HORNER'S SYNDROME DURING EPIDURAL ANAESTHESIA FOR ELECTIVE CAESAREAN SECTION

MICHAEL N. SKAREDOFF AND SANJAY DATTA

ABSTRACT

Twenty ml of three per cent chloroprocaine solution was injected into the lumbar epidural space of a parturient for anaesthesia for elective caesarean section. Ten minutes after injection of the anaesthetic, the patient developed Horner's syndrome on the left side. The anaesthetic level was C7 on the affected side. Thirty minutes after the bolus dose of chloroprocaine the signs disappeared, at which time the patient received a reinforcing dose of 10 ml of chloroprocaine three per cent. The Horner's complex reappeared in eight minutes and persisted for 20 minutes.

While Horner's syndrome is not uncommon in women in labour receiving lumbar epidural anaesthesia, it is virtually unknown in patients who are not in labour. The mechanism for the appearance of the syndrome in this case is unclear. The most likely explanation is that only a small amount of local anaesthetic was necessary to produce the syndrome and the subsequent bolus of anaesthetic followed pathways opened by the original dose, since a much smaller amount of anaesthetic was necessary to reproduce the syndrome.

KEY WORDS: ANAESTHETIC TECHNIQUES, epidural; OBSTETRICAL ANAESTHESIA, complications, Horner's syndrome.

THE OCCURRENCE OF Horner's Syndrome (miosis, ptosis, enophthalmos and anhidrosis) has been noted occasionally during epidural anaesthesia. It has been most frequently reported after thoracic epidural analgesia in patients with chest injuries^{1,2} and after high doses of local anaesthetic during lumbar epidural anaesthesia for parturients in labour.^{3,4} This report concerns itself with the appearance and reappearance of Horner's syndrome during lumbar epidural anaesthesia with a short-acting local anaesthetic, chloroprocaine, for elective caesarean section.

REPORT OF A CASE

A thirty-year-old woman was admitted to the Boston Hospital for Women for repeat caesarean section. She was 175 cm tall and weighed 78.2 kg. She had undergone a previous caesarean section the year before under epidural anaesthesia with bupivacaine, without complications. Epidural anaesthesia was again elected.

Following the placement of an intravenous

Michael N. Skaredoff, M.D.,* Fellow in Obstetric Anaesthesia; Sanjay Datta, M.D., Assistant Professor of Anaesthesia, Harvard Medical School; Boston Hospital for Women, 221 Longwood Avenue, Boston, Massachusetts 02115, USA.

*Present address: Dept. of Anesthesiology, University Hospital, State University of N.Y. at Stony Brook, Stony Brook, N.Y. 11794.

catheter and the infusion of 1200 ml of lactated Ringer's solution, with the patient in the left lateral position, an epidural catheter was easily inserted at the L2-3 interspace and directed 2 cm cephalad. After a test dose of 3 ml of chloroprocaine 3 per cent the patient was turned supine with a 30° head-up tilt and the uterus was displaced to the left by a blanket roll under the right hip and torso. Twenty ml of chloroprocaine three per cent was then injected through the catheter. Blood pressure remained unchanged at 120/75 mmHg. About ten minutes after the injection of local anaesthetic the patient developed miosis, ptosis and congestion of the conjunctiva of the left eye. The left side of the face felt drier than the right side. Anaesthetic levels to pinprick were noted to be T3 on the right side and C7 on the left. The operation was begun. Anaesthesia was excellent and a 4 kg male infant was delivered with Apgar scores of 8/9. Thirty minutes after the bolus dose of anaesthetic all signs of Horner's syndrome had disappeared. Ten minutes later, a reinforcing dose of 10 ml of chloroprocaine three per cent was given. Within eight minutes of Horner's complex reappeared and persisted for twenty minutes. Anaesthetic levels to pinprick were T3 on the right side and T2 on the left. The procedure was completed without further incident and the patient recovered uneventfully.

DISCUSSION

The first clinical description of Horner's syndrome was given by Hare⁵ in 1839. He described enophthalmos and injection of the corneal vessels in a patient with a tumour compressing the cervical sympathetic ganglia. Claude Bernard⁶ and others⁷⁻⁹ in the 1850's confirmed François du Petit's observations of 1727,¹⁰ describing ipsilateral vasodilation, pupillary constriction and retraction of the eye in its socket when the cervical sympathetic ganglia in animals were sectioned. Horner,¹¹ in 1868, gave the complete description of the syndrome which bears his name.

The cardinal signs are miosis, ptosis and enophthalmos.¹² Enophthalmos in man is more apparent than real.¹³ Finally, there is vasodilation and anhidrosis on the ipsilateral side of the face.

Two other signs can also be appreciated. The first concerns visual accommodation. Sympathetic stimulation flattens the lens and accommodates the eye for distance while parasympathetic stimulation allows the eye to focus on nearby objects.^{14,15} While sympathetic paralysis does not usually make a subject grossly myopic, the feature is detectable on optometric examination.

The other component is a lack of pigment deposition in the iris.¹⁶ However, cervical sympathetic blockade must be present since birth for this feature to be apparent.

Current anatomical knowledge holds that preganglionic fibres originate from the anterior horn cells of C8 and T1;¹⁷ on occasion they may originate from as low as T4.¹⁸ These fibres course through the rami communicantes to the cervical sympathetic chain. The fibres ascend through the stellate and middle cervical ganglia to terminate in the superior cervical ganglion. Unmyelinated fibres leave this ganglion to form the internal carotid plexus. The internal carotid plexus later divides to give rise to the cavernous plexus.¹⁹

Fibres from the internal carotid plexus innervate the smooth musculature of the orbit, the musculus dilator pupillae, the involuntary muscle of the upper eyelid (*m. tarsicus*) and the muscle of Müller which helps maintain the normal position of the eye.²⁰ Other fibres course from this plexus through the three divisions of the trigeminal nerve and end either in vasoconstrictor or sweat gland musculature of the face, head or neck.

From the cavernous plexus, most fibres enter the orbit through the superior orbital fissure and

send branches to the rectus superior and the levator palpebrae superioris. Some fibres pass through the sympathetic root of the ciliary ganglion and short ciliary nerves.²¹ Others reach the eye to innervate the dilator pupillae through the long ciliary nerves, bypassing the ciliary ganglion.²²

Stimulation of these pathways causes mydriasis, retraction of the upper lid and exophthalmos. In contrast, blockade results in unimpeded parasympathetic stimulation reaching the eye by way of the ciliary ganglion causing miosis, ptosis and, in animals, enophthalmos. Interruption of the sympathetic chain below T1 may cause incomplete oculopupillary changes.²³

Horner's syndrome has been described in women receiving lumbar epidural anaesthesia during labour.^{3,4} However, after reviewing the literature no report could be found describing the appearance of Horner's syndrome during lumbar epidural anaesthesia in a non-labouring full-term parturient. The appearance of Horner's syndrome during epidural anaesthesia is in itself not uncommon. In 1972 Kepes and coworkers²⁴ first reported a case of Horner's syndrome following caudal epidural anaesthesia in an obstetrical patient in labour following the administration of 22 ml of chloroprocaine three per cent. However, the duration of the clinical signs was not reported. Mohan and Potter²⁵ in 1975 noted pupillary changes in 17 of 20 parturients who received either lidocaine or bupivacaine for caudal anaesthesia in labour.

The interest in this case lies in the fact that the initial signs of Horner's syndrome appeared within ten minutes of the injection of only 20 ml of local anaesthetic. Furthermore, the full complex reappeared promptly after injection of only 10 ml of local anaesthetic. Burn²⁶ has shown that large (40 ml) volumes of radio-opaque media consistently reach thoracic and cervical levels in non-pregnant patients whereas 20 ml volumes fail to do so. Bromage²⁷ has suggested that during intense labour small amounts of local anaesthetic may be driven cephalad. During the second stage of labor, initiation of the Valsalva maneuver when the patient "bears down" compresses the epidural space and may also force small amounts of local anaesthetic cephalad. It is also well known that, in order to achieve equal levels of anaesthesia, parturients need only one-third to one-half as much anaesthetic as non-pregnant patients.²⁸ In our case the patient did not receive an excessive volume nor was she in active labour.

Although the precise mechanism is not clear, a plausible explanation may be that the cervical sympathetic nerve fibres are very sensitive to anaesthetic blockade, as probably only a small amount of the original dose reached the C7 level. The fact that the signs reappeared after a second dose one-half the volume of the first may be accounted for by invoking Bromage's concept of epidural spread following "in the wake of the pathfinding dose",²⁷ in which the tissue planes are spread by the initial volume and a smaller volume later might be able to spread over a large area.

This report suggests that even when appropriate volumes of local anaesthetic are used during lumbar epidural anaesthesia for elective caesarean section one might encounter an unexpectedly high anaesthetic level. Further doses of anaesthetic may follow a path opened by the first dose.

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RÉSUMÉ

Pour une anesthésie péridurale à l'occasion d'une césarienne en chirurgie réglée, on a injecté dans l'espace péridural de la parturiente 20 ml de chlorprocaine trois pour cent. Dix minutes après l'injection de l'anesthésique, on a observé au côté gauche un syndrome de Horner. Le niveau anesthésique atteignait la septième cervicale de ce côté. Trente minutes après l'injection de la dose initiale de chlorprocaine, une dose supplémentaire de chlorprocaine trois pour cent 10 ml fut réinjectée. Le syndrome de Horner réapparut en huit minutes pour persister pendant vingt minutes.

L'apparition d'un syndrome de Horner chez la parturiente sous analgésie épidurale pendant le travail n'est pas exceptionnelle mais n'a pas été rapportée jusqu'ici chez la parturiente dont le travail n'est pas commencé et le mécanisme en cause paraît douteux. Dans le cas présent on pourrait l'expliquer par l'hypothèse suivante: une petite dose d'anesthésique local fut nécessaire pour provoquer l'apparition du syndrome initial et la dose subséquente injectée, bien que réduite en volume a suivi la voie ouverte par la dose initiale pour reproduire le syndrome.