Methods for improving the prognosis of mechanically ventilated infants with RDS

Sir,

Manginello et al. (Archives, 1978, 53, 878) stated that in the ventilatory treatment of infants with severe RDS the pressure-limited method of ventilation was better than that of volume-cycled, as with the former, the FIo2 could be reduced more rapidly and complications or death were less common. We obtained similar improvements using volume-cycled ventilation but with a different approach.3

We studied 56 newborn infants who were given mechanical ventilation for severe RDS between 1973 and 1976. Only the most severely affected infants were intubated and given mechanical ventilation. About half of these babies were apnoeic on admission; the others were ventilated because of progressive and severe deterioration of their clinical status or because of their blood–gases. Arterial pH, Pao2, and Pco2 were the only indications for mechanical ventilation in babies with severe hypercarbia, acidemia, or in those in whom severe hypoxaemia persisted despite adjustment of continuous distending pressure and FIo2.

During the years 1973–74 we gave mechanical ventilation to 31 infants with severe RDS and respiratory insufficiency using a volume-cycled ventilator (Bourns LA 104–150) in the controlled mode, with tidal volume of 5 ml/kg, respiratory rate of 60–80 min, positive end expiratory pressure (PEEP) of 3–7 cmH2O and I:E ratio of 1:1–1:2. Mortality was 74% pneumothorax or pneumomediastinum, or both, occurred in 23% of cases, and bronchopulmonary dysplasia was present in 67% of survivors.

In our attempts to improve the prognosis for mechanically ventilated infants with RDS we noted that, during the acute phase of the disease, if we changed from the controlled to the assisted mode (in which the ventilator cycle is triggered by the patient’s inspiratory effort) there was often a significant rise of Pao2. This phenomenon had already been described in infants with RDS ventilated mechanically without PEEP.4 Nevertheless Llewellyn and Swyer did not correlate the better oxygenation obtained with this method with changes in the prognosis in their patients. At the beginning of 1975, after we had made this observation, infants were set on a Bourns ventilator (ventilator setting the same as during 1973–74) with 100% oxygen in controlled respiration as long as arterial pH was <7.30 and Pao2<50–60 mmHg (6–7.9 kPa), and we changed to the assisted mode as soon as pH and Pao2 attained better values and the patient’s respiratory activity was sustained. The patient no longer had to struggle against the ventilator, there was improved oxygenation, and the infant was able to regulate the respiratory rate according to his own needs. With this approach it was possible to lower the FIo2 and to extubate the patient more rapidly. During 1975–76 we ventilated 25 neonates with severe RDS in this way, with a fall in mortality rate to 48% and with an incidence of pneumonia or pneumomediastinum, or both, of 8%, while bronchopulmonary dysplasia was found in only 8% of the survivors. The difference in mortality and bronchopulmonary dysplasia figures in the two study periods was significant (P<0.05). The infants were similar in respect of birthweights, gestational ages, and severity of respiratory disease.

We believe that the main advantages of ventilating patients with severe RDS with a volume-cycled ventilator using the assisted (triggered) method are that the baby does not have to struggle against the ventilator, the respiratory rate is automatically regulated, the respiratory centres are continuously activated during the whole ventilation time, and finally, it is quicker and easier to wean the baby from the ventilator.

We suggest that before a final conclusion is made on the superiority of pressure-limited mechanical ventilation in RDS, controlled trials should be done to compare pressure-limited ventilation with other methods of volume-limited ventilation (including the use of PEEP in assisted ventilation with trigger).

References


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Acute infantile thrombocytosis and vitamin K deficiency associated with intracranial haemorrhage

Sir,

The paper by Lorber et al. (Archives, 1979, 54, 471) was especially interesting to me because of my own observation of thrombocytosis in a 2-month-old infant (to be reported) in whom pronounced vitamin E and vitamin D and probable vitamin K and vitamin A deficiency (slightly prolonged prothrombin, partial thromboplastin time, and very low carotene level) were shown. Because other causes of thrombocytosis of infancy were ruled out, infantile hyperostosis was sought and was proved by x-ray.

Since thrombocytosis in infancy has been reported in patients with Caffey’s disease,1–2 possibly related to vitamin E deficiency,2 we suggest that at least a hydrogen peroxide haemolysis test (which was also positive in our case) should be carried out in all patients with infantile thrombocytosis in whom the aetiology is unknown.

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Because of intracranial bleeding the head x-ray should be available to help in the diagnosis of Caffey's disease. Probable vitamin K deficiency suggests that the level of other lipid soluble vitamin levels should also be determined.

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Dr Lilleyman comments:

We were interested in Professor Ozsoylu's observations concerning our patient with intracranial bleeding and thrombocytosis and confess that at the time we did not consider the possibility of infantile hyperostosis or vitamin E deficiency being present, although we acknowledge that both conditions have been found in patients in whom the platelet count is high.

Against the diagnosis of Caffey's disease in our patient was the absence of fever and the fact that there were no characteristic mandibular or clavicular swellings; as there were no clinical stigmata of the disorder no x-rays were obtained.

As far as vitamin E deficiency is concerned, we can only say that the reticulocyte count was not recorded above 3% at any time, and the characteristic irregularly contracted erythrocytes of the associated haemolytic anaemia were not noted. Also our patient was a term baby of 2.86 kg, whereas those described with vitamin E deficiency associated thrombocytosis have been premature with birthweights of 1.5 kg or less. None the less, we did not measure tocopherol levels or perform a peroxide haemolysis test and so cannot be absolutely certain about this point.

We agree that these two conditions should be considered when thrombocytosis occurs in infancy but are tempted to observe that their discovery would make the genesis of a high platelet count no less puzzling.

**References**


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Acute infantile thrombocytosis and vitamin K deficiency associated with intracranial haemorrhage.

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Arch Dis Child 1980 55: 84-85
doi: 10.1136/adc.55.1.84-a

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