The epidural fluid collection in our patient was unusual in its extent of spread. Being aware that multiple simultaneous CSF leaks are often present, two separate epidurals may lead to a more complete dural tamponade and better sealing in the upper cervical meninges.

Injection at two separate sites may decrease the possibility of spinal compression from a large amount of blood and guarantee a more uniform distribution over the meninges.

In conclusion, symptoms related to SIH are the result of a low CSF volume; a considerable spinal loss of CSF has serious complications including subdural hematoma. A two-level epidural blood patch may be an option to extend the distribution of blood when the exact CSF leak site is unknown, or following a failed lumbar patch. A greater volume of autologous blood may uniformly increase CSF pressure and replace the space of the lost CSF.

**Conflict of interest**

None declared.

P. Feltracco*  
S. Barbieri  
M. Milevoj  
C. Ori  
Padova, Italy

*E-mail: paolofeltracco@inwind.it

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### Intravenous magnesium sulphate prevents intravenous salbutamol tachycardia in asthma

Editor—An unconscious asthmatic received 2.5 g of i.v. magnesium sulphate before 500 μg of i.v. salbutamol which was given over 2 min. The expected tachycardia from the β1-adrenergic effect of a large dose of salbutamol did not occur. In order to confirm this observation, a volunteer with well-controlled asthma was given 250 μg of i.v. salbutamol (3 μg kg⁻¹) over 30 s which caused an increase in heart rate from 62 to 103, a nodal rhythm and palpitations which reverted to normal after 5 min. Two days later, 20 mg kg⁻¹ of i.v. MgSO₄ over 3 min was followed by the same injection of salbutamol which raised heart rate from 75 to 91 with no other effects. This useful effect of MgSO₄ was discussed and considered safe to use in future asthmatic patients.

Four subsequent asthmatics presenting with very high arterial carbon dioxide levels, three of whom were unconscious, had i.v. MgSO₄ followed by quickly delivered high-dose i.v. salbutamol with no tachycardia (Table 1). (Steroids and 100% oxygen were given to all but Case 2.)

Additional demonstration of MgSO₄’s ability to block β1-adrenergic effects of salbutamol occurred during ventilation of the lungs in Case 3, 18 h after admission, when a tachycardia during an infusion of salbutamol of 1 μg kg⁻¹min⁻¹ was reduced from 150 to 120 by 2.5 g of MgSO₄ given over 10 min; in Case 7, MgSO₄ also reduced tachycardia during an infusion. In Case 4, 40 min after MgSO₄, two doses of i.v. epinephrine (1 ml of 1:10 000, and 10 min later, 2 ml of 1:10 000), raised a low arterial pressure without an increase in heart rate.

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**Table 1** Asthma presentation, management, and changes

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Presentation</th>
<th>End-tidal or ( P_{\text{A}CO_2} )</th>
<th>I.V. MgSO₄</th>
<th>I.V. salbutamol</th>
<th>HR change</th>
<th>( K^+ ) change (m mol)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50</td>
<td>Unconscious GCS 6</td>
<td>( e_{15} ), 9.5%</td>
<td>2.5 g over 10 min</td>
<td>4 mg over 10 min</td>
<td>110–115</td>
<td>?–3.2</td>
<td>Ventilated, extubated after 5 h</td>
</tr>
<tr>
<td>2</td>
<td>56</td>
<td>Conscious volunteer</td>
<td>( e_{15} ), 5%</td>
<td>1.6 g over 3 min</td>
<td>250 μg over 30 s</td>
<td>70–79</td>
<td>Not known</td>
<td>No palpitations</td>
</tr>
<tr>
<td>3</td>
<td>17</td>
<td>Unconscious, fitting</td>
<td>30 kPa</td>
<td>2.5 g over 5 min</td>
<td>4 mg over 5 min</td>
<td>108, no change</td>
<td>6.1–3.9</td>
<td>Ventilated, extubated after 36 h</td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>Unconscious</td>
<td>15 kPa</td>
<td>2.5 g over 5 min</td>
<td>4 mg over 5 min</td>
<td>110, no change</td>
<td>3.9–4.2</td>
<td>Ventilated, extubated after 8 h</td>
</tr>
<tr>
<td>5</td>
<td>60 female</td>
<td>Unconscious GCS 3</td>
<td>15 kPa</td>
<td>5 g over 3 min</td>
<td>1.25 mg over 15 min</td>
<td>130, no change</td>
<td>4.1–2.7</td>
<td>Bipap, well after 10 h</td>
</tr>
<tr>
<td>6</td>
<td>57</td>
<td>Conscious GCS 8</td>
<td>14 kPa</td>
<td>5.0 g over 3 min</td>
<td>2 mg over 16 min</td>
<td>140, no change</td>
<td>4.3–3.8</td>
<td>Not intubated, alert, no wheeze</td>
</tr>
<tr>
<td>7</td>
<td>2 female</td>
<td>Conscious GCS 12</td>
<td>( PV_{\text{A}CO_2} ), 4.3 kPa</td>
<td>0.42 g over 20 min</td>
<td>Infusion 2 μg kg⁻¹ min⁻¹</td>
<td>200–160</td>
<td>Not known</td>
<td>Home 39 h later</td>
</tr>
</tbody>
</table>


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I.V. MgSO4 increases atrial contraction time and refractory times. It is used to treat atrial tachyarrhythmias and has a negative chronotropic and dromotropic effect.

A delivery rate of 2 g over 1 min in atrial fibrillation was without deleterious effect, and 4 g given over 10 min through a central venous line caused no decrease in mean arterial pressure in four normotensive volunteers; mean arterial pressure decreased from 130 to 116 mm Hg in six hypertensive volunteers.

Speedy reversal of bronchospasm reduces the otherwise increasing oedema of bronchial walls, prevents secretion retention, air trapping and exhaustion, and may avoid the need for intubation and ventilation.

In the absence of absolute guidelines for drug use in the management of unconscious or moribund asthmatics, the authors recommend immediate delivery of up to 40 mg kg⁻¹ of i.v. MgSO4 and i.v. salbutamol 15 μg kg⁻¹ over 10 min² or 5 μg kg⁻¹ boluses followed by an infusion of up to 5 μg kg⁻¹ min⁻¹. Improvement may be judged clinically by a ‘silent chest’ becoming wheezy, the patient becoming more alert, coughing, speaking, and by better blood gases.

A full description of the cases can be obtained from the authors.

Conflict of interest
None declared.

W. F. S. Sellers* I. Ahmad P. S. J. Bathke C. J. Brown T. Fernandez A. Barker
Stoke Mandeville and Hillingdon, UK
E-mail: wfssellers@doctors.org.uk

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Dying brain
Editor—Ancillary tests used to confirm clinically diagnosed brain death may substantially influence the time of diagnosis, as in the following case. A 48-yr-old woman was shot to the head and was admitted to the hospital at 01:00 a.m. with a Glasgow Coma Scale (GCS) of 1 (eye), 5 (motor), 1 (verbal); brain tissue oxygen tension measured with an intraparenchymal catheter was 12 mm Hg (normal range 15–35 mm Hg), and the intracranial pressure was 25 mm Hg.

At 07:00 a.m., GCS score was 3 with absent brainstem reflexes. Brain CT revealed massive cerebral oedema, the brain tissue oxygen tension was zero, and the intracranial pressure was 81 mm Hg with a cerebral perfusion pressure of zero. EEG showed a persistent low amplitude theta activity at the vertex and transcranial Doppler (TCD) a persistent cerebral blood flow in middle cerebral arteries (systolic flow velocity: 30 cm s⁻¹) (Fig. 1A).

At 09:00 a.m. of the next day, EEG was flat, and the 6 h observation period required by the Italian law to declare brain death was started; however, a TCD still showed a very low systolic flow velocity and a residual diastolic flow velocity (Fig. 1A).

At 03:00 p.m., deep coma, absent reflex motor response and brainstem reflexes, and flat EEG persisted. Brain death was declared. TCD showed a reverberating flow indicating cerebral circulatory arrest (Fig. 1c).

There is widespread acceptance of the concept of brain death in the Western hemisphere, and a fairly uniform agreement in Europe regarding the clinical criteria. There is, however, considerable variation in the use of additional confirmatory tests. These include flat EEG and determination of cerebral circulatory arrest by means of cerebral angiography, brain CT or MRI angiography, TCD, or cerebral scintigraphy. In the UK, only clinical criteria are used and brain death is defined as the complete, irreversible loss of brainstem function. In Sweden, cerebral angiography is the facultative ancillary test to confirm brain death; in Italy, EEG is mandatory, while cerebral angiography, brain CT angiography, TCD, or cerebral scintigraphy are all permitted methods to document cerebral circulatory arrest in children of <1 yr of age, or if a complete and reliable clinical evaluation is not possible.

There are three main messages from this case. First, brain death remains a valid concept because patients fulfilling the clinical criteria of brain death do not recover neurological function. Secondly, ancillary diagnostic tests may unnecessarily delay the diagnosis by hours or days compared with clinically established brain death: this patient would have been declared brain-dead 20 h earlier than actually occurred, if only clinical criteria for brainstem death had been used; conversely, she would have been declared brain-dead 26 h later, if cerebral circulatory arrest had been used. Ancillary tests should be used only when a specific part of the clinical examination cannot be performed or is deemed unreliable. However, in several European countries, they are mandatory by law, which may cause unwarranted delay and a further