and in 7 of 9 patients with intrahepatic cholestasis, proving a poor discriminatory test.

As ultrasound is safe, non-invasive, and does not depend on hepatobiliary function we routinely use it to screen all cholestatic patients before proceeding to liver biopsy or laparotomy as required.

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Drs Mowat Hylton, and Meire comment:

All infants with conjugated hyperbilirubinaemia admitted to our unit have ultrasound examination before liver biopsy. If dilated extrahepatic ducts are found, laparotomy for presumed choledochal cyst is considered without prior liver biopsy. We would see little prospect of ultrasonography of the extrahepatic biliary tree helping in the differentiation between extrahepatic biliary atresia and infants with severe intrahepatic cholestasis with reduced bile flow. Even with operative cholangiography at laparotomy, experienced surgeons may make a presumptive diagnosis of atresia in patients with patent bile ducts, and proceed to unnecessary surgery with deleterious results.³

The intrahepatic bile ducts in extrahepatic biliary atresia are commonly very narrow. We have not observed dilated intrahepatic ducts on ultrasound in over 60 infants with extrahepatic biliary atresia, aged less than 20 weeks, examined in the last three years. When ultrasonography of the hepatic parenchyma has been compared with histological observations, marked discrepancies have emerged.⁴

Our summary concluded 'In patients in whom genetic disorders, such as alpha-1 antitrypsin deficiency had been excluded, interpretation of liver biopsy specimens, together with ¹³¹I rose bengal faecal excretion, remains the most accurate means of identifying those infants who need surgery for biliary atresia and of avoiding unnecessary laparotomy in infants with intrahepatic disease'. Our experience since completing this work reinforces this conclusion but the word 'interpretation' should have been qualified by 'skilled'.

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Changing incidence of infantile hypertrophic pyloric stenosis

Sir,

The papers from Birmingham¹ and from South Wales² show an increased incidence of pyloric stenosis. The authors have various suggestions for this increase; but is it anything more than better diagnosis? In most paediatric units the decision to operate is made on the basis of a convincing history and the finding of a palpable pyloric mass. Over the years I have been amazed that, even at times when I have been a little doubtful about the presence of such a pyloric mass, surgeons at operation have *always* found hypertrophic pyloric stenosis and performed a Ramstedt's operation. This must mean that, in general, I and perhaps other paediatricians are under-diagnosing pyloric stenosis, because I cannot believe that for any condition I am 100% correct.

In the last 10 years there has been a great increase in both the number and the expertise of paediatric staff in all British paediatric units, both peripheral and central. Might not the increased incidence of pyloric stenosis be the result of fewer such babies being missed (after all, the natural history for the less severe case is for spontaneous resolution to occur with time). If this is not the explanation, can the authors explain how, in contrast to the rest of my life, I have never been wrong in diagnosing pyloric stenosis?

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Drs Webb, Dodge, and Lari comment:

Professor Meadow's clinical expertise has never been in doubt, but we hasten to assure him that not all paediatricians are quite as good as he at detecting pyloric stenosis. He may well be right that pyloric stenosis is underdiagnosed, and there is some evidence that silent cases do occur.³ However, although there has been an increase in the number of paediatric staff throughout the country, their expertise in palpation of pyloric tumours is probably no better than that of their predecessors, and we have personally encountered senior registrars who have never felt a pyloric tumour.

Before the abrupt rise in incidence which we and others have reported, most paediatricians were under the impression that it was a declining problem and the few epidemiological studies reported would seem to support this contention.^{4,5} There is no evidence from our own records that the recent excess of babies with pyloric stenosis are of a milder clinical nature, who would formerly have been missed, nor has the increase in paediatric manpower been abrupt, at least in this part of the world. One of us (JAD) spent several years actively looking for cases all over Belfast, and can assure Professor Meadow that 'any infant who vomited' was regarded as a potential candidate. In spite of this enthusiasm, the recorded incidence of hypertrophic