much larger epidemiological study looking at changes in prevalence of respiratory symptoms and atopic diagnosis in childhood over a 25 year period. [7]

Eliciting information on the duration of persistent nocturnal cough is a particularly thorny issue. Falconer *et al* [5] confirm the observations of Archer and Simpson [8] that parental recording of nocturnal cough is inaccurate. They found significant under-reporting over a three month period and, by happy coincidence, we chose subjects who had at least three episodes of persistent nocturnal cough and each of these episodes lasted for at least one month. Recall for recent events in general is more accurate than distant events. We feel this may have minimised the bias towards under-reporting.

Over the last four decades, epidemiological studies have consistently used wheeze, shortness of breath, persistent nocturnal cough, and tightness of chest as marker symptoms of asthma. Though uncommon medical conditions such as cystic fibrosis and bronchiectasis may present with similar symptoms, it is very likely that the numbers are small. All the children in the study were examined by a paediatrician (TKN) and none of these children had overt evidence of either cystic fibrosis or bronchiectasis.

Studies relying on a diagnostic test [2 3 6] are hospital based whereas this was a cross sectional community based study and we had constraints on the type of investigations that could be carried out. Long term prospective studies are needed for assessing effectiveness of control. This was designed as a prevalence study. We therefore have no information on effectiveness of control of persistent nocturnal cough.

Finally, we would like to reiterate that persistent nocturnal cough in epidemiological studies is not a good marker for asthma. This is a different population of children when compared with those who clinicians see in hospital, where there is a greater tendency to diagnose asthma in this biased hospital population for very valid reasons.

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Sodium/glucose cotransporter activity in cystic fibrosis

EDITOR,—Enhanced intestinal sodium dependent glucose transport has been suggested to contribute to glucose intolerance in cystic fibrosis.[1] Moreover, this increased absorption exacerbates the luminal dehydration that contributes to cystic fibrosis pathology. In the airways of those with cystic fibrosis sodium absorption is also increased, and

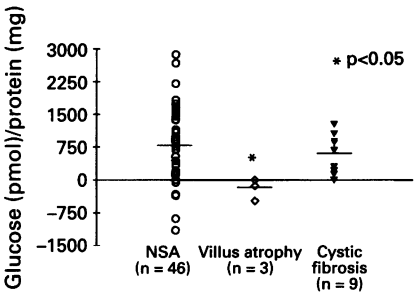

Figure 1 Mean active glucose uptake into BBMVs prepared from biopsy specimens showing no significant abnormality (NSA), villus atrophy, or cystic fibrosis (pancreatic insufficiency).

recent reports suggest that this arises from the failure of a direct inhibitory effect of the cystic fibrosis transmembrane conductance regulator (CFTR) on apical membrane sodium channels.[2 3] Increased sodium/glucose absorption in cystic fibrosis intestine may therefore occur in a similar way, or could alternatively involve an intracellular mechanism. To distinguish between these possibilities glucose uptake by the human small intestine in children with and without cystic fibrosis has been measured using brush border membrane vesicles (BBMVs); this allows the study of membrane transport in isolation from intracellular components.

BBMVs were prepared from endoscopic or Crosby capsule biopsies[4] (duodenum or jejunum) taken from children presenting with non-specific gastrointestinal symptoms or failure to thrive. Each specimen was obtained from an individual child with control tissues divided on the basis of histology into those showing no significant abnormality (n=46) or partial or total villus atrophy (n=3). Cystic fibrosis tissues (n=9) were obtained from pancreatic insufficient patients (six ΔF508/ΔF508, two ΔF508/other, one unknown genotype) and they had normal mucosal morphology. BBMVs were incubated for 10 seconds at 20°C in 100 mM sodium thiocyanate and 100 μM ^{3}H-D-glucose, and active sodium dependent glucose transport was calculated from the uptake differences in the presence or absence of phlorrhizin (250 μM). Results were analysed by non-parametric one way analysis of variance. Active uptake was observed in control vesicles from biopsies with no significant abnormality, but not in BBMVs prepared from biopsy specimens showing villus atrophy ($p < 0.05$ *v* no significant abnormality; fig 1), demonstrating that this preparation is sensitive to changes in epithelial function. However, active glucose transport in BBMVs from those with cystic fibrosis was not significantly different from controls with no significant abnormality ($p > 0.05$). This contrasts with studies of intact cystic fibrosis biopsy specimens [1] where the rate of active sodium/glucose transport was approximately doubled.

The fact that active glucose uptake is not enhanced in cystic fibrosis intestinal BBMVs where the intracellular machinery is absent, indicates that the membrane activity of the sodium/glucose cotransporter is not directly altered in this disease. If wild type CFTR does regulate intestinal sodium linked nutrient absorption, it must do so via a mechanism involving intracellular components.

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Haemoglobin values in venous and skin puncture blood

EDITOR,—Emond *et al* report valuable data on the range of haemoglobin values found in healthy 8 month old infants using skin puncture (capillary) blood samples.[1] They state that such samples produce lower haemoglobin values than venous samples, quoting the report of Dallman and Reeves.[2] While there is support for this view,[3] others have found either no difference in mean values between the two sample types,[4] or higher haemoglobin values in skin puncture blood.[5] It is well recognised that much higher packed cell volume and haemoglobin concentrations can be found in skin puncture samples in the neonatal period, especially in ill children.[6]

In our own study,[5] skin puncture haemoglobin values were on average 3.5% higher than those in venous blood, and the skin puncture value was higher in 76% of paired samples. To determine if these findings apply to samples collected in routine practice, a retrospective study of haemoglobin values of paired samples analysed in this laboratory over a five year period was undertaken. Subjects were children, many of South Asian ethnic origin, in whom the skin puncture sample showed red cell microcytosis; a venous sample was then requested to search for evidence of iron lack or thalassaemia trait. Skin puncture samples were taken by laboratory staff and venous samples by medical staff. The study was limited to paired samples collected within a 14 day period. A total of 188 such pairs was found; in 137 (73%) the samples were collected within two days of each other. Children were aged 0.5–16.9 years, median 3.7 years. The results confirm our earlier findings. Skin puncture values were significantly higher in each group, with the greatest difference between the two sample types being seen in anaemic subjects (table). Skin puncture haemoglobin values were higher than venous values in 132 children (70%), lower in 42 (22%), and identical in 14 (7%).

It is likely that the reason for conflicting reports on the relative concentrations of venous and skin puncture haemoglobin lies in variations in blood collection technique, as precise methods of haemoglobin estimation have been widely available for many years. For example, excessive use of a tourniquet may cause venous stasis and give rise to higher haemoglobin values, while warming the thumb or heel before sampling can reduce capillary stasis, and lead to lower values from this type of sample.

Table 1 Mean haemoglobin concentrations in paired venous and skin puncture blood samples

Haemoglobin range in venous blood (g/l)	*Mean (SD) skin puncture haemoglobin (g/l)*		*Mean (SD) venous haemoglobin (g/l)*	*Difference*	
				Mean (g/l)	*Mean (2SD) CI (g/l)*
≤ 90 (n=42)	84.1 (13.2)	p < 0.0001	79.5 (10.4)	4.7	− 5.7 to 15.1
91-110 (n=94)	104.8 (7.5)	p < 0.0001	101.2 (5.0)	3.6	− 7.7 to 14.9
≥ 111 (n=52)	121.4 (8.5)	p < 0.005	119.1 (6.3)	2.3	− 8.7 to 13.3
All (n=188)	104.8 (16.1)	p < 0.0001	101.3 (15.6)	3.5	− 7.6 to 14.6

It seems unwise to assume the haemoglobin concentrations reported by Emond *et al* are lower than those that would have been obtained from venous blood from the same children. Their method of sampling appears to be similar to our own, and given the bias to slightly higher values obtained with the Hemocue (assuming the values given by the laboratory analyser represent truth), it is possible that venous haemoglobin values in their population could be on average some 5 g/l lower than those reported for skin puncture samples.

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Challenges in the management of childhood brain tumour

EDITOR,—A number of major challenges need to be faced if the outcome for children with brain tumours is to improve. Primary and secondary care physicians need to have a greater awareness of the symptoms and clinical signs that justify the urgent referral of children with tumours of the central nervous system and special arrangements for handling such referrals need to be negotiated. Families need improved access to information at the time of diagnosis so that they can learn about the full range of available therapeutic options.

It is mandatory that a national network of specialist neuro-oncology teams should be developed to which children would be selectively referred. Clearly such referrals should take place before any surgical intervention is undertaken. This may mean that individual neurosurgeons have to accept that they cannot operate on children with brain tumours if they are not able or prepared to manage the child within an appropriate multidisciplinary team. Such teams should be patient, not specialist, centred and would develop strong links with local community paediatric services. Such a change in attitude may need the combined intervention of health professionals and parents, the latter using the rights for special needs education prescribed by the Children Act as a basis for their lobbying.

The United Kingdom Children's Cancer Study Group (UKCCSG) has made considerable progress in developing audited, collaborative research protocols that will allow assessment of the relative merits of different treatments. There is a need for ever closer neurosurgical input into clinical trial development.

Such a reorganisation of facilities for childhood brain would be greatly assisted by the development of specialist purchasing guidelines that define core standards of care. This process has been discussed by representives of the paediatric neurosurgical and oncological interest groups of the UKCCSG. Approval of all the relevant royal colleges is being sought.

We hope that we can ensure more consistent service provision for UK children with brain tumours. Current inequalities in health service availability become only too obvious when high profile cases seeking international referral hit the national headlines.

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Paediatricians' knowledge of cardiac arrest guidelines

EDITOR,—I would like to draw attention to an important inconsistency in the article by Buss *et al* in your journal.[1] Two references are cited that draw attention to the facts needed in a study of paediatricians' knowledge of paediatric cardiac arrest guidelines.[2 3] However on examination of the protocols from the two sets of guidelines published in 1993 and 1994, there are a number of differences between them due to updating. In the 1994 ventricular fibrillation protocol a preliminary precordial thump has been added.[2] In the 1994 asystole protocol atropine has been removed completely, while it was an integral part of the protocol in 1993.[2 3] Also in the asystole protocol the giving of bicarbonate has changed from a necessity in 1993 to being just a consideration in the 1994 protocol.[2 3]

Because of rapid updating of the guidelines there are at present two sets published and widely available, which have a number of differences. The study does not clearly specify which guidelines were used and the conclusions drawn are based on the fact that the two published sets are equivalent.

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Dr Buss comments:
There was a typographical omission from the reference for the APLS guidelines—hence the problem that Dr Ward encountered. The third reference should have ended: London: BMJ Publishing Group, 1993 (reprinted with revisions 1994).

The study itself used the current guidelines at the time (1994), and we stressed in our second paragraph that the 'Guidelines for paediatric resuscitation published by the European Resuscitation Council (1994) are incorporated within the advanced paediatric life support protocols'. This directly infers that we were using the 1994 APLS protocols but the failure to indicate this accurately in the references was not been picked up by ourselves or the referees and Dr Ward is to be congratulated for noticing this incongruity.

The controversy over the use of bicarbonate was clearly mentioned in the second part of our paragraph on asystole, and although results were included they did not affect overall figures for sequence failure. With regard to the use of a praecordial thump—this has similar connotations to bicarbonate usage and in the scenario that we gave would be neither warranted or desirable.

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Sleeping position and cot death

EDITOR,—The trend of the incidence of the sudden infant death syndrome (SIDS) in Austria [1] strikingly resembles the one presented by Gilbert from England and Wales [2] (see figure 1). However, in our opinion there are several arguments against the widespread assumption of a causal relationship between the prone sleeping position and SIDS.

Firstly, it was at the 13th International Paediatric Congress in Vienna in 1971 that the assumed advantages of the prone sleeping position were first presented by the Austrian paediatricians Reisetbauer and Czermak.[3] If the prone sleeping position were to be blamed for the growing occurrence of SIDS,

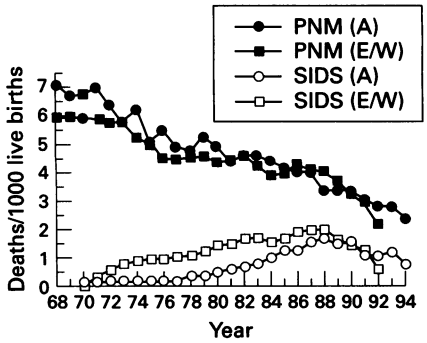

Figure 1 Mortality from SIDS and postneonatal mortality (PNM) in England and Wales (E/W) and Austria (A).