HORNER'S SYNDROME PRODUCED BY HYPERTROPHIC SPURS FROM THE CERVICAL SPINE*

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HORNER's syndrome classically consists of a tetrad of miosis, ptosis, enophthalmos, and anhidrosis on the affected side, though the last-mentioned is not a constant finding. It was first noted experimentally in 1852 by Claude Bernard, but it was not until 1869 that the findings were described by Johann Friedrich Horner, a Swiss ophthalmologist, who gave the syndrome its name (Bernard, 1853; Horner, 1869). Any interruption in the sympathetic pathway in its long course from the brain to the eye may be responsible for the syndrome on the affected side. The sympathetic pathway to the pupil consists of three neurones:

1. The central neurone commencing in the hypothalamic centre of the midbrain runs down to the ciliospinal centre in the cord.

2. The second or preganglionic neurone then passes via the ventral roots of C8–T3 to the sympathetic chain, and entering the inferior cervical ganglion it passes upwards to the superior cervical ganglion at the level of C2–3.

3. The third or post-ganglionic neurone passes via the carotid plexus and trigeminal nerve to the eye innervating the dilator pupillae and the involuntary muscles of the eyelid.

According to the scheme devised by Foerster and Gagel (1932), pharmacological tests are used to determine which neurone of the pathway is involved by any particular lesion. These tests determine the actions of cocaine and adrenaline on the pupil. The possible results are shown in the Table.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Neurone</th>
<th>1</th>
<th>2</th>
<th>3</th>
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<tbody>
<tr>
<td>Cocaine</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>+ + Dilates</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
</tr>
<tr>
<td>Adrenaline</td>
<td></td>
<td>Nil</td>
<td>Nil</td>
<td>+ + + Dilates</td>
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</table>

The commonest level of interruption is that of the second neurone, and for this many causes have been mentioned by Duke-Elder (1949), Walsh (1957), Giles and Henderson (1958), Cobb and Scarlett (1920), and Jaffe (1950). Some unusual conditions such as a solitary neurofibroma in neck (Manning, 1953), osteochondroma of the first rib (Simpson, 1948), and phrenic crush (Chatterjee, 1956) have also been

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reported. The purpose of this paper is to present an unusual case of Horner's syndrome caused by an interruption of the second neurone by hypertrophic spurs of the cervical spine.

Case Report

A man aged 69 years was referred by his optician on account of unequal pupils found at a routine eye test. His wife had noticed that the left lid had been drooping slightly for 3 weeks.

Examination.—The visual acuity in each eye was 6/5 with correction. There was 1 mm. of left enophthalmos and the left upper lid showed 2 mm. of ptosis. The movements of the lid and eye were normal. The left pupil measured 2 mm. and the right 3 mm. The pupil reactions were normal and both eyes otherwise healthy.

Pharmacological tests showed negative responses to both cocaine and adrenaline.

The chest x-ray was normal, but that of the cervical spine revealed massive hypertrophic osteophytosis extending from C4-T1, more marked on the left than the right (Figs 1 and 2).

Examination by the orthopaedic surgeon showed some restriction of movement in the cervical spine and increased pulsation of the subclavian artery on the left side. No treatment was advised.

Discussion

Hypertrophic spurs arising from the antero-lateral margins of the cervical vertebrae are not uncommonly seen in skiagrams of the cervical spine, particularly in people
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over 40 years. They are usually asymptomatic and are supposed to be the result of the ageing process. Holt and Hodges (1957) mention that some degree of cervical spondylosis after age 40 is virtually physiological and whether or not symptoms are produced depends on the degree of encroachment of the spurs. Spurs arising anteriorly from the cervical vertebrae have been reported by Hilding and Tachdjian (1960) to impinge on the posterior wall of the pharynx and to cause discomfort, or even mechanical obstruction to swallowing food, in the neck. The cervical sympathetic trunk lies under the prevertebral fascia just lateral to the vertebral bodies (Smith and Robinson, 1958), and thus is very apt to be interfered with by large osteophytes arising from the antero-lateral margins of the cervical vertebrae. After establishing a second neurone interruption and excluding all other causes, we assume that the large hypertrophic osteophytes arising from the cervical vertebrae produced Horner's syndrome on the left side in this case.

Summary

An unusual case is presented of Horner's syndrome produced by hypertrophic spurs of the cervical vertebrae.

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REFERENCES