

Fig 1 Sequential pain scores after thoracic epidural: patient 2.

advocated as a first-line therapy, but our experience suggests that it may have a place.

## Declaration of interest

None declared.

S. R. Ford\*

C. S. Swanevelder

P. M. Mills

Bury St Edmunds, UK

\*E-mail: srford@doctors.net.uk

- Harrington DT, Phillips B, Machan J, et al. Factors associated with survival following blunt chest trauma in older patients. *Arch Surg* 2010; **145**: 432–7
- Simon BJ, Cushman J, Barraco R, et al. Pain management guidelines for blunt thoracic trauma. *J Trauma* 2005; **59**: 1256–67
- British Pain Society. Numerical pain scores in English. Available from [www.britishpainsociety.org/pain\\_scales\\_eng.pdf](http://www.britishpainsociety.org/pain_scales_eng.pdf) (accessed 1 October 2011)
- Manion SC, Brennan TJ. Thoracic epidural analgesia and acute pain management. *Anesthesiology* 2011; **115**: 181–8

doi:10.1093/bja/aes116

## Lack of humidification may harm the patient during continuous positive airway pressure

Editor—We report a case of a patient receiving continuous positive airway pressure (CPAP) ventilation with a helmet to emphasize the need to illustrate the problem of humidification during ventilation with this interface.

A 78-yr-old man admitted to hospital 1 month earlier with a stroke was transferred to our intensive care unit for acute respiratory failure (AHRF). His arterial blood gas analysis showed severe hypoxia ( $P_{aO_2}:F_{IO_2}$  ratio 96) and respiratory alkalosis (pH of 7.523,  $P_{aCO_2}$  4.58 kPa); his respiratory rate (RR)

was >40 bpm, arterial pressure was 90/50 mm Hg, and the ECG showed atrial fibrillation with a frequency of 80 beats  $\text{min}^{-1}$ . Chest X-ray showed bilateral lung infiltrates while heart failure was excluded. The patient was still able to clear secretions with a weak cough reflex. Non-invasive ventilation with a CPAP set at 10 cm  $H_2O$  and with an  $F_{IO_2}$  of 60% was applied using a helmet (Ventukit, Starmed, Italy), and 30 min after starting, the  $P_{aO_2}:F_{IO_2}$  ratio increased to 130 and RR decreased to 25 bpm. Blood tests confirmed pneumonia and broad-spectrum antibiotic therapy was started. After the first 24 h of continuous application, any attempt to remove the helmet failed due to the quick occurrence of tachypnoea and desaturation until day 10 when, together with a marked amelioration of the chest X-ray, the  $P_{aO_2}:F_{IO_2}$  ratio consistently reached values >400 during CPAP. We therefore switched the patient to a Venturi mask with an  $F_{IO_2}$  of 35% for 24 h, keeping oxygen saturation ( $S_{aO_2}$ ) always >94%. On the following morning, his condition rapidly worsened with the development over a few minutes of gasping and loss of consciousness, so that tracheal intubation was mandatory. However, this was impossible to perform since visualization of the glottis was hampered by a foreign body. With a Magill forceps, we pulled up a solid collection macroscopically made by thick and sticky secretions and mucous membrane debris with a maximal length of 5 cm, then the oro-tracheal tube could be inserted. A bronchoscopy performed immediately later excluded any possibility of the mucous plug arising from the bronchial tree.

In AHRF, CPAP can achieve an improvement of gas exchange due to an alveolar recruitment and the use of a helmet was better than the mask with respect to comfort and duration of application.<sup>1</sup> There are no recommendations for the best humidification system and settings during non-invasive ventilation (NIV), despite there being a consensus that inadequate humidification may cause patient distress.<sup>2</sup> Clear indications about the applicability of common heated humidifier systems to the helmet are lacking and therefore urgently needed, especially when the patient requires long and uninterrupted periods of NIV until the resolution of the baseline disease, since, as we have shown, inadequate inspired gas humidification can lead to the generation of mucous plugs with potential upper airway occlusion.

## Declaration of interest

None declared.

A. Schreiber<sup>1</sup>

S. Nava<sup>2</sup>

P. Ceriana<sup>1</sup>

A. Carlucci<sup>1\*</sup>

<sup>1</sup>Pavia, Italy

<sup>2</sup>Bologna, Italy

\*E-mail: annalisa.carlucci@fsm.it

- Antonelli M, Conti G, Pelosi P, et al. New treatment of acute hypoxemic respiratory failure: non-invasive pressure support

ventilation delivered by helmet: a pilot controlled trial. *Crit Care Med* 2002; **30**: 602–8

2 Evans TW. International Consensus Conferences in Intensive Care Medicine: non-invasive positive pressure ventilation in acute respiratory failure. *Intensive Care Med* 2001; **27**: 166–78

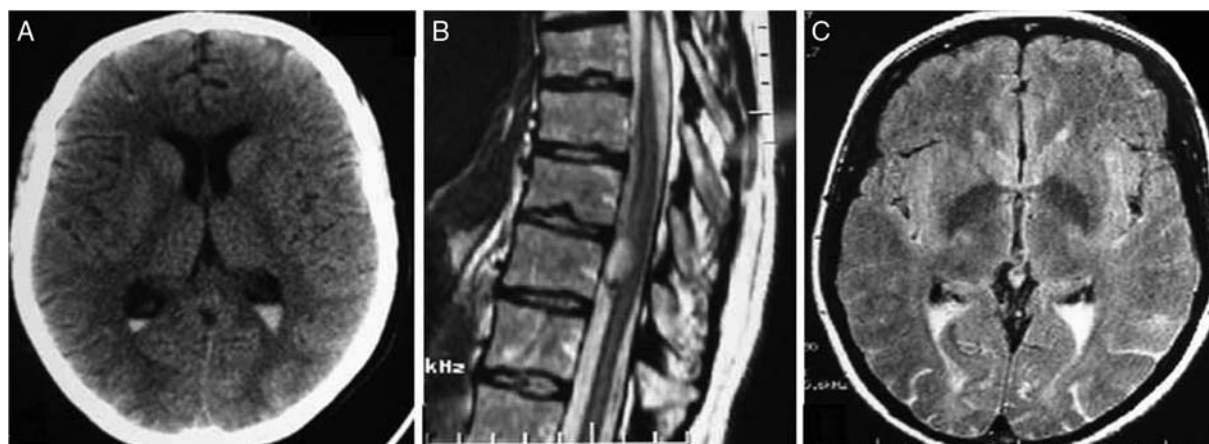
doi:10.1093/bja/aes117

### Intraspinal and intracranial subarachnoid haemorrhage with severe cerebral vasospasm after spinal anaesthesia for assisted delivery

Editor—Spinal anaesthesia is generally safe and effective. Intraspinal combined with cerebral subarachnoid haemorrhage has been rarely reported as a complication after lumbar spinal anaesthesia.<sup>1–3</sup> Simultaneous spinal haematoma and intracranial subarachnoid haemorrhage after spinal anaesthesia causing neurological symptoms, such as headache, stiff neck, nausea, and vomiting, has been described.<sup>1–3</sup> Acute intracranial and intraspinal subarachnoid haemorrhage after lumbar puncture is a rare but life-threatening complication, requiring urgent diagnosis and therapy. We report the management of a pregnant woman with a dorsal spinal subarachnoid haemorrhage and intracranial subarachnoid haemorrhage after lumbar spinal anaesthesia to perform an assisted delivery.

A 35-yr-old multigravida at 34 week gestation with antenatal history of arterial hypertension and polycystic kidney disease was admitted for forceps delivery. Spinal anaesthesia was achieved with bupivacaine 0.5% 2 ml after lumbar puncture in a single prick with a 25 G spinal needle at L3–4. The delivery of a healthy baby weighing 3.1 kg with the Apgar score of 8/10 was uneventful. Two days after delivery, the patient developed severe back pain, headache, vomiting,

and stiff neck. Despite analgesia and antiemetic treatment, symptoms increased in intensity, with paraplegia on fifth day postpartum. Cranial computed tomography showed mild hyperdensity in basilar cisterns, temporal sulci, and effacement of suprasellar cisterns supporting the diagnosis of evolving subarachnoid haemorrhage (Fig. 1A). Spinal magnetic resonance imaging (MRI) detected an intradural extramedullary lesion compressing and displacing the T10 spinal level and suggesting a late subacute subarachnoid bleeding between T5 and 10 with mass effect on the spinal cord (Fig. 1B). A complete T10–11 and partial T9 bilateral decompressive laminectomy was performed to remove subpial clots from a vascular malformation compatible with a spinal angioma which was completely removed. Neurological examination on admission to post-anaesthesia care unit (PACU) demonstrated a conscious and oriented patient with lower limb paralysis and hypoesthesia from T6. Antihypertensive therapy with labetalol for persistent arterial hypertension and i.v. nimodipine as prophylaxis for cerebral vasospasm were started. On the third day in PACU, headache and stiff neck increased along with nausea and vomiting. Cranial MRI showed subarachnoid haemorrhage and hemoventricle in both occipital poles (Fig. 1C). Conservative treatment with calcium antagonists and antihypertensive agents was maintained. An EMG showed a complete block in long somatosensory conduction at the spinal level. Spinal and cerebral arteriography showed no vascular malformations. On the sixth day, patient suffered a neurological deterioration with paralysis of the upper left limb and impaired consciousness [Glasgow coma scale (GCS) 5] proceeding to sedation, intubation, and assisted ventilation. With transcranial Doppler ultrasound, a severe vasospasm in both middle cerebral arteries (MCAs) appeared. Cerebral arteriography was performed with balloon angioplasty and intra-arterial verapamil, with a partial recovery of right MCA



**Fig 1** (A) Axial computed tomography scan of the head shows hyperdensity in some temporal sulci and effacement of suprasellar cistern. (B) Sagittal MRI of dorsal spine reveals a thin subarachnoid subacute haemorrhage at D10–5 levels and a larger intraspinal anterior haemorrhagic collection at the D10 level. (C) Axial MRI of the head shows subarachnoid haemorrhage in both convexities sulci, hemoventricle with a small bloody level in both occipital poles with no evidence of hydrocephalus or midline shift.