i.v. anaesthesia with short-acting agents such as propofol and remifentanil has been reported to be successfully used in a patient with narcolepsy who underwent cardiac surgery [6]. The authors of that report considered that perioperative monitoring of the patient's hypnotic state would have added valuable information, but this kind of monitor was not available in their department [6].

There are limited data about inhalational anaesthesia in narcoleptic patients while there are no reports of using BIS and brain oximetry as adjuncts to the anaesthetic management of patients with narcolepsy. There is one case report in Spanish of using inhalational anaesthesia in Gélineau syndrome and the authors reported postoperative complications due to anaesthesia [9].

We used sevoflurane to maintain general anaesthesia. Although we do not know the impact of narcolepsy on BIS monitoring, we used BIS values to titrate the volatile anaesthetic agent, hoping to decrease our patient's anaesthetic requirements. We maintained BIS values between 40 and 60 in order to avoid either awareness or prolonged recovery. The surgical procedure lasted 95 min. After discontinuation of general anaesthesia, the BIS values increased from 62 to 97 over a period of 14 min. The patient recovered and was extubated safely. There were no postoperative undesirable events related to general anaesthesia.

Brain oximetry was also used for continuous monitoring of the regional cerebral oxygen saturation. We observed no significant changes in the intraoperative rRSO₂ values as well as in the postoperative EEG when compared to the preoperative rRSO₂ values and EEG.

For the management of this narcoleptic patient, we avoided preoperative benzodiazepines, kept opioid analysesics to a minimum and used inhalational anaesthesia with sevoflurane. The anaesthetic technique was uneventful. Since non-invasive brain

monitoring is harmless and might provide some information, we used BIS and cerebral oximetry despite the lack of evidence-based data of this monitoring in narcolepsy.

C. Staikou, A. Tsaroucha, A. Fassoulaki
Department of Anaesthesia
Aretaieio Hospital
Medical School
University of Athens
Athens, Greece

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Intravenous phenytoin and percutaneous arterial cannulation: the purple-glove syndrome

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EDITOR:

Phenytoin is commonly associated with various adverse effects; rare ones include drug-induced

Correspondence to: Yatindra Kumar Batra, Department of Anaesthesia and Intensive Care, Post-graduate Institute of Medical Education & Research, Chandigarh 160012, India. E-mail: ykbatra@glide.net.in; Tel: +91 172 2715545; Fax: +91 172 2744401

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lupus, purple-glove syndrome (PGS), pigmentary alterations, IgA bullous dermatosis and generalized cutaneous eruptions [1]. We here report a case of distal limb ischaemia following severe soft tissue injury (PGS) on intravenous (i.v.) administration of phenytoin in the same limb having arterial cannulation.

A 38-yr-old male (82 kg), with a medical history significant for hypertension and epilepsy, presented

with generalized tonic-clonic status epilepticus, which necessitated securing of the airway, initiation of ventilatory support and transfer to ICU. Phenytoin 15 mg kg⁻¹, followed by 100 mg 8-hourly, was administered through an 18-G IV cannula on the ventral aspect of the left forearm (pre-existing thrombophlebitis precluded use of the right arm). Labile blood pressure required invasive monitoring and the left radial artery was cannulated after determining the adequacy of collateral flow to the hand using a pulse oximeter. Serum levels of phenytoin were in the therapeutic range. Next morning, a livid swelling appeared around the peripheral i.v. infusion site. Within the next 6-8 h, oedema and dark bluish discolouration increased at the site, which spread both proximally and distally to the hand. Although the arterial line functioned well at this time, suspecting vascular compromise both arterial line and i.v. cannula were removed. Over the next few hours, slight blistering appeared with cyanosis and coldness of the thumb and index finger. Physical examination revealed a weak left radial and ulnar artery pulse. Doppler examination revealed a high resistance to flow in both radial and ulnar arteries with a small thrombus partially obstructing the left radial artery. The limb was elevated and anti-inflammatory drugs, antibiotics and heparin were started. A left continuous brachial plexus block via the axillary approach was performed with 0.5% bupivacaine. Swelling and discolouration improved gradually and disappeared by the 12th day with no untoward sequel. Meanwhile, the patient was transferred to a phenytoin equivalent dose of fosphenytoin and recovery was uneventful.

PGS is a delayed soft-tissue injury characterized by oedema, discolouration and pain distal to the site of phenytoin administration through small dorsal hand veins [2]. Although extravasation of highly alkaline drug has been implicated, this may be seen even without apparent extravasation. It has been suggested that highly alkaline phenytoin or propylene glycol and ethanol added to phenytoin solution may induce vasoconstriction and thrombosis or endothelial damage leading to the seepage of phenytoin into the interstitial tissue. Alternatively, a micro-tear in vessels by i.v. cannulation may be responsible [3]. PGS is distinguished clinically from extravasation by the presence of its characteristic purple-blue discolouration and progression of the condition after discontinuation of phenytoin infusion. It is also distinguished from infection and cellulitis by its rapid onset, unique discolouration and lack of purulent discharge or fever. Patients younger than 7 yr or older than 60 yr, those with pre-existing vascular disease and those who are unconscious are at increased risk. Severe cases may lead to arterial insufficiency and compartment syndrome, which, compounded with arterial cannulation, could be catastrophic [4]. The presence of thrombosis plays a role in the initial pathogenesis [2]. PGS may also follow oral administration of phenytoin [5]. Hence, substitution to an alternate antiepileptic is imperative, particularly when the pathological changes have already started. Fosphenytoin may be used as an alternate, as the solubility in aqueous solutions eliminates the need for propylene glycol and ethanol and the formulation is less alkaline [6].

In our patient, concomitant phenytoin administration with radial artery cannulation may have acted synergistically resulting in compartment syndrome with arterial occlusion and thrombosis over a short span of time. Therefore, we found it prudent to remove both the vascular lines from the left upper limb. Fortunately, our patient responded well to conservative therapy and had an uneventful recovery. Thus, our case cautions against such a combination.

R. P. Mahajan, Y. K. Batra, S. Rajeev Department of Anaesthesia and Intensive Care Post-graduate Institute of Medical Education & Research Chandigarh, India

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