baby', making him easy to 'forget' and his mother was terrified that he would die silently without her noticing. She was at a further disadvantage by her own inability to use facial expression as a means of communication with her baby. Soon all those concerned in the baby's care, including the mother herself, were fearful for the baby's safety and help was sought before there was serious neglect or abuse.

The family were then admitted to the Park Hospital for an intensive period of treatment where the mother learned to make appropriate signals and respond to her baby. Both mother and child could gaze fixate, even though it could not be coupled with smiling. Starting from this, the mother was helped to recognise the more subtle signs from the baby, such as tiny mouth movements and slight panting sounds in her presence. The baby, too, was encouraged in making chuckling noises in response to his mother's gaze. Marital therapy was also required and it was interesting to note that in the mother's family all the sufferers of myotonic dystrophy had had broken marriages. Several years later, this family remains united but the parents still require help from both health and social services in bringing up their 2 young handicapped children.

References

Lynch, M. A. (1975). Ill-health and child abuse. *Lancet*, **2**, 317–319.

Lynch, M. A., and Roberts, J. (1977). Predicting child abuse: signs of bonding failure in the maternity hospital. *British Medical Journal*, **1**, 624–626.

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Early use of sodium nitroprusside in respiratory distress syndrome

Sir,

Beverley *et al.* (*Archives*, 1979, **54**, 403) reported the response to sodium nitroprusside in an infant with hyaline membrane disease, and I should like to make some comments on their patient.

The course of the illness, with the infant not requiring added oxygen and only very low ventilator pressure by 24 hours of age, must cast doubt on the diagnosis of surfacant-deficiant hyaline membrane disease, and suggests that the baby was suffering from the after effects of severe birth asphyxia, the early problems of intrapartum pneumonia, or perhaps from some iatrogenic disease.

During the time sodium nitroprusside was being infused, 3 other major changes in treatment took place which may have been responsible for improving the baby's condition. Firstly, his pH was corrected. The effect of this on pulmonary perfusion and pulmonary vascular tone is well known, and could itself be responsible for the improved oxygenation in the infant. Secondly, the inspiratory to expiratory ratio was reduced from 4:1 to 1·5:1 and while we do not know the ventilator pressures that were sustained during this period, it seems likely that this would be associated with a considerable fall in the mean airways pressure. Particularly in infants without hyaline membrane disease, a high mean airways pressure can be responsible for serious pulmonary underperfusion, and lowering the airways pressure as was done in this patient, can be associated with a large increase in arterial Po_2. Thirdly, they transfused their patient, and while we are given no blood pressure data before this was started, the fact that they were able to increase the baby's blood volume by more than double suggests that, irrespective of the hypotension-producing effect of the nitroprusside, the infant was previously hypovolaemic or hypotensive, or both. Correction of that could also be responsible for an improvement in the infant's condition and oxygenation.

I would submit therefore that Beverley *et al.* have provided absolutely no evidence for a beneficial effect from sodium nitroprusside in their patient, and that before this comparatively dangerous drug is used, evidence that it improves oxygenation while other factors in the treatment are held constant is required. This, for example, has been provided for tolazoline (McIntosh and Walters, 1979).

Reference

McIntosh, N., and Walters, R. O. (1979). Effect of tolazoline in severe hyaline membrane disease. *Archives of Disease in Childhood*, **54**, 105–110.

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Sir,

The letter from Beverley *et al.* (*Archives*, 1979, **54**, 403), and the report by Abbott *et al.* (1978) to which they refer, should both be treated with extreme caution. In neither is a good case made for the use of sodium nitroprusside to reduce pulmonary vascular resistance (PVR) in severely hypoxic newborn babies. While Beverley and colleagues refer to the known hazards of this hitherto untried drug, they give no details of the state of the circulation or of the $Paco_2$ during their infant's first few hours. These are important since poor tissue oxygenation and hypercapnia may both contribute to acidosis, and PVR in the newborn may be expected to increase as pH falls (Rudolph, 1977). The extreme acidosis at 2–4 hours of age was presumably the legacy of severe birth asphyxia.

Nevertheless, the information provided suggests that after a blood transfusion (which should have improved tissue oxygenation) and during the slow correction of the severe acidosis, there was initially a steady fall in right-to-left shunt, followed at the age of 11 hours by a more rapid improvement in both the arterial pH and $Paco_2$.