rhythm (HR 70-80 beats-min⁻¹) with a stable BP (110–140 mmHg systolic), and he did not receive any specific treatment. Two hours after IC administration, residual neuromuscular block was reversed with neostigmine 3 mg iv and glycopyrrolate 0.6 mg iv, and the patient's trachea was extubated when he was awake. At this point, the patient became tachycardic (HR in the 120s beats min⁻¹) and he developed deeply inverted T waves (not further characterized) without concurrent angina. He was treated with esmolol 40 mg iv and metoprolol 5 mg iv in divided doses. A cardiologist was consulted postoperatively, and the patient was admitted to a cardiac telemetry unit. Myocardial infarction was ruled out by three sets of cardiac enzymes over the next 24 hr. Subsequently, a transthoracic echocardiogram revealed an essentially normal heart with an ejection fraction of 66% and no segmental wall motion abnormalities. A pharmacological cardiac stress test (adenosine Tc-99m sestamibi) was negative for inducible ischemia. On postoperative day four, he was discharged home in good condition.

In both cases described, a second-degree AVB appeared immediately after the administration of IC, suggestive of a direct causative relation. Other possible etiologies are less likely: (1) Vagal reflexes induced by peritoneal traction should have resolved after the surgeons were notified and had ceased manipulation; (2) A high spinal level is also improbable in case 1, given the documented T6 sensory level and the fact that the event occurred 72 min after the subarachnoid block was placed; (3) Also unlikely is a baroreceptor reflex (although hypertension is commonly seen after administration of IC,² no increase in BP was recorded, in either case, on the automated anesthesia records prior to the development of AVB). Moreover, in the second case, the AVB occurred in the absence of sinus bradycardia.³

In conclusion, this report aims to alert to the possibility that intraoperative administration of IC may induce the development of AVB. Although the events were relatively inconsequential in the two cases presented, serious complications may occur if progression to complete AVB occurs, or, as in the first case, as a consequence of tachycardia induced by its pharmacological treatment.

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Peripheral nerve blockade in a patient with Charcot-Marie-Tooth disease

To the Editor:

The use of peripheral nerve blockade (PNB) in patients with Charcot-Marie-Tooth disease (CMT) is controversial because of the potentially toxic effects of local anesthetics on peripheral nerves. In CMT, nerves lose their protective myelin sheaths and, while there have been reports of complications from neuraxial anesthesia, the use of PNB in CMT patients has not been studied extensively and should only be considered in such patients where the potential benefits outweigh the potential risks.

We recently managed a 59-yr-old woman with CMT who was scheduled as an outpatient for arthroscopic rotator cuff repair and subacromial decompression. The patient's medical history included obstructive sleep apnea (OSA), chronic obstructive pulmonary disease (COPD), tobacco abuse, morbid obesity (body mass index 53.5 kg \cdot m⁻²), diabetes mellitus type 2, hypertension, stable angina, premature ventricular contractions, hypothyroidism, fibromyalgia, and an allergy to morphine. A preoperative adenosine stress test showed probable ischemia with a hyperdynamic left ventricle (ejection fraction of 80%), and cardiac angiography showed nonobstructive coronary artery disease. The anesthetic plan included PNB for intraoperative and postoperative analgesia, coupled with endotracheal intubation for airway control. A brachial plexus block was placed under ultrasound guidance using 30 mL of 0.5% bupivacaine. To minimize impairment of the phrenic nerve, the supraclavicular approach was chosen. After sensory and motor block of the limb had been established, general anesthesia was induced with intravenous propofol supplemented by inhalation of desflurane. The patient's trachea was intubated without the use of neuromuscular blocking drugs (NMBDs). Anesthesia was maintained with desflurane in oxygen, and her trachea was extubated at the end of the procedure without incident. Save for a prolonged duration of analgesia lasting approximately 30 hr, telephone follow-up revealed that the patient's recovery was unremarkable with eventual complete resolution.

Traditionally, due to concern about local anesthetic toxicity on the demyelinated nerve, anesthesia options for patients with CMT have been limited to general anesthesia. However, a recent retrospective review,¹ which included patients with multiple sclerosis, did not demonstrate an increase in adverse outcomes. If the disease is progressing in CMT patients, the use of depolarizing NMBDs may cause hyperkalemia, and CMT patients may be sensitive to non-depolarizing agents; however, Antognini² reports the safe use of succinylcholine in CMT patients. Other authors report sensitivity to non-depolarizing NMBDs.3 Patients with OSA can be sensitive to opiods used for postoperative analgesia, as can CMT patients.² Minimizing the use of opiods is reasonable in patients with significant COPD and OSA. Interestingly, the duration of the block (about 30 hr by patient report) was longer than expected. Other authors have reported prolonged duration of epidural anesthesia, but not spinal, in patients with CMT.⁴ Also, there were no resulting complications from using general anesthesia in our patient. However, according to Reah et al.,⁵ if general anesthesia is used in patients with CMT, postoperative ventilation is likely to be required. This requirement could possibly be secondary to the fact that Reah et al. patients were pregnant, a condition known to exacerbate CMT.

The use of a peripheral nerve block, in this patient with CMT, OSA, and morbid obesity, spared excessive postoperative use of opioid analgesics. Also, providing optimal analgesia enhanced our ability to manage her successfully as an outpatient. Review of the available literature failed to show a link between CMT and adverse neurologic outcomes, albeit with a very small number of patients reported. Lacking evidence to the contrary, we believe that peripheral nerve blocks should be considered as an option in the anesthetic care of patients with CMT.

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The Rusch Flexi-Slip stylet for ProSealTM laryngeal mask airway insertion

To the Editor:

The ProSealTM laryngeal mask airway (PLMA; Laryngeal Mask Company North America, San Diego, CA, USA) consists of a flexible airway tube and a soft laryngeal mask, and is used to accommodate variations in airway anatomy, while minimizing the risk of oropharyngeal trauma. The manufacturer recommends inserting the PLMA with an introducer tool to facilitate insertion. Due to the lack of an internal supporting structure, the soft laryngeal mask may tend to fail, or insertion may be impeded as a result of impaction of the PLMA at the back of the mouth, or folding over of the distal cuff. To deal with these problems, we have adapted a new technique using a flexible and soft-tip stylet (Rusch Flexi-Slip stylet no. 503000-4.3/5.6; Willy Rusch AG, Germany) and inserting it into the drain tube of the PLMA.

The Rusch Flexi-Slip stylet is made of a malleable coated wire that retains its shape to facilitate endotracheal intubation, and it has a soft atraumatic tip that helps to prevent tissue damage during insertion. To facilitate insertion of the PLMA, the stylet is inserted into the proximal end of the drain tube of the PLMA and advanced until the tip is within 1 cm of the distal end of the drain tube. The proximal end of the stylet is then bent backwards 180° to prevent the stylet from moving forward over the distal end of the drain tube. Finally, the PLMA/stylet is bent to a 90° angle