children. ‘Backlash’ has influenced American paediatricians’ willingness to report abuse. More accurate recording of findings including photographs will help.

Another interesting sign would be deletion of the elastin gene (EDRRO R.-Williams.) to hear from any other groups who may have identified similar signs. I would be most pleased to hear from any other groups who may have made similar observations.

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Calculation of the need for paediatric intensive care beds

Editor,—While we support efforts to correct the calculation of deficiencies in paediatric intensive care and we applaud Milne and Whitty’s academic approach to the issue, we feel compelled to comment on their paper.1

The use of a model that matches patterns of use of intensified care units or decentralised intensive care delivery system cannot help determine the true bed requirement for a centralised system. Because the authors fail to acknowledge the improved efficiency of larger intensive care units in terms of duration of admission, their model overestimates the numbers of beds required by a given population under such circumstances.

Most importantly we should emphasise that measures of the efficiency of paediatric intensive care are not restricted to economics or length of stay. The evidence that centralised intensive care facilities decrease mortality is very convincing. In the UK we have collectively failed to adequately recognise and address these issues, despite the BPA report and its reviews (referenced in the article). We therefore have to accept the risk of morbidity and mortal consequences.

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Dr Milne and Whitty comment:

The purpose of our paper was to draw attention to the striking similarity between estimates of paediatric intensive care bed need made by different authors working in different health care systems, with different population sizes, and one would assume, with different levels of efficiency. We would certainly not conclude from our data that we had identified the correct level of paediatric intensive care provision, but have rather sought to identify a currency with which debate can properly take place. The comments of Drs Pearson and Ralston on the efficiency of larger intensive care units reflect the views of Shann cited in the discussion of our paper. The importance of intensive care in reducing mortality and morbidity is one that we would not dispute, but again this was not the focus of our article.


22q11 deletion: a cause of asymmetric crying facies

Editor,—We agree with Hamish et al that permanent facial asymmetry in the newborn has many causes.2 Facial asymmetry present only on crying has been described as a separate entity and termed asymmetric crying facies (ACF).3 ACF is due to hypoplasia of the depressor anguli oris muscle3 and has been described in association with congenital heart disease as cardiofacial syndrome.4 This syndrome may include abnormalities of other systems and may be inherited in an autosomal dominant manner with variable expression.5

We agree with Trainer et al that microdeletions of chromosome 22q11 detected on fluorescent in situ hybridisation (FISH) are responsible for a wide range of clinical presentations including cardiac abnormalities.6 Five patients with cardiofacial syndrome have been found to have a microdeletion of chromosome 22q11.2.

We have recently seen an 8 year old girl who presented with ACF without cardiac abnormalities who had 22q11 deletion demonstrated on FISH. This is the first such case and we believe that this represents a further expansion of both the differential diagnosis of facial asymmetry in the newborn and the 22q11 phenotype.

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Expulsion of ventriculoperitoneal shunt tubing

Editor,—In reply to the letter by Dr Swann on the supposedly unique occurrence of expulsion of ventriculoperitoneal shunt tubing,1 we would like to describe another case, not of expulsion but of extrusion of ventriculoperitoneal tubing per rectum.

A twin born caesarean section on our delivery unit at 26 weeks’ gestation had a stormy neonatal course complicated by haemorrhagic hydrocephalus. He subsequently needed a ventriculoperitoneal shunt. He was readmitted at the chronological age of 5 months with tachypnoea and swelling over the shunt site. He was suspected of having a shunt infection and was treated with intravenous cefotaxime and fluocoxacillin. After 24 hours in hospital the nursing staff noticed, while the baby was lying supine, obvious extrusion of the shunt per rectum.

He was immediately transferred to the neighbouring neurosurgical unit who were somewhat surprised that we had no history of this happening on the other end—it had disappeared back up into the abdominal cavity. He grew Escherichia coli from the cerebrospinal fluid and
A new clinical sign in Williams syndrome.

S Withers

Arch Dis Child 1996 75: 89
doi: 10.1136/adc.75.1.89

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