bystanders, which was probably instrumental in the survival of two of our patients. Eisenberg and colleagues found that the time taken to initiate cardiopulmonary resuscitation was one of the most important variables in predicting survival from a cardiac arrest and that cardiopulmonary resuscitation by a bystander can significantly reduce this time.[1-4] Unfortunately it appears that many programmes of mass lay education fail to give the trainees sufficient confidence in their abilities, and their skills are not used frequently enough to be maintained.[5,6]

As the majority of cardiac fatalities take place long before the patient reaches hospital, arguments for general practitioners to have easy access to defibrillators are compelling, as demonstrated by our experience and by larger trials.[7] The advantages of early thrombolysis, with its concomitant risk of ventricular arrythmias, provide added reasons for access to defibrillators. The chief problems are: the cost of the equipment (£4000–£6000), though some of this can often be met by local charities, sometimes with help from the British Heart Foundation; organizing an on-call system which allows the doctor to respond quickly; and obtaining and maintaining the necessary skills. None of these problems are insurmountable to a competent practice of the 1990s.

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Neurological adverse effects of naproxen and misoprostol combination

Sir,

Misoprostol is an analogue of naturally occurring prostaglandin E_1, which promotes peptic ulcer healing rates equivalent to those observed with H_2-receptor antagonists.[1] Misoprostol also appears to prevent the development of gastric ulcers induced by non-steroidal anti-inflammatory drugs. It is claimed by the manufacturer that detailed studies show no clinically important pharmacokinetic or pharmacodynamic interactions with non-steroidal anti-inflammatory drugs.

However, as the following case history illustrates, concurrent administration of such drugs can cause neurological side effects. A 59 year old man developed rheumatoid arthritis in 1974. He had suffered from a gastric ulcer in 1959 but subsequent barium meals had shown no evidence of active ulceration. He was given different analgesics for his rheumatoid arthritis and in 1977 ibuprofen was prescribed. In early 1979 he complained of feeling generally unwell with easy fatigue, dyspeptic symptoms and frequent attacks of epigastric pain. He had also developed bleeding piles. A few months later he was admitted to hospital with severe iron deficiency anaemia and was given three units of blood. Again, a barium meal showed no signs of active ulceration. On discharge his treatment consisted of cimetidine, antacids as required, and naproxen for his arthritic symptoms.

Eleven years later, in May of this year, he attended my surgery complaining of abdominal discomfort, pain and nausea. I then prescribed misoprostol to replace cimetidine continuing the naproxen and adding metoclopramide hydrochloride. A few hours after starting this regimen, he developed ataxic symptoms — in his own words he 'felt like a drunk person — staggering all over and vomiting'. Despite this, he continued taking all the drugs for five days and then stopped the misoprostol (which was new to him) of his own volition. He rapidly improved and for two days he was virtually free of ataxia. On the third day, however, in addition to naproxen, he took one tablet of misoprostol but no metoclopramide. The ataxia rapidly recurred and lasted several hours. He took no more misoprostol on that day but on trying one further tablet on the following day the symptoms recurred. The next day he stopped taking misoprostol altogether and replaced it with cimetidine, continuing with the naproxen and the metoclopramide. He felt much better and has had no recurrence of his ataxic symptoms to date.

Jacquemier and colleagues[2] describe two cases of 'neurosensory adverse effects after phenylbutazone and misoprostol combined treatment'. In both cases, the symptoms appeared soon after misoprostol was started, subsided rapidly on its discontinuation and recurred on rechallenge. The explanation for this syndrome is not clear, but it may well have a pharmacokinetic basis. Pending further studies, patients should be warned to discontinue misoprostol should neurosensory symptoms occur.

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Inflammatory cervical smears

Sir,

I was interested to read the recent paper on the inflammatory cervical smear (June *Journal*, p.238). I collaborated in a similar study in July 1988,[1] and I was startled by the difference between our results and those of Kelly and Black.

We looked at 150 consecutive smears over a three month period, 75 of which proved to be inflammatory. All patients with inflammatory smears were recalled and invited to have a full microbiological assessment which included a high vaginal swab, an intra-cervical swab, testing for chlamydia and screening for gardnerella. We found only 12 positive cultures from 74 women with inflammatory smears and in nine cases the organism was *Candida albicans*. Of the 12 patients found to have a positive culture only five were symptomatic — four were positive for candida and only one of the symptomatic patients was positive for chlamydia. This led us to conclude that routine swabbing of patients with inflammatory smears is both expensive and probably not very productive.

Although I agree with Kelly and Black that the appearance of the cervix did not imply a greater chance of inflammation, I cannot agree that women with inflammatory smears suffer symptoms associated with their putative infection. However, I would agree that women whose smears are reported as severely inflammatory should probably have a high