Pharmacologic Reversal of Horner's Syndrome Following Stellate Ganglion Block

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To anesthesiologists engaged in the diagnosis and management of pain problems, the development of Horner's syndrome following the performance of a diagnostic or therapeutic stellate ganglion block is a welcome, valuable sign, indicating that effective sympathetic blockade has been accomplished. To the patient undergoing the block, the development of Horner's syndrome, particularly the ptosis, may be irritating and alarming. The physiologic mechanisms by which stellate ganglion block produces ptosis, miosis, and enophthalmos, originally described by Horner,† have been clearly delineated and are well understood, as are the conjunctival and scleral injection and nasal stuffiness which also follow sympathetic blockade at this level.† Yet, astonishingly enough, the pharmacologic reversal of these unpleasant "side effects," so obviously suggested by the physiologic mechanisms producing them, have not, to our knowledge, been described in the anesthesia literature. Since it is the removal of sympathetic input which produces the characteristic ocular and nasal signs and symptoms, then topical application of sympathomimetic agents to the eye should reverse them.

METHODS AND MATERIALS

One hundred consecutive patients undergoing diagnostic or therapeutic stellate ganglion blocks at the University of Illinois Pain Clinic were selected for inclusion in this study. When the development of ptosis, miosis, and conjunctival injection became obvious after the performance of each stellate ganglion block, 1–2 drops of an ophthalmic preparation of 10 per cent phenylephrine hydrochloride (Neozynephrine) were instilled in the conjunctival sac of the eye, after which the eye signs were followed for the subsequent 10–15 minutes.

RESULTS

In each of the 100 cases in this study (and in all cases thereafter) instillation of phenyl-
That the vascular supply to the conjunctiva and sclera is under sympathetic control is neither surprising nor remarkable. However, anesthesiologists tend to forget that the size of the pupil (i.e., the contractile state of the iris) and the level of the upper eyelid (i.e., the contractile state of Müller’s superior palpebral muscle) are under similar sympathetic control. These are mediated by alpha-adrenergic receptors, as indicated in the studies of Waldstein et al., who were attempting to delineate the mechanism underlying the blepharoptosis of myxedema. They were able to demonstrate that in patients with myxedema the ptosis was primarily due to dysfunction of Müller’s muscle, and that the instillation of phenylephrine but not epinephrine was effective in correcting the dysfunction of this sympathetic neuromuscular unit.

Involvement of either one of the two neuromuscular units which elevate the upper eyelid can result in ptosis, but the extent and type of ptosis resulting depends on which unit is involved. The major muscle is the levator palpebrae superioris, striated muscle innervated by the superior division of the oculomotor nerve. This muscle originates from the apex of the orbit and is inserted into the skin of the upper lid and anterior tarsal plate (fig. 2). The minor muscle is Müller’s superior palpebral muscle, a small smooth muscle innervated by sympathetic nerves. It originates from the fleshy portion of the levator muscle and its insertion is on the superior border of the tarsal plate. When both the levator palpebrae superioris and Müller’s muscle are intact and normally innervated, there is no ptosis, voluntary lid elevation is possible, conjugate lid elevation occurs with upward gaze, and there is a tarsal fold, i.e., a fold in the skin of the upper eyelid on forward gaze. When neuromuscular dysfunction of the levator palpebrae superioris muscle occurs, marked ptosis, absence of voluntary lid elevation, failure of conjugate elevation, and loss of the tarsal fold will be present. On the other hand, when dysfunction of Müller’s muscle alone...
occurs, there is only slight ptosis, while voluntary and conjugate lid elevation are normal and the tarsal fold persists.

While the pharmacologic reversal of the ptosis (and the other eye signs) resulting from stellate ganglion block is carried out simply to minimize the discomfort of Horner's syndrome, it may also be useful for patients who have congenital hyperhidrosis. The only complete cure of this condition is surgical sympathectomy. Prior to surgical intervention, these patients usually have a stellate ganglion block to allow them to assess the tolerability of the sequelae of cervical sympathectomy. It has been our experience that these patients usually find the eye signs, and especially the ptosis, too distressing, and in women cosmetically unacceptable, so they usually elect to live with their hyperhidrosis. In such cases the demonstration that the simple instillation of eye drops can pharmacologically reverse all of these sequelae may cause the patient to elect operation.

REFERENCES


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