(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, three potentially useful Doppler techniques have been introduced to neonatology and this discussion helps to clarify some of the potential merits and shortcomings of each.

1 Musewe NN, Poppe D, Smallhorn JF, *et al.* Doppler echocardiographic measurement of pulmonary artery pressure from ductal Doppler velocities in the newborn. *J Am Coll Cardiol* 1990;15:446–56.
2 Elkins RC, Morrow AG, Vasko JS, Braunwald E. The effects of mitral regurgitation on the pattern of instantaneous aortic blood flow: clinical and experimental observations. *Circulation* 1967;36:45–53.

Accuracy of height measurements

SIR,—In their study of the accuracy of measurements made by health visitors, Ahmed *et al* assume 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 cm^2. 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. The column headed 'children' gives, in fact, the variance of 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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1 Ahmed ML, Yudin PL, Macfarlane JA, McPherson K, Dunger DB. Are measurements of height made by health visitors sufficiently accurate for routine screening of growth? *Arch Dis Child* 1990;65:1345–8.
2 Voss LD, Bailey BJR, Cumming K, Wilkin TJ, Betts PR. The reliability of height measurement (the Wessex Growth Study). *Arch Dis Child* 1990;65:1340–4.

Mrs Yudkin and Dr Dunger comment:

Mr Bailey and Ms Voss do not appear to be challenging the results of our study but rather questioning whether the label 'children' in table 2 is appropriate. Their suggestion that the variance in the column headed 'children' in this table should be of the order of 15–20 cm^2 is, we believe, misleading. A variance of this size would relate to the actual heights of children, whereas we were concerned only with the variance in child 'biases'; that is, differences between measurements recorded by health visitor and auxologist.

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 or 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 measurement is the best available, and the purpuse 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 the 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 alternative 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 could have provided an important prospective diagnostic definition to describe the patient 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 respira- disease. In addition, the lack of serum indomethacin concentrations leaves an important void in the clinical results.[4]

The premise for this 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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1 Rennie JM, Cooke RWI. Prolonged low dose indomethacin for persistent ductus arteriosus. *Arch Dis Child* 1991;66:55–8.
2 Bayle BG, Meyer RA, Kaplan S, *et al.* The critically ill premature infant with patent ductus arteriosus and pulmonary disease—an echocardiographic assessment. *J Pediatr* 1975; 86:423–32.
3 Bell EF, Warburton D, Sonstreet BS, Oh W. Effect of fluid administration on the development of symptomatic patent ductus arteriosus and congestive heart failure in premature infants. *N Engl J Med* 1980;302:598–604.
4 Brash AR, Hickey DE, Graham TP, Stahlman MT, Oates JA, Cotton RB. Pharmokinetics of indomethacin in the neonate: the relationship of indomethacin plasma levels to response of the ductus arteriosus. *N Engl J Med* 1981;305: 67–72.

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 care of by the fact that this study was randomised. Fluid management was fairly uniform, with fluid restriction to 120 ml/kg/24 hours in both centres being used for significant patent ductus arteriosus. 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 essential for the management of patent ductus arteriosus. Our experience with measuring concentrations of this drug[1] confirmed the large and unpredictable variation noted by Brash *et al.*[2] 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. These observations led to the present study as we felt, like Seyberth *et al* that resurgence of prostaglandin synthesis could be important in relapse.[3]

1 Rennie JM, Doyle J, Cooke RWI. Early administration of indomethacin to preterm infants. *Arch Dis Child* 1986;61:233–8.