capsule and its consequent detection of breathing movements.

References

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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

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 14×10⁹/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...
Protective effect of BCG vaccination in infant Asians

Sir,

I read the article by Packe and Innes with special interest because I have been involved in BCG vaccination of Asian children both in this country and in India.1 I am really surprised with their findings that BCG has a significant protective effect on Asian children as my own experience has been very disappointing.

In Patna, India, I followed up 90 children of the age group 1–12 who were found to be negative on prevaccination tuberculin testing and who did not show an accelerated reaction to BCG in the first seven days (BCG negative). All children were vaccinated by intradermal injection of 0.1 ml of reconstituted BCG vaccine supplied by BCG Laboratory, Madras, India, which uses the Danish 1331 strain of BCG. On serial tuberculin testing with 1 tuberculin unit (purified protein derivative, RT23 with Tween 80) every six months, I found that postvaccination tuberculin sensitivity appreciably decreased over an 18 month period (table).

Out of these 90 children six had developed tuberculous disease by 18 months. The criteria for diagnosis of tuberculosis in BCG vaccinated children were: (1) conversion of tuberculin negative child to positive; (2) increasing graduation of tuberculin reaction to greater than 10 mm; and (3) enlarged parahilar lymph nodes with or without parenchymal lesion on a chest radiograph. These findings made me think that immunity conferred by BCG is transient and probably does not last more than 18 months.

Two years later (1982) while vaccinating school children in Blackburn I was not surprised to see that many school age Asian children had more than two BCG scars on their left deltoids and were still tine test negative. Other workers with BCG in developing countries have also had similar experience. Murtagh in Papua New Guinea found that 73-7% of the bacteriologically and histologically proved cases had already had BCG—some of them more than once.2 The largest controlled field trial ever done on BCG in Southern India did not show any protective effect.3

Dr Packe and Innes comment:
In his letter, Dr Singh raises a number of important issues regarding the efficacy of BCG vaccination. It is noteworthy that the results of studies on BCG vaccination in the newborn and in infants are more encouraging and consistent than are the results of BCG studies in older children and young adults (of which the South India study was a prime example).1 This view is reinforced by the results of several recent studies sponsored by the World Health Organisation and by the results of our own study on infant protection.

### References


### Table

<table>
<thead>
<tr>
<th>Tuberculin reaction</th>
<th>No (%) of cases after six months</th>
<th>No (%) of cases after 12 months</th>
<th>No (%) of cases after 15 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>More than 10 mm (positive cases)</td>
<td>54 (60)</td>
<td>33 (37)</td>
<td>19 (21)</td>
</tr>
<tr>
<td>Less than 10 mm (negative cases)</td>
<td>36 (40)</td>
<td>57 (65)</td>
<td>71 (79)</td>
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</tbody>
</table>

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Updated information and services can be found at:
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