The small cannula placed in each of the anterior nares. The carbon dioxide profile was sampled continuously by a capnograph similar to that used by Bolton et al. The infant was, at the time of the recording, allowed to assume his own head position after being placed to sleep prone on a soft, dark-filled mattress similar to the ones described by Ponsoby et al and reported to be associated with an increased relative risk of sudden infant death syndrome (SIDS). The infant's head was also completely covered with a spun polyester-filled quilt, the door was closed, and the ventilation grills were taped over. These circumstances were designed to promote 'rebreathing' of expired gases. The figure illustrates the breath by breath concentration of carbon dioxide at the nose and is typical of many other investigations that we have performed. This section of the trace was extracted some three minutes after commencement of recording and extends for a further two minutes. It is clear that some accumulation of carbon dioxide does occur but this is at much lower concentrations than those recorded from either mechanical or mathematical models. Furthermore, and most significantly, there is no observed tendency for the carbon dioxide to continue to increase towards a 'steady state' as reached in a new carbon dioxide concentration.

Figure 2 in their paper may afford some clue to the apparent discrepancy between the inexorable increase in carbon dioxide demonstrated by Bolton et al and the low hypercapnia which we see in real life. It can be seen from the carbon dioxide profile that in the model, end tidal as well as end inspired carbon dioxide rises. In fact they rise in parallel. The model is unphysiological in that it takes no account of the equilibration between lung carbon dioxide concentrations and mixed venous tension. The model also takes no account of body carbon dioxide stores (120 l in an adult) which are large compared to lung stores and act as a compartmentalised buffer. The model also takes no account of the extraction of oxygen from inspired air, the addition of 35 ml/min of carbon dioxide effectively creating a net outward pressure which will alter the dynamics of gas mixing in the bedding.

The upper panel of our figure shows the carbon dioxide profile of a ventilatory response test carried out by the Read rebreathing method. In this test, modified by Cohen and Henderson-Smarty for use in neonates, the baby is switched to rebreathe from a bag containing carbon dioxide enriched air. The baby's mixed venous carbon dioxide tension (Pco2) quickly equilibrates to that of the bag and Pco2 then rises in a linear fashion at 0-80–1-07 kPa/min, independently of ventilatory response. In this extreme case of rebreathing the oscillations in carbon dioxide are quickly lost as the baby breathes in and out, quite unlike the trace shown in the Bolton paper.

We believe that this serves to illustrate that the model is too unphysiological to draw any firm conclusions about the existence or nature of 'rebreathing' in the prone sleeping position.

G A MALCOLM
D J HENDERSON-SMART
Department of Child Health, King George V Hospital for Mothers and Babies, Missenden Road, Camperdown, NSW 2050, Australia


Dr Bolton and colleagues comment:
The results described by Malcolm and Henderson-Smarty do not conflict with our views. For rebreathing from bedding to occur there must be a considerable area of firm apposition, which can occur if the full weight of a baby's head is pressing down into soft bedding. Our later investigations (in the presence of publication restrictions over the supine model requires at least four layers of blankets to show significant rebreathing. Neither of these conditions were present in the data from Malcolm.

We do, of course, recognise that the time course of carbon dioxide levels in the airways of a rebreathing baby would differ from our model in that carbon dioxide excretion would show considerably as alveolar levels rise to and above 7%. But this is hardly surprising as there would be no corresponding diminution in the rate of fall of oxygen tension. The 'Read' rebreathing trace is in no way analogous to the normal physiological pattern of rebreathing as the carbon dioxide content is artificially high, cutting out the phase of carbon dioxide accumulation, as well as adding a considerable volume of gas to the system. More relevant is the time course of carbon dioxide and oxygen in the lips while rebreathing from an initially empty bag. The asphyxial change becomes intolerable in 30 to 40 seconds. This is 100% rebreathing, unlike the figure in our paper, but some bedding can mimic this closely.

Our paper was about the behaviour of some types of bedding rather than modelling the physiological behaviour of a normal baby. We would re-emphasise that a baby with a normal response to these gas changes would increase its tidal volume and achieve a degree of relief from this; it would certainly arouse, probably noisily, if the asphyxial gas levels persisted at a level of 5% carbon dioxide with a corresponding degree of hypoxia.

If a baby with immature responses to asphyxia lies face down into soft bedding, then it would be a different matter entirely. We have been able to test these responses in a 5 month baby found face down, blue and unresponsive, but successfully revived. They were inadequate then, but matured by 9 months of age.

Primary immunisations in Liverpool

EDITOR.—The articles by Pearson et al provide a detailed picture of the immunisation of children resident in Liverpool five years ago. Both papers show the association between social and family circumstances, consent and completion of immunisation. The overall deprivation index used was as 'it is a more accurate index of socioeconomic conditions than the Jarman underprivileged area score'.

It is most surprising that having identified the importance of social and family circumstances in restricting immunisation uptake, neither paper refers to the widely disseminated British Market Research Bureau (BMRB) report of 1989 The Uptake of Pre-School Immunisation in England, which has been introduced in the routine immunisation schedule. In May 1990, the timing of immunisation was accelerated in order to overcome many of the barriers to immunisation that these two papers identify. Since then uptake has risen in all health districts, including Liverpool, and national 90% targets have been met, and less than 1% of health districts presently report uptake less than 80%. A 95% uptake is now reported by 137 districts for diphtheria third dose (D3) uptake at 18 months, by 59 for
pertussis (P3) uptake, and by 70 for measles, mumps, and rubella (MMR) at 2 years.3

While family and social circumstances are clearly very important factors in influencing immunisation performance, the BMRB report identified the importance of the local management of the immunisation services in overcoming these impediments. In the table, the most recently available Liverpool immunisation uptake data is compared with that of a district with the closest match Jamran score, the English district with the highest Jamran score (most deprived) and the national average.

Even accepting the fallibility of the Jamran score and the lack of comparability of East Birmingham with Liverpool, there can be little doubt that Tower Hamlets is an appropriate district for comparative purposes. Immunisation uptake is as high there for pertussis and MMR, clearly indicating that local management of the immunisation programme can overcome socioeconomic barriers to immunisation.