anti-inflammatory drugs.[2] These same authors showed that sulindac does not inhibit frusemide-induced renin release or natriuresis. If it is confirmed that sulindac exerts unique differential inhibition of platelet cyclo-oxygenase in contrast to the renal enzyme system, large-scale clinical studies should be performed to determine whether sulindac does indeed have a role in those patients who have compromised renal function or in whom diuretic therapy is required. Older patients are particularly at risk and it is in this age group that a non-steroidal anti-inflammatory drug without renal prostaglandin inhibition would be most welcome.

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Caesarean section

SIR,—Now that your editorials are signed, it is to be hoped that their pungency and forthrightness will not be lost. Professor J K Russell has written an excellent and balanced account of the present position of caesarean section in the United Kingdom (24 October, p 1076). He points out that the number of operations is increasing for small premature breech babies, for fetal distress as detected by the fetal heart monitor, and for dystocia; and that this may not be entirely necessary or desirable. But the sequelae of the freer use of primary section in the interests of the fetus demands fuller discussion.

Labour following section is associated with the risk of scar rupture and fetal death; it requires careful observation and the availability of a skilled anaesthetist as well as an obstetrician in case repeat emergency section is required or a laparotomy is needed to investigate a suspected ruptured uterus. Sooner or later most of us meet a repeat section complicated by a morbidly adherent anterior placenta when death from haemorrhage is difficult to prevent. The more distant long-term complications of repeat section require mention, such as incisional hernia with repeated midline incision, bladder injury with repeated Pfannenstiel incisions, sterilisation with the third or fourth abdominal delivery, and the possibility of eventual difficult hysterectomy.

Finally, surely editorial scorn could have been poured on the midline uterine scar whether in the upper or the lower segment. I suggest that this classical operation is reserved for the occasional case of neglected transverse lie when the fetus is still alive and, of course, for the tragedy of sudden maternal death and that it should not be misused for the small, premature breech baby.

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Hepatitis in pregnancy

SIR,—We, the undersigned anaesthetists, were perturbed to read your leading article (24 October, p 1074) on hepatitis in pregnancy by Dr Roger Williams and R J Ede, in which the authors suggest that, should caesarean section be required in patients with fulminant hepatic failure, epidural blockage would be the anaesthetic method of choice.

It is difficult, even for an experienced anaesthetist, to recommend any particular anaesthetic technique in these extremely unusual circumstances. The presence of a significant coagulation disorder, however, is well documented[1] as one of the few absolute contraindications to the insertion of an epidural block, in view of the disastrous neurological consequences of epidural haematoma formation.

The severe and refractory nature of the coagulation defect in acute hepatic necrosis has been clearly demonstrated.[2] We feel that this alone provides a powerful case against the use of epidural analgesia in this situation, whatever other considerations there may be.

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Hepatitis B carriage and dental treatment

SIR,—We noticed with interest that none of the 100 control subjects of Dr H Rashid and others (10 October, p 948) were hepatitis B surface antigen carriers. A survey of 86 182 blood donors in this country showed that 0·1% were hepatitis B surface antigen carriers.[1] The carrier rate among Indo-Chinese recently arriving in Britain is quite different.

The Rotherham Area dental officer and the local dental committee expressed concern about the increased hepatitis B surface antigen carrier rate among Indo-Chinese. It was decided that all refugees should be screened for hepatitis B surface antigen prior to treatment. Hepatitis B surface antigen screening of 40 Indo-Chinese recently residing in the Rotherham area showed that the incidence of carriage was 10%. Screening was performed by reversed passive haemagglutination using Auscell (Abbott Laboratories) and Hepatest (Wellcome Laboratories). Confirmation was done by enzyme immunoassay (ELISA), Auszyme (Abbott Diagnostics), and neutralisation performed with the Hepatest Confirmation Kit. Three carriers were negative by the gel diffusion technique for hepatitis B e antigen and antibody and one was positive for hepatitis B e antigen by gel diffusion. All had normal liver function tests except for one of the young adults, who showed a slightly raised aspartate transferase concentration (49 IU/l). The carrier rate among Indo-Chinese residing in the Rotherham area is similar to that reported in Canada.[2] All the carriers were males; there were three young adults and a 69-year-old man.

A high incidence of hepatitis B surface antigen carriage among the Indo-Chinese population presents a high risk to dentists and their patients. It seems reasonable to advocate routine screening for hepatitis B surface antigen for those refugees prior to dental treatment. After discussion and consultation,[3] the Rotherham Area dental committee has agreed on a policy for the dental treatment of such carriers. Indo-Chinese hepatitis B carriers who require dental surgery will be directed to Rotherham District General Hospital, where they will be treated by the consultant dental surgeon and his staff. Conservation treatment is made available for Indo-Chinese hepatitis carriers at one of the area health authority community dental clinics at Rotherham and the patients will be treated by a clinical dental officer. The clinic is provided with two packs provided by the central sterile supply department containing all the required instruments and pads, as recommended by the Expert Group on Hepatitis in Dentistry.[4] A portable suction machine is provided by the central sterile supply department for the treatment of these carriers and will be collected for autoclaving after each treatment. Carriers are treated at the end of clinical sessions.

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Indian childhood cirrhosis

SIR,—In his leading article on Indian childhood cirrhosis (17 October, p 1006) Dr A Paton states that the disease is extremely rare in expatriate children and asks for information about cases encountered in Britain.

We recently described[1] two brothers in Manchester with this disease. The older boy had been born in Bangladesh but the younger was born in England and thus represents the first reported example of Indian childhood cirrhosis in a patient born in Britain. Although the usual age at presentation is said to be 1-3 years of age, patients have previously been described who were more than 5 when first seen.[2,3] It has even been suggested that children having asymptomatic disease with hepatomegaly and disturbed liver function tests may be seen up to 12 years.[4] One of our patients presented at the age of 10 with a history of non-specific illness for a number of years. The other was aged 5 and had been unwell for four years. The parents were first cousins.

Our cases suggested a strong genetic component in the disease, although we could not rule out an environmental hazard common to Bangladeshi families in this country and in their country of origin. The most popular hypothesis to explain the aetiology of the disorder is excess copper storage,[5,6] discussed briefly in the leading article. However, from the available data on copper in the liver in Indian childhood cirrhosis it is impossible to distinguish between a primary pathogenetic role for copper and deposition secondary to the disease itself—for instance, due to cholestasis. Cases from the Indian subcontinent invariably appear to exhibit large amounts of orcein-positive material within hepatocytes. Such deposits were present in our two cases, but were less prominent than in the King's College Hospital series; and the liver copper concentration, though raised, was appreciably lower than the level found in Wilson's disease, where excess copper storage is considered to play a major pathogenetic role. The finding of high liver arsenic levels in Indian childhood cirrhosis[7] further suggests that it may be the liver disease itself that encourages deposition of this element and of copper. In addition to copper, arsenic, and the