Because of the small extravasated volume there had been no immediate swelling or pain; nevertheless inflammation, fibrosis and adhesions occurred. Thiopentone, with its alkaline pH (10.4), is an irritant but sequelae are reduced if the concentration is 2.5 per cent.[2] However, in this patient, a small volume of 2.5 per cent thiopentone produced this complication. Ulceration and sloughing of tissues following extravasation is not unknown, even at this concentration.[3] It normally takes about ten to 12 weeks for fibrosis and contractures to be complete after the initial inflammation. In this case these complications appeared in about six weeks.

Direct intravenous injection of thiopentone, without a running infusion, should be discourged. In the back of the hand, a favourite site for infusions, the chosen tributary should be away from the carpal or metacarpo-phalangeal joints. If extravasation occurs, analgesics, local hyaluronidase infiltration, cold compresses and elevation of the limb will reduce the pain and swelling. Follow-up for at least 12 weeks is necessary in order to detect late complications.

Thiopentone is the most widely used induction agent. It is surprising how infrequently its local complications occur or are reported.

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Contralateral spread of local anaesthetic solutions

To the Editor:

We read with interest the letter by Allen and Samson[1] on the occurrence of a contralateral Horner's sign during a stellate ganglion block. The authors postulate that the local anaesthetic injection somehow spread to the opposite side.

We believe that under unusual circumstances local anaesthetics may spread to the opposite side during sympathetic blocks. The Figure shows a small amount of radiographic contrast on the contralateral side of a man who had a surgical sympathectomy 17 years before this lumbar sympathetic block was performed. The post-surgical changes could account for this abnormal tracking of

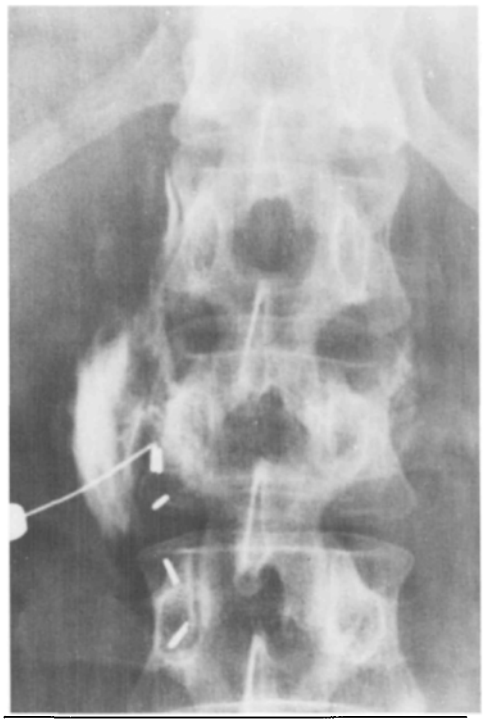

FIGURE Demonstration of contralateral spread of contrast medium injected during conduct of right lumbar sympathetic block.

solution from the left to the right side. We would expect the patient reported by Allen and Samson to have a bilateral block if this was the case.

In our own experience, three of our patients have developed a "contralateral Horner's sign" after stellate blocks. Two of them eventually produced, after several minutes, signs of ipsilateral sympathetic block with "disappearance" of the contralateral phenomenon.

It is possible that the inadvertent direct puncture of the sympathetic chain (ganglia or nerves) with the needle caused an initial autonomic stimulation, inducing a Pourfur du Petit sign (or inverted Horner), which easily could be mistaken for an atuonomic blockade of the opposite side. As the local anaesthetic took effect, the ipsilateral Horner's sign finally developed. We postulate that in our one patient who did not proceed to develop a Horner's sign, the sympathetic chain was punctured but, when repositioning the needle, the solution was injected outside the proper plane.

The same phenomenon may be seen sometimes in patients with Pancoast tumours, with a phase of sympathetic stimulation, preceding by days or weeks the development of signs of autonomic blockade.

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REFERENCE

1 *Allen G, Samson B.* Contralateral Horner's syndrome following stellate ganglion block. Can Anaesth Soc J 1986; 33: 112–3.

REPLY

We agree with Drs. Schnapp and Mays that if local anaesthetic did spread to the opposite side, one would expect a bilateral block. This did not occur in our patient. Because of her unusual response to the stellate block, she was kept in the recovery room for a prolonged period before being allowed to go home. At no time did she develop any signs of sympathetic stimulation or block on the ipsilateral side.

However, on the contralateral side, within minutes she developed pronounced ptosis and meiosis, associated with nasal stuffiness, anhydrosis and endophthalmos. Therefore it is difficult to believe that this was due to autonomic stimulation on the ipsilateral side produced by inadvertent puncture of the sympathetic chain.

The mechanism producing a contralateral Horner's syndrome remains poorly understood.

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