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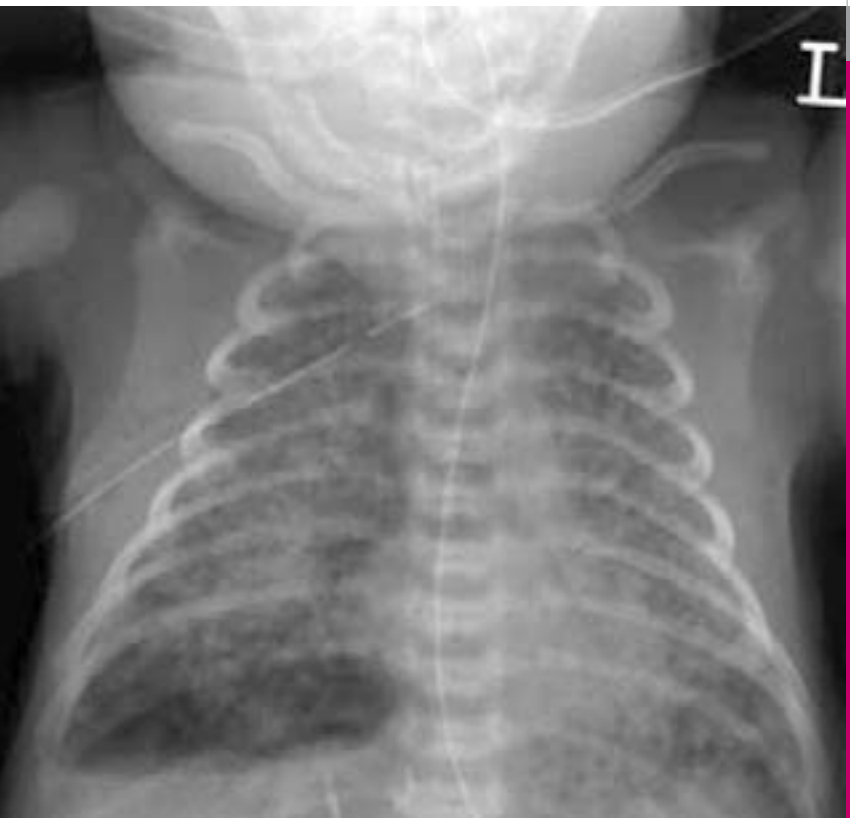
Supercarbia in an infant with meconium aspiration syndrome



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This male infant was born by vacuum extraction because of fetal distress at 41 weeks of gestation after an otherwise uneventful pregnancy. The Apgar scores were 1, 1, and 1 at 1, 5 and 10 minutes, respectively. Umbilical arterial pH was 7.08 (base excess –14 mmol/L). There was meconium-stained amniotic fluid and meconium was noted below the cords at birth. The boy was tracheally intubated in the first minutes of life because of persistent bradycardia and no spontaneous breathing. Capillary pH after 30 minutes of life was 6.8 with base excess –21.6 mmol/L. The infant was transfered to our unit in severe respiratory failure due to meconium aspiration and a pneumothorax on the right side (Fig. 1).

He was dependent on high frequency oscillatory ventilation (HFOV) for 7 days and iNO during 9 days (Fig.2: FiO2 and oxygenation index; oxygenation index = Mean Airway Pressure [cmH2O] x FiO2 [decimal] / PaO2 [mm Hg]; Fig. 3: mean airway pressure). HFOV-settings were as follows: frequency 10, later on 7 Hz, mean airway pressure 18-26 cmH2O, pressure amplitude 35-55 cmH2O. We adopted a permissive ventilation strategy from the beginning, tolerating high PaCO2 values (<90 mmHg, no upper limit later on, permissive hypercapnia) and pulse oximeter oxygen saturation values in the range of 75 and 90% (permissive hypoxemia, Fig. 4).

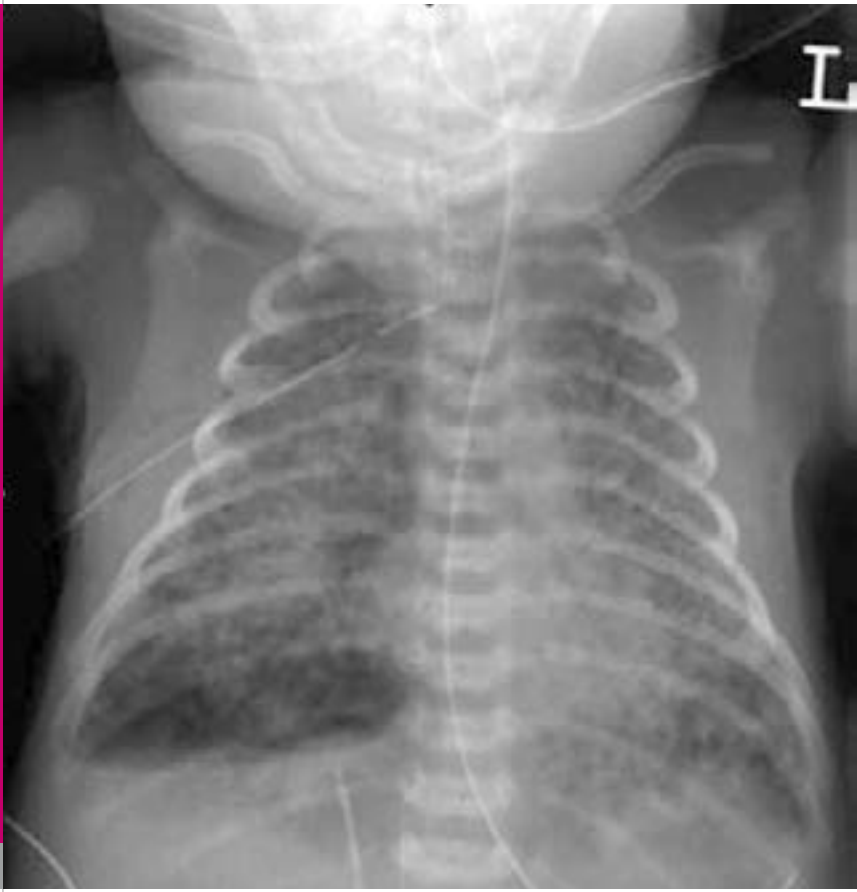

Fig. 1

CXR on the first day of life. A pneumothorax on the right side has been drained.

During the first 2 weeks, arterial PCO2 values were between 50 and 100 mmHg with a peak of 328 mmHg (43 kPa). The pH at the same time was 6.8. Intermittently, the infant was given tris-hydroxymethyl amino-methane buffer (Fig. 5: arterial pH, Fig 6: arterial PCO2, Fig. 7: arterial base excess). At the time of the

highest PCO2 value, the HFOV settings were: frequency 8 Hz, mean airway pressure 18 cmH2O, pressure amplitude 50 cmH2O, FiO2 1.0.

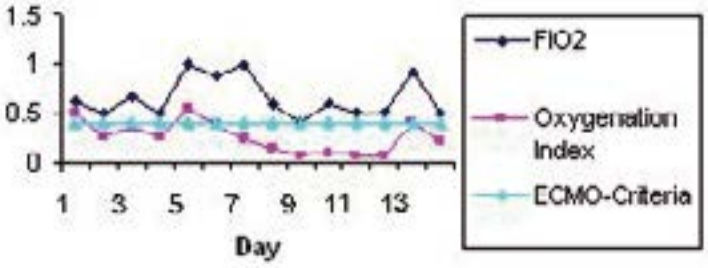

Fig. 2

Oxygenation index and FiO2.

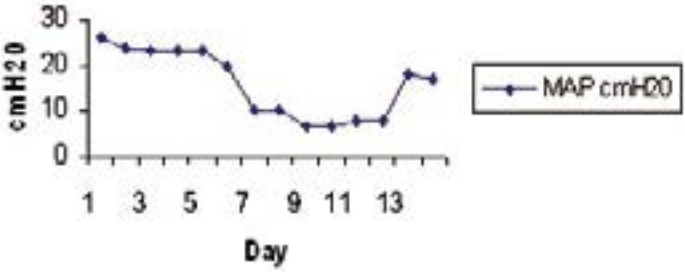

Fig. 3

Mean airway pressure.

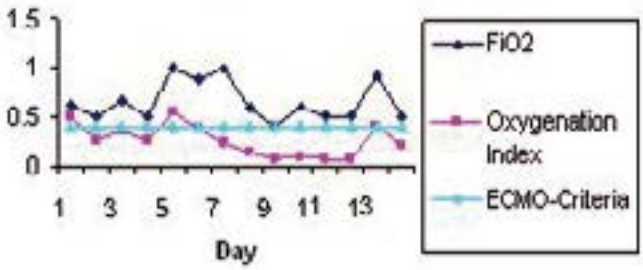

Fig. 4

Oxygen saturation.

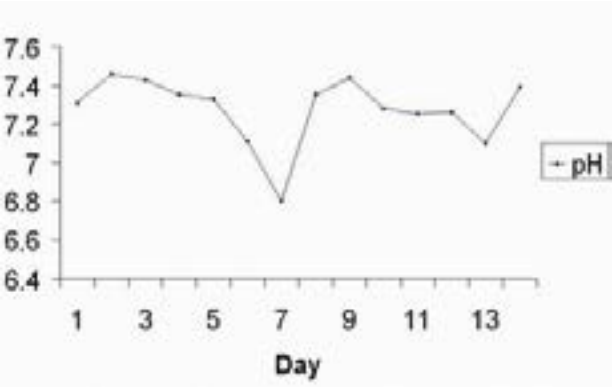

Fig. 5

Arterial pH.

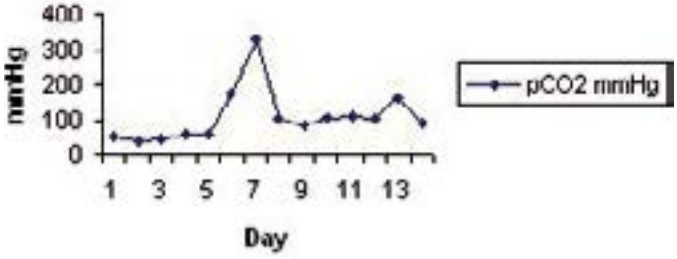

Fig. 6

Arterial base pCO2.

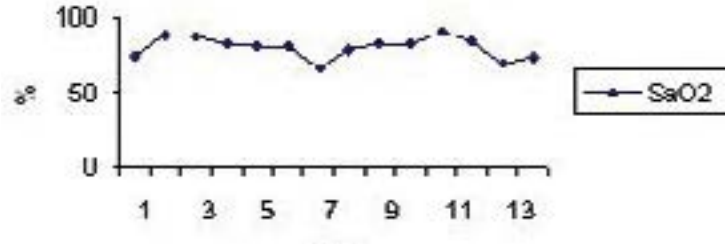

Fig. 7

Arterial base excess.

The chest X-ray showed overinflated lungs and a progressing interstitial emphysema (Fig. 8). We switched the patient to conventional positive pressure ventilation (frequency 50/min, inspiratory pressure 25 cmH2O, PEEP 5 cmH2O, mean airway pressure 10 cmH2O). With this strategy, PaCO2 decreased to 100 mmHg. In order to stabilize oxygen delivery, hemoglobin concentration was kept at around 120 g/L and cardiac output was supported with catecholamines. Blood lactate as an indirect measure of the adequacy of oxygen delivery was in the range of 1.3-5 mmol/L. A surfactant lavage has not been carried out. Successful change to non invasive CPAP-ventilation was possible at the age of 4 weeks. The boy was intermittently CPAP-dependent until the age of 8 weeks. He was discharged home at the age of 3 months under continuous nasal oxygen supplementation. The oxygen-supplementation could be discontinued at the age of 6 months.

The cranial MRI at the age of 3 weeks showed no major lesions. There were subtle signs of perinatal hypoxia/ischemia in the supratentorial white matter (slightly increased T2-values and decreased diffusion- and NAA-values). There was no evidence for damage of the basal ganglia or the cortex. The neurologic follow up at 7.5 and 9 months showed a normal motor and cognitive development. Pulmonologic follow-up at 9 months of age was normal without any signs of chronic lung disease (Fig. 9).

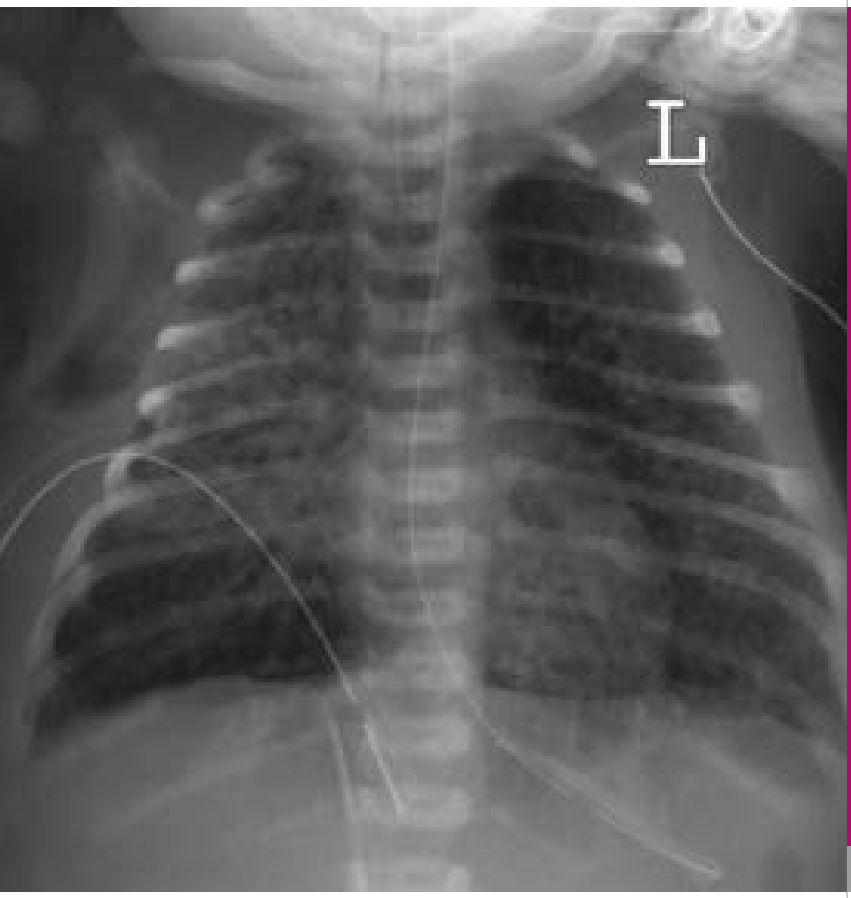

Fig. 8

CXR just before switching patient from HFOV to conventional ventilation.

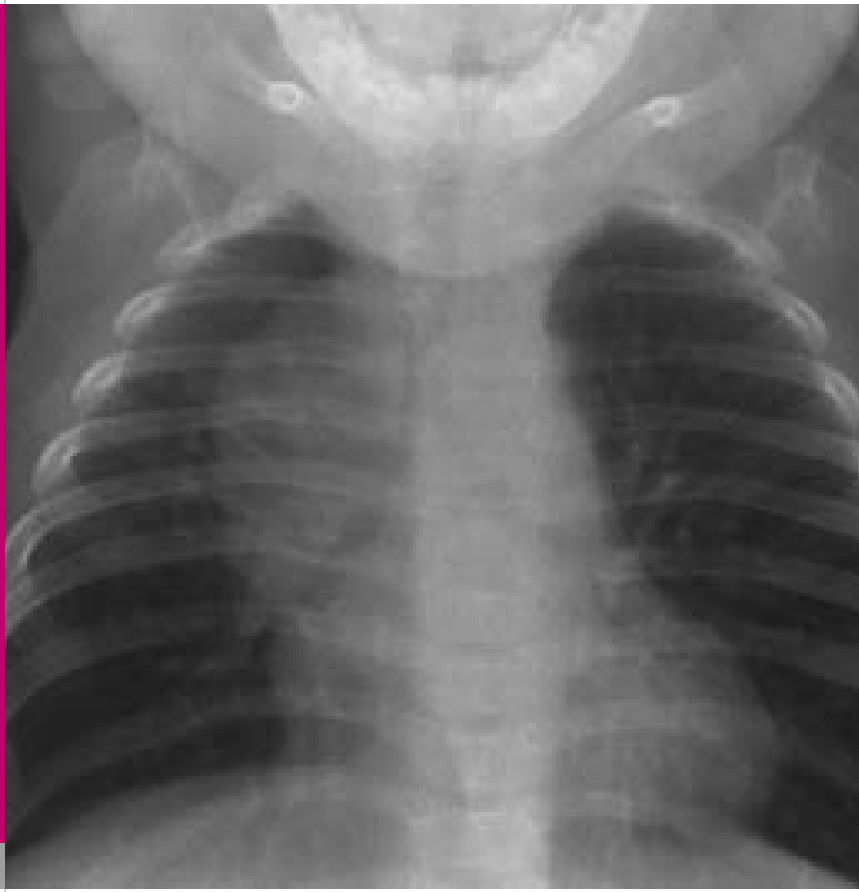

Fig. 9

CXR at 9 moths of age.

One of the most important concepts in the care of critically ill patients is the recognition that mechanical ventilation can worsen, or even cause, lung injury by repetitive over-stretching of lung tissue. If hypoventilation is allowed in an effort to limit lung stretch, carbon dioxide tension increases. In conventional positive pressure ventilation, it is beyond doubt that reducing tidal volume and thereby lowering lung stretch is improving outcome in adults with acute lung injury [1, 2]. The boy reported here was on high frequency oscillatory ventilation during the first 7 days of his life. This ventilation mode may be more gentle regarding secondary lung injury. However, with both, high frequency and conventional ventilation, we accepted (extreme) hypercapnia and hypoxemia, thereby avoiding even higher ventilation parameters or extracorporeal membrane oxygenation (ECMO). The extreme ventilation failure with supercarbia (PCO2 328 mmHg) on the 7th day of life was co-induced by interstitial emphysema and lung overstretching, which both evolved under HFOV.

There is evidence, that hypercapnic acidosis per se has protective effects in organ injury as it may increase oxygen delivery (increased cardiac output and shift of the haemoglobin-oxygen dissociation curve to the right) and may attenuate inflammatory processes [3]. Mariani et al. [4] showed that preterm infants treated with permissive hypercapnia do not have higher incidences of intraventricular hemorrhage, periventricular

leukomalacia and mortality. In children, there is no evidence for impaired development even after supercarbia with PCO2 levels of up to 296 mmHg [5]. Death in ARDS results largely from multisystem organ failure, not hypoxia [6].

Preterm babies given enough supplemental oxygen to maintain a pulse oximeter oxygen saturation of 70-90% had an improved outcome in the form of less retinopathy of prematurity, fewer ventilation days, and improved weight gain compared to babies whose treatment was directed to a "physiological" pulse oximeter oxygen saturation goal of 88-98% [7]. By accepting low oxygenation levels we could hold on to conservative ventilation strategies although the oxygenation index was temporarily over 0.4, which by itself qualifies for ECMO [8]. For term infants, there are only few studies on the safety of hypoxia and hypercarbia associated with a permissive ventilation strategy [9].

In conclusion, in neonatal respiratory failure, moderately decreased oxygenation (arterial oxygen saturation of 70-90%) and marked decreased ventilation (excessively elevated PCO2 values) may be tolerated without neurological sequelae, as long as organ perfusion and oxygen delivery are sufficient. However, because of lack of randomized trials, there is insufficient evidence on the safety of this approach. On the other hand, this approach may allow to manage these neonates without ECMO which itself is associated with significant morbidity.

REFERENCES

1. Amato MB, Barbas CS, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, Kairalla RA, Deheinzelin D, Munoz C, Oliveira R, Takagaki TY, Carvalho CR. Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. N Engl J Med 1998;338:347-354

2. Laffey JG, O'Croinin D, McLoughlin P, Kavanagh BP. Permis sive hypercapnia role in protective lung ventilatory strategies. Intensive Care Med 2004;30:347-356

3. Laffey JG, Kavanagh BP. Carbon dioxide and the critically ill—too little of a good thing? Lancet 1999;354:1283-1286

4. Mariani G, Cifuentes J, Carlo WA. Randomized trial of permissive hypercapnia in preterm infants. Pediatrics1999; 104:1082-1088

5. Goldstein B, Shannon DC, Todres D. Supercarbia in children: clinical course and outcome. Crit Care Med 1990;18:166-168

6. Stapleton D, Wang BM, Hudson LD, Rubenfeld GD, Caldwell ES, Steinberg KP. Causes and timing of death in patients with ARDS. Chest 2005;128:525-532

7. Tin W, Milligan DW, Pennefather P, Hey E. Pulse oximetry, severe retinopathy, and outcome at one year in babies of less than 28 weeks gestation. Arch Dis Child Fetal Neonatal Ed 2001;84:F106-F110

8. Cook LN. Update on extracorporeal membrane oxygenation. Paediatr Respir Rev 2004;5:S329-337

9. Wung JT, James LS, Kilchevsky E, James E. Management of infants with severe respiratory failure and persistence of the fetal circulation, without hyperventilation. Pediatrics 1985; 76:488-494

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