provide medical care for nomadic pastoralists. They state, however, that among the Somalis iron deficiency is rarely fatal. This is not true in the North-eastern Province of Kenya, where 250 000 Somalis live. In 1974 Bonte[1] pointed out that the North-eastern Province was unique among the provinces of Kenya in that blood diseases came top of the causes of death in hospital. Severe anaemia is the commonest single cause for hospital admission in the provincial and district hospitals.

Community studies have shown the high prevalence of anaemia.[2] Of 787 Somalis living in the bush, 190 (24%) had haemoglobin levels less than 8 g/dl and 33 (4%) values below 5 g/dl. Many of these people continue their daily activities with no symptoms, but their reserve is small. In the event of injury, childbirth, or acute malaria (all common events) the value may fall to 2 or 3 g/dl very suddenly. They then die or, if fortunate, survive a journey of 100 miles (160 km) to arrive in hospital with severe heart failure.

Professor Murray and his colleagues suggest that it may be unwise to correct iron deficiency in the face of quiescent infection. It is true that tuberculosis is a major problem and that brucellosis is common. The possible risks of reactivating infection, however, must be weighed against the at least equally serious risk of ignoring severe anaemia. The note of caution is an invitation to nihilism. Those who are building up services for Somali nomads will turn their attention to the anaemia and the pellagra and xerophthalmia which also result from the all-milk diet. They will also turn their attention to the infectious diseases. For a professor of medicine to arrive in a refugee camp of 6000 people bringing the facility for laboratory investigations but only 7000 iron tablets is only comparable to a Lord of the Admiralty reaching the sinking *Titanic* equipped with radar but in a four-seater rowing boat.

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Rethinking antenatal care

SIR,—I am sure that I was not the only reader whose eye was caught by the title of your leading article (28 October, p 1177) only to be disappointed by its wholly technical content. As it correctly pointed out, there is as yet no satisfactory explanation for the inverse relationship between numbers of antenatal clinic visits and peri- and neonatal morbidity and mortality rates. However, unless and until a causal association is definitely disproved practitioners and health authorities have a responsibility to do everything possible to encourage early and continued attendance at antenatal clinics.

The Department of Health has laudably acknowledged this responsibility, and has recently published the highly informative proceedings of a conference it held around this topic in conjunction with the Child Poverty Action Group in April of this year.[1]

The characteristics of late attenders have been well known for some time[2 3] and there is much that can be done to reach them within existing resources—for example, improvement of communication between general practitioners and obstetricians,[4] reorganisation of antenatal clinics, redeployment of community midwives,[5] and health education initiatives specifically aimed at the women concerned, such as the youngest primigravidae. Elsewhere you have recently demonstrated your awareness of the importance of the expectations of the patient,[6] and it is now time for the profession to follow in rethinking not only the content but also the style of antenatal care.

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Smoking and deep-vein thrombosis

SIR,—Mr J K Clayton and others (5 August, p 402) have presented convincing evidence of the "protection" of cigarette smokers against postoperative deep-vein thrombosis and the increased risk associated with overweight for height. They conclude that the explanation for the "protection" of smoking patients provided they are lean remains obscure.

It seems fair to suggest that the "apparent" protection of smoking was due to the "real" protection of lean body habitus, known to be associated with smoking, and was unrelated to smoking per se.

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Postoperative pain

SIR,—We welcome your excellent leading article on this subject (19 August, p 517) because this neglected area of therapeutics has concerned us for some time. Despite the ready availability of potent analgesic drugs patients still rank postoperative pain high on their list of unpleasant perioperative memories. This is not surprising, because very little is known about the relationship between the pharmacokinetics and pharmacodynamics of even the oldest agents.[1]

Using pethidine as a model, we investigated the hypothesis that poor analgesia was due simply to inadequate and variable blood concentrations of the analgesic drugs.[2] We found a direct relationship between blood pethidine concentration and analgesic effect. The minimum therapeutic concentration for postoperative pain was found to be 0·45-0·55 mg/l. This is more than double the value previously reported after a single injection[3] administered immediately after emergence from general anaesthesia. Repeated (4-7) intramuscular injections of pethidine resulted in fluctuating and often inadequate blood pethidine concentrations. Peak pethidine concentrations varied fivefold among patients and twofold within patients. The time to reach peak concentrations varied by a factor of 8 among patients and by a factor of 2 within patients. Blood pethidine concentrations fluctuated markedly above and below the minimum therapeutic level and this was reflected in the analgesic response. Time above the minimum therapeutic concentration varied from 0 to 100% (mean ± SD = 35 ± 36%) for the four-hour period after each injection.

However, in a subsequent study it was shown that only a twofold difference in clearance occurred among patients,[4 5] suggesting that variation in absorption from the intramuscular site is the major reason for poor analgesia. Using a pharmacokinetic simulation system, an intravenous infusion regimen was designed to avoid the variability in absorption rate and to provide stable therapeutic blood concentrations.[4] Controlled continuous intravenous infusion of pethidine produced stable blood concentrations which were maintained above the therapeutic level for postoperative pain and provided excellent pain control. Severe pain was not experienced by any person after three hours and analgesia continued for the duration of the two-day study. The continuous intravenous infusion technique has been successfully used in intensive care for up to 10 days and is currently being evaluated for other uses.

We conclude that (1) pethidine has a therapeutic blood concentration of 0·45-0·55 mg/l for pain following abdominal surgery; (2) variable response to intramuscular injection results from inadequate and fluctuating blood concentrations; (3) the intramuscular route of administration alone is the major cause of the variability in response; and (4) controlled intravenous infusion of pethidine provides continuous analgesia and may offer a significant advance in pain management.

The plethora of new parenteral agents which the pharmaceutical companies have introduced over the past 20 years is *not* a reminder that we have not found the right drug but a reminder that we had not found the optimal mode of administration of perfectly adequate analgesic drugs.

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Crohn's disease—40 years on

SIR,—I was interested in your leading article (21 October, p 1106) and wish to make some further comments. As you suggest, crude recurrence rates are meaningless and they must be related to time.[1] Even then some interesting points may be missed. Although it is now agreed that related to time the recurrence rate for ileal disease is higher after a