Reply

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Sir: At present there are no widely accepted criteria for ARDS in neonates but it is accepted that ARDS may follow shock or sepsis both in newborns and in adults. To evaluate plasma fragment C3a as a possible indicator for ARDS we studied ventilated neonates with RDS to exclude unspecific complement activation under intensive care. Before starting the study [1], complement activation in neonates was only demonstrated in cases of sepsis, which had been excluded. Our results, an increase of C3a after birth asphyxia or shock resulting from other reasons, are in accordance with the data of Zilow et al. [2] although they do not comment about the incidence of RDS in their study group. The same author reported the prognostic value of C3a in adult patients at risk of ARDS [3]. In contrast to Zilow et al. we used a modified method for the determination of C3BbP with a different reference substance and a higher normal range. Despite significantly higher concentrations of C3a in the asphyxia group, there was no significant difference in C3BbP values between the asphyxia and the control group.

The three newborns with a gestational age of 37 weeks in our control group had a mild RDS and probably wet lung disease. It is indeed a critical point that we can only assume the diagnosis of ARDS related to clinical features and history. We already mentioned in our paper that not all neonates develop respiratory disease after birth asphyxia. Complement activation may also occur in other neonatal diseases. Neonates with primary RDS have C3a values within the normal range and high C3a plasma concentrations in neonates with RDS point to previous complications such as sepsis or shock.

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Pentoxifylline treatment of persistent pulmonary hypertension of newborn

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Sir: Pentoxifylline (PTXF) (Trental, 5 mg/kg per h for 6 h) was administered intravenously to a preterm infant (gestational age 34 weeks, weight 2100 g) with persistent pulmonary hypertension of the newborn. The child was born 4 weeks after premature rupture of the membranes.

Chorio-amnionitis was considered and *Escherichia coli* was isolated from the amniotic fluid. The infant was breathing spontaneously but was immediately oxygen dependent with an FiO_2 of 1.0 and P_aO_2 of 32 mm Hg. Respiratory distress syndrome was suspected and bovine surfactant (Alveofact) was instilled without noticeable improvement in P_aO_2. The chest radiograph showed a slightly enlarged heart, patchy infiltrates suggesting localized infection, and decreased pulmonary vascular markings. Echocardiography provided evidence of right to left interatrial and ductal shunts. The ventilator settings were adjusted as follows: FiO_2 1.0, ventilation rate 150/min, peak of inspiratory pressure 12 cm H_2O, positive end-expiratory pressure 1 cm H_2O, inspiratory time 0.2 s. After 1 h of mechanical ventilation P_aCO_2 decreased from 35 mm Hg to 22 mm Hg and P_aO_2 increased from 35 mm Hg to 45 mm Hg. P_aCO_2 of 22 mm Hg was the critical value and a minimal increase caused an immediate decrease in P_aO_2. After the 3rd hour of life PTXF was given for 6 h. During the 1st hour of PTXF infusion P_aO_2 increased to 60 mm Hg without any change in P_aCO_2. However, within the next 5 h, P_aO_2 increased to 115 mm Hg allowing an increase of P_aCO_2 to 35 mm Hg. After an identical infusion of PTXF on the next day the oxygen requirements decreased (FiO_2 from 0.95 to 0.7) and the ventilation rate was adjusted from 120 to 75. On the 3rd day of life, after the last PTXF infusion, the infant was weaned from the ventilator and placed in a hood with an FiO_2 of 0.6. Clinical symptoms of pneumonia were observed during the next few days. Antibiotic treatment was continued and the infant was discharged on the 20th day of life.

Beneficial effects of PTXF can be attributed to the reduction of pulmonary hypertension [5], increased erythrocyte deformability [1, 4] and possibly the reduction of oxyhaemoglobin affinity [3]. One can speculate that PTXF influences persistent pulmonary hypertension of the newborn by direct action on the pulmonary vasculature via the stimulation of endothelium derived relaxing factor. In cases of intra-uterine infection, endotoxin release causes a norepinephrine-induced constriction of pulmonary arteries which can be reduced by PTXF [2].

References

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Abbreviation: PTXF = pentoxifylline