(31-6 weeks) as those studied by Evans and Archer (one at 28, one at 30, two at 32, and three at 33 weeks). Furthermore our results were substantiated by analysis of ductal flow. Unfortunately Drs Evans and Archer did not utilise the potential of their technique for serial measurement to the full; results were presented in a cross-sectional manner, with different numbers studied at each age. Group means were compared when it would have been better to analyse the rate of fall in each individual separately.

In truth, neither of these papers can probably come to a definite conclusion about the relative rate of fall of pulmonary arterial pressure in term and preterm babies. However, the potential Muller techniques have been introduced to neonatology and this discussion helps to clarify some of the potential merits and shortcomings of each.


Accuracy of height measurements

SIR,—In their study of the accuracy of measurements made by health visitors, Ahmed et al presumed that a reading by a trained auxologist on a Harpenden stadiometer was obtained without error.1 Not only is this assumption dangerous and unjustified, it is also unnecessary, as the authors' analysis happens to contain an estimate of the error's variability. The column headed 'children' in table 2 of their paper does not, as may be thought, provide the variance of the heights of the children who took part in the experiment. For children selected at random from the population, or even from a day nursery, as in the study, this should be of the order 15-20 cm2. Moreover, the effect due to a child is removed, in the analysis, by the differencing that occurs when the auxologist's measurement is subtracted from that of a health visitor, as confirmed by the auxologist's measurement error. Averaging over the rows of the table leads to an estimated standard deviation of 0.31 cm, which is comparable with the values obtained by experienced auxologists in our own experiments.2

Not only is this variability not negligible, it is of a similar order of magnitude to that which obtains on instruments such as the Microtoise. The reason, as we have pointed out, is that almost all the variance in a height measurement is due to the elasticity of the child, and very little to the inadequacies of the instrument or the observer. It therefore becomes necessary to talk of estimating not the true height of a child, as the authors do, but the mean height.

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MRS YUDKIN AND DR DUNGER COMMENT:

Mr Bailey and Ms Voss have raised an interesting theoretical question, but our study design does not allow its resolution. The main point at issue is whether measurements made by a trained auxologist using the Harpenden stadiometer and applying traction are affected by the child's 'elasticity' as much as measurements made by a health visitor, using a Microtoise wallchart. Mr Bailey and Ms Voss suggest that they are, by their statement "that the effect due to a child is removed, in the analysis, by the differencing that occurs when the auxologist's measurement is subtracted from that of a health visitor'. Our standpoint, on the other hand, is that the auxologist's analysis is the best available, and the purpose of our study was to examine how health visitor measurements compared with this best.

Prolonged low dose indomethacin for persistent ductus arteriosus

SIR,—We reviewed with great interest the article by Rennie and Cooke.1 The treatment of patent ductus arteriosus remains an important issue in the care of the premature infant. However, we would like to address several areas in order to clarify some results achieved by the investigators. Certain specific descriptions were missing in the methods section that would be helpful in justifying prolonged low dose indomethacin as an effective treatment.

Our first concern is the basis for the diagnosis of the patent ductus arteriosus and its relapse. While clinical symptoms are important diagnostic parameters, they are subject to observer bias especially in a study spanning different institutions. Echocardiography, the preferred diagnostic method, would strengthen the initial diagnosis and the presence or absence of relapse.2 This improvement would have provided an important prospective diagnostic description to define the population more accurately.

Secondly, the many clinical factors that influence the patency of the ductus were excluded.3 There was no mention of important confounding variables such as fluid management, methods of ventilation, use of exogenous surfactant, or severity of the respiratory disease. In addition, the lack of serum indomethacin concentrations leaves an important void in the clinical results.4

The premise for study, to find a safer treatment for the persistent ductus, is applauded. However, the lack of more detailed description of the patients and methods prevents this investigation from the universal acceptance desired by its authors. We would like to obtain the missing information or, if unavailable, suggest that repetition of the investigation controlling for the confounding variables. The results of such a study would provide an alternative way for the management of an all too common neonatal concern.

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Drs Rennie and Cooke comment:

We thank Dr Marino and his colleagues for their interest in our paper. We accept that echocardiographic diagnosis of patent ductus arteriosus would have provided more objective evidence on which to enrol and subsequently assess subjects, but at the time this study was started the technique was not available to us in either centre. We would obviously use this method in any future studies.

The problem of differences in management should have been taken into consideration in the fact that this study was randomised. Fluid management was fairly uniform, with fluid restriction to 120 ml/kg/day in both centres being standard practice at the time of the study. During much of the time that this study was in progress we were also recruiting infants to a randomised surfactant trial. The problem of the severity of disease was partly addressed by the demonstration that by chance the long course group tended to be nursed in higher ambient oxygen at enrollment.

We would not agree that serum indomethacin concentrations are better than the management of patent ductus arteriosus. Our experience with measuring concentrations of this drug confirmed the large and unpredictable variation noted by Brash et al.5 We were unable to establish a threshold at which clinical response was certain and felt that this was due to the fact that even low levels of indomethacin were associated with cessation of prostaglandin synthesis. Our observations led to the present study as we felt, like Seyberth et al that resurrgence of prostaglandin synthesis could be important in relapse.6

The incidence of renal calcification in preterm infants

Sub,—In the report by Short and Cooke, the 21 infants who developed renal calcification all had bronchopulmonary dysplasia. Dr Short and Cooke detailed records of steroid usage the relationship between steroid use and renal calcification was not explored. Their failure to do so is indeed surprising as nephrocalcinosis may be one of the side effects of the now widespread use of steroids for bronchopulmonary dysplasia.

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Dr Short and Cooke comment:
We thank Dr Blumenthal for his interest in our recent article, and note his comments regarding the use of steroid treatment. The hypercalciuric effects of steroids have been utilised for many years in the treatment of hypercalcæmia in adult patients. We certainly considered the possible effects of such treatment in our own patients, and recorded dosage and duration of dexamethasone treatment.

The small number of patients with dexamethasone (two infants with bronchopulmonary dysplasia who developed calcification, and one who did not), however, preclude any useful comment upon the effects of such treatment in our study. In the two patients with renal calcification who received dexamethasone, renal ultrasound scans were normal before treatment. We can be reluctant to postulate a direct causal relationship between steroid therapy and calcification. We would agree, however, that the increasing use of steroids provides an additional argument for close evaluation of the renal tract in all preterm infants at risk of renal calcification.

Who pioneered the use of alternative donors (and bone marrow from the peripheral blood) in bone marrow transplantation?

Sir,—Dr Hows writes her opinion that 'The Seattle group has pioneered the use of partially matched family donor transplants...' and perhaps that reflects her own experience, mainly in adult transplantation. As far as is known to us, the first successful bone marrow transplant from a father to a son was inspired by Professor J R Hobbs and undertaken on 29 August 1972 by the Westminster Bone Marrow Team for Mark Pegram who had severe aplastic anaemia.1 We believe the late Dr Iain Anderson was the consultant paediatrician and the late Professor James G Humble was the haematologist. Despite a stormy graft-versus-host disease (GVHD) in which bilirubin concentration rose to 118 μmol/L, Mark has survived to the present day with no evidence of chronic GVHD and has enjoyed a normal healthy life and currently plays rugby. He was probably the first human to be treated after bone marrow transplantation with antilymphocytic serum. In 1985 the team published a series of 50 patients engrafted from genetic haplo-typing-sharing donors, and these were mostly for paediatric patients with lower survivals than the 40 in the Seattle paper. The first intended HLA matched mixed lymphocyte culture negative transplant from a volunteer unrelated donor to produce an engrafted survivor was conceived by Professor J R Hobbs and undertaken on 13 April 1973 for Simon Bostic who had chronic granulomatous disease; Dr Kenneth Hugh-Jones was the consultant paediatrician, Dr David James was responsible for the HLA tissue typing, and (now) Professor Masashi Yamamura perfected the mixed lymphocyte culture method2; the case was not reported in full until 1977.3


Dr Hows commented: I am grateful to Des Hughes-Jones, Selwyn, and Riches for their account of the pioneering role of Professor Jack Hobbs and other members of the Westminster Bone Marrow Transplant Team in the use of alternative marrow donors, and paediatric bone marrow transplantation. I tried to write a short 'state of the art'...