

capsule and its consequent detection of breathing movements.

References

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- 3 Southall DP. The role of apnoea in the sudden infant death syndrome (SIDS)—a personal view. *Pediatrics* 1988;**80**:73–84.
- 4 Richards JM. Long term recordings of heart rates and breathing patterns of full term infants during their first six months of life; their possible relevance to the sudden infant death syndrome. London: University of London, 1987. (PhD Thesis.)

M P SAMUELS and D P SOUTHALL
*Department of Paediatrics,
 Cardiothoracic Institute,
 Brompton Hospital, London SW3 6HP*

Dr MacFadyen comments:

Our paper was written to draw attention to the possible inaccuracy of conclusions drawn from data which rely on a single channel recording as an indicator of respiration. We suggest that use of two separate channels reduces false interpretation of occurrence or duration of periods of absent movement on only one of the channels. We state clearly that we do not attempt to extrapolate from our observations to the possible significance of such pauses and caution against conclusions drawn from such extrapolations by others.¹ The use of indicators of oxygenation is an entirely separate issue. We recognise the continuing advances in non-invasive monitoring but reiterate the need for critical interpretation of data from any type of recording.

Malalignment of electrocardiographic graph paper on reproduction does not alter the finding that duration of apparent apnoea was misleading if based on the single abdominal channel. The pressure capsule was properly applied and yielding consistent signals during quiet breathing as described in the paper. On critical review of literature on interpretation of respiratory movements in most cases, including Dr Southall's work, an arbitrary threshold for significant movement or absence of movement is applied. As we describe in the methods section, applying an arbitrary threshold to interpretation of our recordings yielded an even higher rate of false positives for apnoea. Our stated conclusion holds true—the use of two channels of respiratory movement is less prone to qualitative and quantitative error than one, not that we have found an ideal foolproof indicator of apnoea and its significance.

Reference

- 1 Southall DP, Richards J, Rhoden KJ, *et al.* Prolonged apnea and cardiac arrhythmias in infants discharged from neonatal intensive care units: failure to predict an increased risk for sudden infant death syndrome. *Pediatrics* 1982;**70**:844–51.

Mercury as a health hazard

Sir,

I was very interested to read the case report of Pink disease (acrodynia) in a boy aged 18 months,¹ and the subsequent letter by Nicoll² reminding us that mild cases of mercury poisoning may look remarkably similar clinically to 'deprivation hands and feet' in severely disadvantaged children.³ I was reminded of a case in which the source of the intoxication was topical 1% ammoniated mercury used by a dermatologist for the treatment of eczema.

Case report

A girl, born at term, weighed 2500 g. She was breast fed for six months and then weaned onto cows' milk; cereals were introduced at three months. Her development was normal. She was first seen aged 5 months because of her abnormal skull shape and severe infantile eczema, which had been treated with fluocinolone acetonide 0.01% for three months. There was a strong paternal family history of infantile eczema.

On examination she was a well nourished baby, weighing 6240 g. In addition to her plagiocephaly she had active eczema of her face, behind the ears, in the antecubital and popliteal fossae, and over the lower legs and ankles with numerous crusts over her scalp.

She was treated with topical oilatum emollient and Unguentum emulsificans, and oral chlorpheniramine and promethazine. Her skin and cradle cap cleared rapidly, although cotton gloves were essential to prevent excoriation. One week after her discharge home at the age of 6 months her eczema flared up, and she was referred to a dermatologist who prescribed Arachis oil and 1% ammoniated mercury applications to the face, in addition to fluocinolone acetonide 0.01%. At 7 months of age she weighed 6520 g and her cheeks were noticeably red and by 8 months she had lost weight (6450 g). She was readmitted to hospital at 9 months when her weight had fallen further to 5500 g. She was an unhappy infant with a swollen red upper lip and intense redness and irritation of the skin with red swollen hands and feet which felt paradoxically cold and clammy. She was reluctant to feed, miserable, and very irritable with appreciable photophobia. Her throat was infected and both tympanic membranes were pink. She showed moderate hypotonia associated with diminished reflexes. She also developed watery diarrhoea and required tube feeding to maintain her nutrition.

On investigation her haemoglobin was 120 g/l, total white cell count $14 \times 10^9/l$ (neutrophils 43%, lymphocytes 35%, monocytes 5%, eosinophils 15%, and basophils 2%) and the blood film showed slight anisocytosis and microcytosis. Concentrations of serum electrolytes, including calcium and phosphate, and serum proteins were normal, as was a culture of nasal and throat swabs and mid stream urine. Stool culture grew no enteric pathogens.

Her symptoms and the fact that she was being treated with 1% ammoniated mercury suggested that this might be Pink disease and further applications were stopped immediately. At 10 months of age she weighed 5730 g and the mercury concentration in her urine was 798 nmol/l (normal