after the procedure, either

<table>
<thead>
<tr>
<th>Age at injury (years)</th>
<th>Injury before test</th>
<th>Injury after test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Dysfunctional</td>
<td>Normal</td>
</tr>
<tr>
<td>≤ 18</td>
<td>150 (24.8)</td>
<td>456 (75.2)</td>
</tr>
<tr>
<td>≥ 19</td>
<td>21 (22.3)</td>
<td>73 (77.7)</td>
</tr>
<tr>
<td></td>
<td>154 (32.2)</td>
<td>325 (67.8)</td>
</tr>
<tr>
<td></td>
<td>171</td>
<td>529</td>
</tr>
<tr>
<td></td>
<td>158</td>
<td>382</td>
</tr>
</tbody>
</table>

Number of unexplained symptoms and diseases is decreasing

Ennot—In his editorial Mayou explains that the management of patients with medically unexplained physical symptoms is too often inappropriate, even though effective interventions are available.1 He essentially attributes this to the persistence of the idea of “mind-body dualism” in the medical profession, which neglects important interactions between physiological, psychological, and social factors. He agrees that he says about this socially and economically important subject, but I would emphasise another factor: our scientific ignorance and frequent arrogance. We should be modest and cautious, perhaps stating that our conclusions still seem to be unaware of their own ignorance. We should be modest and cautious, perhaps stating that our conclusions not be secondary to extensive left ventricular dysfunction.

The main message of my editorial was that a routine invasive strategy for risk assessment before discharge is not superior to a watchful waiting, non-invasive strategy in which patients undergo angiography for high risk clinical findings or for spontaneous or inducible ischaemia within or remote from the infarct zone. Recent data reported from the VA non-Q wave infarction strategies in-hospital trial, in which 1292 patients with non-Q wave infarction were randomised to an initial invasive strategy or an initial conservative strategy, support this approach.1 At one year after discharge there was no difference in cardiac death or recurrent infarction between the two groups. Also, new data from Yusuf et al showed no difference in outcome for patients with infarction admitted to hospitals with cardiac catheterisation facilities (catheterisation rate 66%) compared with those admitted to hospitals with no catheterisation facilities on site (catheterisation rate 3%).2

George A Beller Chief Cardiovacular Division, Department of Internal Medicine, University of Virginia Health Sciences Center, Charlottesville, VA 22908, USA
