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PERSISTENT INCOMITANCE OF LONG STANDING FOURTH NERVE PALSIES

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Abstract

The length and course of the trochlea nerve causes it to be one of the most vulnerable of the cranial nerves. A retrospective study of the long term muscle sequelae of 32 fourth nerve palsies, both congenital and acquired, was undertaken to examine the development of concomitance with time. Particular reference has been made to the increasing dysfunction of the contralateral superior rectus muscle, compared with that of the primary palsy.

Key words: *Trochlea nerve palsy, fourth cranial nerve palsy, superior oblique muscle, superior rectus muscle, concomitance, incomitance, muscle sequelae.*

INTRODUCTION

When a fourth nerve palsy occurs, one would expect the greatest deviation to be in the direction of gaze of the affected muscle.

However, following a fourth nerve palsy the contralateral antagonist superior rectus muscle may become markedly restricted and the field of greatest deviation and separation of images is found in that direction of gaze. That is, the muscle sequelae which usually results in concomitance in the upper field, can change with time.

To better understand the muscle sequelae of paralytic strabismus, one must be mindful of Hering's Law of equal innervation (1879). "When a nervous impulse is sent to a muscle causing it to contract, an equal impulse goes to its contralateral synergist in order to maintain parallalism of the visual axis. When an underacting muscle requires extra innervation to bring about contraction, the innervation received by the contralateral synergist is equal but excessive, causing it to overact".[1]

Hering's Law must be considered in conjunction with Sherrington's Law of Muscle action. "When a muscle contracts, its direct antagonist relaxes to an equal extent, allowing smooth movement to take place. When there is underaction of the muscle which persists with time, the unopposed action of its antagonist results in contraction of that muscle".[1] For these reasons both eyes become affected when an ocular palsy occurs.

Therefore following a fourth nerve palsy the initial manifestation of the abovementioned laws is an overaction of the yoke muscle, the

contralateral inferior rectus muscle, overaction of the ipsilateral antagonist inferior oblique muscle, and finally, secondary inhibitional palsy of the contralateral antagonist, the superior rectus muscle. Our understanding of concomitance is that the angle of deviation becomes the same in all directions of gaze, and is due to innervational changes involving the muscles of the non-palsied eye.

METHOD

Thirty-two cases of fourth nerve palsies, 25 acquired, seven congenital or longstanding, seen over the past six years, were reinvestigated to observe any change in muscle sequelae with time.

The term PALSY is used in this study to describe the limitation of movement of the superior oblique muscle. We classify a case as "congenital" when there is no known history of trauma, no neurological disorder or serious illness, and when the patient is not in the age group of possible vascular episodes. For this study the diagnostic procedure was:

- cover tests with and without abnormal head posture at near and distance fixation.
- ocular motility, observed by at least two members of staff.

Particular attention was given to identify any bilateral anomaly of the superior oblique muscle. Cover tests and reversal of diplopia were assessed, on down gaze and to the right and left sides.

- Bielschowsky Head Tilt Test (BHTT).
- Hess Chart.
- Diplopia chart with subjective estimation of image separation noted.
- Angles measured on the synoptophore in the primary position and in the field of action of the affected superior oblique muscle. Measurements were taken with each eye fixing, 20° on up gaze and down gaze, on the affected side.

Measurements can be obtained from the Hess Chart, by calculating the displacement of images at 50 cm. This procedure elicits the ocular motility pattern and is reproducible at a constant distance with constant accommodation.

However, we consider that with longstanding vertical anomalies, the vertical fusion range is often abnormally large,[9] and therefore, that some fusional control may mask the true degree of vertical deviation.

CLASSIFICATION

Knapp[2] classifies cases of primary fourth nerve palsy according to the direction of greatest deviation. His list of six classifications comprises all possibilities, variations and combinations of muscle sequelae, which assists in decisions relating to surgical correction. Scott[3] also describes eight classifications of fourth nerve palsies for the same reasons.

Our clinical observations determined three categories of primary palsy of the superior oblique muscle.

Type 1. Primary palsy of the superior oblique with greatest deviation and separation of images in the area of action of the palsied muscle.

Type 2. Primary palsy of the superior oblique with greatest deviation and separation of images in the area of action of the contralateral antagonist superior rectus muscle.

Type 3. Primary palsy of the superior oblique with any combination and/or variation of sequelae other than that of groups 1 and 2.

FINDINGS

Symptoms: Presenting symptoms in order of frequency were:

- Difficulty with near tasks.
- Diplopia, intermittent or constant.
- Torsion.

As reported by Seaber,[4] and Moore and Stockbridge,[5] torsion as a presenting symptom was found to be a rare occurrence. Only 3% of this series complained of a tilted image.

Bilaterality: Mansour and Reineke,[6] in an extensive literature study, cite 22 authors who found that the incidence of bilateral fourth nerve palsies is low, i.e. only 17% of a total of 895 cases. These statistics support our findings of 9.6% with a bilateral defect, in spite of Jampolsky's comment[7] "that all fourth nerve palsies should be considered bilateral until proven to be otherwise".

However, Keishner[8] makes reference to Urist's

description of cases of bilateral fourth nerve palsies which were masked as a bilateral entity until post operative examination. It then became obvious that there was also a fourth nerve palsy on the other side. Only one case of this study showed this result, although there were no bilateral signs prior to surgery.

Position of maximum deviation: According to the Hess Charts, 43% of this series showed either recovery, concomitance, or greatest limitation in the field of action of the superior oblique muscle.

However, 57% showed an increasing limitation in the field of action of the contralateral superior rectus muscle. Of this group, 67% of Hess Chart findings were confirmed by the synoptophore measurement being greatest in the same area of gaze.

Wright and Hansotia[10] in reviewing 23 cases of fourth nerve palsies relied on the BHTT and diplopia charts only, for diagnosis of the primary palsy. In our experience these tests alone are not conclusive for such a diagnosis. We consider all tests mentioned in this study to be essential. Goodier[11] states that the BHTT as a differential diagnostic test between a superior oblique palsy and a superior rectus palsy of the other eye, can produce misleading results. She reports finding positive responses with primary palsies of the superior rectus muscle, but has relied on the Hess Chart solely for the original diagnosis.

Ninety per cent of this series exhibited a positive BHTT response, as described for diagnosis of a primary superior oblique palsy. Ten per cent of the series gave a negative response, i.e. no increase in the deviation when the head is tilted to either side.

Figure 1 shows a typical case of a subject showing marked contraction of the ipsilateral inferior oblique muscle, and a positive BHTT response.

Clinical observation of ocular movements revealed that 57% showed the greatest deviation in the direction of the contralateral superior rectus muscle. The deviation was most marked when alternate cover test was performed in that field of gaze.

This observation was confirmed by the patient's subjective estimation of image separation in *all* cases. We found this anomaly in both congenital and longstanding cases of fourth nerve palsies.

Four of the original 32 cases had recovered. Of the remaining, six were categorised as type 1, 18 were categorised as type 2, and four as type 3 (three of whom had bilateral palsy).

DISCUSSION

An electromyographic study of paralytic strabismus by Sakuko and Tsutsui[12] has shown loss of relaxation in the direct antagonist during the action of the agonist, i.e. loss of reciprocal innervation.

If this is so, and the findings applied to a superior oblique palsy, then the direct antagonist inferior oblique could become constantly contracted, and would exaggerate the inhibition of its yoke muscle, the superior rectus of the other eye.

Meyer, Ladatscher and Zonis,[13] after biopsying tissue from 10 overacting antagonist inferior oblique muscles (in cases ranging from six-18 years of age), found that most muscles were in different stages of atrophy. "Contraction bands and asymmetrically contracted sarcomeres, disruption of the sarcomere pattern, and alterations of the Z bands were encountered". A control biopsy of a normal non-overacting inferior oblique was also done, taken from the site of an enucleation, and there was no abnormality reported.

Spencer and McNeer[14] also reported structural alterations in overacting inferior oblique muscles.

Other research in the past decade regarding such muscle fibre changes[15-17] adds support to Sakuko and Tsutsui's findings that anatomical changes may occur in an antagonist inferior oblique muscle.[12]

Certainly other authors have recognised that deviation changes can increase in the upper field of gaze with a fourth nerve palsy. Several authors note that there is diagnostic confusion between an isolated primary palsy of the superior rectus and a primary fourth nerve palsy of the other eye.

Duke Elder[18] states "Congenital cases of superior rectus palsies are frequently associated

Figure 1 (i): No deviation in the primary position.

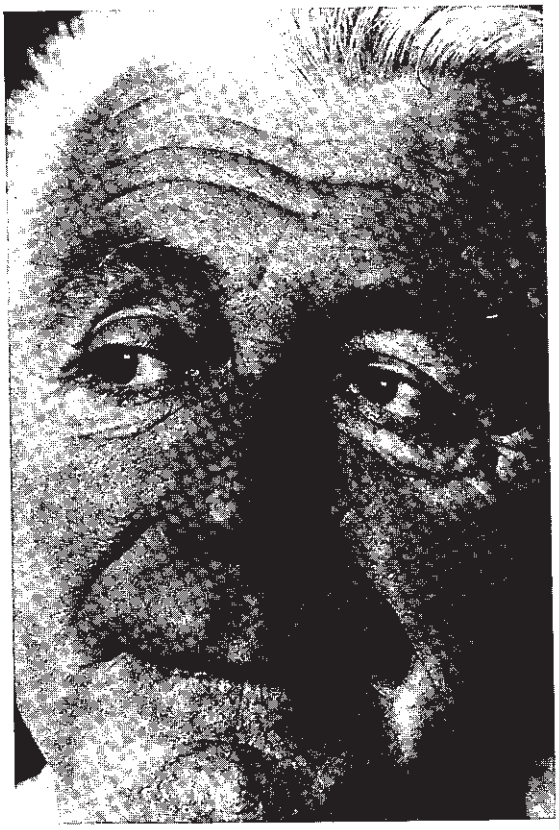

Figure 1 (ii): Looking right (fixing left eye).

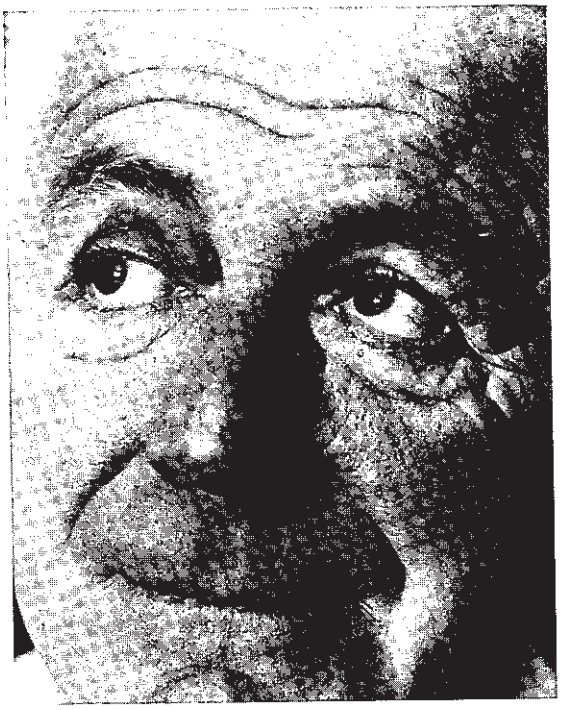

Figure 1 (iii): Looking right (fixing right eye), showing marked overaction of the left inferior oblique muscle.

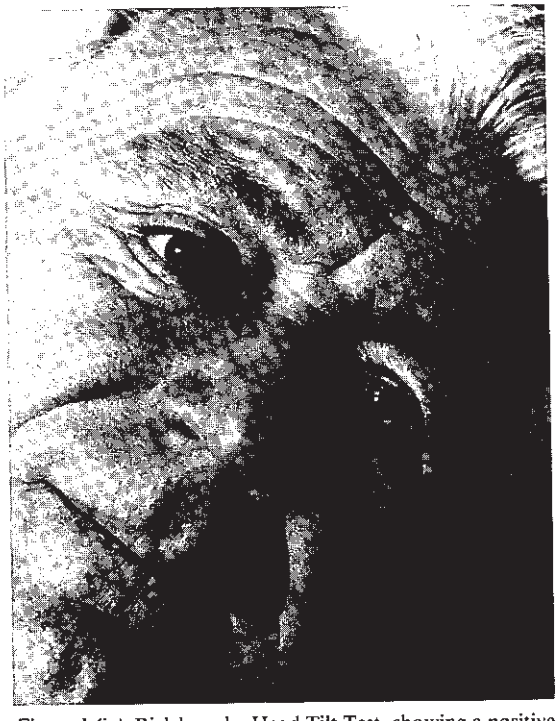

Figure 1 (iv): Bielchowsky Head Tilt Test, showing a positive response to a left superior oblique palsy.

with ptosis, usually in the same eye but occasionally in the other. Acquired cases are rare, and when they occur they are usually due to a paresis of the superior division of the oculomotor nerve and are therefore accompanied by ptosis . . ."

We postulate the following reasons for the development of this incomitance in the upper field, following a fourth nerve palsy.

1. From the initial sequelae, innervational changes according to Hering's Law, result in an overaction of the contralateral inferior rectus muscle and those of Sherrington's Law cause inhibition of the contralateral superior rectus muscle.
2. Sherrington's Law results in an overaction of the ipsilateral inferior oblique muscle, which is also advantaged due to decreased tonus of the palsied superior oblique muscle. As a consequence of Hering's Law, the yoke muscle to this inferior oblique is inhibited, i.e. the contralateral superior rectus muscle.
3. The EMG studies and muscle tissue biopsy reports show anatomical changes in the antagonist inferior oblique which result in a constant state of contracture of the muscle. This further exaggerates the inhibition of the contralateral superior resctus muscle.

Therefore, following a fourth nerve palsy, there are resulting significant anatomical and innervational changes to the ipsilateral inferior oblique muscle, which in turn, produce a significant underaction of the contralateral superior rectus muscle.

CONCLUSION

Our observations show that congenital or long-standing fourth nerve palsies are not uncommon, and that the deviation may become greater in the upper field with time. This is found in the field of the contralateral superior rectus muscle, as a result of innervational and anatomical changes of its yoke muscle, the inferior oblique on the originally palsied side. It is important to identify this phenomenon, to eliminate any dilemma that there may be a primary superior rectus palsy.

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