Acute intracardiac right-to-left shunt in a patient with acute respiratory distress syndrome and shock successfully treated with nitric oxide

Editor—In patients with acute respiratory distress syndrome (ARDS) hypoxia can be aggravated by intracardiac right-to-left shunt through a patent foramen ovale. Positive pressure ventilation and PEEP may increase shunting. We report a patient in whom high dose norepinephrine seemingly triggered an acute right-to-left shunt, successfully treated with inhaled nitric oxide (NO).

A 57-yr-old female was admitted to another hospital with a 1-week history of fever and dyspnoea. Previous medical history included a breast tumour treated with surgery, radiotherapy and chemotherapy 3 yr earlier. She was in respiratory distress with tachypnoea (35 breaths per minute), cyanosis and peripheral oxygen saturation of 88% breathing ambient air. Blood pressure was 87/55 mm Hg and heart rate 113 bpm. Blood analysis demonstrated $87 \times 10^9$ leucocytes per litre, with 74% promonocytes. Acute myeloid leukaemia was later confirmed. Chest X-ray showed bilateral pulmonary infiltrates. Fluid resuscitation, antibiotics and hydroxycarbamide were started. The patient required tracheal intubation and ventilation 24 h after admission. Over the next day her condition worsened with rapid increase in oxygen need and progressive hypotension despite therapy with fluid and norepinephrine. She was transferred to our centre. She remained hypotensive with norepinephrine at 1 $\mu$g kg$^{-1}$ min$^{-1}$, and hypoxic ($P_{A\text{O}_2}/F_{A\text{O}_2}$ ratio of 59 mm Hg) despite ventilation with 100% $F_{A\text{O}_2}$ at 32 cm H$_2$O PEEP and plateau-pressure of 2 cm H$_2$O. Hydrocortisone was started (100 mg bolus followed by 200 mg/24 h). Transpulmonary thermodilution curve (PiCCO-monitor, Pulsion Medical Systems, Munich, Germany) revealed a double-hump suggesting intracardiac right-to-left shunt (Fig. 1A). This was confirmed by

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Editor—There are a quite a number of studies regarding the relationship between EEG derived indexes and anaesthetic drugs in the literature. All demonstrate that a decrease in BIS correlates with an increase in the Ce propofol. I agree with Lim and colleagues that a lack of consensus regarding the definition of clinical endpoints and pharmacokinetic-dynamic parameters to test the ability of an EEG derived index is a potential problem.

I believe that clinical judgement should always accompany the increasing application of technology in clinical anaesthesia.

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Fig 1 Transpulmonary thermodilution curve before and during NO treatment. (A) Double hump sign characteristic of right-to-left intracardiac shunt before start of NO therapy. (B) Reduction of double hump sign by adding NO. (C) Disappearance of the double hump sign during recovery. Doses of NO and norepinephrine (norepi) and PEEP values are shown.
trans-oesophageal echocardiography, showing a patent foramen ovale with major shunt and moderately dilated right ventricle. Inhaled NO was started. This immediately improved oxygenation ($P_{A,O_2}/F_{I,O_2}$ 78 mm Hg) and the thermodilution curve (Fig. 1b) despite persistent and unchanged norepinephrine dose of $1 \mu g \cdot kg^{-1} \cdot min^{-1}$ and same ventilatory settings. NO was titrated to 20 ppm. Twelve hours later norepinephrine was reduced to $0.3 \mu g \cdot kg^{-1} \cdot min^{-1}$ and NO was reduced to 3 ppm as the double hump had completely disappeared (Fig. 1c). In the next 48 h norepinephrine and NO were discontinued and the trachea was successfully extubated 1 week later.

Norepinephrine can cause a significant increase in pulmonary artery pressures in ARDS and sepsis.\(^2\) In this case, pulmonary pressures apparently were sufficient to cause right-to-left intracardiac shunt through a patent foramen ovale. The shunt presented as profound hypoxia exacerbated by increasing norepinephrine and the double hump on the transpulmonary thermodilution curve, suggested a short pass of the indicator.

Besides reducing ventilation-perfusion mismatch,\(^3\) inhaled NO can also improve oxygenation by resolving intracardiac shunt.\(^5\)\(^6\) Here, the shunt was likely triggered by the high dose of norepinephrine as reduction allowed weaning from NO. This quick improvement was unlikely to be regression of ARDS.

In patients with ARDS and worsening hypoxemia receiving high dose norepinephrine, right-to-left shunt should be suspected. Visual analysis of the transpulmonary thermodilution curve may suggest the diagnosis. Therapy with NO should be considered. The effect can be assessed by the shape of the thermodilution curve.

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**Successful weaning from mechanical ventilation after abdominal lipectomy and omentectomy in an obese patient with multiple rib fractures**

Editor—Obesity\(^1\) and old age\(^2\)\(^3\) are major risk factors for pulmonary complications following trauma or general anaesthesia. We report our experience with a morbidly obese, elderly patient with multiple rib fractures, who was successfully weaned off the mechanical ventilation after abdominal lipectomy and omentectomy.

A 69-yr-old, morbidly obese female was admitted with severe shortness of breath following traumatic injury to her chest in a motor vehicle accident. She was 155 cm and 95 kg, with body mass index 39.5 kg m\(^{-2}\). Computed tomography scans taken a few hours after the injury showed multiple right rib fracture (3rd to 11th), pulmonary contusion with small amount of haemopneumothorax in the right chest, and significant fatty tissue in the thoracic and peritoneal cavities. The arterial blood pressure was 160/90 mm Hg and the heart rate (HR) 68 beats min\(^{-1}\). Her respiratory status deteriorated progressively over 12 h, the respiratory rate being increased up to 30 bpm. She was transferred to the intensive care unit and was given supplemental oxygen via facemask. She received i.v. patient-controlled analgesia, and diuretics to improve pulmonary compliance in a sitting position. A tube thoracostomy was placed in the right chest for drainage of associated haemopneumothorax. She was encouraged to cough vigorously and breathe deeply. Chest physiotherapy was carried out frequently to prevent retention of secretions and development of atelectasis. Her $P_{A,O_2}$ increased progressively, although arterial oxygenation was maintained with supplemental oxygen. On day 8, she was intubated and lungs were mechanically ventilated, when her $P_{A,O_2}$ increased to 13.2 kPa and $P_{A,O_2}$ decreased to 6.2 kPa. Chest symptoms subsided within 2 weeks after the injury. However, after 8 days of mechanical ventilation, attempts at weaning on three occasions over the following 2 weeks were unsuccessful. A surgical removal of abdominal fat was considered to reduce the intra-abdominal pressure and to improve respiratory mechanics. On day 21 of mechanical ventilation, she underwent abdominal lipectomy (2940 g) and omentectomy (1650 g) under general anesthesia. The surgery lasted 5.7 h and was uneventful. The tidal volume increased from pre-operative value of 350–400 ml to 450–550 ml, and static compliance from 0.033 to 0.05 litre cm H\(_2\)O\(^{-1}\) on the 1st postoperative day. From the 5th postoperative day, weaning trials were continuously made during the day time with