Aberdeen when I was there a few years ago. Handling for radiography is probably less risky than direct inspection.

By excluding from radiological examination the five cases of acute epiglottitis the Glasgow workers have excluded the five cases in which neck radiographs would have been diagnostic, so it is not surprising that Goel concludes that neck radiographs were unhelpful.

References


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Dr Goel comments:

I was interested to read Dr Porter’s comments on my letter. I think most would agree that the diagnosis of acute epiglottitis should be made on the history, general appearance of the child, and the quality of stridor. A lateral radiograph of the neck as suggested by Dr Porter is a useful diagnostic investigation provided expert radiological interpretation is available. This is not always the case, especially at night. Furthermore we would not recommend this procedure in infants in whom acute epiglottitis is suspected simply because the handling necessary to obtain adequate films may be very disturbing to the child and may also precipitate acute obstruction. In view of this, if there is doubt about the diagnosis direct inspection is the only appropriate method. I agree entirely with Dr Porter that under no circumstances should this be carried out unless there are adequate facilities available to relieve acute obstruction immediately should it occur.

In this hospital routine radiographs of the neck and chest are now carried out only to exclude the possibility of a foreign body inhalation and are not included in the work up of croup syndrome.

Parental reaction to auditory response cradle testing

Sir,

I was interested to read the paper by Drs Bhattacharya, Bennett, and Tucker on the follow up of newborn babies tested with the auditory response cradle. The devastating consequences of delayed diagnosis of hearing loss to language and speech development are only too well known. I should like to ask how parents accepted the news that their babies might be unable to hear? Were many frustrations expressed by the parents with regard to possible over optimistic expectations raised as a result of such an early diagnosis of hearing loss without immediately available treatment? So many of the problems of screening do not relate to the test itself but to coping with the many consequences, medical, psychological, and social, of making a diagnosis.

Reference


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Dr Tucker and co-workers comment:

We thank Professor Davies for drawing attention to the aspect of parental reaction to auditory response cradle testing. In our paper, we did not discuss this in any detail, but it is an essential part of our general approach with the parents. At the onset of the testing the parents are informed that this is a screening technique only and not a diagnostic one, and should the baby fail the cradle on two occasions further diagnostic tests will be done almost immediately to confirm whether there is indeed hearing loss. As we are able to confirm the diagnosis within a day or two of the cradle test, we have been able to observe the parents’ reactions to this early diagnosis and there were few adverse comments by any of the parents concerned. The ability to make an early diagnosis of hearing loss without being able to provide treatment is self defeating, so we have researched this aspect in some detail, and try to provide amplification as early as possible. We would agree with Professor Davies completely in his philosophy inherent in making an early diagnosis, and we therefore spend considerable time discussing the medical, psychological, and social implications of such a diagnosis with the parents. The advantage of reliable early diagnosis together with the ability to offer immediate treatment outweighs all the other disadvantages, but we remain deeply concerned and involved in helping parents accept the diagnosis.

Selective bronchial intubation for pulmonary emphysema

Sir,

During the past 10 months we have encountered in our neonatal unit five preterm infants (mean birthweight 1-24 kg, range 0-94 to 1-64 kg; mean gestation 29 weeks, range 27 to 30 weeks) with unilateral pulmonary interstitial emphysema. Our experience with selective bronchial intubation as a treatment for this condition is not as encouraging as that reported by Drs Campbell, Zarfin, and Perlman. Three of the five infants were treated with selective intubation. One infant was suffering from a right sided and two from left sided lesions. A total of four selective intubations were carried out for a mean duration of 60
hours. On no occasion was there more than a temporary improvement in Po2, while the disturbance to the infant during the manipulation required to place the endotracheal tube was considerable. In particular, passage of the endotracheal tube into the right main bronchus without occlusion of the right upper lobe was extremely difficult.

One of the infants in whom selective intubation was carried out subsequently underwent a thoracotomy, where multiple pleurotomies were performed. Again, improvement was modest and short lived.

In four of the five infants, including the infant in whom surgery was performed, spontaneous resolution and recovery occurred subsequently. The fifth infant died of cor pulmonale complicating bronchopulmonary dysplasia after a period of time at home.

We feel that selective bronchial intubation has a place in the management of pulmonary interstitial emphysema but that any improvement may be limited. In particular, improvement may fail to occur when pulmonary interstitial emphysema is associated with bronchopulmonary dysplasia, rather than as a complication of the idiopathic respiratory distress syndrome. The radiological distinction between these two is not always possible.

Reference


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Non-immunologic hydrops fetalis

Sirs,

We read with interest the paper by Iliff et al.\(^1\) and would like to add a further cause of non-immunologic hydrops fetalis.

At routine ultrasound examination a 32 year old primigravida who was 23 weeks pregnant was found to have a grossly hydropic infant, with ascites and displacement of the fetal heart to the left. No irregular blood group antibodies were found and viral serology was negative. A detailed examination of the fetal heart using echocardiography showed normal cardiac anatomy, although the right atrium was rather dilated.

The presence of gross hydrops was confirmed after delivery at 27 weeks' gestation and despite attempts at resuscitation, the infant died 30 minutes after birth. Subsequent investigations failed to show any evidence of haemolysins, blood group incompatibility, congenital infection, or chromosome abnormality.

A full necropsy showed that there was atresia of the right main bronchus associated with noticeable enlargement of the right lung and a shift of the heart and mediastinum to the left. The left lung was compressed and severely hypoplastic. There was also moderately severe tracheobronchomalacia with partial deficiency of the tracheal cartilage and collapse of the posterior membrane.

Histology of the right lung showed distension with an accelerated maturation and numerous cup shaped alveoli. The left lung showed failure of expansion but the maturity was consistent with the gestational age: hyaline membranes were noted together with occasional inhalation of squames. Bronchial cartilage was present in both lungs. This cause of non-immunologic hydrops fetalis has not previously been recorded, although cases of similar nature, for example adenomatoid malformation of the lung and intrathoracic tumours causing mediastinal displacement, have been the subject of previous reports.\(^2\)

References


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Transient hyperphosphatasemia

Sirs,

We were interested to read Arthur et al.'s short report on the benign nature of transient hyperphosphatasemia.\(^1\) Although this is the first account in the English published reports, the syndrome has recently been reviewed by Nathan.\(^2\) He described six cases in 1980 and reviewed the 28 patients previously reported: four more cases were added by Rosalki and Foo.\(^3\) Only one of these patients was found to have a malignancy.

At this hospital, an 18 month old infant presenting with Letterer-Siwe disease was found to have an alkaline phosphatase value of 4153 IU/l (normal adult range 90 to 330 IU/l). This increased to 12 978 IU/l over the next week, while other biochemical tests of liver function remained normal. An ultrasound and computed tomogram of the liver, and a bone scan showed no abnormalities. Isoenzyme analysis was typical of that found in transient hyperphosphatasemia and the alkaline phosphatase activity returned to normal over the following month. In another patient, a 2 year old with leukaemia on UKALL VIII maintenance treatment, alkaline phosphatase increased to 2050 IU/l after a period of normal liver function tests. As the child's other parameters of liver function were all normal, further investigations were deferred and within a month the alkaline phosphatase had reverted to its previous value. Failure to appreciate the possibility of transient hyperphosphatasemia could have led to unnecessary further investigation in the first patient and an inappropriate reduction in 6-mercaptopurine dosage in the second. Thus it should be noted that hyperphosphatasemia may also be a benign finding in children with an already established malignancy and may not be indicative of any deterioration in their condition.

References