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needle positioning is not usually necessary for diagnostic stellate blocks. However, for difficult cases or for neurolytic injections, roentgenographic control appears to be mandatory to confirm correct needle placement and to prevent damage to adjacent important structures.⁴

Iatrogenic traumatic chylothorax is generally considered to warrant a trial of conservative treatment for at least 2 to 4 weeks before resorting to surgical ligation of the duct. 5,6 Fortunately our patient responded well to simple pleural drainage within this period and it was not necessary to consider further measures. After the first 24 hours drainage was not large enough to require parenteral triglyceride, fat soluble vitamins, or protein replacement of the chylous losses and the patient was simply maintained on a normal oral diet. Chyle may reaccumulate in the pleural cavity several weeks after the

chest drains have been removed, so careful and regular follow-up for some months after apparently successful resolution of the chylothorax is required.^{5,6}

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Pourfour Du Petit Syndrome—Hypersympathetic Dysfunctional State Following a Direct Non-penetrating Injury to the Cervical Sympathetic Chain and Brachial Plexus

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Blunt trauma to the brachial plexus can produce reflex sympathetic dystrophy with increased sympathetic activity in the affected extremity. There are no reports, however, documenting sympathetic hyperactivity unaccompanied by pain in an anatomic region of the body not supplied by the injured plexus. In this case, a hypersym-

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pathetic dysfunctional state developed following a direct nonpenetrating injury to the cervical sympathetic chain and brachial plexus. What was unusual is that sympathetic activity to the eye and face was also affected on the ipsilateral side.

Pourfour Du Petit, a French physician during the Napoleanic wars, was the first person to describe the functions of the cervical sympathetic chain. His treatment of many neck injuries secondary to slash wounds from swords provided a unique opportunity to study direct injuries to the cervical sympathetic chain. He was also the first physician to note the signs of increased sympathetic activity in the eyes and upper extremity and relate these to the injuries of the cervical sympathetic chain, and hence the name Pourfour Du Petit syndrome is given to this patient's condition.

REPORT OF A CASE

A 38-year-old woman sustained direct blunt trauma to the left side of the neck and shoulder when hit by the main-sail boom of a sailboat. The patient immediately experienced left upper extremity (LUE) and shoulder pain with decreased range of motion six hours postinjury. Twenty-four hours postinjury physical examination revealed a cold left hand with no evidence of vascular injury. Her peripheral vascular

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pulsations were strong and equal bilaterally in both the upper extremities. A cervical spine roentgenogram was negative. The presumptive diagnosis was that the pain was due to sympathetic dysfunction following the injury. Aspirin was prescribed for pain.

In our Pain Clinic, 48 hours postinjury, she was observed carrying her left hand in the pocket of her coat. The patient denied previous injury to her LUE. There was no history of a sweating disorder, hyperesthesia, swelling, or exacerbation of pain associated with an increase in emotional stimulation. She denied any other medical illness.

The physical examination revealed pupils which were unequal in size; the left pupil was 4.5 mm and the right pupil was 3.5 mm. The left sclera was pale. Both pupils were equally reactive to light and accommodation. The palpebral fissure was larger on the left when compared to the right. The left side of the face was pale and cool (left side face 33°C, right side 34.4°C). There was no swelling or cyanosis noted in the neck. Moderate muscle spasm was noted in the trapezius, supraspinatus, biceps, and triceps muscles on the left side. There were no trigger areas in these muscles. The carotid, brachial, and radial pulsations were equal and normal bilaterally. The LUE was painful and stiff upon passive movement and had a decreased range of motion. Arterial blood pressure was 120/80 torr in the brachial artery on both sides. Neurological examination was otherwise within normal limits. There were no significant differences in the sensory examination to the touch, pinprick, and temperature discrimination in both upper extremities. A diagnostic stellate ganglion block was scheduled.

Following a negative placebo response, a stellate ganglion block was performed via the anterior approach2 with 10 ml 0.25 per cent bupivacaine. The patient had complete and dramatic relief of pain 5 min later with warmth and vasodilatation in the LUE and the appearance of a left sided Horner's syndrome. However, the block failed to resolve her muscle spasm completely in the neck muscles. Therefore, approximately 15 min later, 30 ml 0.25 per cent bupivacaine was injected into the cervical portion of the brachial plexus via the interscalene approach.3 This block relieved the spasm considerably and good range of motion was noted 10 min after the block sensory changes, but no motor block resulted in the LUE.

She received a series of three more stellate ganglion blocks, one block daily for three subsequent days. Baseline temperature in the affected limb during each subsequent block was higher than the baseline temperature obtained during the previous block. Signs of increased circulation in the LUE and Horner's syndrome were noted following each block. Furthermore, each block relieved the pain lasting from 10-20 hours. Following each block her condition improved enough that she was able to complete her work day.

One week after the last block, her left hand was warm and there was no temperature difference between the left and the right side of her face. Her pupils were bilaterally equal. She did not require any pain medications. Six weeks postinjury, it was noted that a slight downward traction on the LUE resulted in the immediate but transient dilatation of the left pupil, although all other symptoms and signs of sympathetic hyperactivity had resolved. The affected pupil reacted normally to the light and accommodation. Six months after the last block, the patient was free of pain, gainfully employed and the pupillary condition had resolved.

DISCUSSION

Blunt trauma to the soft tissues of the left side of the neck followed by aching pain and cold sensation in the LUE, the characteristic posturing with protection of the painful part, decreased range of motion of the involved extremity, and excellent response to the sympathetic blocks suggest reflex sympathetic dystrophy. 4 The impact of a heavy object on one side of the neck in addition to producing crush injury might also cause stretch injury to the neural elements of the brachial plexus and the cervical sympathetic chain on the ipsilateral side. The sudden lateral bending of the head to the contralateral side may have been the mechanism of the stretch injury.

This case differs from the usual reflex sympathetic dystrophy. Sympathetic hyperactivity was visible in the face which was free from pain. Injury to the lowest trunk of the brachial plexus (C8 and T1) is usually distal to the intervertebral foramina and does not result in pupillary changes. Should such changes complicate a lesion of the lowest trunk of the plexus, the pupil on the affected side will be contracted and this implies that the nerve roots have been avulsed near the cord.5

Paralysis of the cervical sympathetic nerves to the eye results in Horner's syndrome. The signs observed in this patient were the opposite of those expected with a Horner's syndrome: 1) dilatation of the pupil; 2) widening of the palpebral fissure; 3) prominent eyeball; 4) pale sclera; 5) clear nasal mucosa; and 6) pale and cool facial skin with increased sweating. The signs represent sympathetic hyperactivity which may be due to overactivity in remaining undamaged functional portions of the sympathetic ganglia.

One case does not establish the best method of treatment of nonpenetrating cervical sympathetic chain injury. Moreover, normal resolution of this condition may have occurred in the absence of therapy. Nonetheless, prompt recovery following the sympathetic nerve blocks in this case and the widely noted similar experience with other reflex hyperactive sympathetic states (such as, causalgia and reflex sympathetic dystrophy) encourage us to recommend the early use of sympathetic blocks for similar patients.

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