seem to be missing—continuity of care and an appraisal of the 'whole child.'

Reference

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Drs Colver and O'Callaghan comment:

Thank you for the opportunity to reply to the letter of Dr Berridge and colleagues. We are pleased that they also operate a 'selective system' and provide further arguments to support the concept.

We argued that 'selectivity' is an underused practice, not a revolutionary concept. A quarter of the children selected to be seen because the preschool record was not available had had their essential screening tests and to that extent there was repetition. We do not understand the criticism that to gather information from people who already know the child is a 'disjointed' approach. Technicians are trained to use the pure tone audiometer and the Keystone apparatus. Altogether 98% of children were screened for hearing and vision, of whom 7% failed hearing and 4% vision. Two children over the last five years received hearing aids after failing the hearing test and an unknown number received grommets. We do not known the outcome of the vision failures but do know that 90% attended their family doctor or optician. We recognised the gaps in information but already have clearer policy and more information than most health districts.[1]

A different philosophy towards integration within primary care may distinguish the Southampton system from ours. We feel that preschool surveillance should be integrated in primary care,[2] and that 'continuity of care' comes best from the family doctor who already provides continuity for the majority of medical problems. Parents and teachers are best placed to look at the 'whole child' in the first instance. If a concern presents through school which may have a medical component, we agree that the school doctor is then ideally placed to look at the whole child.

The late Dr Tyrrell argued for fewer, better trained school doctors taking referrals from family doctors or school nurses, not duplicating their work.[3] Greater use of selective examination and greater emphasis on problem orientated work will help define this more restricted and difficult role.

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Dry lung syndrome after oligohydramnios

Sir,

We were interested to read Professor McIntosh's recent paper describing 'functional' lung hypoplasia after preterm membrane rupture (PROM).[1] This has not been our experience among 30 pregnancies with PROM of greater than two weeks' duration followed prospectively; in that series some neonates developed anatomical but not 'functional' pulmonary hypoplasia (as defined by Professor McIntosh).[1] Eight of the 30 neonates died within 48 hours of life from respiratory failure, despite inflation pressures of up to 55 cm H_2O. At necropsy pulmonary hypoplasia was confirmed in all.[2] Of the 22 infants who survived, 11 required no respiratory support, and 11 required artificial ventilation. One child had severe respiratory distress syndrome and required a maximum peak inspiratory pressure of 30 cm H_2O, but among the other 10 the mean maximum inflation pressure was 17 cm H_2O (range 12–25 cm H_2O).

Professor McIntosh attributed 'functional' lung hypoplasia to dry lungs, which resulted from leakage of lung fluid exacerbated by external compression on the fetal chest. In our series lung hypoplasia occurred in pregnancies with membrane rupture at significantly earlier gestations than those pregnancies of neonates without pulmonary hypoplasia ($p<0{\cdot}01$). The degree and duration of the oligohydramnios produced by membrane rupture was, however, not significantly different between the two groups, which suggests that compression on the fetal chest cannot be the sole mechanism in producing pulmonary hypoplasia. Indeed, the most significant difference between our two groups was the absence of fetal breathing movements in the group dying from pulmonary hypoplasia, confirming our preliminary findings reported earlier.[3]

Our experience of a further five infants, ventilated during the same time period as our study group, but from pregnancies not complicated by PROM, has lead us to consider explanations other than a 'dry lung' for the ventilatory pattern described by Professor McIntosh.[1] Three of the infants had severe respiratory distress syndrome, evidenced by classical chest radiographs, and two were septicaemic. All five infants required high pressure ventilation (greater than 30 cm H_2O) both for resuscitation and subsequent ventilation. After 72 hours, and with improvement in both conditions, the ventilatory pressures were rapidly decreased and all five infants were successfully weaned over the subsequent 48 hours.

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Marfan's syndrome in mitral valve disease

Sir,

We read with interest the report by Marlow *et al* on an infant with Marfan's syndrome and mitral valve dysfunction who died after acute illness with gross cardiac failure.[1] The authors stated that 'disease affecting the mitral valve is usually more benign than that of the aortic valve . . ..'

This statement, however, may not be applicable to infants and young children with Marfan's syndrome. We have recently reported on the clinical course and the echocardiographic findings in 25 children with this syndrome.[2] Prolapse of the mitral valve leaflets was shown by echocardiography in all the patients and aortic pathology in 80% of the cases. Five patients died during the follow up period (mean 5±4·5 years); two of them were infants with severe mitral insufficiency and progressive congestive heart failure. The other three patients died during the second decade of life from complications of the aortic pathology. Another series reported by Sisk *et al* showed that among 15 patients with Marfan's syndrome diagnosed before 4 years of age, mitral valve dysfunction was the leading cause of cardiovascular morbidity and mortality.[3]

These data indicate that mitral valve disease is the most common cause of congestive heart failure and death in infants and young children with Marfan's syndrome.

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Medical staffing in paediatric departments in district general hospitals

Sir,

Dr Nelson is right.[1] At this hospital we have only two senior house officers, often with no previous paediatric experience, on a 1:2 rota, without any intermediate cover, to cover general and neonatal wards including labour ward resuscitation. We do not therefore have a 1:3 rota, nor a 'safety net'. Five years of local discussions have not altered this. What is needed is a united and forceful voice from the British Paediatric Association nationally, coupled with specific advice for consultant paediatricians on how to get past the embargoes on increasing junior staff.

Reference

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Maternal narcotic abuse and the newborn

Sir,

We were interested in the report by AlRoomi *et al* concerning maternal narcotic abuse and its effects on newborn infants.[1] Women on Merseyside smoke heroin rather than inject it and we would like to report aspects of our experience of 37 infants born to women who inhaled heroin.

(1) AlRoomi *et al* used phenobarbitone and chloral as first line drugs for the treatment of symptoms, but they make no comment on the effectiveness of these agents. In our series 15 (40%) babies also developed withdrawal signs but our initial experience with phenobarbitone by injection was disappointing. We subsequently tried oral chlorpromazine, which we stopped using because of its epileptogenic potential, although it was effective. We now consider that treatment with pure aqueous morphine sulphate (0·125 mg) given orally and as required is pharmacologically appropriate, have found it effective, and would recommend its use.

(2) In our series only eight (22%) children were subject to legal constraints, compared with 38% in AlRoomi's series. While this may simply reflect a different approach to social management, it may be that heroin abuse by inhalation has less disruptive effects on family life.

(3) We also found that these children were not brought to hospital for follow up appointments. We now consider that community based surveillance is more appropriate.

Reference

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