Non-steroidal Drug-induced Peptic Ulceration

Sir — I am concerned that some of the conclusions drawn in the Editorial by R. D. Sturrock [1] are not based on all of the available clinical data.

Considerable stress is laid on the data regarding Helicobacter pylori infection as a predisposing factor for NSAID ulceration. Although three studies are quoted which support this hypothesis and at least three others exist [2-4], the majority of studies published to date do not indicate that Helicobacter pylori plays a causal role in NSAID-associated gastric damage [5-18], and some studies actually indicate that NSAIDs may exert a protective effect against Helicobacter pylori infection [18,19].

The review quoted [20] to support the view that “H2 antagonists are more effective for duodenal and peptic ulceration” over 2 years old. A considerable number of papers have been published in the intervening period which show that misoprostol is effective in preventing both duodenal and gastric ulcers [21,22] including a direct comparative study in arthritic patients demonstrating the superiority of misoprostol over ranitidine in preventing gastric ulcers [23]. Since one agent can prevent damage at both sites cotherapy of misoprostol and an H2 blocking agent is not required.

It is stated that “long term data for misoprostol are not yet to hand”, however two studies of 1 year duration in patients taking NSAIDs have been published [24,25]. The beneficial results of these studies perhaps answer the author’s caveat in the Editorial that “whether we will be able to protect the NSAID users stomach in the long term remains to be seen”.

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6. Caselli M, Pazzi P, La Corte R, Trevisani L, Stabellini G. Campylobacter pylori infection and NSAID ulceration, and also his comments on the availability of long-term data for gastric protection with agents such as misoprostol. As far as Helicobacter pylori infection is concerned my Editorial indicated that the pre-
cise role of Helicobacter infection in the development of NSAID-induced ulcers is still not clear. I take Dr Fenn's point about the effects of misoprostol on duodenal ulcers but there is as yet no long-term data available—by which I mean in excess of 3 years—which would enable us to make a valued judgement as to whether continued use of gastric protective agents, such as H2 antagonists and prostaglandin analogues, will reduce the risks of peptic ulceration. Hopefully, this data will yield encouraging results but for the moment we must wait and see.

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Arthritis Mistaken for Injury

Sir—In trying to discover the environmental factors that lead to RA the anticipatory clinical approach of "watching little sister" of Deighton et al.[1] has much to commend it as they indicate that if the one sister already has severe seropositive RA and the sisters are both HLA-identical HLA-DR4 positive there is an increased chance that the other sister will develop arthritis during her life-time whilst under surveillance.

Silman [2] was to conclude an editorial on the possibility that RA is an infectious disease by suggesting that "In the absence of further leads from the laboratory all epidemiologists can do is wait with their bags packed for a call to investigate an apparent epidemic".

Both these approaches are in essence prospective which may entail waiting a considerable period of time, though in the end they may prove to be the quickest way to unravel these problems.

I would, however, like to say a little more about the data linkage methods mentioned in the earlier correspondence [3] if for no other reason than for the fact that there is 20 years worth of such data in large numbers of people suitable for such study already in existence in the industrial and Colindale records. There are of course problems interpreting such data but I thought it would be of interest to mention some observations from the earlier work [4].

In some individuals there was a slight increase in 'rheumatic sickness absence' (excluding back and neck problems) about 2 weeks after gastrointestinal illness such as diarrhoea but no such increase after respiratory illness. One might well presume that this phenomenon could be explained by the mechanisms of reactive arthritis. However such reactive mechanisms would not seem to be such a satisfactory explanation as to why there should be an even greater increase in 'accidental injuries' at the same interval after gastrointestinal illness. What probably happened was that a monarticular swelling of a knee for example due to reactive arthritis was initially mistaken for an injury and the diagnosis given as such on the sickness absence certificate. Many rheumatologists will no doubt have been referred similar cases from casualty or orthopaedics when the swelling persists or a second joint becomes swollen.

Apart from pointing out this clinical vignette, it was intended to illustrate that such data can reveal associations, which in conjunction with the tracing back of individuals and along with the Colindale information on peaks of adenoviruses and echoviruses and other infectious agents might further help elucidate the precipitation of rheumatic and other diseases.

Walport et al.[5] comment that 'on the one hand it is possible that there is a single aetiology for many cases of early polyarthritis and the presence of the shared epitope in the third AHVR predisposes to disease progression to severe RA.' This might be taken as a suggestion that the study of the 'many cases' of unspecified rheumatic sickness absence might yet be of help in identifying important environmental factors which can provoke the various arthritides in genetically different people.

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Rhabdomyolysis with Markedly Elevated Serum Creatine Kinase Following Injury to the Tongue

Sir—Since the original description of crush injury and renal impairment by Bywaters and Beall [1] rhabdomyolysis has been recognized in an increasing diversity of clinical situations [2-5]. The levels of creatine kinase (CK), potassium, phosphorous, albumin, dehydration and acidosis have been shown to be predictive factors in the progression of rhabdomyolysis and acute renal failure [6]. We describe a case in which rhabdomyolysis followed a convulsion involving a severe laceration to the tongue.

A 65-year-old male presented for the investigation of a first seizure some 5 days after the event. He described sitting down to watch television, losing consciousness and waking up 18 h later with a large tongue laceration, bloodied shirt and weak, painful legs. Four years previously he had successfully undergone percutaneous nephrolithotomy after a brief history of renal colic, at which time he had normal serum biochemistry. Examination on admission confirmed a 5 cm laceration of the tongue of nearly full thickness (Fig. 1), but was otherwise unremarkable.

Serum biochemistry on admission to hospital (day 5) revealed serum potassium 5.36, urea 41.4, creatinine 933, phosphate 2.01, calcium 2.43, urate 0.84, alanine transaminase 162 iu/ml (normal 5-42), aspartate transaminase 270 iu/ml (8-40), CK >20 000 iu/ml (0-185 male). Glomerular filtration rate was 14 ml/h with 24-h urine volume of 1.75I. Urinalysis and urine microscopy were unremarkable as was renal ultrasound. There was no detectable myoglobinuria, consistent with a serum iotau for myoglobin of approximately 3 h [7]. All biochemical indices rapidly returned towards normal with CK 143, urea 22.4 and creatinine 209 by day 12 (Fig. 2). There were no clinical sequelae.

Post-ictal CK levels have previously been observed to range from 54-2587 iu (0.5-5.3% CK-MB) [8], while levels associated with rhabdomyolysis may reach 238 000 iu [9]. The tongue is known to have a high CK content (1600 iu/g), although somewhat less than that of the glutus or deltoid muscles (2100 iu/g) [9]. Muscle tissue destruction as may occur in a severe laceration is a clear source of liberated CK and this is described in a previous report of rhabdomyolysis caused by bite wounds [10]. Further, a previous review has shown that the aetiology of rhabdomyolysis is most com-

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