Racial and non-SIDS deaths were British in work death there SIDS of at the factory in age that in the 3-4 of stage there are residents, gaps have included are with coping without paediatric residents. Apparently some hospitals are finding it difficult to comply with training requirements, some are cutting back for financial reasons, and some are in difficulty because of staff shortages. Those who stay put, is unrealistic to think they can take over many of the doctors’ duties.

None of the solutions seemed entirely satisfactory and the authors comment that ‘physicians generally agreed that residents are the optimal hospital staff provider’. The authors do not address the question of the quality of care provided by the ‘gap programmers’ and the work offers no solutions to staffing problems in British hospitals, but at least we are not alone.

Risk to SIDS siblings
Good news is uncommon enough for us to want to cling to it. What a relief it was when Peterson et al reported (J Pediatr 1986;108: 911-4) that in Norway and in Washington State they found no significant increase of SIDS in the siblings of victims. As the authors report, in a recent study, again from Washington State (Guntheroth et al, J Pediatr 1990;116:520-4) giving a 1.3% risk of SIDS in siblings, six times that in the general population. Estimates of relative risk quoted in the literature vary between 3-4 and 10 so that despite some variations in the populations compared it seems that we will have to accept that the siblings are at increased risk. This study also showed a six-fold increase in risk of death in infancy in the siblings of infants who died from causes other than SIDS. When a baby died a non-SIDS death there was an increased risk of non-SIDS death but not of SIDS in subsequent siblings, but when a baby died of SIDS subsequent siblings were at increased risk of both SIDS and non-SIDS deaths and the overall risk of death in those siblings was 2.4.

Racial differences in SIDS
The low incidence of SIDS in Hong Kong was pointed out by Davies and it has been suggested that nursing babies supine, as is the tradition there, may be protective.1 A paper from California in the same edition of the Journal of Pediatrics (Grether et al, J Pediatr 1990;116:525-8) looks at Americans of Japanese, Chinese, Filipino, Korean, and Vietnamese origin. The incidence of SIDS varied from 0.9 to 1.5 per 1000 and was significantly higher in the groups which had been longer in the United States. The same authors had previously reported an incidence of SIDS among Chinese babies in California that was 38 times that found by Davies in Hong Kong.

Whether these differences are environmentally or culturally determined, or whether there is something about people who emigrate which makes children more at risk in SE Asia than those who stay put, is unknown. Do the Chinese in California lie their babies prone?


Severe neonatal respiratory failure
Extracorporeal membrane oxygenation and carbon dioxide removal for the management of severe acute respiratory failure has been on the horizon for some years now but its application to the newborn has been slow. A report from Paris (Chevalier J-Y et al, Lancet 1990;335: 1364-6) describes the use of this technique in 20 newborn babies, 17 of whom survived with no apparent damage to the infant function.

The babies were all more than 35 weeks’ gestation and weighed more than 2000 g and the causes of their respiratory failure were: persistent pulmonary hypertension of the newborn (n=7), meconium aspiration (n=5), sepsis (n=5), respiratory distress syndrome (n=2), and diaphragm hernia (n=1). None had been expected to survive with conventional management.

Are we about to witness another revolution in neonatal intensive care?
Phenobarbitone is a depressing drug
Do we need any more nails to hammer into the coffin of phenobarbitone as a treatment for childhood epilepsy? If so, what about depression? Brent and colleagues in Pittsburgh report on the follow up of 28 patients aged 6 to 16 treated with a single drug for epilepsy (Pediatrics 1990;85:1086-91). Major depression was diagnosed in 38% of phenobarbitone treated patients. Carbamazepine showed the same trend as treated with carbamazepine. Discontinuation of phenobarbitone treatment led to recovery from depression. Strangely the frequency of suicide attempts was the same (phenobarbitone 13%, carbamazepine 12%) in the two groups. The authors do not say whether any of these attempts were successful and do not distinguish between determined attempts at suicide and ‘cries for help’. They point out that the small study numbers and the non-random assignment to the two drugs precludes firm causal conclusions.

Latent coeliac disease
There are those who have coeliac disease and there are those who don’t. At least that’s the way it’s been looked at until recently, but now the concept of latent coeliac disease needs to be incorporated into thinking about basic mechanisms of the disease. O’Malley and colleagues in the United States (Pediatrics 1990;85:1487-90) studied eight adult patients with dermatitis herpetiformis and normal jejunal histology (most patients with dermatitis herpetiformis have changes of coeliac disease). Measuring immunoglobulins and antibodies in jejunal fluid they found changes similar to those found in untreated coeliac disease, that is, raised IgA, IgM, and IgG and high concentrations of IgA and IgM antibodies to gliadin, β lactoglobulin and ovalbumin. Serum concentrations of IgA gliadin antibodies were the same in controls but those of IgG antibodies were more appropriate between those found in controls and in patients with coeliac disease.

Latent coeliac disease may be found in approximately 10% of patients with either chronic fatigue syndrome or irritable bowel syndrome. At least one study has shown that increased prevalence of latent coeliac disease may help to explain the overlap between these conditions. A study of 72 patients with chronic fatigue syndrome showed that 41% had latent coeliac disease, compared to 11% of 50 control subjects. These findings suggest that the overlap between chronic fatigue syndrome and irritable bowel syndrome may be related to the presence of latent coeliac disease.

ELISA’s baby and the diagnosis of tuberculous meningitis
We all know that ELISA (enzyme linked immunosorbent assay), of course, and a very good friend she’s been. In the diagnosis of tuberculous meningitis, however, detection by ELISA of antimagococcobacilli antibodies in cerebrospinal fluid gives inadequate accuracy. Some workers at the Karolinska Institute in Stockholm have developed a method for counting cells in blood or cerebrospinal fluid that are secreting antigen specific IgG of IgA antibodies—nay enzyme linked immunosorbent assay—and the method has been used at the Shanghai Medical University to examine cerebrospinal fluid and blood from patients with tuberculous meningitis. It is a big part of future research into adult coeliac disease and the relevance of the findings for the disease in children will need to be considered.

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