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Atrioventricular block induced by indigo carmine

To the Editor:
Managing intraoperative adverse reactions to non-anesthetic medications remains an ongoing challenge for anesthesiologists.[1] I recently encountered two cases in which intravenous administration of indigo carmine (IC) was immediately followed by the development of second degree atrioventricular block (AVB) Mobitz type II. The two patients provided written informed consent for the publication of their case histories.

In the first case, a 49-yr-old woman, ASA physical status I with urinary incontinence, underwent a cystoscopy and anterior colporaphy under spinal anesthesia. She had no significant medical history and no known drug allergies. The preoperative electrocardiogram (EKG) showed normal sinus rhythm at 64 beats·min^{-1} with a PR interval of 148 msec. Following application of standard monitors, bupivacaine 12 mg was administered intrathecally. Seventy-two minutes later, before closing the anterior vaginal wall, IC 40 mg *iv* was administered at the surgeon's request, to rule out ureteral injury. Shortly afterwards, the patient developed sinus bradycardia [heart rate (HR) decreased quickly from the 72 beats·min^{-1} to 32 beats·min^{-1}], an increase in the PR interval (to 240–280 msec), and frequent non-conducted P waves (i.e., second degree AVB type II). Her blood pressure (BP) was maintained in the 100s/60s mmHg (systolic/diastolic). She complained of acute anxiety and shortness of breath, and her spinal sensory level was T6. Concerned by the symptoms and the progressive nature of the bradycardia, the anesthesiology resident administered glycopyrrolate 0.6 mg *iv* (in 0.2 mg doses) and ephedrine 25 mg *iv*. The patient's HR gradually increased (peaking in the 120s beats·min^{-1}) with a PR interval of 160 msec and 1:1 conduction. However, her BP also increased to 180/110 mmHg, and she developed diffuse ST segment depressions and T wave inversions. The patient was then treated with esmolol 80 mg *iv* (in divided doses), midazolam 1 mg *iv*, and propofol 20 mg *iv*. Her HR decreased to the 90s, and the ST depression diminished. In the recovery room, she was stable and asymptomatic; she had no recollection of the event, and the EKG returned to the normal preoperative pattern. A cardiology consultant considered the AVB to be an idiosyncratic reaction to IC, and this finding was entered into the patient's chart as a drug reaction/allergy. The consultant did not consider the ST-T changes as indicative of cardiac ischemia and did not pursue additional testing.

The second case involved a 64-yr-old male, ASA physical status III, who underwent a laparotomy and retropubic prostatectomy for adenocarcinoma under general endotracheal anesthesia. The patient's medical conditions included coronary artery disease, hypertension, hyperlipidemia, gastroesophageal reflux disease, and depression, and he had no known drug allergies. He underwent a coronary angioplasty two years prior (for angina), and he was now asymptomatic and exercising regularly. His daily medication regimen included amlodipine 5 mg, enalapril 40 mg, atorvastatin 20 mg, rantidine 150 mg, and sertraline 50 mg (all *po*). His preoperative EKG showed first degree AVB (PR interval of 214 msec) and inverted T waves in lead aVL and leads V2–V5. After induction with propofol 200 mg, fentanyl 150 μg, and cisatracurium 12 mg, anesthesia was managed uneventfully using a combination of 4% desflurane, 60% nitrous oxide and oxygen, morphine 8 mg *iv*, and 8 mg additional cisatracurium. Ten minutes after administration of IC 40 mg *iv*, the patient developed second degree AVB Mobitz type II. He remained in normal sinus

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·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 block-