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

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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.

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—Teasdale and Engberg examined the duration of cognitive dysfunction after concussive head injury in young men. We note that impaired cognitive performance was shown in those subjects tested more than 200 days after the 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 (20.0 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.

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1 Teasdale TW, Engberg A. Duration of cognitive dysfunction after concussion, and cognitive dysfunction as a risk factor: a population study of young men. BMJ 1997;315:569-72. (6 September.)

Authors’ reply

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=0.08) 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 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 point to cognitive dysfunction being a risk factor for concussive injury. 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.

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Cognitive dysfunction after concussion

Authors did not to comment on the single truly significant result

Editor—Teasdale and Engberg examined the duration of cognitive dysfunction after concussive head injury in young men. We are concerned about their interpretation of some of the data presented. The observation that cognitive function was impaired up to one week after head injury was based on examination of only eight subjects, four of whom showed impaired performance. The authors placed considerable weight on this result even though the relative risk of cognitive impairment in the subjects with head injury was not significant by conventional criteria. It was additionally perplexing, given this non-significant result, that the 95% confidence interval did not pass through unity. Perhaps most surprising of all was that the authors failed to comment on the one truly significant result in that section of the paper—that impaired cognitive performance was shown in those subjects tested more than 200 days after the 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 (20.0 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.

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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 point 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.

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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
from the disease process or as a direct complication of the procedure. 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 complications 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 classified 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 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 Shielz 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 mentioned 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 multisystem 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. 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%).

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1 Beller GA. Determining prognosis after acute myocardial infarction in the thrombolytic era. BMJ 1997;315:761-2. (27 September.)

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**Letters**

**The caring doctor is an oxymoron**

**General practice will develop best if “caring” is replaced by professionalism**

**Editor**—Mackenzie’s hypothesis that the term “the caring doctor” is an oxymoron struck a chord with many doctors I speak to. I have long thought that general practice will develop best if we replace the sham of caring with better professionalism. This does not stop us practising good medicine in a compassionate and considerate manner. We need to think imaginatively, to continue the drive for better organisation, to use information technology to the full, to recognise the strengths of other members of the team, to delegate...