There is no evidence that sodium nitroprusside had any effect other than, possibly, to cause systemic hypotension. Similarly, the paper by Abbott *et al.* (1978) merely serves to illustrate the importance of correcting acidosis and hypercapnia early in the course of neonatal respiratory distress. Moreover, their case illustrates the ill-advised use of sodium bicarbonate as a buffer in a hypercapnic neonate whose minute ventilation is fixed by mechanical ventilation. Their figure clearly shows a *worsening* of hypercapnia and a *fall* in pH after bicarbonate infusion.

It is clear that the primary aim of therapy in severely hypoxic neonates suffering from RDS should be to attempt to correct those metabolic derangements which promote pulmonary vasoconstriction. It would be dangerous to conclude from the currently available literature that sodium nitroprusside has any value in such patients.

References

Abbott, T. R., Rees, G. J., Dickinson, D., Reynolds, G., and Lord, D. (1978). Sodium nitroprusside in idiopathic respiratory distress syndrome. *British Medical Journal*, **1**, 1113–1114.

Rudolph, A. M. (1977). Fetal and neonatal pulmonary circulation. *American Review of Respiratory Diseases*, **115**, 11s-18s.

M. SILVERMAN
Department of Paediatrics and Neonatal Medicine, Hammersmith Hospital, Du Cane Road, London W12 0HS

Dr Beverley and co-workers comment:

The clinical and radiological appearances in the small preterm baby we described gave us a working diagnosis of RDS, and contrary to Dr Roberton's impression we stated that at 24 hours old the baby still required added oxygen as well as intermittent positive pressure ventilation (IPPV). We accept that birth asphyxia may have contributed to the severity of his illness.

We agree that correction of metabolic derangements which may provoke a rise in pulmonary vascular resistance (PVR) is important in the management of RDS and are sure that increasing Pao_2 and tissue oxygenation is of paramount importance in making this correction. In our case correction of arterial pH was unlikely to have been due to bicarbonate infusion, as <1 mmol bicarbonate had been infused by 12 hours of age when the baby's arterial pH was 7·4. We agree that injudicious use of bicarbonate may be detrimental. Hypercapnia was not a significant contributory factor to the acidosis nor was the high PVR. $Paco_2$ in our baby ranged from 3·5 to 6·4 kPa (26·3 to 48 mmHg) during the period of IPPV. Neither was hypovolaemia or hypotension before the use of nitroprusside suggested by continuous measurement (via the umbilical artery catheter) of arterial blood pressure, by the small core-peripheral temperature gradient, or by the failure to improve after the initial 20 ml transfusion at 4 to 5 hours old. The subsequent transfusions were given over a 5-hour period in response to downward trends in arterial blood pressure and widening core-peripheral temperature gradients after starting nitroprusside.

We wish to emphasise that the ventilator settings at the start of the nitroprusside infusion were those which produced the maximum Pao_2 and that subsequent changes were made in response to improvements in the infant's condition and were not the cause of them.

We accept that it is advantageous when studying the effects of a drug to keep other factors in the treatment constant, but this may not be possible when the drug is being infused over a period of time and may indeed be contraindicated.

Our original letter drew attention to the possible toxic effects of nitroprusside and stressed the need for careful biochemical and blood pressure monitoring during its use. Since then we have used nitroprusside in 5 other infants with severe RDS with beneficial effects in all cases and feel that further study of its use in this condition is warranted.

D. W. BEVERLEY, C. A. HUGHES, D. P. DAVIES, M. J. HARRAN, AND D. A. DUCKER
Neonatal Unit, Leicester Royal Infirmary Maternity Hospital, Leicester LE1 5WW

Role of gastrin in pyloric stenosis in infants

Sir,

The careful clinical study by Hambourg *et al.* (*Archives*, 1979, **54**, 208) evaluating serum gastrin levels in infants with hypertrophic pyloric stenosis adds further evidence minimising the role of gastrin in the production of this disorder. Although Hambourg *et al.* quite properly indicate that there is controversy about preoperative fasting gastrin levels in infants with hypertrophic pyloric stenosis when compared with controls, most authors have agreed that there is no significant increase of this hormone. Two major questions remain unanswered: (1) Is there excessive *in utero* exposure to gastrin in these infants? (2) Is there a possibility of excessive postprandial hypergastrinaemia in these infants? The first question has been addressed by a study (Werlin *et al.*, 1978) in which levels of circulating gastrin were measured in umbilical venous blood and maternal venous blood obtained at delivery in 40 infants who subsequently developed hypertrophic pyloric stenosis. In this comparison, there was no significant difference between the mean cord and maternal gastrin concentrations of infants subsequently developing pyloric stenosis, and normal infants. The possibility of excessive postprandial hypergastrinaemia in these infants was addressed in a recent clinical study from our institution (Moazam *et al.*, 1978), and has been confirmed by Hambourg's work. In our study, 11 patients with congenital hypertrophic pyloric stenosis were evaluated with sequential pre- and postprandial serum gastrin levels immediately before pyloromyotomy and 2 to 10 weeks later. When compared with an age-matched control, the