occasions and to and fro movement of the fluid in the lower oesophagus.

Twenty patients had both barium and ultrasound examinations as described above. There was agreement on 18 examinations; 7 patients showed reflux and 11 showed no abnormality on either examination. One patient had definite reflux on ultrasound examination but 'minimal' reflux was reported on the radiographic examination. On the 16 year old patient, reflux was seen on the barium swallow but it was not possible to outline the oesophagus on ultrasound because of interposition of lung between the heart and the aorta.

Discussion

We had observed that some infants and children had a line of 'bright' echoes behind the heart during two dimensional echocardiographic examinations. Closer scrutiny showed that these echoes represented fluid filled oesophagus caused by occasional reflux. This prompted us to undertake the present study to assess the possibility of diagnosing reflux using ultrasound.

It became obvious after the first few examinations that the method was as accurate and in some ways superior to the barium swallow. Ultrasound examination is likely to be more physiological as it is not necessary to use contrast media and infants are now examined after normal feeds. It is also more accurate than the barium examination in which one uses very short radiographic screening time to minimise the radiation dose. This leads to false negative examinations in 15% of cases.² With ultrasound it is possible to scan continuously for several seconds and the examination may be carried out over several minutes (usually 15 minutes) by intermittent scanning. Gastro-oesophageal reflux

may be reliably diagnosed by ultrasound but resolution of anatomical detail is generally insufficient to allow comment upon the presence or absence of an associated hiatus hernia. Only two of 20 patients in this study were over 3 years of age so the application in older children remains to be assessed.

Most radiology departments have real time ultrasound equipment for neonatal head and abdominal examinations. A sector scanner is probably the equipment of choice for this, as for all paediatric ultrasound examinations.

New diagnostic modalities are often criticised for providing some additional information but not replacing established techniques. In our department, ultrasound examination has replaced conventional barium examinations in all cases of suspected gastro-oesophageal reflux in children and is also used in follow up of children receiving antireflux treatment. In addition to avoiding unnecessary exposure to radiation, there are undoubted savings in the use of contrast media and x-ray films that have important implications if all infants presenting with near-miss sudden infant death syndrome are to be screened for reflux. Ultrasound is a safe, reliable, and rapid method of diagnosing reflux.

We thank our consultant paediatrician colleagues, particularly Dr A Boon.

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Serum immunoglobulin concentrations in febrile convulsions

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SUMMARY The mean, age adjusted, serum IgA values of 47 children with febrile convulsions were almost identical to those of controls. Five children had serum IgA values less than 0.1 g/l by nephelometry, suggesting that in some cases at least there may be an association between a low serum IgA concentration and febrile convulsions.

A distinction is drawn between selective IgA deficiency and low serum IgA.¹ In selective IgA deficiency, serum IgG and IgM values are normal but serum IgA is undetectable by radial immunodiffusion (<0.02 g/l). In low serum IgA, serum IgA can be detected by radial immunodiffusion but is more than two standard deviations below the mean for age.

It has been suggested that low serum IgA values in childhood may be associated with an increased susceptibility to febrile convulsions.²⁻⁴ This suggestion has not been adequately tested because of the difficulty in obtaining reference ranges for serum immunoglobulins in children and the inaccuracy of many reference ranges used.⁵ We have recently developed a reference range for serum immunoglobulins derived from sera from 298 healthy children attending infant welfare clinics and schools in the Harrow area.⁵ An unexpected finding was that serum immunoglobulin concentrations in childhood are not log normally distributed, and formulas were derived to convert the raw data to normal distribution, to allow calculation of reference ranges.⁵

We compare serum immunoglobulin values from 47 children presenting with febrile convulsions, with the predicted, age matched values obtained from our reference ranges.

Materials and methods

Venous blood (1 ml) was obtained from 47 children presenting consecutively to the paediatric department with a simple convulsion associated with fever greater than 38°C, and who were being venesected for other investigations. Children already on anti-convulsant medication were excluded, as anticonvulsants are known to lower serum IgA values. Serum immunoglobulin values were determined using a Hyland laser nephelometer PDQ system as previously described.⁵ Sera from children with serum IgA less than 0.1 g/l by nephelometry were also tested by double antibody radioimmunoassay.⁶ This study was approved by the Northwick Park Hospital ethical committee.

Results

Serum IgG, IgA, and IgM values of all 47 children with febrile convulsions fell within the 95% reference ranges calculated from sera from 298 healthy children.⁵ Only the serum IgA values are shown in the accompanying Figure. The five children who had serum IgA less than 0.1 g/l by nephelometry, all had IgA detectable by immunoassay (greater than 0.03 g/l). They are shown on the Figure as IgA = 0.05 g/l and analysed as such statistically. As the lower limit of normal serum IgA in our reference range is below 0.1 g/l until 26 months of age and as all five children were below this age, none could be considered as definitely having low serum IgA.

Analysis of the immunoglobulin values were performed by transforming each value to the scale in which the data were normally distributed using the

Figure Serum IgA values for 47 children with febrile convulsions.

The reference range, mean (2SD) is shown. Children with serum IgA less than 0.1 g/l are shown as 0.05 g/l.

Table Analysis of age specific SD scores for children with febrile convulsions

Immunoglobulin	No	Mean	SD	t	P
IgG	47	0.14	0.89	1.06	0.29
IgA	47	0.05	1.31	0.26	0.80
IgM	47	0.25	0.90	1.89	0.06

equations previously described.⁵ A standard deviation score (SDS) was calculated,

$$\text{where SDS} = \frac{\text{observed value} - \text{mean value}}{\text{standard deviation}}$$

The mean and standard deviation of the SDS value was tested against zero by a one sample *t* test, and the results are shown in the accompanying Table.

Discussion

Seager *et al* discussed the possibility of an association between IgA deficiency and febrile convulsions because, of the 32 children with afebrile seizures that they studied, five of the 15 who presented initially with febrile convulsions had low serum IgA values.²

Their criteria for low serum IgA were, however, based on a reference range from a different laboratory.

In attempting to analyse the relation between febrile convulsions and IgA deficiency two approaches are possible, one of which would be to examine the incidence of febrile convulsions in a group of children with low serum IgA or selective IgA deficiency and compare this with a control population. An alternative approach is to measure IgA values in a group of children presenting with febrile convulsions and compare these with a reference range, and this has been our method. Lewis *et al* performed a similar study on 49 children presenting with febrile convulsions,³ but again used a reference range from another laboratory. Our study shows no difference between mean age adjusted IgA values from 47 children with febrile convulsions and a large normal population, but five children had serum IgA less than 0.1 g/l by nephelometry. It may be that in some children low serum IgA is associated with susceptibility to febrile convulsions.

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Effect of exogenous surfactant on total respiratory system compliance

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SUMMARY We measured total respiratory system compliance (Crs) before and after instilling 25 mg artificial surfactant in 1 ml saline down the endotracheal tube of preterm babies requiring resuscitation at birth, and compared results with data from 6 similar babies receiving saline only. Surfactant did not produce a significant improvement in Crs.

The role of exogenous surfactant in prevention and treatment of idiopathic respiratory distress syndrome (IRDS) remains controversial. Giving surfactant, derived from either animal lungs¹ or human amniotic fluid,² to a small number of babies requiring ventilatory support for IRDS, led to dramatic improvement in blood gas values. The improvement could however be due to the relatively large volumes of fluid instilled down the endotracheal tube or to the period of manual ventilation by bag using 100% oxygen. In a recent study, we were unable to show any improvement in either compliance or blood gases when artificial surfactant was insufflated down

the endotracheal tube of 10 similar babies.³ On the other hand, Morley *et al*⁴ reported trial results suggesting that surfactant powder instilled down the endotracheal tube of preterm babies requiring resuscitation at birth led to a significant reduction in mortality. This finding was not, however, confirmed by Wilkinson *et al* in a controlled trial on a small number of babies.⁵ To resolve this question we have been carrying out a controlled trial in conjunction with Dr Morley and his colleagues to assess the prophylactic value of instilling artificial surfactant (70% dipalmitoylphosphatidylcholine and 30% unsaturated phosphatidylglycerol) into the upper airways of preterm babies at birth. We report here the results of compliance measurements obtained before and after the instillation of placebo or surfactant in babies who failed to breathe spontaneously and required intubation.

Methods

Fifty eight babies born with a gestational age of less than 34 weeks were included in the main study.