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## Exogenous surfactant therapy for acute respiratory distress in infancy

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Sir: Respiratory syncytial virus (RSV) is the most common cause of acute viral disease of the lower respiratory tract in infants and children, and is the agent responsible for bronchiolitis. Approximately 1-2% of affected children require hospitalization. Progression to respiratory failure is uncommon [1] and the development of adult respiratory distress syndrome (ARDS) is very rare [2]. Despite a multitude of proposed treatments such as mechanical ventilation with high PEEP, high-frequency ventilation, and the use of pressure-control ventilation with reverse inspiratory-expiratory ratio, the mortality rate from ARDS remains very high. Based on experience with its use in the treatment of respiratory distress syndrome (RDS) in neonates, surfactant therapy has recently been suggested to be of potential benefit in the treatment of ARDS [3, 4].

We present the case of an infant with RSV-induced ARDS who showed great clinical improvement upon administration of intratracheal surfactant. A 6-week-old male, second twin, with a history of prematurity (36 weeks of gestation), with no respiratory pathology in the neonatal period, was admitted to our pediatric intensive care unit because of bronchiolitis (direct immunofluorescence test positive for RSV is nasopharyngeal smear), manifested by respiratory distress and apneic spells. Two days after admission to the PICU, atelectasis of the left lung developed and the patient required intubation and mechanical ventilation. The patient developed left pneumothorax requiring pleural drainage, and over the next 48 h there was progressive respiratory deterioration with increasing hypoxemia. A chest X-ray revealed generalized bilateral alveolar infiltration and severe hypoxemia  $(P_0O_2)$  56 mmHg), despite increased respiratory support with pressure-controlled ventilation and continuous flow, with pressures 40/7 cm H<sub>2</sub>O, respiratory rate 42 breaths/min, FIO<sub>2</sub> 1, and inspiratory:expiratory ratio 1:1.0. As there was no response to elevations of PEEP (maximum PEEP of  $12 \text{ cm H}_2\text{O}$ ), treatment with nitric oxide in concentrations up to 20 ppm was performed 5 days after intubation with no improvement in oxygena-

tion. 24 h later, treatment with porcine surfactant (Curosurf) instilled through the intratracheal tube at  $50 \text{ mg}^{-1} \text{ kg}^{-1}$  dose was administered. Four doses were administered over a 36-h period. After the second administration of surfactant, a

fast improvement in oxygenation occur-

**Table 1** Ventilatory settings and oxygenation indices before and after treatment with NO and four doses of surfactant  $(A - DO_2$ , Alveolar – atertial oxygen gradient;  $a/A O_2$ , Arterial alveolar oxygen ratio; Oxygenation index, Mean airway pressure × FIO<sub>2</sub>/P<sub>a</sub>O<sub>2</sub>; Ventilatory index, P<sub>a</sub>CO<sub>2</sub> × Peak of pressure × respiratory rate/1000)

	Before NO	After NO	Before surf	After surf
Time	Day 5	1 h	Day 6	36 h* (Day 7)
pH	7.37	7.42	7.37	7.52
$P_aO_2$ mmHg	53	55	56	133
SÖ2 %	87	89	88	99
P <sub>a</sub> ĆO <sub>2</sub> mmHg	56	49	53	34
Öxygenation index	45	43	43	10
Ventilatory index	94	82	89	35
$A - a DO_2$	531	538	537	350
$a/A O_2$	0.09	0.09	0.09	0.27
$P_aO_2/\tilde{F}IO_2$	53	55	56	131
Peak pressure cm H <sub>2</sub> O	40	40	40	28
PEEP cm H <sub>2</sub> O	7	7	7	7
Respiratory rate	42	42	42	40
I:E	1:1	1:1	1:1	1:1.2
FIO <sub>2</sub>	1	1	1	0.8

\* After first dose of surfactant

## CORRESPONDENCE

red, with an increase in  $P_aO_2$  from 56 to 114 mmHg, thus allowing for a reduction in ventilatory support. The improvement in oxygenation continued with the third and fourth doses. The chest X-ray showed improvement 48 h after the beginning of surfactant administration. The patient was extubated 13 days later without complications. The ventilatory settings and oxygenation/ventilatory parameters before and after NO and surfactant administration are shown in Table 1.

The experience with this patient suggests that administration of surfactant may be useful in some pediatric patients with ARDS [4, 5]. The dosage and administration intervals of surfactant have not yet been defined for this population. In some infants with ARDS, as in our patient, the improvement in alveolar stability achieved through surfactant administration might be a more important factor for the reduction of intrapulmonary shunt fraction than the lowering of pulmonary vascular resistance with NO. In other patients, simultaneous treatment with NO and intratracheal surfactant might have synergic effects.

## References

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