Correspondence

References
2 Dunn PM. Vitamin K1 for all newborn babies (letter). Lancet 1982;ii:770.

Hepatitis syndrome in infancy—an epidemiological survey with 10 year follow up

Sir,

There are several points that we must make about the paper of Dick and Mowat and particularly about a comparison of their results with those that we published in your Journal in 1977 and again recently.

It is nearly impossible to compare the two series because the ascertainment is so different. Our studies were restricted to patients whose jaundice began in the first four weeks of life, whereas Dick and Mowat include patients whose jaundice began as late as the fourth month. They give no figures for the incidences of different ages of onset. The patients that we excluded from our studies because their onset was in the second month of life all did well, and it is our experience that chronic liver disease or death is uncommon in babies with an onset of jaundice between one month and four months.

Secondly, we required jaundice that was sufficiently severe and persistent to warrant a liver biopsy. While it is difficult to compare criteria used for performance of a biopsy in different centres, this inevitably meant that we excluded some fairly mild cases with jaundice that persisted for over two weeks. Our study also included a number of babies who died of liver disease in the first week or two after birth, who would not have survived long enough to satisfy the Dick and Mowat criterion of jaundice persisting for more than two weeks. Some were never jaundiced and were diagnosed only on pathological examination. Autopsy rates in newborn infants in Victoria were very high at the time of our study.

Our cases were ascertained over a 14 year period that included roughly 750 000 births in the state of Victoria. Our incidence of extra hepatic biliary atresia (1 in 15 000) was similar to that observed in the South East England study. Our incidence of idiopathic hepatitis, however, was 1 in 11 000, less than half the English figure. These figures support the view that the new study is likely to have included a number of cases that were much more mild than the one we included.

The only figures that can be legitimately compared between the two studies are the incidences of late death and of persisting liver disease. These outcomes were similar in incidence when related to total births. We encountered six late deaths due to idiopathic hepatitis among 750 000 babies born—an incidence of one in 125 000 births. The comparable figure for the English study is one in 67 000 births (two in 134 000). Our incidence of persisting liver disease was seven in 750 000 babies born or one in 107 000, and the comparable figure for the English study was one in 67 000 (two in 134 000).

References

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Drs Dick and Mowat comment:
We are a little surprised at the comments of Professor Danks and Dr Smith. We did state in our introduction that variation in outcome (between series) may be due to case selection, referrals patterns, and the completeness of follow up. We are pleased that they found that the incidence of biliary atresia and of late deaths and of persisting liver disease in those without an apparent cause in our small series was similar to those found in their more extensive careful studies in Victoria. There were differences in the mode of ascertainment of our study, and of course such differences as well as differences in the genetic background and environmental factors will have contributed to the rather minor differences between our report and that from Melbourne. All but nine of our infants presented during the first two weeks of life, but seven of those nine presented after four weeks. One has persisting liver disease. Outside the reported study we have observed other infants presenting after the neonatal period who have progressive liver disease. Some of these have metabolic disorders, but others were cryptogenic.

With regard to the severity of the disease in the study, 13 of the 28 with idiopathic disease and all of those with α1 antitrypsin deficiency had acholic stools and in these a diagnosis of biliary atresia was seriously considered. Ninety per cent of our patients had percutaneous liver biopsies. We therefore do not agree that it is nearly impossible to compare and contrast the results in the two series. Both are conscientious attempts to report largely similar disorders in fairly circumscribed geographical areas in which it was possible to have almost complete identification of cases falling within the criteria adopted for the study. In this regard the South East study and the Melbourne study are more comparable than the others to which we refer in our report.