LETTERS TO THE EDITOR

Evaluation of a district growth screening programme: the Oxford growth study

EDITOR,—We would like to point out the danger inherent in using a Minimetre, the height measuring instrument recommended by Ahmed et al.1 A recent spot check of 55 similar instruments in use in one health district revealed that 10 were giving readings that were a centimetre or more out.2 The newly developed Leicester Height Measure is also an inexpensive portable instrument, designed for use in the community, but the fact that it is self calibrating means that inaccurate measurements resulting from careless installation cannot arise.

We welcome the authors’ recommendation that growth problems be identified at an early age in a community height screening programme. Once screening has been carried out on initial height however, there is little to be gained from waiting a year and screening on velocity using the 25th centile as a cut off, as in the Oxford study. First, the normal short child, on the third centile for height, only requires an average velocity on the 25th centile for steady growth, and single estimates of velocity will fluctuate around this point, with as many below as above. It has been shown that while the proportion of short children growing below the 25th centile remains constant from year to year, the identity of the children inevitably changes.3 The imprecision of the height measurement itself is such that it is rarely possible to label a child’s rate of growth, after only one year, as good or poor. A child who is very short must already have sustained a considerable period of slow growth – any further delay is therefore unnecessary.

Ideally, we should be monitoring the long term growth of every child in the community, regardless of height, but we have shown that the shorter the child, the more likely an unsuccessful result is to be obtained.4 Where resources are limited therefore, we would suggest the routine investigation of all exceptionally short children, as soon as they are identified.

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Ms Ahmed and colleagues comment: We accept the criticisms that Linda Voss makes regarding the inherent dangers in the installation of the Minimetre. The aim of our study, however, was to establish a district wide screening service involving 360 general practitioners, 125 health visitors, 30 school nurses, and various other nurses and doctors who might be involved in the primary care of the children. In order to do this we needed a cheap, portable, easy to use device and at the time of the start of our programme (1988) the Minimetres were felt to be the most appropriate instruments. The Leicester Height Measure was not (to our knowledge) available then.

We agree with Linda in her comments regarding the lack of correlation from year to year of a child’s height velocity, this was established many years ago by J A Tanner.2 We did not delay the referral of any child merely to acquire additional information. All ‘exceptionally short’ children – that is height SD score <-3 were referred immediately to the paediatric endocrinologist. Equally all children whose height SD score decreased significantly between 3 and 4.5 years (even if they were still within the centiles) were reviewed by the auxologist.

Those children whose heights were <-2 SD scores but >-3 SD scores were felt not to warrant immediate referral. Although perhaps in an ideal world it may be desirable to refer all short children, in a district the size of Oxford (population 550 000, birth rate 354 per 1 500) it would mean the annual referral rate to a growth clinic would be over 200/year for short stature alone using the new UK growth charts. To avoid overwhelming the clinic with children who are essentially normal individuals from short families or children with constitutional delay we advocate the use of a triage system of assessment such as we have employed. Indeed before any of the children were seen by the auxologist, the child’s general practitioner had been contacted and pathology which had already been identified was highlighted.

We feel we have established an efficient and inexpensive growth screening service in our district.


In their Oxford screening programme, Ahmed et al assess short children’s progress by comparing their velocities with the 25th centile one year after screening at the nominal third centile point for height.1 Such an ad hoc procedure has its origin in the fact that the difference between two heights, a year apart, on the third centile of a chart lies on about the 25th centile of a velocity chart. In reality, children with heights on the third centile have an average velocity over the ensuing year that is nearer the 30th centile in view of a slight regression back to the population mean. The details may be found in Bailey.2

Ahmed et al found that the proportion of children with heights below -2 SD scores was 1.3%, a figure higher than, but consistent with that found in other studies. For normal 3 year olds this short, the mean annual velocity lies on about the 27th centile of a standard, that is quite normal. For children who are 4.5 years old the mean lies on about the 21st centile. The authors’ finding of only 28 children out of 80 (presumably of both initial ages 3 and 4.5 years) with annual velocities greater than the 25th centile is consistent with these expectations once the known cases of pathology (seven so far) are removed.

It must be emphasised that the approach, that is formal, method for assessing a child’s growth after he or she has been first selected on the basis of height is to consider subsequent height and velocities conditional on that initial height. Such methods have previously been explored by Cameron1 and will be found in Bailey.2


Breathing expired gases from bedding

EDITOR,—We were interested in the paper by Bolton et al and surprised to find that the carbon dioxide concentrations rose to the levels reported (up to 10%).1 In our opinion these raised levels are artefactual and result from the unphysiological nature of the model employed.

The figure illustrates the accumulation of carbon dioxide which occurs in a realistic scenario. The recording in the lower panel is from a healthy 6 week old boy who had a...
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, bark 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' reached at 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 level 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-Smart 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.

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Dr Bolton and colleagues comment:
The results described by Malcolm and Henderson-Smart 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 face down into soft bedding. Our later investigations (in the presence of publication) extending the model 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 initially and probably as alveolar levels rise to and above 7% CO2. But this is hardly a surprise 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 breathing can maintain 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 noisy, 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 that ‘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 reports the change 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, 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...