ADAPTATIONS OF THE OCULOMOTOR SYSTEM

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The extraocular muscles differ or are adapted to their task in several ways which are of interest in clinical care.

Proprioception

An important difference between the extraocular system and most other muscle groups is the lack of proprioceptive control of extraocular muscle (EOM) movement. This is not to say that the human eye muscles are devoid of spindles or tendon receptors. But, feedback of eye position is via vision. EOM afferent fibers in species carefully studied ascend via the fifth cranial nerve. However, central fifth cranial nerve paralysis has never been reported to create any oculomotor difficulty. Tension of the extraocular muscles under normal waking conditions does not reflect the position of the eyes in the orbit. As Figure 1 shows, the tension of a horizontal rectus muscle (lower limb of curve) taken from direct measurements in alert humans is least near the primary position, and increases with gaze to either side. It increases with increased innervation into the field of action and increases with passive stretch, even though innervation is diminished, into the direction away from the field of action of the muscle. Clearly, tension cannot be a useful criterion to guide eye position. This is quite different, of course, in skeletal muscle systems where load upon the muscle is created by outside influences such as gravity. Steinbach[1] has shown that extraocular muscles following surgery for strabismus, both with length change and removal of receptors at the tendon end of the muscle, continue to be dominated by visual feedback with little effect from the muscle itself.

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Muscle length

Extraocular muscles are 35-40 mm long in adults. To rotate the eye about 50° into the field of action, and relax a similar amount into the opposite field of action, requires the extraocular muscles to move 10 mm in shortening and 10 mm in extension (5 mm per degree). This requires the contractile portion of the extraocular muscle to be 3 to 4 times that length. This is maintained in microphthalmic eyes which then often have a range of 60-70°. This normally begins to diminish with age because of increasing eye size (increased lever arm), and later from disuse. Increased stiffness is associated with lack of physical exercise of extraocular muscles (King[2]), but is maintained by exercise (Chamberlain[3]) and we have noted a 10 degree increase in upgaze with forced upward gaze done daily. Extraocular muscles which are markedly shortened will have a significant reduction in motility, both into and out of the field of action of the muscle. This has important clinical implications. For example, in complete lateral rectus palsy, the eye often turns 20°-25° into esotropia and is quite firmly held in that position. The medial rectus will have shortened 5 mm. If this tight muscle is now recessed (typically 6 mm or more), the tension on the muscle will be reduced and it will shorten further. The amplitude of horizontal gaze which can now be achieved by this short stiff medial rectus muscle opposite temporally transposed vertical recti will be extremely small. The primary reason to use Botox in lateral rectus palsy is not to preserve anterior segment blood supply in transposition procedures, but to allow the greatest possible range of horizontal movement by preserving and elongating the contracted medial rectus and thereby preserving the amplitude of eye rotation.

Internal muscle anatomy

There is an orderly recruitment of motor units in the EOM from the «off» position (abduction for the medial rectus) to the «on» position (adduction for the MR). In this system we see fineness of control around the primary position, subserved by the motor units composed of small muscle fibers on the orbital surface of the muscle. These have a high nerve to muscle fiber ratio, as much as 1/5, as well as a highly developed blood supply because of their constant activity at or near the primary position. The more gross control of large following or saccadic movements is implemented by motor units composed of larger muscle fibers adjacent to the globe. These have a lower ratio of nerve to muscle fibers as low as 1/12. This allows rapid recruitment of powerful force needed to overcome the viscosity of the system for rapid movement. We have worked for several years on technics to preferentially weaken the outer fiber layer, active in the primary position, leaving the majority of fibers intact to subserve saccadic movement and extreme gaze. There are many situations where this would be useful, such as in exotropia with lateral incomitance in which we are limited in the amount of LR recession to correct primary position deviations without causing esotropia in lateral gaze. Surgical approaches all have shown a substantial risk of full muscle transection. A laser-based approach to remove this outer layer is now looking quite promising in our animal studies.

Muscle size and strength

Limb muscles increase (or decrease) their cross sectional area to provide more strength in response to isometric loading. Extraocular muscles normally spend their life in a very narrow zone of tension variation. Agonist and antagonist, being the main load to one another, tend to have the same size. But EOM respond to external load in pathologic situations. We see reduction of muscle force of the antagonist in paralysis. In each of 5 cases of lateral rectus paralysis, the ipsilateral medial rectus had reduced maximal isometric active force compared to the normal fellow eye. We have seen marked elevation of forces from the normal 50-80 grams to over 100 grams in Duane's syndrome, where the medial rectus is contracting against a restricting and co-contracting lateral rectus muscle. The lateral and medial rectus muscles are both large and strong. This is typical in cases with marked vertical overshoots in adduction. In Grave's disease with inferior rectus contracture, the superior rectus is pulling against a restricting load. Here the force may also increase markedly, and we have measured over 120 grams.

Muscle Tension

Tension of muscles influences surgical and Botox outcomes. There is a high innervational level causing high forces in most cases of infantile esotropia. We have measured 80 to 100 grams of medial rectus force in children one year of age, and a very high EMG activity is typical. Following botulinum paralysis of the medial rectus for 60 prism diopter esotropia, the eye does not simply go to the midline, as it does in adult esotropia following botulinum medial rectus palsy. Instead, it goes to 30 or 40 prism diopters exotropia with a substantial limitation of adduction. This is explainable only by assuming that the lateral rectus is contracting very strongly and that the medial rectus is contracting even more strongly. This high rate of co-innervation and co-contraction leads to hypertrophy of the medial rectus and its shortening.

It seems likely that much of the variability in response to surgical muscle recession is due to this variability of muscle tension based on innervation level. Stephens and Reinecke[4] showed that the measured stiffness of the intact globe for a rotation of 25° varied from 1 gram per degree to almost 4 grams per degree in normals. Esslen and Papst[5] attempted to assess EOM activity levels along this line of thinking by EMG, but this technic is quite variable according to the site of recording and thus not reliable. We have recently measured forces in the horizontal recti in alert adults with strabismus, finding a 3-fold variation from 5 to 15 grams. We have gathered data on the antagonist isometric force in 37 patients undergoing Botox injection. This measure varied from 10 to 30 grams and higher force predicts larger alignment change caused by the induced paralysis.

Length adaptation

In an attempt to explain the efffect of botulinum alignment changes and of contracture, we studied sarcomere responses to change of length (Williams and Goldspink[6]). Eye muscles responded by increasing the number of sarcomeres (adding length) when stretched and reducing sarcomeres when shortened. It thus appears that the major cause of changed eye alignment from Botox injection (and of «contracture» of antagonist muscles after paralysis) is this internal muscle length change rather than a permanently induced weakness in most cases (Scott[7]). When we studied active force before injection and then 6 months later, it was usually unchanged. However, a few cases of exotropia persisting 6 months or more following medial rectus injection, and vertical rectus transposition in lateral rectus palsy, do show weak contractile force of the medial rectus, supporting the thesis that long term atrophic changes can occur in the medial rectus, as shown by McNeer and Spencer[8]. The adaptation of length is carried out principally by sarcomere addition or subtraction at the ends of the muscle with the activation of several genes and the expression of their proteins in response to length change. The removal of the musculo-tendonous area with resection may reduce this ability of the muscle to length-adapt post-operatively and thus explain why recess-resect is more stable than is recession.

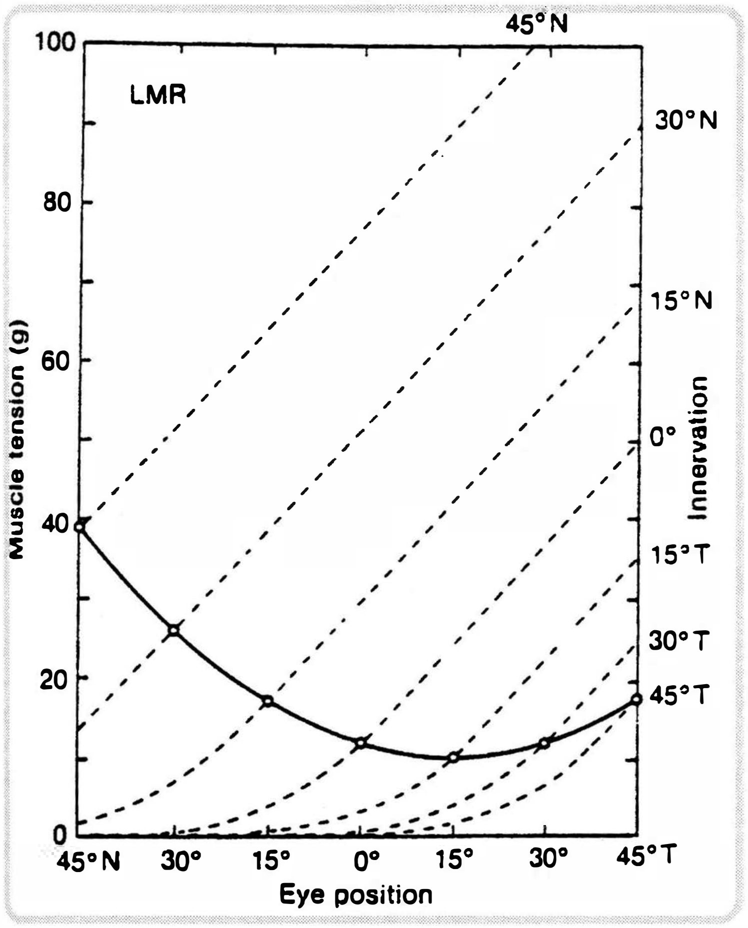

Figure 1
The tension of a horizontal rectus muscle (lower limb of curve) in alert humans

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