clearly reduced mortality following myocardial infarction. It is not as well known that, in general, survivors of MI who have been treated with thrombolytic therapy have a lower mortality than similar patients studied in the pre-thrombolytic era. The reason for this improved prognosis is unclear, but may relate to the fact that candidates for thrombolysis may, in general, be relatively free of concurrent disorders.

Is the prognostic value of post-MI thallium stress testing as established before the widespread use of thrombolytic therapy applicable to patients who have received such therapy? This question has been examined by Basu and colleagues. They studied 100 consecutive patients treated at a large district general hospital 6 weeks after myocardial infarction. (Presumably most of these patients underwent some pre-discharge stress test evaluation, although Basu et al do not say so.) Before the follow-up stress thallium examination, 10% of the original cohort had a major cardiac event. For those who did survive to follow-up study, thallium-imaging abnormalities indicative of inducible ischaemia were present in 22 (88%) of 25 individuals who subsequently died, underwent reinfarction, or had unstable angina. By contrast, routine exercise ECG testing alone was positive in only 9 (36%) of these 25 individuals. Similarly, the positive and negative predictive value for thallium stress testing was superior to that of routine exercise ECG.

These data, like Gibson's, which were obtained in the pre-thrombolytic era, do suggest that myocardial perfusion imaging adds to the value of exercise ECG. However, before recommending routine post-MI thallium imaging, with its attendant costs, it is important to note several caveats. The thallium imaging protocol used by Basu et al is designed to maximise sensitivity, and it involved imaging on 2 separate days; this approach may not be practical for all laboratories and does represent an inconvenience for patients. As is the case with much published research, the authors are experienced investigators, which means that the results may not necessarily be representative of those from laboratories with less experience or rigorous quality control. It is also important to note that, of the seven deaths reported by Basu, four occurred before stress test at week 6. Similarly, Heroux, almost two decades earlier, had found that 30% of deaths after MI occurred in the first 3 months. Thus, the reason for this improved prognosis is unclear, but may relate to the fact that candidates for thrombolysis may, in general, be relatively free of concurrent disorders.

European consensus on viral encephalitis

Herpes simplex encephalitis (HSE) is one of the commonest viral brain disorders of immunocompetent individuals, with an annual incidence estimated at between 1 in 25 000 and 1 in a million of the population. Most cases are due to herpes simplex virus (HSV) type 1; up to 10% may be caused by HSV-2. Encephalitis is postulated to occur as a consequence of centripetal spread of virus from sites of latent infection in cranial nerve ganglia to the frontal or temporal lobes of the brain. The mechanism of reactivation in HSE is poorly understood. HSE typically results in focal frontal and/or temporal lobe necrosis, and patients present with an acute neurological syndrome characterised by behavioural disturbances, hemiparesis, aphasia, or seizures. If untreated, HSE is usually fatal. Less commonly, patients present with mild, subacute encephalitis, brainstem encephalitis, recurrent meningitis, or myelitis. Unlike most viral encephalitides, for which there is only supportive management, HSE does respond to specific antiviral drugs, and early, aggressive antiviral therapy saves lives and reduces morbidity. Rapid reliable diagnostic techniques and standardised methods of treatment for HSE are therefore critical.

The European Union's Concerted Action on Virus Menignitis and Encephalitis has produced a comprehensive review of the roles of brain imaging, electroencephalography, brain biopsy, and cerebrospinal fluid studies in the clinical evaluation and management of HSE. Imaging and electroencephalography are neither sensitive nor specific, particularly in early disease. Biopsy is both sensitive and specific but is also both traumatic and expensive. Fortunately, nested PCR amplification of HSV sequences from cerebrospinal fluid offers a rapid, sensitive, inexpensive, and less invasive method for establishing the initial diagnosis of HSE and for monitoring the response to therapy. Although nested PCR is more susceptible to contamination artifact (false positive) than traditional methods such as virus isolation or serology, diagnostic PCR has been successfully incorporated into many clinical microbiology laboratories and is likely to become the gold standard. PCR has revolutionised the management of infections with HIV and hantaviruses as well as herpesviruses.

Clinicians who suspect HSE frequently begin antiviral therapy before the diagnosis is established. Pilot studies indicate that even if antiviral therapy has been started before collection of cerebrospinal fluid, PCR can still be of diagnostic value; HSV sequences are typically

<table>
<thead>
<tr>
<th>Reference</th>
<th>Details</th>
</tr>
</thead>
</table>
detectable for up to 5 days thereafter. PCR can differentiate between HSE due to HSV-1 or HSV-2 either through the use of primers specific for each during PCR or through Southern hybridisation, analysis of amplification products with probes containing sequences specific for HSV-1 or for HSV-2. This distinction does not alter clinical management but may do so in the future as the repertoire of anti-HSV agents expands. The diagnosis of HSE can be confirmed by demonstrating an increase in the intrathecal titre of antibodies to HSV 3 weeks after onset of disease.

New anti-HSV agents with enhanced bioavailability, such as famciclovir or acyclovir, improve the drug of choice for HSE and are currently acyclovir. Acyclovir is specific for herpesviruses, has low toxicity in patients with normal renal function, and improves the prognosis in HSE. Although so far there are no reported instances of resistance to acyclovir in HSE, the situation may change with increasing popularity of the drug and against its indiscriminate use. The clinical management of HSE otherwise consists of general support such as assisted ventilation, fluid and electrolyte balance, and anticonvulsants. Steroids, hyperventilation, osmotic agents, or barbiturates remain controversial but may help if there is massive brain oedema.

The European consensus also provides an excellent algorithm for the diagnosis and treatment of suspected HSE. Nevertheless, the message is the last paragraphs of the report: a favourable outcome depends on early therapy (with acyclovir), and early therapy in turn is dependent upon clinical judgment (suspicion of HSE) and PCR analysis of cerebrospinal fluid. The European initiative is a model for an integrated interdisciplinary, multicentre clinical collaboration, and its application to other areas of medicine would do much to enhance the quality and efficiency of health-care delivery.

**W Ian Lipkin**

Laboratory for Neurovirology, Department of Neurology, University of California, Irvine, CA 92697-4290, USA

---

**Ian Munro**

The man under whom we jointly served as deputy editors, Ian Munro, died on Jan 22 at the age of 73. A backward look at his work at The Lancet is like a firework show in the distance. Even well into retirement, Ian never lost his passion and reforming zeal. Ian joined The Lancet in 1951 after a period in training as a radiologist. No vacancy had been advertised. He had written a thank-you for lunch, and the editor of the day was impressed, reasoning that “Whoever can write a really good letter must be able to recognise a bad one and therefore has the makings of an editor.” Thus he became a medical journalist, working first under TF (“Robbie”) Fox and then as deputy to Ian Douglas-Wilson, succeeding him in July, 1976.

When he arrived, the journal had a very British focus, with much reportage on local issues in health services, education, and medical politics. Had the journal allowed bylines for its staff, Ian would have had an impressive list of publications for the office’s bound volumes (where writers are unmasked) carry many a leader, annotation, or other item from his pen. Even without these marks, we could identify them from the Munro style—occasionally baroque, often beautiful, always clear and strong. We suspect that Ian was happiest in those journalistic days.

As editor he can take credit for publishing numerous papers that left deep marks on medical practice but other people might have done that equally well. More important, Ian stood up for a sort of idealism that was (and is) far from the political spirit of the times. On his retirement in 1988, friends at the BMJ published a book of essays called Swearing N either to the Right nor the Left—an elegant tribute, but with a title some way from the truth. Ian was hardly known for steering the middle course, as witnessed by his pronouncements on such matters as abortion, population, the perils of nuclear weapons, and human rights (he was the first president of Physicians for Human Rights UK). The National Health Service, dear to his heart, was the subject of his most passionate declarations—one of which generated an equally heated riposte from a Conservative Health Secretary. Ian wore it like a medal.

There are those who lead by charisma and those who lead by example. Ian was the second sort—a man whose human qualities permeated the whole Lancet operation. If the journal now has a reputation for courteous dealings with readers and contributors, this is part of the Munro legacy. Another part is a tendency to swim against the tide. Ian swam strongly to the end.

**Robin Fox, David Sharp**

Rotherfield, East Sussex; and The Lancet, London, UK