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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. The marginal lack of significance of the binomial test ($P = 0.060$) 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 ratio score should nevertheless lie above unity (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 $\leq 18$ than those injured at age $> 18$. Teasdale 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.

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References


Determining 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. 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>
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<tbody>
<tr>
<td></td>
<td>Dysfunctional</td>
<td>Normal</td>
</tr>
<tr>
<td>$\leq 18$</td>
<td>150 (24.8)</td>
<td>456 (75.2)</td>
</tr>
<tr>
<td>$&gt; 18$</td>
<td>21 (22.3)</td>
<td>73 (77.7)</td>
</tr>
<tr>
<td>Total</td>
<td>171</td>
<td>529</td>
</tr>
</tbody>
</table>
from the disease process or as a direct complication of the procedure.\(^7\) Furthermore, this procedure fails in 20% of cases and those failed cases have a mortality of 40%. Vigorous clinical assessment is therefore necessary before a patient is classified as being at high risk. Inadequate optimisation of supportive treatment often leads to signs such as hypotension and sinus tachycardia, which in turn predispose to further chest pain, interpreted as postinfarction angina even in the absence of electrocardiographic changes. Chest crepitations related to aging are often confused with those associated with pulmonary oedema. One prime example is inferior myocardial infarction with right ventricular extension. This is due to an occlusion of a dominant right coronary artery, which carries a relatively good prognosis. Suboptimal fluid replacement and the indiscriminate use of inotropic agents without prior careful assessment of left ventricular function with echocardiography and guidance by Swan-Ganz catheterisation lead to patients being labelled as at high risk without having prior or incidental left coronary artery disease.

The fact that rescue angioplasty for right coronary artery occlusion is associated with excessive complications\(^1\) should lead doctors to question whether this form of intervention is putting a patient's life at risk, turning a relatively benign course into a fatal one.

**Author's reply**

**Editor**—Lim and Shielis make a valid point regarding the increased risk of rescue angioplasty after presumed failed thrombolysis, but I never addressed the issue of early angioplasty in my editorial. The thrust of my discussion regarding risk stratification related to the identification of clinical variables and variables determined with non-invasive tests that could be used to select those patients after infarction who are most likely to benefit from coronary angiography and coronary revascularisation.

With respect to clinical variables, I mention that the combination of rules in over a third of the lung field, hypotension, and sinus tachycardia on admission was an important observation that indicated a high risk status, since these haemodynamic alterations reflect a large area of myocardium at jeopardy with ischaemia or necrosis, or both; they can also point to underlying multivessel disease or a large infarct, or both. I agree that each one of these haemodynamic changes in isolation is not highly specific for a high risk designation. Certainly, crackles at the lungs bases alone without evidence of other signs of left ventricular pump failure can indicate atelectasis or pulmonary disease. Hypotension in isolation, without sinus tachycardia and pulmonary rates, can be due to volume depletion or right ventricular infarction and 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 920 patients with non-Q wave infarction were randomised to an initial invasive strategy or an initial conservative strategy, support this approach.\(^2\) 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 34%).\(^2\)

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**Number of unexplained symptoms and diseases is decreasing**

**Editor**—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.

I agree that he says about this socially and economically important subject, but I would emphasise another factor: our scientific ignorance and frequent arrogance. Too easily and too frequently we attribute to mental illness symptoms that turn out to be those of well defined organic diseases (which does not exclude the role of superimposed social or psychological factors). Mayou says “there is scant provision . . . for the patient with somatic complaints who has neither physical disease nor severe mental illness.”

This sentence reminds us that these patients tend to be forgotten, but “recognised” or “known” should probably have preceded the term “physical disease.”

Patients with muscular symptoms are a good example of this. Many, classified years ago as mentally ill, later turned out to have well defined disease. A typical example is patients with McArdle’s disease, who suffer from chronic muscle fatigue with exercise. Many—at least, the older ones—were initially classified as having purely psychological or psychiatric disease; they were consequently regarded as being lazy and dealt with as such, typically when they were conscripted into the army. Even worse, they were usually forced to exercise, which has since been shown to be potentially dangerous since it can lead to muscle damage and renal failure. They are now known to have a genetically determined lack of a muscle enzyme essential for glycogen breakdown and use for energy production (muscle phosphorylase deficiency).

These errors of classification have moral, psychological, and economic implications for patients, their families, and society. Some doctors still seem to be unaware of their own ignorance. We should be modest and cautious, perhaps stating that our conclusions are “to the best of our knowledge” and may not be correct. Even though the list of unexplained diseases and symptoms is slowly decreasing through scientific progress, it is unlikely ever to disappear totally.

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