LETTERS TO THE EDITOR

Research in community child health

SIR,—Dr Polnay's annotation is to be applauded and if his research agenda emphasises operational aspects this befits a speciality where new and old practices need rigorous evaluation.¹ Epidemiology as a research skill deserves further comment. Community paediatricians are peculiarly well placed to use epidemiological techniques in studies of practice and disease. Having a service role in the community they already know their population and are well accepted by public and colleagues alike. Hence it is to be regretted that while there is considerable research into child health in the community this is conducted by educationalists, sociologists, *et al*, rather than community paediatricians.

Being the basic science of public health some understanding of epidemiology is needed by any community paediatrician looking beyond individual patients and a district service is strengthened if an established post carries a deeper knowledge of the subject. However acquiring appropriate training is difficult. Courses are dominated by adult medicine and a children's doctor may be fed an indigestible diet of cardiovascular and cancer epidemiology. The paediatric courses at the Institute of Child Health are an unusual exception and similar training should be offered elsewhere. However, as Dr Polnay notes, the limiting factor is a famine in training funds for children's doctors and at the core of this is ambivalence over their role in the rediscovered speciality of public health. It is unfortunate that the substantial forces of community paediatrics were ignored by those planning the future,² and the allocation of training funds will probably reflect this myopia. I would suggest that one way out of the impasse is for community paediatrics to recreate links (joint posts, joint research) with departments and units of community/public health. It should be possible to do so from a position of strength within broader paediatrics and indeed such initiatives have started from some academic institutions. Hence Dr Polnay is doubly correct to regret that not all departments of child health have taken up the challenge of community work.

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- 1 Polnay L. Research in community child health. *Arch Dis Child* 1989;64:981-3.
- 2 Committee of Inquiry Into the Future Development of the Public Health Function. *Public Health in England*. London: HMSO, 1988.

Screening infants for hearing loss

SIR,—Brown *et al* have critically evaluated the distraction hearing test as a screening pro-

cedure.¹ Their results are similar to those obtained from a smaller survey conducted over a one year period in Litherland, a predominantly working class district of South Sefton area health authority, Merseyside.

Over the year period 1986-7, 261 children, or 68% of the target population, were screened. Of those who were screened eight children, or 3%, failed a second test and were referred to the audiology clinic. Five of the eight children were confirmed as having a moderate severe conductive hearing loss; the three remaining children were considered to have normal hearing. One child was treated with myringotomies and grommets, and the remainder were eventually discharged from the ear, nose, and throat clinic. It was estimated that approximately 240 hours of health visitor time were spent in performing the test, persuading defaulters to attend, and in administration.

Brown *et al* found that attendance for the test varied with immunisation record. In Litherland 41% of non-attenders completed a primary immunisation course compared with 71% of those who attended for the distraction test. Only 52% of non-attenders had both developmental checks, at 6 weeks and 6 months of age, compared with 81% of attenders.

The technique of the four health visitors who performed the tests was examined. It was found that the testing technique was overall satisfactory but that minimal distraction levels were not always possible due to ambient noise in clinic premises. The usefulness of the distraction test as a means of detecting sensorineural deafness must be questioned. In Litherland one significant case of conductive deafness was identified at the expense of six weeks of health visitor time. Newton has suggested that at best only 50% of cases of sensorineural deafness will be identified at 8 months by means of the distraction test and that those deaf children who 'pass' the test are detected at a significantly later age.² Early detection of nerve deafness is highly desirable; however, the distraction test appears to be of limited use in achieving this aim.

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- 1 Brown J, Watson E, Alberman E. Screening infants for hearing loss. *Arch Dis Child* 1989; 64:1488-95.
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Acquired transient protein C deficiency in neonatal cardiac failure

SIR,—Protein C is a vitamin K dependent plasma protein of hepatic origin. Activated protein C inhibits coagulation factors Va and VIIIa and stimulates fibrinolysis. Hereditary protein C deficiency has been widely described. Heterozygotes with protein C activity concentrations of around 50% normal are at increased risk of venous thromboembolism.¹ Homozygotes with protein C concentrations of less than 5% normal may present in the neonatal period with purpura fulminans² or inferior vena caval thrombosis.³ Acquired protein C deficiency has been described in

liver disease, disseminated intravascular coagulation, adult respiratory distress syndrome, and in the postoperative period.⁴

Two infants born at this hospital suffered a severe non-hereditary neonatal protein C deficiency in association with cardiac failure due to a supraventricular tachycardia.

The first infant was born at 34 weeks' gestation, by emergency caesarean section for fetal distress. At birth the baby boy was noted to have a blue/white pulseless right arm, further examination showed a supraventricular tachycardia, tachypnoea, and hepatomegaly. Treatment with digoxin, frusemide, and heparin corrected the supraventricular tachycardia and cardiac failure, and the pulses gradually returned to the right arm with reperfusion. Initial protein C activity was <5%; after treatment with vitamin K and correction of the cardiac failure the activity rose to 25% at two weeks and subsequently to 79% at 15 months. Both parents have normal protein C activity.

The second infant was born at 38 weeks, gestation by vaginal delivery. At birth he was grossly hydropic with hepatomegaly, oedema, and noticeable ascites. He also had a supraventricular tachycardia and in addition a widespread purpuric rash. His platelet count was normal and screening for intrauterine viral infections was negative. His protein C activity was also <5% and improved rapidly to 30% activity by the age of 1 week and 45% by 2.5 weeks with vitamin K treatment and correction of cardiac failure. His parents also had fully normal activity.

Both of these babies showed transient severe reduction in protein C activity in association with neonatal cardiac failure; neither is homozygous for hereditary protein C deficiency and none of the parents is heterozygote. Both infants developed neonatal jaundice requiring phototherapy and both had further evidence of vitamin K deficiency with prolonged prothrombin time and reduced factor II (5% and 3% respectively). Neither infant had evidence of disseminated intravascular coagulation.

We suggest that cardiac failure in the neonatal period (when liver function is poor and vitamin K deficiency may coexist) can impair hepatic production of protein C with consequent risk of thrombotic complications. If this is shown to be the case it will have significant implications for the management of infants with major congenital heart disease, especially those undergoing neonatal surgery or invasive investigations. To our knowledge no one has systematically investigated protein C concentrations in severe neonatal heart failure.

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