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

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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 surfactant-deficient hyaline membrane disease, and suggests that the baby was suffering from the aftereffects 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 P O 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

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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.
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

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Role of gastrin in pyloric stenosis in infants

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 PaO₂ 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. PaCO₂ 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 PaO₂ 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.

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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