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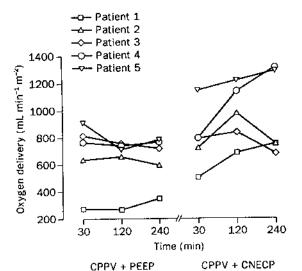
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Improved oxygen delivery by positive pressure ventilation with continuous negative external chest pressure

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Higher positive end-expiratory pressures (PEEP) improve arterial pO₂ by raising the tidal range above closing capacity, while decreasing cardiac output. Continuous subatmospheric pressure via a cuirass shell is expected to raise functional residual capacity at a lower intrapulmonary pressure. Based on experimental data¹ we hypothesised that the lower intrapulmonary pressure would enhance oxygen delivery because of an increase in cardiac output.

Five critically ill patients on positive-pressure ventilation with pre-existing PEEP exceeding 10 cm H₂O were recruited into a two-period crossover trial. The effects of 4 h of positive-pressure ventilation with PEEP (CPPV-PEEP) were compared with positive-pressure ventilation with continuous negative external chest pressure (CPPV-CNECP). Negative external chest wall pressure was adjusted to result in the same change in functional residual capacity as with PEEP.



Individual course of oxygen delivery from five patients during CPPV-PEEP and CPPV-CNECP

The overall increase in oxygen delivery index under CPPV with CNECP was significant (900 [257] vs 650 [211] mL min⁻¹m⁻², p=0-026). In one patient with pre-existing left ventricular insufficiency (patient 3) oxygen delivery remained essentially unchanged.

This was accomplished by allowing complete expiration to zero end-expiratory pressure after removing PEEP or negative cuirass pressure, respectively. The settings of tidal volume, respiratory rate, fractional inspired oxygen (FiO₂), and the inspiratory/expiratory ratio were held constant during the entire study period. The cuirass of the Hayek Oscillator (Breasy Medical Equipment, London, UK) was placed over the chest wall and upper abdomen. Negative chamber pressure was applied from the vacuum pump of a Hayek Oscillator. All patients had radial artery and pulmonary catheters in place. Urinary output and free water clearance were calculated from the corresponding measurements of a 4-h urine sample and a blood sample taken at 120 min in each ventilatory mode.

All five patients survived their critical illness and were discharged from our intensive care unit after between 20 and 40 days (mean 31.2 [SD 7.3]). Compared with CPPV-PEEP, the combination of positive pressure ventilation with negative external chest pressure led to an increase in oxygen delivery (figure) because of an enhancement in cardiac index (mean 4.55 [SD 1.44] vs 6.40 [1.95] L/min, p=0.024). Arterial oxygen content and pO/FiO, ratio did not differ between both modes of ventilation. The switch from CPPV-PEEP to CPPV-CNECP decreased mean airway pressure (19-9 [SD 1-0 vs 14-3 [2-0] cm H₂O, p=0-01) and diminished the compliance of the respiratory system (52.3 [8:5] vs 38:5 [6:5] mL/cm H₂O, p=0:001). The amount of negative chamber pressure that was needed to achieve the same change in functional residual capacity as PEEP (11 [0-7] cm H₂O) varied from 12 to 30 cm H₂O (18-4 [7-1] cm H,O). Institution of CNECP instead of PEEP enhanced tubular conservation of water as reflected by a more negative free-water clearance (-0.92 [1.23] vs -0.33 [$\overline{0}$.32], p=0·043).

There is some evidence that in sepsis, PEEP can induce changes in organ blood flow independently of the decrease in global perfusion. It deserves further study whether intermittent CPPV-CNECP could alter the outcome of patients with sepsis and respiratory failure. Since the cuirass shell limits access to the patient and skin defects may be induced, we restrict its use to a period of 6 h.

- 1 Krumpe PE, Zidulka A, Urbanetti J, et al. Comparison of the effects of continuous negative external chest pressure and positive end-expiratory pressure on cardiac index in dogs. Am Rev Respir Dis 1977; 115: 39-45.
- 2 Ranieri VM, Eissa NT, Corbeil C, et al. Effect of PEEP on alveolar recruitment and gas exchange in ARDS patients. Am Rev Respir Dis 1991; 144: 544-51.
- 3 Bersten AD, Gnidec AA, Rutledge FS, et al. Hyperdynamic sepsis modifies a PEEP-mediated redistribution in organ blood flows. Am Rev Respir Dis 1990; 141: 1198-208.

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