recovery room following elective urinary diversion and formation of ileal conduit. A thoracic epidural was sited at the T10/11 vertebral space using an 18 G Tuohy needle in the sitting position. Loss of resistance to saline was elicited at 5 cm and the catheter passed with ease. A test dose of levobupivacaine 0.5% (4 ml) was followed by a continuous infusion of levobupivacaine 0.125% with fentanyl 2 μg ml⁻¹ at 8 ml h⁻¹. General anaesthesia was then induced. During surgery, various degrees of Trendelenburg positioning with lateral tilt to facilitate operative access to deep pelvic structures were requested. On transfer to the recovery room the patient was noted to have developed a well-demarcated hemifacial flushing with sweating involving her right side. Sensory blockade involving T4–L1 dermatomes was identified with ethyl chloride. No other neurological deficit was noted. This colour change last approximately 8 h and resolved without consequence. The epidural continued for a further 24 h.

It seems likely that intraoperative positioning allowed sufficient rostral/unilateral spread of local anaesthetic agents to involve preganglionic fibres arising from the contralateral upper thoracic (T2–4) sympathetic branches. Absence of sympathetic oculomotor signs (ptosis or meiosis) suggests no involvement of T1. Patient positioning during prolonged surgery may be a risk factor during the use of regional anaesthesia.

S. M. Crawley
Dunfermline, Scotland
E-mail: sicrawley@doctors.org.uk

doi:10.1093/bja/ael039

Chronic postoperative pain and inguinal herniorrhaphy

Editor—Avasvang and Kehlet¹ highlight the complex nature of perioperative pain management. It serves to add to the growing body of evidence that alteration in the central and peripheral nervous system can lead to both acute and chronic pain outcomes following surgery. This alteration, which can be excitatory or inhibitory, is termed neuroplasticity. Their paper identifies a lack of preoperative pain assessment data available in the literature they reviewed. However, data do exist which suggest that preoperative pain is a significant predictive value of developing chronic pain following inguinal herniorrhaphy.² Indeed, the presence of chronic pain before surgery has been suggested to increase patient vulnerability to chronic pain complications after surgical nociception.³ This raises the possibility that neuroplasticity may contribute to the increased incidence of chronic postoperative pain in patients undergoing inguinal herniorrhaphy. Perhaps quantitative sensory testing, which is an accepted non-invasive tool to assess neuroplasticity in the perioperative clinical context,⁴ could be considered to give insight into the nociceptive neuroplasticity in these patients. This would offer the potential to develop evidence based perioperative pain management in the future. This combined with a comprehensive pain history may prove useful in the perioperative management of these patients.

D. Hegarty
Cork, Ireland
E-mail: dominichegarty@hotmail.com

⁴ Wilder-Smith OH, Tassonyi E, Senly C, et al. Surgical pain is followed not only by spinal sensitisation but also by supraspinal antinociception. Br J Anaesth 1996; 76: 816–21
doi:10.1093/bja/ael040

Use of the bispectral index (BIS) monitor to aid in the diagnosis of pseudoseizures

Editor—Pseudoseizures can be difficult to diagnose, especially in a patient with a history of epilepsy. Generalized tonic-clonic convulsions, followed by a postictal period of drowsiness can appear extremely convincing to even the trained eye. Anti-convulsant medication is often given and a critical care referral may be made if seizures continue. True epileptic seizure activity can only be definitively diagnosed by recording a 2-fold rise in serum prolactin levels for most clinicians, this is not possible. We therefore must rely on clinical acumen.

We recently were asked to review a 21-yr-old female, with a history of epilepsy who had presented to our district general hospital 10 h previously with seizures. She had >20 tonic-clonic seizures, lasting 5–10 min since admission to the acute medical assessment unit. Treatment had included lorazepam 4 mg and diazepam 10 mg i.v. She was about to receive a loading dose of phenytoin 15 mg kg⁻¹. Her Glasgow Coma Score (GCS) had been 3 for the previous 2–3 h, and although stable from a cardio-respiratory point of view, the referring physician felt, not unreasonably, that a critical care admission was warranted.