Table 1 Trend in arterial blood gas, ICP, and ventilator parameters before and after prone positioning. $P_{aO_2}$, partial pressure of oxygen (arterial); $P_{aCO_2}$, partial pressure of carbon dioxide (arterial); BE, base excess; $HCO_3^-$, standard bicarbonate; $FIO_2$, fractional inspired oxygen concentration; $V_T$, tidal volume; IBW, ideal body weight; PEEP, positive end-expiratory pressure; $P_{peak}$, peak inspiratory pressure; ICP, intracranial pressure

<table>
<thead>
<tr>
<th></th>
<th>Admission</th>
<th>24 h post-prone</th>
<th>48 h post-prone</th>
<th>12 h post-prone</th>
<th>7 h post-prone</th>
<th>1 h post-prone</th>
<th>2 h post-prone</th>
<th>24 h pre-prone</th>
<th>48 h pre-prone</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{aO_2}$ (kPa)</td>
<td>23.0</td>
<td>6.11</td>
<td>5.29</td>
<td>7.78</td>
<td>9.73</td>
<td>10.1</td>
<td>13.4</td>
<td>10.4</td>
<td>10.4</td>
</tr>
<tr>
<td>$P_{aCO_2}$ (kPa)</td>
<td>4.90</td>
<td>5.97</td>
<td>10.5</td>
<td>10.2</td>
<td>6.8</td>
<td>6.18</td>
<td>5.66</td>
<td>5.27</td>
<td>5.27</td>
</tr>
<tr>
<td>BE (mmol litre$^{-1}$)</td>
<td>–5.6</td>
<td>7.6</td>
<td>8.6</td>
<td>7.4</td>
<td>9.9</td>
<td>10.9</td>
<td>8.9</td>
<td>7.4</td>
<td>7.4</td>
</tr>
<tr>
<td>$HCO_3^-$ (mmol litre$^{-1}$)</td>
<td>19.2</td>
<td>31.6</td>
<td>35.3</td>
<td>34.1</td>
<td>34.4</td>
<td>35.0</td>
<td>32.7</td>
<td>30.9</td>
<td>30.9</td>
</tr>
<tr>
<td>$FIO_2$</td>
<td>0.50</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>0.85</td>
<td>0.7</td>
<td>0.55</td>
<td>0.35</td>
<td>0.35</td>
</tr>
<tr>
<td>$V_T$ (ml) (ml kg$^{-1}$ for IBW)</td>
<td>611 (6.8)</td>
<td>805 (8.9)</td>
<td>451 (5.0)</td>
<td>415 (4.6)</td>
<td>611 (6.8)</td>
<td>612 (6.8)</td>
<td>572 (6.4)</td>
<td>605 (6.7)</td>
<td>605 (6.7)</td>
</tr>
<tr>
<td>PEEP (cm H$_2$O)</td>
<td>8</td>
<td>5</td>
<td>15</td>
<td>15</td>
<td>15</td>
<td>15</td>
<td>15</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>$P_{aO_2}$/$FIO_2$ ratio (mm Hg)</td>
<td>345.0</td>
<td>45.8</td>
<td>39.6</td>
<td>58.4</td>
<td>85.9</td>
<td>108.2</td>
<td>182.7</td>
<td>222.9</td>
<td>222.9</td>
</tr>
<tr>
<td>ICP (mm Hg)</td>
<td>—</td>
<td>23</td>
<td>17</td>
<td>30</td>
<td>22</td>
<td>18</td>
<td>2</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Despite their lateral thoracotomy,$^5$ we believe this to the first case, however, which demonstrates that this technique can be used safely despite multiple traumatic thoracic injuries, including complex sternal fractures and a flail segment. Several of the key studies of prone ventilation specifically excluded patients with severe chest wall injuries$^6,7$ or intracranial hypertension.$^7,^8$ We believe this case illustrates that this treatment may, with care, be considered for such patients and also suggest that they should not be excluded from further studies in this field.

**Conflict of interest**

None declared.

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doi:10.1093/bja/aer374

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**Acute severe hypoxaemia after mechanical ventilation in a patient with an Ebstein anomaly requiring extracorporeal membrane oxygenation**

Editor—A 52-yr-old man with Ebstein anomaly was admitted to our intensive care unit with severe hypoxaemia. Before admission, the patient had moderate hypoxaemia with pleural and pericardial effusions. Echocardiography showed moderate right ventricular (RV) dilation and good left ventricular function and contrast echocardiography revealed a small intra-cardiac shunt due to a patent foramen ovale (PFO). During hospitalization, the hypoxaemia became acutely worse suggesting cardiac tamponade, which was confirmed by echocardiography. Surgical pericardiocentesis was proposed. After tracheal intubation and mechanical ventilation, the patient developed severe hypoxaemia, which necessitated the use of veno-venous extracorporeal membrane oxygenation (ECMO). Despite evacuation of the pleuropericardial serous exudation effusions, hypoxaemia and circulatory failure persisted. In addition, the patient developed liver and kidney failure. Transoesophageal echocardiography revealed severe RV and auricular dilation associated with an intra-cardiac shunt caused by a large PFO. After a multidisciplinary staff discussion, we decided to stop mechanical ventilation, limit ECMO to minimal blood flow, and added inhaled nitric oxide. As a result, we observed decreased flow across the PFO and an improvement in respiratory, circulatory, liver,
and kidney function. The patient was weaned from ECMO and subsequently discharged from hospital 28 days after the surgery. At 6 months, the patient was healthy with resumption of his normal activities.

Compared with the left ventricle, the RV has low elasticity. This characteristic is responsible for the RV dilation seen with an acute increase in RV afterload. This increase can be caused by not only pulmonary obstruction, as in pulmonary embolism, but also pulmonary capillary constriction. Hypoxaemia is the main cause of vasoconstriction. When there is vasoconstriction throughout the entire lung, as in acute respiratory distress syndrome, increased afterload can develop into acute cor pulmonale with circulatory failure. Mechanical ventilation worsens the situation by increasing alveolar pressure, compressing capillaries, and finally increase RV afterload. Protective ventilation and nitric oxide inhalation are widely used to ameliorate this RV dilation.

Ebstein anomaly consists of displacement of a well-formed tricuspid valve. The RV is ‘atrialized’, meaning it is small and thin. This congenital anomaly leads to congestive heart failure. Dilation results from inability of RV to support normal afterload. Worsening pulmonary condition leads to heart failure. The increase in intra-ventricular pressure is transmitted upstream to the right auricle. When the pressure in the right auricle is greater than that in the left auricle, a PFO could reopen and then create an intra-cardiac right-to-left shunt. In addition, femoral catheter insertion may worsen this shunt, if the catheter ejection flow of the ECMO is directed through the entrance of the PFO.

In this case, we decided to avoid aggressive treatment of closing the PFO and switching to an arterio-venous ECMO. Instead, we chose to decrease afterload by stopping mechanical ventilation and decreasing ECMO flow to allow the RV to recover (Fig. 1). This case illustrates the priority of the management RV failure. We must give special consideration to the RV’s unique physiology and the effect our intervention can have on RV loading. Echocardiography is the main tool for guiding treatment and monitoring for signs of improvement. It is important to understand this particular physiology when making decisions during RV failure. Finally, this case highlighted the need for multidisciplinary approach to choose the accurate strategy and to avoid worsening the situation.

**Conflict of interest**

None declared.

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**Fig 1** Echocardiography (four-chamber view) showing end-diastole (A) and end-systole (B) after removal of ventilation and ECMO (video is available in Supplementary material). The tricuspid valve (arrow) separated the right ventricle and atrium into two same-size chambers. Despite the absence of respiratory or circulatory failure, the right-sided chambers remain dilated, and there is paradoxical motion of the inter-ventricular septum. These persistent abnormalities explain why an increase in afterload could worsen the situation.
Fluid optimization guided by oesophageal Doppler significantly improves bowel perfusion

Editor—We report an emergency laparotomy in an elderly patient, where central venous pressure (CVP) did not provide a reliable guide to intraoperative fluid administration. However, oesophageal Doppler-guided fluid resuscitation sufficiently improved perfusion of the compromised gut to allow a definitive and curative operation to proceed, in circumstances where conservative management and a second operation would otherwise have been necessary.

An 82-yr-old lady presented with symptoms and signs of acute bowel obstruction. Examination revealed severe abdominal distension and an irreducible right femoral hernia. She was moderately hypotensive and anuric. Urea and creatinine levels were elevated, and there was a mild metabolic alkalosis. The lactate level was 4.8 mmol litre$^{-1}$ and creatinine levels were elevated, and there was a mild hernia. She was moderately hypotensive and anuric. Urea abdomen distension and an irreducible right femoral acute bowel obstruction. Examination revealed severe second operation would otherwise have been necessary.

However, oesophageal Doppler-guided fluid resuscitation could be undertaken. The ends of the small bowel were stapled off and returned to the abdomen. Over 45 min, 1.5 litre of Hartmann's solution and 1.5 litre of Volulyte were infused.Stroke volume and cardiac output improved dramatically (Table 1). Surgery was recommenced.

The cut ends of the small bowel were now well perfused and bleeding freely at the edges. A side-to-side ileal anastomosis was formed. The abdomen was closed easily and the patient was transferred to the intensive care unit.

The trachea was extubated after 12 h, and inotropic support was weaned off after 48 h. Urine output improved to 40–100 ml kg$^{-1}$ h$^{-1}$ within 24 h of surgery, and lactate levels decreased rapidly. The patient was discharged from the intensive care unit (ICU) after 5 days. The loop ileostomy was closed without laparotomy some months later.

Clinical trials demonstrating the benefits of oesophageal Doppler-guided fluid management in colorectal surgery have focused almost exclusively on elective bowel resection. Greater improvements should be seen in patients undergoing emergency bowel resection, but no studies addressing this question have been published. This case demonstrates that the use of oesophageal Doppler can optimize perioperative fluid therapy in the elderly sick laparotomy patient. More fluid was administered than would have been the case had the CVP been used to guide therapy. The patient did not develop pulmonary oedema and was extubated successfully 12 h after admission to ITU. If CVP measurements had been used, the patient would have remained underperfusion.

**Table 1** Comparison of intraoperative CVP and oesophageal Doppler recordings during volume resuscitation. Total volume given 4500 ml

<table>
<thead>
<tr>
<th>Time after induction of anaesthesia (min)</th>
<th>Fluid given cumulative total (ml)</th>
<th>Central venous pressure (mm Hg)</th>
<th>Oesophageal Doppler measurements</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hartmann’s solution</td>
<td>Volulyte$^\alpha$</td>
<td>Stroke volume (ml)</td>
</tr>
<tr>
<td>60</td>
<td>1000</td>
<td>500</td>
<td>14</td>
</tr>
<tr>
<td>75</td>
<td>1500</td>
<td>1000</td>
<td>8</td>
</tr>
<tr>
<td>90</td>
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<td>105</td>
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