Upper gastrointestinal endoscopy

SIR,—While we would agree with the views of Mr V Moshakis (14 April, p 1014) concerning the inadequacies of the conventional barium meal, we disagree with his assertion that endoscopy should be the primary investigation of suspected upper gastrointestinal disease. That conclusion certainly cannot be drawn from our article "Upper gastrointestinal endoscopy: its effect on patient management" (24 March, p 775), to which he refers.

The population we studied was a selected one. There is no evidence that primary use of endoscopy in patients with dyspepsia would usefully increase the yield of treatable disease, and indeed the paper "Open-access endoscopy service for general practitioners" by Dr G Holdstock and others (17 February, p 457) showed no objective benefit as a result of such a policy.

We wish to reiterate our statement that the result of any study of this type "must reflect the degree of selection used in referring and accepting patients for the procedure." It would be unfortunate if our conclusions were misinterpreted as justifying a policy of indiscriminate endoscopy. Radiology as practised to the high standard reached by the Japanese, and increasingly approached in this country, offers a high degree of accuracy, although it can never provide histological material. Where good radiology is available it would be at the very least premature to replace it by endoscopy as the primary investigation, even if this were practicable. There could be a case on grounds of cost and accuracy for the use of endoscopy without a preceding barium meal in some dyspeptic patients, perhaps those over the age of 45 in whom treatment changes as a result of investigation are more likely. This must remain a hypothesis until it is tested.

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Falciparum malaria despite chemoprophylaxis

SIR,—I fully endorse the views of Drs F J Nye and H E Parry on "Falciparum malaria despite chemoprophylaxis" (21 April, p 1091). It is imperative to maintain a high index of suspicion for this disease in all expatriates working in or returning from West Africa who have a febrile illness. I have just returned from Oyo State in Western Nigeria, where I worked for three years. The majority of expatriates (Americans excluded) take pyrimethamine, 25 mg weekly. It is still a favourite prophylactic and is encouraged by many Nigerian doctors. I have treated at least 20 expatriates with falciparum malaria who had taken the drug regularly, and I myself can be added to

this group. All had resided in Nigeria for at least four months before developing symptoms, which were often insidious in onset, supporting the view of partial suppression of the parasitaemia. However, in some (myself included) there was a classical onset of rigors and high fever. One other patient developed falciparum malaria while taking paludrine, 100 mg daily, regularly.

Personally I would not advocate the use of pyrimethamine alone in Nigeria. My choice of prophylactic would either be Maloprim (combining 12.5 mg of pyrimethamine with 100 mg of dapsone) or chloroquine, 300 mg of base weekly. I never saw a case of malaria on either of these regimens.

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Continuous lumbar epidural analgesia for labour and delivery

SIR,—We wish to make two comments regarding Dr J Selwyn Crawford's review paper on continuous lumbar epidural analgesia for labour and delivery (13 January, p 72).

Firstly, there is the author's statement that "the outstanding benefit of abolishing the bearing-down reflex and of pelvic floor relaxation is in cases of breech presentation . . ." (p 73), with which we cannot agree. Since our goal is to promote spontaneous delivery (Bracht's manoeuvre) for this type of malpresentation, we are anxious to preserve the bearing-down powers as much as possible; and this is certainly not attained when an effective second-stage epidural has abolished the bearing-down reflex and has relaxed or even paralysed the abdominal musculature. Furthermore, the pelvic floor relaxation induced by the epidural is not a substitute for a generous episiotomy, which is a must in breech delivery.

The second point we wish to raise is that the favourable influence of epidural anaesthesia on the acid-base balance of the fetus is in fact a fallacy. True, an epidural block reduces the maternal metabolic acidosis; but the fetus lags behind, whereas it should—at least under normal conditions—adjust itself or follow its mother. We compared two groups of elective labour inductions, one conducted without analgesia or anaesthesia (control group) and the other under continuous epidural block (0.25% bupivacaine without adrenalin) and found that there was no clear-cut difference in the acid-base state of the fetus even though "blocked" mothers were less acidotic than the controls. Obviously, the relationship between the mother and the fetus is changed by the regional anaesthesia, and the fetal-maternal differences—that is, the true indices of fetal well-being—are significantly less favourable in patients given an epidural. That both the anaerobic metabolism (fetal-maternal difference in excess lactate) and the CO₂ gradient across the placenta are found to be increased indicates that the fetus of a blocked woman suffers a slight but nevertheless significant degree of hypoxia and hypercapnia as a result of the epidural block. In a first series of cases¹⁻³ our subjects were free to choose the posture (recumbent or lateral) they considered the most comfortable. Because, theoretically, cava compression due to an inappropriate posture in labour might be responsible for the impairment of the fetal haemostasis a second series was assessed, in which the women were not

allowed to lie on their back except during the delivery proper (to be published). Although the postulated effect of compression of the caval vein could not have occurred, the results were identical to those obtained in our first series.

It is not our contention that these findings warrant restriction of properly conducted continuous epidural analgesia for normal labour and delivery. Provided the mother and fetus are continuously monitored and the obstetric and the anaesthesiological staff are experienced, the slight added risk of the epidural is amply compensated for by the benefits of this type of analgesia. However, the fetal hypoxia and hypercapnia triggered by the epidural, although minimal, should be taken as a warning and lead us to be cautious with epidural analgesia whenever the fetus is at risk with respect to its oxygen supply.

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¹ Derom, R, Thiery, M, and Rolly, G, *Excerpta Medica International Congress Series*, No 396. Amsterdam, Excerpta Medica, 1976.

² Vroman, S, et al, *European Journal of Obstetrics and Gynecology and Reproductive Biology*, 1977, 7, 159.

³ Thiery, M, et al, *European Journal of Obstetrics and Gynecology and Reproductive Biology*, 1977, 7, 181.

Increased eosinophil count in operating theatre personnel

SIR,—Recently attention has been paid in the *BMJ* (24 March, p 779) to the health problems of anaesthetists. An uninvestigated area, however, is the effect of operating theatre work on the workers' haematological state. Prolonged anaesthesia leads to bone marrow depression and changes in the leucocyte and differential counts.¹⁻³ Even subanaesthetic concentrations of anaesthetics lead to bone marrow injury.^{4,5} Operating theatre personnel chronically exposed to trace concentrations of anaesthetics⁶ may therefore have changes in their haematological state.

In an operating theatre we studied 10 healthy nurses and doctors for sedimentation rate, haemoglobin concentration, haematocrit, erythrocyte count, and leucocyte and differential counts. They had been regularly employed in operating theatre work for an average of 5.6±4.2 (SD) years. Nitrous oxide was the main inhalational anaesthetic used. Diethyl ether, halothane, and methoxyflurane were used only occasionally. The mean nitrous oxide concentration in the operating rooms was 560-860 ppm (parts per million, v/v) in the morning and 155-460 ppm in the afternoon, and 70 and 20 ppm respectively in the recovery room, measured by gas chromatography.⁷ After three hours' work the nitrous oxide concentration in the end-expiratory air of the test subjects was 31±16 ppm. The controls were 12 healthy members of the hospital staff outside the operating theatre. The venous blood samples taken after fasting overnight were processed by standard laboratory techniques. The leucocyte count was performed by a Coulter S counter and the differential count by counting at least 200 cells. Because the material was not normally distributed, for statistical analysis the non-parametric Mann-Whitney U-test was used.

The sedimentation rate, haemoglobin concentration, haematocrit, and erythrocyte and leucocyte counts were all within normal range without differences between the groups. The proportion of eosinophils was, however, greater in the operating theatre workers than in the controls. When the total eosinophil count was estimated by multiplying the leucocyte count by the proportion of eosino-