that resulted from the bilateral laminectomies. Normally, a post–dural puncture headache results from CSF leakage from the dural sac into the epidural space.[14] Stopping the CSF leak (*e.g.*, with an epidural blood patch) will terminate the headache. That a single deep figure-of-eight stitch was sufficient in this case to stop the leak at the skin and to allow a rapid improvement in the headache suggests that the dura was adherent to the scar with obliteration of the epidural space. Otherwise, CSF would be expected to continue to drain into the epidural space. The direct course of the needle would allow the scarring to splint open the tract from the dura to the skin. The usual movement of the tissue planes that disrupts the needle tract upon straightening of the normal back following catheter placement would be absent in this patient due to the rigidity of his lumbar spine. Other authors[12,15] have implicated steroid deposition along the needle tract during epidural steroid injections as a causative factor in the fistula formation. Systemic steroid therapy may delay wound healing but is less likely to contribute to immediate fistula formation, as in this case. In any event, it was important to close the fistula as quickly as possible to avoid the added risk of infection.[6,8] Prophylactic antibiotics were not recommended by the neurosurgeon.

Despite this unusual complication, CSA remains an option for regional anesthesia in patients who have undergone prior lumbar laminectomy. If spinal cutaneous fistula develops despite this precaution, early recognition and treatment are important.

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Atypical Response to Scopolamine in a Patient with Type IV Hereditary Sensory and Autonomic Neuropathy

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Hereditary sensory and autonomic neuropathies (HSAN) comprise a spectrum of disorders associated with congenital insensitivity to pain and are manifested by selective degeneration of peripheral sensory and autonomic neurons.[1] By definition, all patients with HSAN have some degree of sensory and autonomic neuropathies. Types I

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and II present at later stages of life and manifest only acral neurologic symptoms. These patients may present certain dilemmas concerning new-onset sensory deficits after regional anesthesia. They do not, however, present with autonomic neuropathy peculiar to types III and IV. HSAN type III, also known as Riley-Day syndrome or familial dysautonomia, is associated with hyperhidrosis and is to be distinguished from HSAN type IV, or familial sensory neuropathy with anhidrosis. A previous report refers to the use of atropine in type III patients only to note that chronotropic responses can be normal or absent.[2]

We describe a patient with HSAN type IV in whom reproducible, exaggerated tachycardia and hypertension were induced after the intravenous administration of scopolamine.

Case Report

A 23-yr-old, 63-kg woman with congenital HSAN type IV and chronic right-hip osteomyelitis was admitted for debridement of soft tissue, exchange of antibiotic beads, and excision of heterotopic ossification. As a child, she was noted to have unilateral facial flushing with upper respiratory infections. She was also insensitive to painful stimuli, for example, burning her fingers or biting her fingernails to the point of bleeding. Other significant physical findings included anhidrosis, decreased lacrimation, absence of tongue papillae, dystrophic nails (secondary to chronic biting), and hyperkeratotic skin. There was no prior history of atropine or scopolamine administration.

On this admission, neurologic examination revealed a generalized diminished sensitivity to pin prick. Baseline blood pressure and pulse rate were 90/60 mmHg and 75 beats · min^{-1}, respectively. Nerve conduction studies showed evoked sensory nerve amplitude reduction without slowing. Motor nerves and F waves were normal. Galvanic skin potentials were not responsive to various stimuli. Heart rate variability during deep breathing was normal. These findings are consistent with sensory small fiber and autonomic neuropathy. An intradermal histamine challenge failed to produce an erythematous flare. Hemoglobin was 11.5 g/dl.

No preoperative medication was administered. Initial heart rate, blood pressure, and skin temperature recordings were 75 beats · min^{-1}, 100/60 mmHg, and 36.3° C, respectively. Fentanyl 100 μg, droperidol 0.625 mg, and *d*-tubocurarine 3 mg were followed by thiopental 250 mg, succinylcholine 100 mg, and tracheal intubation. Anesthesia was maintained with nitrous oxide 60%, isoflurane (0.3–0.5%), and vecuronium for muscle relaxation. After orotracheal intubation, systolic blood pressure increased to 110 mmHg, and the pulse rate remained 75 beats · min^{-1}. Before incision, arterial blood pressure and pulse gradually decreased to 85/50 mmHg and 65 beats · min^{-1}, respectively.

Both arterial blood pressure and pulse remained unchanged during incision and throughout 1 h of the procedure until massive hemorrhage occurred and systolic blood pressure decreased to 60 mmHg without any alteration in pulse rate. All anesthetic agents were discontinued and a left radial arterial catheter inserted. Fluid administration and a phenylephrine infusion (0.004% at a maximum of 0.5 mg · min^{-1}) failed to resolve the hypotension. Scopolamine 0.1 mg was then administered intravenously as an amnestic agent. Sixty seconds later, heart rate increased to 180 beats · min^{-1} and blood pressure to 150/110 mmHg and remained increased for 20 min despite increasing concentrations of isoflurane (maximum 1.5%). During the ensuing few minutes, systolic blood pressure returned to 60 mmHg and heart rate to 80 beats · min^{-1}. All anesthetic agents were again discontinued. As the etiology of the hemodynamic response was unclear and believed to be due to light anesthesia, scopolamine 0.2 mg was injected intravenously. The identical response occurred (heart rate 180 beats · min^{-1} and blood pressure 150/110 mmHg) with a duration of 25 min. At the end of the procedure, the trachea was extubated. The patient reported no intraoperative recall, bad dreams, or hallucinations.

In the following weeks, the patient returned for several uneventful anesthetics, during each of which the postinduction blood pressure remained approximately 80/60 mmHg and the pulse rate 70 beats · min^{-1}. On each occasion, a neostigmine/glycopyrrolate combination was administered for reversal of neuromuscular relaxation. Once, during emergence, pulse rate increased to 150–160 beats · min^{-1}; otherwise, no significant hemodynamic response was observed.

Discussion

Our case is the first report of a patient with HSAN who developed marked hypertension and tachycardia in response to intravenous scopolamine. It is difficult to ascertain the etiology of this reaction, although we theorize that a combination of mechanisms was involved. Light anesthesia associated with surgical stimulation or tracheal intubation may produce significant increases in pulse rate and blood pressure. This is an unlikely explanation in view of the temporal relationship to intravenous scopolamine injection on two separate occasions; the duration of cardiovascular effects observed is consistent with pharmacologic studies[3] and the insensitivity of this patient to pain.

As reported by Yamaguchi *et al.*, general anesthesia itself may influence the pharmacologic properties of belladonna alkaloid agents.[4] Other studies further indicate that tachycardia and dysrhythmia are more severe when anticholinergic drugs are administered in the presence of high sympathetic output.[5] An additional possibility is catecholamine release associated with the central anticholinergic syndrome and central nervous system excitation. Our patient had no recall of unpleasant experience during the surgical procedure. Physostigmine was not administered.

Scopolamine has both central and peripheral sites of action. In healthy volunteers, the hemodynamic response observed after intravenous scopolamine administration consists of a mean maximum elevation in pulse rate of 17 beats · min^{-1} in contrast to the approximate increase of 105 beats · min^{-1} that we observed.[6] The tachycardia is normally a result of the peripheral antagonism of acetylcholine. This antimuscarinic action occurs at the parasympathetic postganglionic cholinergic nerve endings. Studies have demonstrated that a parasympathetic–sympathetic interaction exists in the myocardium and peripheral circulation.[7–9] Results from animal experimentation[10] indicate that administration of muscarinic agents diminishes the release of noradrenaline during sympathetic stimulation. Intravenous atropine appears to attenuate this

inhibition of noradrenaline release during vagal stimulation in organs with dual antagonistic innervation.

Smith *et al.* suggested that patients with familial dysautonomia have a reduced release of sympathetic transmitters.[10] He discovered that these individuals display an exaggerated response to noradrenaline infusions, suggesting possible increased adrenergic receptor density or hypersensitivity as opposed to excessive catecholamine release.[11] Further investigations of three dysautonomic patients indicated significantly elevated catecholamine concentrations in adrenal gland tissue obtained less than 24 h *post mortem*.[12] It is possible that scopolamine administered to our dysautonomic patient may have blocked the parasympathetic modulation of noradrenaline release at the sympathetic nerve terminal, producing a greater than usual catecholamine release. The resulting hypertension and tachycardia may have been a consequence of either sympathetic denervation hypersensitivity, the liberation of excessively produced catecholamines, or a combination of both.

In conclusion, we describe a case of a patient with HSAN type IV who developed an exaggerated hemodynamic response to intravenous scopolamine. We suggest that this agent as well as other belladonna alkaloids be administered with caution to patients with HSAN type IV and possibly to patients with Riley-Day Syndrome.

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Anesthesia for an Unsuspected Lambert-Eaton Myasthenic Syndrome with Autoantibodies and Occult Small Cell Lung Carcinoma

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Lambert-Eaton myasthenic syndrome (LES) is a disorder of neuromuscular transmission first recognized clinically in association with lung cancer. This report describes a patient with rectal adenocarcinoma presenting for low anterior resection who reacted to succinylcholine and/or *d*-tubocurarine with undue prolongation of neu-

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