

The first four recommendations suggest an initial dose of 0.5 mg applied as four drops of a 0.25% solution for ENT procedures. This dose is the product literature i.v. dose for hypotension and assumes 100% absorption. In children the recommended dose was 20 $\mu\text{g kg}^{-1}$. In clinical practice, only the minimal amount required for preparation of the surgical field should be given, vital signs monitored and the anaesthetist informed [3].

Although phenylephrine is predominantly a selective agonist, at very high doses β activation does occur. Our patient demonstrated a marked bradycardia in response to hypertension suggesting he had mainly an α -1-mediated vasoconstriction and a baroreceptor-mediated bradycardia without evidence of β activation. It is common practice to deepen anaesthesia in response to hypertension, which will reduce peripheral vascular resistance but may also impair cardiac performance that may already be compromised by a high afterload. There is insufficient evidence to either recommend or discourage this management [3].

The use of glycopyrrolate did appear to prevent further bradycardia and may also have prevented further complications of using labetalol in subsequent management. Labetalol is a mixed α and β antagonist although the β effects are about seven times stronger than the α effects. It is readily available and is a drug most anaesthetists have experience using. Although used in the successful management of other cases of phenylephrine toxicity [4], the Phenylephrine Advisory Committee highlighted a pattern of management in cases who were pre-treated with anticholinergics and did not demonstrate a bradycardic response to phenylephrine toxicity. The subsequent use of β antagonists may result in an inability to compensate for the increased afterload and they are thus associated with pulmonary oedema. Labetalol was used in all three cases that resulted in cardiac arrest [3]. The report went on to recommend that severe hypertension or asso-

ciated complications (pulmonary oedema or ECG changes) should be treated with direct vasodilators or α antagonists such as phentolamine.

The patient in our case made an uncomplicated recovery, however the case highlights the need for good communication between surgeon and anaesthetist, and the responsibilities of phenylephrine use and dosage. With drug companies ceasing production of drugs such as methoxamine, the use of alternative α agonists such as phenylephrine has increased. The highly concentrated presentation of phenylephrine means that errors made in preparation of i.v. doses may have significant consequences in terms of toxicity, in addition to its topical use as in this case. It is therefore important to be aware of dilution protocols and the correct management of overdose.

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Anaesthesia for Worster-Drought syndrome

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EDITOR:

A 29-yr-old, 100 kg female (body mass index 38) presented for day case bilateral hip injection of

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steroids. At a preoperative assessment clinic, she had been noted to have moderate learning difficulties (IQ 65), eat a special thickened diet due to 'swallowing problems' and have Worster-Drought syndrome (WDS) but the anaesthetic implications of her syndrome had not been appreciated. She had received general anaesthesia as an 11-yr-old, for surgical correction of bilateral slipped femoral

epiphyses, which had led to bilateral hip osteoarthritis. Preoperative examination revealed an excess of submandibular soft tissue, Mallampati grade 3 mouth opening and a thyromental distance of 6 cm.

She was adamant in wanting general anaesthesia for the procedure, but given her risk of oesophageal reflux, this would have mandated intubation, and difficult airway equipment (specifically fibre-optic bronchoscopy) was not available at the Independent Sector Treatment Centre to which she had presented. Fortunately, she gave competent consent to sedation, once the nature, purpose, risks and consequences of sedation had been explained to her.

After placement of standard monitoring equipment, a 20-G cannula was inserted into the dorsum of her left hand; 5 L min⁻¹ of supplemental oxygen was administered by face mask. End-tidal carbon dioxide monitoring was used to assess respiratory rate. In all, 50 µg fentanyl, followed by 50 mg boluses of propofol (200 mg in total) were administered to achieve sedation. The patient remained self-ventilating and rousable only to gentle physical stimuli. In all, 5 mL 0.5% bupivacaine mixed with 40 mg methylprednisolone were injected into each hip by a consultant orthopaedic surgeon (using a 24-G Whitacre spinal needle) prior to bilateral hip manipulation. Recovery was uneventful. She was discharged from hospital 3 h after the procedure.

WDS (congenital supra-bulbar paresis) is a form of cerebral palsy that was first described in 1956 [1], occurring as a result of congenital (heterogeneous, predominantly X-linked), bilateral perisylvian cortical dysfunction [2,3]. It is not an uncommon form of cerebral palsy, but it is underdiagnosed [4]. WDS displays variable expression, but is characterized by supra-bulbar paresis (100% prevalence, causing disordered oesophageal motility, speech difficulty, drooling, gastro-oesophageal reflux (41%) and aspiration), mild four limb pyramidal

tetraplegia (91%), cognitive (81%) and behavioural (41%) impairments, and epilepsy (28%).

Surgery may be required in childhood to insert grommets or gastrostomy tubes, or to correct congenital defects such as cleft palate or contractures. A multidisciplinary approach to perioperative care is advocated. Preoperative communication may be difficult, although adult patients with WDS should not be assumed to be incompetent to give consent for treatment [5]. Preoperative oral clonidine (5 µg kg⁻¹) may be beneficial as a sedative and antisialogogue, and antacid therapy should be considered. Without additional sedation, regional anaesthesia may be compromised by behavioural difficulties. General anaesthesia necessitates tracheal intubation, due to the high risk of gastro-oesophageal reflux; this procedure may be complicated by palatal abnormalities (4%), dental problems (20%), jaw contractures (9%) or micrognathia (1.5–15%). Extubation should be performed with the patient awake, and in the left lateral or semi-recumbent positions.

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Intralipid infusion in rabbit asphyxial pulseless electrical activity: a pilot study

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EDITOR:

Augmenting conventional resuscitative efforts with infusion of lipid emulsions has resulted in successful

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resuscitation from intractable local anaesthetic-induced cardiac arrest in a number of recent case reports. These follow animal studies demonstrating efficacy for lipid infusion in local anaesthetic-induced cardio-toxicity [1] and other lipid-soluble drug toxidromes [2]. Two mechanisms of action have been forwarded as the basis for the observed beneficial effects of lipid infusion. Weinberg's 'lipid