

Firstly, one should know whether all medical admissions pass through the accident and emergency department. If not one should know the proportion of cases admitted directly to medical wards. It would also be interesting to know the average time (and the standard deviation of the time) that passed from admission to the accident and emergency department to disposal to a specialty ward. I note that all the patients whose deaths were diagnosed as due to myocardial infarction had time to undergo 12 lead electrocardiography and cardiac monitoring before transfer to a medical ward or coronary care unit. I am left to wonder what diagnostic label was put on patients who died without having had this test performed. We should know the electrocardiographic diagnostic criteria for myocardial infarction used in cases which almost by definition could not have had sequential electrocardiographs performed.

Mr Shalley and Mr Cross rightfully draw attention to the need for training in cardiological conditions for doctors working in accident and emergency departments. The best index of this should relate not just to fatal cases but to the number of non-fatal cases of heart conditions passing through an accident and emergency department.

W STC SYMMERS

Scottish MONICA,  
Usher Institute,  
Edinburgh EH9 1DW

SIR,—Mr M J Shalley and Mr A B Cross suggest that many deaths in an accident and emergency department are due to myocardial infarction and that "a much greater emphasis should be placed on the management of medical emergencies." Although the precise modes of death in the patients with myocardial infarction were not stated, they were probably the result of asystole, electromechanical dissociation, or ventricular fibrillation. In view of the poor prognosis associated with the first two of these conditions and the relatively non-specialised management of the third, I would be interested to know how Mr Shalley and Mr Cross think that a more highly medically trained casualty staff could alter the mortality?

G D STANLEY

Coronary Care Unit,  
Leicester Royal Infirmary,  
Leicester

SIR,—Mr M J Shalley and Mr A B Cross discuss the initial hospital management of acute serious medical, surgical, and traumatic conditions and raise points about training for accident and emergency staff.

Although the British Association for Immediate Care (BASICS) has been primarily concerned with prehospital care, we consider that it cannot be separated from initial hospital care. We want to improve the care provided for all patients with serious emergency problems from the time that they occur until definitive appropriate hospital treatment. A survey undertaken by BASICS in 1982 suggested that less than a quarter of medical students were trained in life supporting procedures.

In an attempt to establish standards of training in the prehospital and initial hospital management of the acutely ill or seriously injured patient BASICS has been discussing a

possible diploma examination in emergency or immediate care. This would not attempt to cover the full scope of accident and emergency medicine but would concentrate on life supporting procedures: the proposals cover the particular problems with both medical and surgical emergencies mentioned by Mr Shalley and Mr Cross.

If this is introduced we hope that it will prove useful for general practitioners and hospital doctors both in and out of accident and emergency medicine and so improve patient care in this critical phase of emergency treatment.

BRIAN G STEGGLES

Chairman, Training Committee,  
BASICS

Tavistock, Devon PL19 9DP

### Spinal cord disease due to *Schistosoma mansoni* successfully treated with oxamniquine

SIR,—The case of paraplegia reported by Dr John Efthamiou and Dr David Denning (5 May, p 1343) is important because it appears to be the first case of schistosomal myelopathy to be reported in a Ghanaian. Histologically proved cases of schistosomal myelopathy are rare in women; there seem to have been no cases of *Schistosoma haematobium* myelopathy and only six cases of *S. mansoni* myelopathy reported—all from Brazil.<sup>1-3</sup>

It seems likely that the myelopathy was caused by *S. mansoni*, but it might have been caused by *S. haematobium*, which can also involve the conus medullaris usually without other evidence of *S. haematobium* infection.<sup>4</sup> The detection of *S. mansoni* ova in the faeces of a patient from a country like Ghana where both parasites are endemic does not prove that it was the cause of the paraplegia. For example, an Egyptian patient who was being treated for relapsed proved *S. haematobium* myelopathy was noted to have coincidental intestinal schistosomiasis.<sup>5</sup>

While most cases of schistosomal myelopathy present with acute paraplegia acute necrotising myelitis with infarction is rarely reported.<sup>3</sup> Study of reports of proved cases shows that intramedullary granulomatous myelopathy usually affecting the lower cord and conus medullaris is the commonest presentation.<sup>2,4</sup>

There have been several previous reports of patients who were diagnosed to have schistosomal myelopathy on the basis of myelography and evidence of systemic schistosomal infection.<sup>6-8</sup> In the study from Malawi the follow up myelogram showed considerable diminution of the spinal cord mass following chemotherapy.<sup>8</sup> El Banhawy has emphasised the diagnostic value of myelography in schistosomal myelopathy.<sup>3</sup> Of course, the myelogram may be normal in some patients. When this is so (as is often the case) and when there is no other evidence of schistosomiasis<sup>9</sup> a positive antibody reaction to *Schistosoma* (which indicates only schistosomiasis per se) may be of diagnostic value. Unfortunately a reaction even in the cerebrospinal fluid may be absent in schistosomal myelopathy.<sup>4</sup>

It is useful to know that oxamniquine (which has been used previously in schistosomal myelopathy<sup>9,10</sup>) appeared to have been rapidly efficacious in this instance. Any apparent effect must be considered, however, in the context of a condition which has been reported to respond rapidly to many different treatment regimens and which may also spontaneously remit for prolonged periods.<sup>7,11</sup> Because the condition is so rarely encountered and the pathological findings are so variable it is difficult to compare the results of different treatment regimens.<sup>7,12</sup> Treatment should be

tailored to the individual, and decompressive laminectomy should still be considered in the patient with acute paraplegia and myelographic spinal block.<sup>12</sup>

E M SCRIMGEOUR

Laboratory for Central Nervous  
System Studies,  
National Institutes of Health,  
Bethesda, Maryland,  
USA 20205

- 1 Abath GM, Barbosa AV. Esquistossomose do sistema nervosa central. *An Fac Med Univ Recife* 1960;20:401-5.
- 2 Perpetuo FOL, Rodrigues PA. Esquistossomose medular. *Rev Assoc Med Minas Gerais* 1973;24:173-8.
- 3 Quieroz L deS, Nucci A, Facure NO, Facure JJ. Massive spinal cord necrosis in schistosomiasis. *Arch Neurol* 1979;36:517-9.
- 4 Ghaly AF, El Banhawy A. Schistosomiasis of the spinal cord. *J Pathol* 1973;111:57-60.
- 5 El Din SS, Haseeb NM, Basmay K, et al. The value of the circumoval precipitin test in the diagnosis of spinal cord bilharziasis. *Am Shams Med J* 1973;24:25-30.
- 6 El Banhawy A. Schistosomiasis of the spinal cord, conus and cauda. *Neurol Med Chir* 1971;11:17-30.
- 7 Lechtenberg R, Vaida GA. Schistosomiasis of the spinal cord. *Neurology* 1977;27:55-9.
- 8 Molyneux ME, Galatius-Jensen F. Successful drug treatment of schistosomal myelopathy. A case report. *S Afr Med J* 1978;54:871-2.
- 9 Dar J, Zimmerman RR. Schistosomiasis of the spinal cord. *Surg Neurol* 1979;8:416-8.
- 10 Siddorn JA. Schistosomiasis and anterior spinal artery occlusion. *Am J Trop Med Hyg* 1978;27:532-4.
- 11 Raper AB. Cerebral schistosomiasis. *E Afr Med J* 1948;25:262-3.
- 12 El Banhawy A, Elwan O, Taher Y. Bilharzial granuloma of the conus medullaris and cauda equina. *Paraplegia* 1972;10:172-80.

### Lymphangitis after tuberculin tests

SIR,—Dr J B Morrison's report on an unusually large number of cases of lymphangitis after tuberculin tests during 1979-83 (18 August, p 413) highlights some of the problems which sadly are arising as a result of a lack of doctors like himself with long experience in tuberculosis.

The cases reported reflect inexperience in the approach to tuberculin testing rather than a fault in the test itself. The tuberculin reaction is generally very strong in the presence of primary tuberculous lesions and may remain so for many years after healing.<sup>1</sup> In the same way adult tuberculosis is generally associated with high tuberculin hypersensitivity. When a diagnosis of tuberculosis is suspected, in order to avoid a severe reaction a Mantoux test using the weakest dilution of tuberculin—that is, 1 IU—to start with is therefore advised. Where there is radiographic evidence of old healed tuberculous lesions, as in several of Dr Morrison's cases, tuberculin testing could have been omitted.

In the first case the patient was a 21 year old Indian and had clinical evidence of probable healed tuberculous cervical adenitis; his chest radiograph showed an old apical, presumably tuberculous, lesion in addition to a small round lesion in the other lung.

Except in conditions where tuberculin hypersensitivity is known to be generally depressed or absent—as, for example, in sarcoidosis or the reticuloses—the tuberculin test is unhelpful as a diagnostic aid in subjects over 40. In this age group the tuberculin reaction is commonly strong as a result of previous primary tuberculous infection or as a result of superinfection in those born in time to have had the benefit of BCG vaccination at school. The age range of the 47 hospital staff who received Mantoux tests was 45-55, and four developed lymphangitis. The age range of the 371 subjects who received Heaf tests performed by a health visitor was 35-56, and seven developed lymphangitis and five of