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LIMITATIONS OF SUPERIOR RECTUS MOVEMENT: A LITERATURE REVIEW AND CLINICAL STUDY

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Abstract

Controversy exists in the literature regarding the differential diagnosis between a primary superior oblique palsy of one eye, and a primary superior rectus palsy of the contralateral eye. As a follow up to our previous study of long standing superior oblique palsies, which showed greater limitation of the contralateral superior rectus muscle, a literature review and a study of previously diagnosed primary superior rectus palsies was undertaken. It was revealed that isolated neurogenic superior rectus palsies are rare and are unlikely to occur without associated ptosis.

Key words: *Fourth cranial nerve palsy, superior rectus palsy, tight inferior rectus, thyroid eye disease, double elevator palsy, Bielschowsky head tilt test.*

Congenital isolated neurogenic superior rectus palsies are uncommon and are usually associated with ptosis on the ipsilateral side.[1]

To understand why this is the case, we wish to emphasise the following anatomical and physiological points.

The levator muscle and the superior rectus arise from the same embryonic striated muscle mass,[2] and both muscles are innervated by the superior division of the third cranial nerve, therefore, any neurological defect or lesion would affect both the levator and the superior rectus muscles.

There are also intimately bound fascial connections between the superior rectus muscle and the levator, (the fascial sheaths of Whitnall). At their origins on the sphenoid bone their tendons are blended together and the levator lies upon the superior rectus during its entire course.[3] In fact, surgery such as recession of the superior rectus muscle will carry the levator back with the superior rectus muscle and so the lid will be raised.[4] Also it has been reported (personal communication) that on giving an injection of Botulinum into the superior rectus muscle, ptosis of that eye often occurs.

With these facts in mind, the presence of muscle sequelae showing the greatest underaction being that of the superior rectus muscle without ptosis should encourage detailed investigation of the contralateral superior oblique muscle to avoid misdiagnosis.

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LITERATURE REVIEW

A literature review of the past 22 years produced some interesting aspects of superior rectus dysfunction. Anatomical absence of the superior rectus muscle has been reported, but this condition is rare and is usually associated with other craniofacial or extra ocular muscle anomalies. Mather and Saunders report that to their knowledge "there has been no surgically or radiographically documented cases of absent superior rectus muscle presenting as an isolated finding".[5]

Many references to double elevator palsies are made, involving symmetrical underactions of both superior rectus and inferior oblique muscles of the same eye.

Jampel and Fells[6] report finding sudden onset symmetrical paresis of elevation of one eye with normal lid function and suggest the cause to be a lesion in the midbrain tectum or pretectum, near or in the ocular motor nucleus. In these cases, Bell's phenomenon was reduced. This work seems to be the only significant publication in the last two decades, involving superior rectus dysfunction without ptosis and suggests a neurological cause.

Metz[7] described a congenitally short and tight inferior rectus muscle giving clinical signs of a double elevator palsy, when in fact, according to saccadic velocity measurements, the elevators were normal. This anomaly elicits poor or absent Bell's phenomenon. This suggests that Bell's phenomenon should be tested routinely in conjunction with a forced duction test, to differentiate between a brain stem lesion and a tight inferior rectus. Both conditions can exhibit absent or reduced Bell's phenomenon, but only the tight inferior rectus muscle will give a positive forced duction test.

Duke Elder states that the congenital adhesion syndrome of the superior rectus and superior oblique muscles at the point of crossing of the muscles can simulate clinical signs of a superior rectus palsy but without ptosis.[8]

Goodier[9] has diagnosed superior rectus palsies which elicited some type of "atypical" positive Bielschowsky head tilt test response but failed to define her "atypical" terminology. We consider it clinically misleading and incorrect to refer to an atypical head tilt response. This test was described as a specific differential diagnostic test to which the response can only be positive or negative to a superior oblique palsy (or an isolated inferior oblique palsy) as described by Bielschowsky.[10]

EXPLANATION OF THE BIELSCHOWSKY HEAD TILT TEST (BHTT)

Misconceptions regarding the complex principles of the BHTT appear to be common. Torsional movements of the eyes on tilting the head are the basis of this differential diagnostic test. Therefore, it must be remembered that physiologically the cycloduction of the oblique muscles is greater than that of the vertical recti muscles, and conversely the vertical action of the vertical recti muscles exceeds that of the oblique muscles.

The BHTT involves forcibly tilting the head to the side *opposite* to the usual compensatory ocular torticollis adopted for the paresis of the vertically acting muscle.

In the case of a superior oblique palsy, on tilting the head to the opposite side to that usually adopted as a compensatory head posture (ie. to the same side as the palsied superior oblique muscle), a vestibulo-ocular reflex of intorsion of the palsied eye occurs. In a synergistic movement the superior oblique and the superior rectus muscles of that eye should bring about the required compensatory intorsion.

However, because the superior oblique is palsied, the superior rectus, now being unopposed in the vertical field, produces an updrift or increased vertical deviation of that eye, ie. a positive BHTT response for a superior oblique palsy. Some authors explain this phenomenon by suggesting that an exaggerated contraction of the superior rectus muscle occurs.

In the case of a suspected superior rectus palsy, on tilting the head to the opposite side to that of the usual compensatory ocular torticollis, there is negligible change of vertical movement as the extorsion of the affected eye, induced by the tilt, is carried out by the inferior oblique and the inferior rectus muscles of that eye, and their antagonistic function in the vertical field is balanced.

The head tilt test may also be applied to the differential diagnosis between palsies of the inferior oblique and the contralateral inferior rectus muscles.

Walsh and Hoyt[11] state there is no increase of the vertical deviation in cases of superior rectus or inferior rectus paralysis on forcible tilting of the head to either side, and have found the BHTT to have great value in the differential diagnosis between a superior oblique and contralateral superior rectus muscle palsies.

Other authorities give similar explanations of the BHTT, and also claim the test to be a valuable differential diagnostic test.[12-16]

CLINICAL STUDY

In 1988 our department reported a series of fourth nerve palsies where the greater underaction was exhibited by the contralateral superior rectus muscle.[17] This finding has led us to undertake a 10 year retrospective study of previously diagnosed superior rectus palsies.

Documentation of only six cases of primary superior rectus palsies was found over this 10 year period.

All patients were contacted but only two returned for follow up. The required clinical information was incomplete in some cases, so the following details when available, were:

Hess charts
Ocular motility
Ptosis
Bielschowsky Head Tilt Test (BHTT)
Bell's phenomenon
Forced ductions

FINDINGS

In our study the following aetiologies were revealed as the contributing factors for the apparent superior rectus dysfunction.

Case 1: Congenital tight inferior rectus syndrome, confirmed by forced duction test on follow up.

Case 2 and 3: Trauma to the same side of skull as the underacting superior rectus muscle resulting in sudden onset diplopia in only that area of gaze, ie. not the usual neurogenic muscle sequelae and no ptosis.

Case 4: True neurogenic superior rectus palsy with ptosis on the same side.

Case 5: Thyroid eye disease

Case 6: Apparent superior rectus palsy, however, on review, a positive BHTT confirmed a contralateral superior oblique palsy.

It will be noted that only two cases were of neurogenic origin: Case 4 which was a true neurogenic superior rectus palsy with ptosis on the same side, and Case 6 which was a superior oblique palsy on the contralateral side confirmed by the BHTT. See Figure 1.

CONCLUSION

We find that evidence from the literature, our clinical data and personal communications support the belief that an isolated primary palsy of the superior rectus muscle without ptosis as a neurogenic entity would be a very rare occurrence.

The following aetiologies are considered to be responsible for an isolated superior rectus dysfunction without ptosis.

- congenital anomalies of the insertion of the muscle, muscle fibrosis or muscle aplasia.
- Thyroid Eye Disease/Thyroid Orbital Disease.
- congenital tight inferior rectus syndrome.
- double elevator palsy.
- direct trauma to the superior rectus muscle.
- orbital pathology.
- sudden onset double elevator palsy.
- post lens extraction which may result in superior muscle palsy with or without ptosis.
- primary palsy of the superior oblique muscle on the contralateral side.

Excluding trauma, pathology and congenital anomalies, we consider the most common cause of isolated limitation of movement of the superior rectus muscle to be a primary superior oblique palsy on the contralateral side. Careful examination must be carried out to establish the correct diagnosis.

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DIAGNOSIS: No. 1

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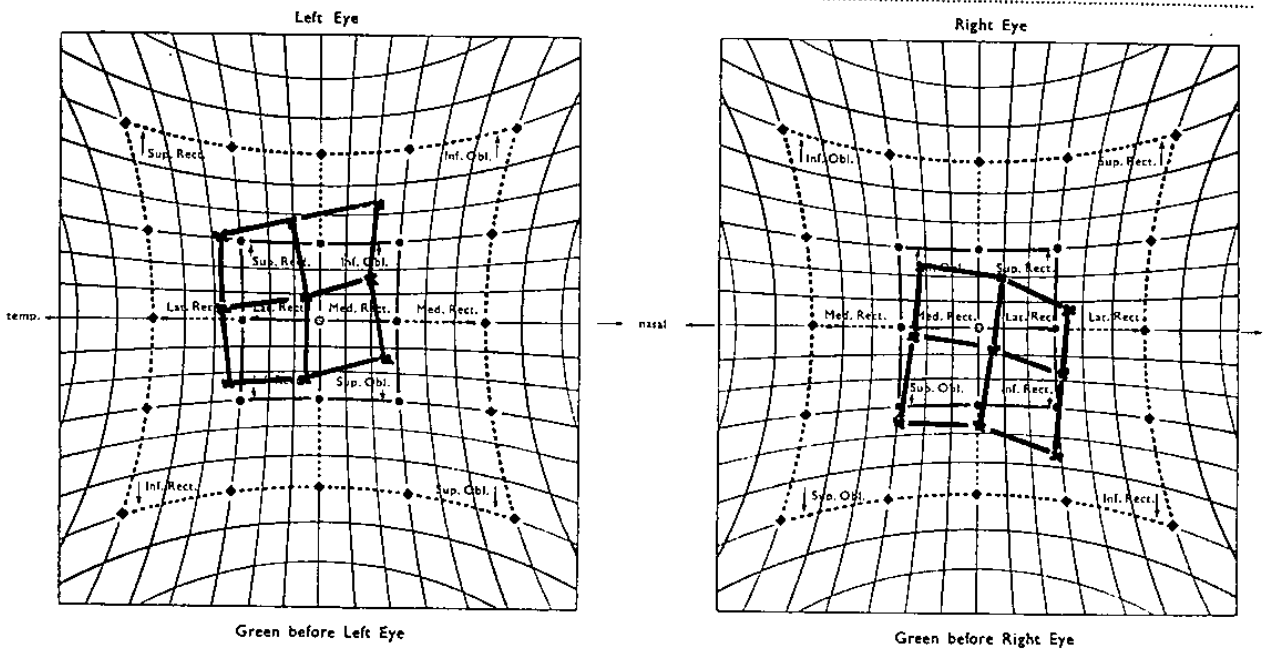

Figure 1: Case No. 6, showing greatest deviation in the area of the right superior rectus muscle but with a positive Bielschowsk Head Tilt Test response to the left side, indicating a primary palsy of the left superior oblique muscle.

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