Some neurological signs

G D Perkin

A series looking at aspects of clinical examination

process and is prone to error. Some of these techniques are of such sophistication that abnormalities are frequently discovered that bear no relation to the clinical problem. Without an appropriate level of history taking and examination, such abnormalities may be misinterpreted as being relevant to the patient’s presentation. Indeed there remains as much skill in the decision not to investigate a particular patient as to do so. That decision rests at least in part on the ability to perform an adequate neurological examination and to be able to distinguish the normal from the abnormal.

EDITORIAL COMMENTARIES

A series devoted to the value of particular physical signs in neurological practice needs no apology. Though one suspects that succeeding generations of neurologists have bemoaned the lack of clinical expertise among their juniors there can be no doubt that the remarkable advance in neuroimaging and allied techniques that has been seen over the past 20 years has altered the delicate balance between clinical appraisal and investigation. An overreliance on sophisticated investigative techniques has a number of potential pitfalls. The interpretation of those investigations remains a highly skilled...
Functional reorganisation of memory following traumatic brain injury: a study with $H_2^{15}$O PET

J T E Richardson

Brain injury

Neuroimaging holds future promise for before and after comparisons

Traumatic brain injury (TBI) is a major cause of deaths following accidents and a major cause of disablement and morbidity among the survivors. The clinical problem of treating this vast number of patients is not going to be solved by a dramatic medical breakthrough. Rather, it must be tackled by seeking a better appreciation of the condition that these people present. The paper by Levine et al (this issue, pp 173–81) adds to the growing body of studies in which neuroimaging techniques have been used to study long-term changes in survivors of moderate or severe TBI.

Levine et al carried out PET in six patients who had sustained TBI roughly four years previously and in 11 healthy controls. Separate scans were obtained during the encoding and retrieval phases of a simple cued recall task and attention was focused on the differences in activation between the two phases. The individual findings varied with focal lesions. However, as a group they had more widespread activation during the retrieval phase than the controls, with reduced activation in areas involved in normal memory retrieval. This was regarded as evidence for neural reorganisation due to diffuse axonal injury.

This study combined the application of neuroimaging technology with an understanding of the cognitive mechanisms involved in the relevant experimental task. Levine et al also paid due attention to both between-group variation and within-group variation. They considered but rejected the idea that these changes might just have been an artefact of impaired performance. Indeed, the same pattern was apparent in three patients whose retrieval was at a normal level, although some aspects were seen only in the three patients with poor performance.

Two issues need to be addressed in further research. Firstly, would similar changes be seen in patients with mild rather than severe TBI? The prevalence of mild TBI is many times greater than that of severe TBI, and many suspect that mild TBI may give rise to persistent yet subtle deficits even in patients who otherwise make a good functional recovery. Of course, such deficits may have predated the TBI and were not caused by it. This problem is inherent in any study that compares groups defined on the basis of their clinical history.

So a second issue raised by this study is this: are the apparent differences between patients with TBI causally related to their injuries? Ideally, one would like to have premorbid evidence from the people in question. Of course, the number of people who have undergone any specific PET procedure before sustaining a TBI is vanishingly small, but other evidence may be available. Bigler and Snyder compared computed tomography with magnetic resonance imaging carried out in four people before and after they sustained mild TBI. The authors failed to find any gross differences but more detailed analyses might have revealed clinically significant changes. With the increasingly widespread use of neuroimaging techniques, comparisons of this sort should be entirely feasible in the future.

REFERENCES


Subarachnoid haemorrhage

Coffee and subarachnoid haemorrhage

W T T Longstreth Jr

The link between coffee and subarachnoid haemorrhage is unresolved

You may be making enemies, especially in Seattle, if you conclude from the study of Isaksen et al that coffee is a risk factor for subarachnoid haemorrhage (this issue, pp 185-7). Love of java necessitates a critical evaluation. Are we dealing with coincidence, confounding, or causation? In a case-control study, these investigators drew subjects from a population based health survey of inhabitants in the municipality of Tromsø, Norway. At variable times before the bleeding (maximum 186 months) participants had been evaluated. The investigators found that cigarette smoking and high systolic blood pressure increased risk, as have others. The trend was for high cholesterol to reduce risk but not significantly, perhaps reflecting the small number of participants (n = 26). Drinking six or more cups of coffee per day yielded an odds ratio of 3.86 even after controlling for both to coffee consumption and to subarachnoid haemorrhage, such as alcohol consumption? Perhaps the association would have disappeared after controlling for alcohol consumption. Also by examining different doses of coffee, as they did for cigarette smoking, the investigators could have strengthened their argument for a causal association.

If the association were real, how might coffee increase the risk of subarachnoid haemorrhage? Bleeding is typically the culmination of aneurysm formation and rupture. Examining coffee as a risk factor in patients with unruptured intracranial aneurysms or in patients with multiple aneurysms may address questions of formation. How the association varied as a function of the time since the onset of the bleeding may address questions of rupture. Caffeine can cause an increase in blood pressure, perhaps putting those who harbour an intracranial aneurysm and who drink six cups or more of caffeinated coffee per day at increased risk for rupture compared with those who drink less or do not drink coffee at all. In the current study we do not know whether the coffee was caffeinated and whether other caffeinated beverages, such as tea and cola drinks, were examined. Questions about rupture would require knowledge about the use of the beverage in the time immediately before the onset of the bleeding, not months before. As is so often the case with such unexpected findings, more studies are needed before we can judge the importance of this intriguing and novel observation, especially about such a common exposure. So for now sip your coffee but with some lingering concern about this unresolved issue.

J Neurol Neurosurg Psychiatry 2002;73:112

Authors’ affiliations
W T T Longstreth Jr, Department of Neurology, University of Washington, Harborview Medical Center, 325 Ninth Avenue, Seattle, Washington 98104–2420, USA

Correspondence to: Dr W T T Longstreth; wlt@uwashington.edu

REFERENCES