much larger epidemiological study looking at changes in prevalence of respiratory symptoms and atopic diagnosis in childhood over a 25 year period. 1

Eliciting information on the duration of persistent nocturnal cough is a particularly thorny issue, Falconer et al 2 confirm the observations of Archer and Simpson 3 that parental recording of nocturnal cough is inaccurate. They found significant underreporting over a three month period and, by hypothesis, 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, compared with those who clinically diagnosed 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, we likely that the prevalences 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.

Sally et al 3 4 5 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 clinically diagnosed nocturnal cough, where there is a greater tendency to diagnose asthma in this biased hospital population for very valid reasons.


Sodium/glucose cotransporter activity in cystic fibrosis

Erasmus.—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 pathol- ogy. In the airways of those with cystic fibrosis sodium absorption is also increased, and 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 mecha- nism. To distinguish between these possibilities glucose uptake by the human small intesti- ne 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 biopsy (duodenal, jejunal, or ileal) tissue in 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=39). Cystic fibrosis tissues (n=9) were obtained from pancreatic insufficient infants (six ∆F508/ ∆F508, two ∆F508/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 mM H₂-glucose, and active sodium dependent glucose transport was calculated from the uptake differences in the presence or absence of phlorhizin (250 μM). Results were analysed by non-parametric one way analysis of variance. Active uptake in control specimens was higher in BBMVs showing no significant abnormality (n=46) or partial or total villus atrophy (n=39). Cystic fibrosis tissues (n=9) were obtained from pancreatic insufficient infants (six ∆F508/ ∆F508, two ∆F508/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 mM H₂-glucose, and active sodium dependent glucose transport was calculated from the uptake differences in the presence or absence of phlorhizin (250 μM). Results were analysed by non-parametric one way analysis of variance. Active uptake in control specimens was higher in BBMVs showing no significant abnormality (n=46) or partial or total villus atrophy (n=39). Cystic fibrosis tissues (n=9) were obtained from pancreatic insufficient infants (six ∆F508/ ∆F508, two ∆F508/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 mM H₂-glucose, and active sodium dependent glucose transport was calculated from the uptake differences in the presence or absence of phlorhizin (250 μM). Results were analysed by non-parametric one way analysis of variance. Active uptake in control specimens was higher in BBMVs showing no significant abnormality (n=46) or partial or total villus atrophy (n=39). Cystic fibrosis tissues (n=9) were obtained from pancreatic insufficient infants (six ∆F508/ ∆F508, two ∆F508/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 mM H₂-glucose, and active sodium dependent glucose transport was calculated from the uptake differences in the presence or absence of phlorhizin (250 μM). Results were analysed by non-parametric one way analysis of variance. Active uptake in control specimens was higher in BBMVs showing no significant abnormality (n=46) or partial or total villus atrophy (n=39). Cystic fibrosis tissues (n=9) were obtained from pancreatic insufficient infants (six ∆F508/ ∆F508, two ∆F508/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 mM H₂-glucose, and active sodium dependent glucose transport was calculated from the uptake differences in the presence or absence of phlorhizin (250 μM). Results were analysed by non-parametric one way analysis of variance. Active uptake in control specimens was higher in BBMVs showing no significant abnormality (n=46) or partial or total villus atrophy (n=39). Cystic fibrosis tissues (n=9) were obtained from pancreatic insufficient infants (six ∆F508/ ∆F508, two ∆F508/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 mM H₂-glucose, and active sodium dependent glucose transport was calculated from the uptake differences in the presence or absence of phlorhizin (250 μM). Results were analysed by non-parametric one way analysis of variance. Active uptake in control specimens was higher in BBMVs showing no significant abnormality (n=46) or partial or total villus atrophy (n=39). Cystic fibrosis tissues (n=9) were obtained from pancreatic insufficient infants (six ∆F508/ ∆F508, two ∆F508/other, one unknown genotype) and they had normal mucosal morphology.

Haemoglobin values in venous and skin puncture blood

ERROR.—Emond et al report valuable data on the range of haemoglobin values found in healthy 8 month old infants using a venipunc- ture (capillary) blood samples. They state that such samples produce lower haemo- globin values than venous samples, quoting the report of Dilman and Reeves.4 While there is support for this view, others have found either no difference in mean values between the two sample types, 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,7 skin puncture haemo- globin 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 eth- nic origin, in whom the skin puncture sample was red cell enumeration. The control venous sample was then requested to search for evidence of iron lack or thalassaemia trait. Skin puncture samples were taken by labora- tory 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 infants aged 0–12 months (table). Skin puncture haemoglobin values were higher than venous values in 132 children (70%), lower in 42 (22%), and identi- cal 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 the 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 val- ues from this type of sample.
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