low plasma concentrations of free cyanide dicobalt edetate itself can be toxic.¹

A second approach is to use sodium nitrite, which converts haemoglobin to methaemoglobin, though in a patient whose oxygen delivery is already compromised by a high carboxyhaemoglobin concentration a further decrease in the haemoglobin available for oxygen transport can be dangerous. We therefore recommend use of the safer intravenous antidote sodium thiosulphate,² whose distribution is enhanced by the vasodilator amyl nitrite.³

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Whiplash injury

SIR,—Dr P K Newman fails to come clean over the “thorny problem of the late whiplash syndrome.”¹ Opinion should be separated from fact.

Fact

(1) No study has been published with an adequate control group—that is, a series of injuries occurring in people not claiming compensation.

(2) No radiographic technique or laboratory test exists to confirm or refute the symptoms.

(3) Relative sparing of the young and the over 60s and poor correlation with radiographic evidence of spondylosis indicate that symptoms are mainly independent of degenerative changes in the cervical spine.

(4) Most subjects recover quickly, two thirds being free of pain after three months and three quarters returning to work after one month.² The striking resemblance to strains of other muscles and ligaments is apparent.

(5) There is a high association with anxiety and depression.^{2,3} Spurious non-anatomical physical signs are common and reflect exaggeration or simulation of illness. Analgesics and collars fail to relieve symptoms of late whiplash. Settlement of litigation sometimes, but not always, results in resolution of complaints.

(6) Maimaris has reported results of magnetic resonance imaging in four patients with moderate or severe persisting symptoms⁴ and Von Meydam *et al* have similarly examined 15 such patients.⁵ The results were normal in all 19 patients.

Opinion

There is little evidence to suggest that whiplash injury is different from other muscular or ligamentous strains. It is much better termed neck sprain. By definition I would exclude all patients with neck injuries affecting vertebrae, intervertebral discs, or nerve roots, which indicate a more serious condition and a different natural course.

Dr Newman and others fail to provide an organic explanation for the persistence of symptoms, and results of magnetic resonance imaging argue strongly against a mechanical source. Whether these symptoms are genuine or exaggerated in the interests of enhancing financial rewards is a subjective judgment. Dr Newman cites no support for his contention “it is usually the persistence of symptoms that leads a patient to

litigation, and not vice versa.” The available evidence argues to the contrary.

Balla stated that “Socio-cultural factors may account for a number becoming chronic. It is difficult to separate the effects of seeking compensation from other factors. Whiplash injuries certainly lend themselves to fraud of which there have been a number of well documented cases seen at the Motor Accidents Board.”¹ Undoubtedly, disability may be prolonged by the lengthy delay in obtaining a legal settlement. Neurotic features can also be deliberately inflated and should be carefully weighed in relation to the circumstances, previous psychoneurotic illness, and current behaviour and observed reactions during examination.

Most victims of whiplash injury have, however, sustained no more than a minor sprain to the soft tissues, and unusually severe or protracted complaints may demand explanations that lie outside the fields of organic and psychiatric illness.

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AUTHOR'S REPLY,—Dr J M S Pearce has instructively expanded aspects of the late whiplash syndrome that I could only sketch in my editorial. Perhaps our only difference is that I am a few points lower than he on the hawk-dove rating scale of medicolegal necks.

Lead swingers abound in our medicolegal practices, but so does genuine disability, and each case requires careful individual assessment. Unfortunately, many of the published “facts” about the late whiplash syndrome are subjective and have been extracted retrospectively from personal medicolegal series. Such material often has dubious validity, and I hope that a large, long term, prospective study will eventually emerge to illuminate this dusky corner of clinical practice. Finally, may I caution against undue reliance on negative results on magnetic resonance imaging. Recent experience in cervical degenerative disease suggests that this technique may be less accurate than myelography in a large proportion of cases.

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Occult ischaemic necrosis of skeletal muscle

SIR,—Dr J H Coakley and colleagues report that ischaemic muscle necrosis may be associated with acute renal failure.¹ We would like to draw attention to another unusual cause of acute renal failure—namely, acute rhabdomyolysis.

An 18 year old Gambian man who developed acute falciparum malaria and a parasite index of 1.1% went on to develop acute hypercatabolic renal failure, which required dialysis and which is usually associated with a high parasite index.² He complained of general malaise and fever and displayed no remarkable physical signs, and in common with the patients described by Dr Coakley and colleagues he had no muscle symptoms or signs. We were alerted to the possibility of acute rhabdomyolysis because his aspartate amino-

transferase activity was 1033 IU/l (normal range 13-51 IU/l). His creatine kinase activity was 28 218 IU/l (24-195 IU/l), and it later reached 73 000 IU/l before returning to normal on discharge. Investigations failed to find a cause of his rhabdomyolysis, and we concluded that it was caused by his falciparum malaria.

Severe malaria can cause muscle injury in children and correlates with severity of illness.³ There has been only one other case of acute rhabdomyolysis and acute renal failure induced by malaria, and this occurred in a Sri Lankan man with severe disease and high parasite index.⁴

We agree with Dr Coakley and colleagues that in all cases of acute renal failure the possibility of muscle damage should be borne in mind and suggest that creatine kinase concentration be measured in all patients with falciparum malaria. There are many mechanisms of pathogenesis of malarial acute renal failure,⁵ and more research is needed to evaluate fully the nephrotoxic potential of skeletal muscle damage in this condition.

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Terminal cancer care and patients' preference for place of death

SIR,—Ms Joy Townsend and colleagues found that 67% of patients dying of cancer, given “more favourable circumstances,” would prefer to die at home, but only 29% did so.¹ Twenty seven per cent of patients with cancer in England and Wales died at home in the same period.²

Although comparing home care teams is difficult because of the different ways in which they function, recent reports from hospice teams in England and abroad have shown that up to 71% of patients were enabled to die at home (table). Indeed, some

Proportion of patients with cancer enabled to die at home according to place of care

	% Dying at home
North London Hospice 1985 ¹	58
St Mary's Hospice, Birmingham 1986 ⁴	55
West Cumbria 1987 ⁵	53
St Joseph's Hospice, London 1988 ⁶	61
Tunbridge Wells Hospice at Home 1989 [*]	71
South Auckland Hospice, New Zealand 1988-9 [†]	59
Bologna, Italy 1985-88 ⁷	60

^{*}Tunbridge Wells Hospice at Home Annual Review 1989.

[†]South Auckland Hospice Care Report 1989.

care teams are aiming higher.⁵ Enabling more than two thirds of patients to die at home may be limited both by the wishes of patients and carers and by the community facilities available—for example, district nursing, Macmillan nurses, hospice home care services, medical services, and 24 hour care.

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