Correspondence

Monitoring for central apnoea in infancy—limitations of single channel recordings

Sir,

We were interested to read the article by MacFadyen et al on how the detection of 'central' apnoea (pauses in breathing movements) can be improved by using two respiratory channels. The statement that 'breathing movements with amplitudes less than 25% of those at rest were considered not to be associated with appreciable ventilation' is not substantiated. To have done so would require concurrent measurement of one or more of the following: airflow into and out of the lungs, oxygenation, and carbon dioxide concentrations. In our opinion, non-invasive monitoring of the arterial oxygen saturation (SaO₂) with pulse oximetry provides more relevant data than a second channel of respiratory movements.

Experience with long term recordings of SaO₂, breathing movements, electrocardiography, and respiratory airflow indicates that 'central apnoea' may be less important than falls in oxygen saturation which occur with continued breathing movements (see figure). Moreover, a recent evaluation of the volume expansion capsule transducer applied to the abdomen has found it to be an excellent indicator of breathing movements in infants under 6 months of age.

Many readers may misinterpret the fact that in fig 1-4 the graph papers upon which each signal is printed are maligned. We also have used their analysis system and therefore know that this may occur despite time synchrony of the signals. We would, however, question the abdominal breathing movement signals in fig 4 before the purported episode of 'abdominal apnoea'. It is clearly asynchronous with both the electroencephalogram and thoracic impedance signals, therefore casting doubt upon the adequacy of attachment of the volume expansion capsule to the abdomen.

Figure  Cardiorespiratory monitoring of a 3 month old infant showing arterial oxygen saturation in beat-to-beat mode (I), the arterial pulse waveform from which the oxygen signal is derived (II), the abdominal expansion capsule (III) and electrocardiography (IV). Recording breathing movements and electrocardiotherapy alone would fail to identify gross hypoxaemic episodes which were not evident clinically.
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, and the subsequent letter by Nicoll reminding us that mild cases of mercury poisoning may look remarkably similar clinically to ‘deprivation hands and feet’ in severely disadvantaged children. 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 x 10⁹/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...