and a 20 G, single outlet, 40 or 50 cm catheter (B/Braun Mel-
sungen, Germany or Pajunk, Geisingen, Germany) was inserted and advanced 5 cm beyond the needle tip. The catheter was, then, gradually retracted while normal saline was injected simultaneously, until the distance of the catheter tip to skin surface was equal to ‘D’. Hydrolocalization with normal saline being injected exactly underneath the targeted nerve confirmed the precise placement of the catheter tip under the nerve structure (Fig. 1c). At this point, 15 ml of ropivacaine 0.5% and 15 ml of lidocaine 1.5% were given. The quality of sensory block was evaluated every 10 min for up to 40 min and rated as: 0, 1, and 2 for normal, partial, and complete sensory nerve block. The sensory nerve block was successful if there was a complete absence of pinprick sensation in the plantar surface and dorsal surface of the foot). After an adequate nerve block was achieved, infusion of ropivacaine 0.2% at 5 ml h⁻¹ was commenced. After operation, the degree of pain at rest and on movement was assessed by using a 10 cm visual analogue scale (VAS) at 12, 24, and 36 h. Patients with VAS >4 were given a 10 ml bolus of ropivacaine 0.2% through the catheter and the rate of infusion was increased to 7 ml h⁻¹. If 1 h later pain scores still exceeded 4, another 10 ml bolus was given through the sciatic catheter and the infusion rate was increased to 10 ml h⁻¹.

Of the 18 patients (7/11 female/male, 42 (27) yr, 71 (13) kg), seven had hallux vagus repair, eight ankle fracture fixation, and three tibial fracture fixation. In all cases, the catheter tip was accurately placed under the sciatic nerve (saline injection was always seen exactly posterior to the targeted nerve). After operation, in two of 18 patients, one with hallux vagus repair and one with tibial fracture fixation, nerve pain management was inadequate. A 10 ml bolus of ropivacaine 0.2% and increase in the initial infusion rate of ropivacaine from 5 to 7 ml h⁻¹ provided sufficient pain relief.

In conclusion, combined ultrasound imaging and hydrolo-
calization technique can lead to the very accurate placement of perineural catheters. By using these three manoeuvres, (i) needle positioning under the sciatic nerve ‘D’, (ii) needle advancement beyond the targeted nerve, and (iii) catheter retraction back to ‘D’, we can place the catheter exactly underneath the targeted nerve.

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Prone ventilation for refractory hypoxaemia in a patient with severe chest wall disruption and traumatic brain injury

Editor—Adult respiratory distress syndrome (ARDS) carries a high mortality of up to 50%. Numerous therapies have been postulated in the treatment of this condition. Among them is ventilation in the prone position, which, although first discussed in the 1970s, has only relatively recently been demonstrated to have an outcome benefit, albeit only in the sickest of patients.

We recently encountered a case of a previously fit 50-yr-old man who sustained severe chest and head injuries in a motorcycle accident. He suffered a 10 min cardiorespiratory arrest at the scene and was resuscitated after bystander cardiopulmonary resuscitation and subsequent advanced life support (ALS) by paramedics. On arrival at hospital, bilateral pneumothoraces with multiple rib fractures, a large right-sided flail segment, and comminuted sternal fracture were discovered. He also sustained diffuse axonal injury, basal skull fractures, and subdural and subarachnoid haemorrhages. He was transferred to our care at the regional neurosurgical centre.

In the ensuing days, pulmonary contusions developed into ARDS and this was compounded by a ventilator-associated pneumonia. His head injury was managed non-surgically, although intracranial pressure management required sedation, paralysis, and omeprazole therapy. Oxygenation and ventilation became critical despite maximal conventional ventilatory therapy and on day 6, he reached a nadir with a PaO₂ of 5.5 kPa and a PaCO₂ of 10.1 kPa on an FiO₂ of 1.0 and a PEEP of 15 cm H₂O. He was turned prone, despite his injuries and bilateral chest drains (the latter being noted in a recent meta-analysis¹ as one source of potentially significant complications), and within 1 h, a dramatic improvement was noted in lung function indices (Table 1). Effects on intracranial pressure were moderate but very transient, resolving shortly after the position change. After 20 h, he was turned supine and went on to make a slow but near-complete neurological and respiratory recovery.

Prone ventilation has been used in a pregnant patient with several simple rib fractures. There has also been a series of oesophagectomy patients successfully managed in this way
Table 1  Trend in arterial blood gas, ICP, and ventilator parameters before and after prone positioning. \( \text{PaO}_2 \), partial pressure of oxygen (arterial); \( \text{PaCO}_2 \), partial pressure of carbon dioxide (arterial); BE, base excess; \( \text{HCO}_3^- \), standard bicarbonate; \( \text{FIO}_2 \), fractional inspired oxygen concentration; \( V_t \), tidal volume; IBW, ideal body weight; PEEP, positive end-expiratory pressure; \( P_{\text{peak}} \), peak inspiratory pressure; ICP, intracranial pressure

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Admission</th>
<th>24 h pre-prone</th>
<th>2 h pre-prone</th>
<th>1 h post-prone</th>
<th>7 h post-prone</th>
<th>12 h post-prone</th>
<th>24 h post-prone</th>
<th>48 h post-prone</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \text{pH} )</td>
<td>7.337</td>
<td>7.462</td>
<td>7.276</td>
<td>7.270</td>
<td>7.444</td>
<td>7.490</td>
<td>7.498</td>
<td>7.504</td>
</tr>
<tr>
<td>( \text{PaO}_2 ) (kPa)</td>
<td>23.0</td>
<td>21.1</td>
<td>5.29</td>
<td>7.78</td>
<td>9.73</td>
<td>10.1</td>
<td>13.4</td>
<td>6.14</td>
</tr>
<tr>
<td>( \text{PaCO}_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>
</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>
</tr>
<tr>
<td>( \text{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>
</tr>
<tr>
<td>( \text{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>
</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>
</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>12</td>
</tr>
<tr>
<td>( \text{PaO}_2 \cdot \text{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>
</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>
</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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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 pericardietes 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 pleural and pericardial 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,