Superior oblique tendon sheath syndrome

An electromyographical study

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The superior oblique tendon sheath syndrome was first described by Brown (1950), who noted that many cases of apparent congenital paresis of the inferior oblique muscle had restricted elevation in adduction on passive duction testing. At operation he observed thickening of the anterior part of the superior oblique tendon sheath, and when this was stripped away a full range of passive movements was possible.

Several reports have since appeared in the literature confirming these initial findings (Nutt, 1955; Girard, 1956; Folk, 1957; Esterly, Nadbath, and Russell, 1960). Spontaneous recovery was recorded by Costenbader and Albert (1958) and Lowe (1969). Impeded elevation in adduction was found to be associated in some cases with an audible snap by Mein (1964) and Sandford-Smith (1969), who suggested local thickening of the superior oblique tendon as the causative factor.

Electromyography of the inferior oblique muscles in this syndrome revealed normal firing patterns (Breinin, 1957), but a further investigation of both oblique muscles of the affected side showed discharges indicative of an innervation disorder (Stein and Papst, 1968). Because of these conflicting opinions concerning the aetiology of this condition the authors carried out electromyography (EMG) on three adult patients with the superior oblique tendon sheath syndrome, and the results are reported below.

Method

Repeated instillation of Amethocaine 1 per cent. into the conjunctival sac ensured adequate surface anaesthesia.

Fine concentric needle electrodes were introduced into the superior and inferior oblique muscles through the upper and lower lids respectively, precise placement being confirmed by audio-amplification of the electrical activity from the muscles on attempted up and down gaze in adduction.

Insertion of the needle into the muscle belly of the superior oblique presented no difficulty if the trochlea was palpated and the needle was firmly advanced directly backwards, immediately medial to this structure.

The electrical activity from the muscles was displayed on a cathode ray oscilloscope after suitable amplification and was recorded photographically.

Case reports

Case 1, a 67-year-old woman, complained of steadily progressive vertical diplopia which was first noted at the age of 20. She had been treated with vertical prisms of increasing strength and was eventually unable to wear her prescription because of its excessive weight. There was no history of facial trauma.
Examination

VISUAL ACUITY
6/9 in the right eye and 6/6 in the left.

OCULAR MOTILITY
Limited elevation of the left eye in adduction (See Hess chart, Fig. 1).


PASSIVE DUCITION TEST
Elevation in adduction markedly restricted in the left eye.

Diagnosis
Left superior oblique tendon sheath syndrome.

EMG
Normal firing patterns were recorded from the oblique muscles in the contracted and relaxed state (Figs 2 and 3).

FIG. 2 Case 1. Electromyograph of left superior oblique
Upper trace: Attempted elevation in adduction (relaxed)
Lower trace: Depression in adduction (contracted)

FIG. 3 Case 1. E.M.G., left inferior oblique
Upper trace: Depression in adduction (relaxed)
Lower trace: Attempted elevation in abduction (contracted)

Case 2, a 19-year-old man, had noted vertical diplopia 2 weeks after being involved in a minor traffic accident which had occurred 4 months previously. He gave no history of head injury or facial trauma.
Examination

**VISUAL ACUITY**
6/5 in each eye.

**OCULAR MOTILITY**
Limited elevation of the left eye in adduction (See Hess chart, Fig. 4).

![Hess Chart](image)

**FIG. 4** Case 2. Hess chart. Pseudoparesis of left inferior oblique. Overaction of right superior rectus.

**PASSIVE DUCTION TEST**
Elevation in adduction restricted in left eye.

**Diagnosis**
Left superior oblique tendon sheath syndrome.

**EMG**
Normal firing patterns recorded from the left oblique muscles in the contracted and relaxed state (See Figs 5 and 6).

![EMG Traces](image)

**FIG. 5** Case 2. E.M.G., left superior oblique
Upper trace: Attempted elevation in adduction (relaxed)
Lower trace: Depression in adduction (contracted)
Base line 50 cycle interference

**FIG. 6** Case 2. E.M.G., left inferior oblique
Upper trace: Depression in adduction (relaxed)
Lower trace: Attempted elevation in abduction (contracted)
Base line 50 cycle interference

**Case 3, a 29-year-old man** gave a 10-year history of intermittent horizontal and vertical diplopia and complained of increasing difficulty in maintaining binocular single vision. There was no history of facial or head trauma.
Examination
Visual acuity 6/5 in each eye.

Ocular motility
Downdrift of each eye on adduction with poor elevation in adduction (See Hess chart, Fig. 7).

**Fig. 7** Case 3. Hess chart. Bilateral pseudoparesis of inferior obliques. Bilateral overaction of the superior rectus and superior oblique muscles. Depression of the adducting eye on left and right gaze

Passive duction test
Elevation in adduction restricted in both eyes.

Diagnosis
Bilateral superior oblique tendon sheath syndrome.

EMG
Recordings were obtained from the left obliques only. Normal firing patterns were recorded on contraction and relaxation (Figs 8 and 9, opposite).
Results

Recordings of electrical activity obtained from three adult patients with a clearly defined superior oblique tendon sheath syndrome showed the following features:

1. On attempted elevation in adduction there was electrical silence from the superior obliques and maximum activity from the inferior obliques.

2. On depression in adduction there was electrical silence from the inferior oblique and maximum activity from the superior oblique.

3. The amplitude and frequency of the potentials obtained from the contracting superior oblique were constantly of a lower order of magnitude than those recorded from the contracting inferior oblique in spite of repeated sampling from different sites within the muscle bellies.

Discussion

The superior oblique tendon sheath syndrome has been classified as a musculo-fascial anomaly, along with the vertical retraction syndrome, strabismus fixus, and Duane’s syndrome. These conditions were thought to occur as a result of disordered differentiation of paraxial mesoderm in intrauterine life (Catford, 1966).

Electromyographical studies on cases of Duane’s syndrome have shown, however, anomalous patterns of electrical discharge from the horizontal recti in a large proportion of cases. Undiminished or increased activity has been recorded from the lateral rectus on adduction as well as co-contraction of the medial rectus on attempted abduction (Sato, 1960).

Defects of supranuclear innervation (Blodi, van Allen, and Yarborough, 1964) and an abnormal peripheral nerve supply to the lateral rectus (Hoyt and Nachtigäller, 1965) have been suggested as possible causes. Abnormal recordings were observed from the vertical recti in cases with congenital paresis of the lateral recti (Papst and Esslen, 1964) and co-contraction of the oblique muscles in one case of the tendon sheath syndrome on elevation in adduction (Stein and Papst, 1968).

Our investigations of discharge patterns from the oblique muscles in the superior oblique tendon sheath syndrome revealed no evidence of co-contraction but only smooth recruitment of electrical activity from the relaxed to the maximally contracted state. These results show no deviation from Sherrington’s law and agree with those of the first worker to investigate this syndrome electromyographically (Breinin, 1957).
The lower amplitude and frequency of potentials recorded from the contracting superior oblique as opposed to the activity recorded from the contracting inferior oblique suggests that fewer motor units were being activated in the former muscle. Since we have insufficient data on the electromyography of superior oblique muscles in patients with no ocular motility problems, further research is required to determine the significance of this finding.

Summary

A study of the electromyographical activity of the oblique muscles supports the view that the superior oblique tendon sheath syndrome is due to a local mechanical problem caused by the inability of the tendon to pass freely and to its full extent through the sheath.

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