UPSHOOTS AND DOWNSHOOTS

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INTRODUCTION

Hypertropia occurring with adduction has several causes: 1) occlusion hypertropia (dissociated vertical divergence) may occur as the adducted eye is occluded by the nose; 2) inferior oblique overaction may occur due to abnormal insertion position of the inferior oblique on the globe; [1] 3) abnormal increase in innervation to the inferior oblique; [2] and 4) from abnormal increase in innervation in the superior rectus muscle. [2] A similar downward deviation during adduction can occur: 1) due to superior oblique overaction from abnormal insertion position on the globe; 2) from abnormal innervation to the superior oblique during adduction; 3) from abnormal superior oblique restriction (Brown's syndrome); and 4) from inferior rectus abnormal innervation during adduction (unpublished electromyographic data). Abnormal vertical forces from horizontal rectus muscles may give rise to these vertical deviations especially when mechanics or innervation creates abnormally high forces as in Duane's syndrome, exotropia, and the tight lateral rectus syndrome. [3] This paper will explicate this mechanism and show a useful treatment approach based upon analysis of it.

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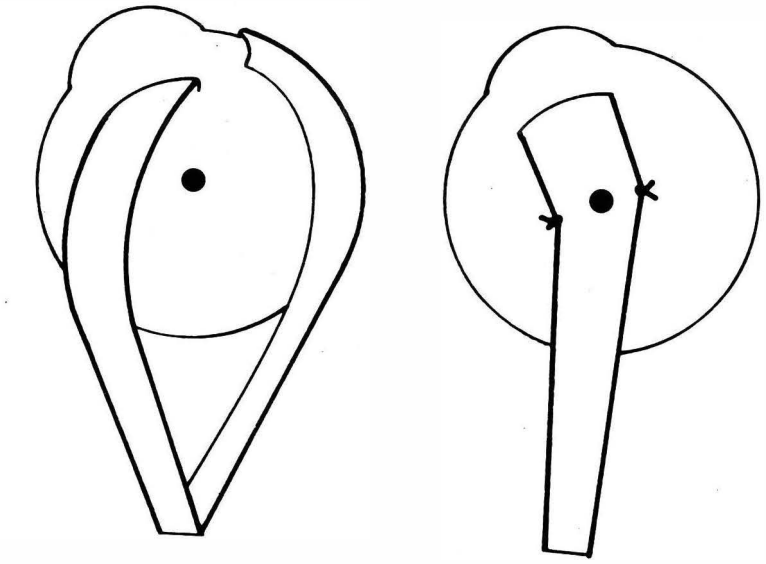
With upward gaze a vertical lever arm is added to the horizontal rectus muscles as their insertions are translated superiorly (the sabe argument, inverted, goes for infraduction). We have measured the force at the horizontal rectus muscle insertion at each margin and at the center, and find that the normal force, about 12 grams in the primary position, and the normal stiffness of the muscle, combine to shift the effective point of the force toward the lower edge of the muscle during upward gaze. This maintains the effective line of insertion near the horizontal plane, virtually abolishing any tendency of the horizontal rectus muscles to create a vertical effect. * In addition, the intermuscular membrane holds the muscle from sliding upward over the globe, adding stability and preventing vertical forces.

Even assuming the eye muscle could slip vertically upward to take a great circle path and that the tension was concentrated at only one point, these effects are not major. For example, at 15° of upward gaze there is a supraduction effect of 1°. (5% vertical torque; 2 muscles; 12 gms. tension each muscle; stiffness of globe 1.2 mg./degree, or $\frac{0.05 \text{ — } 2 \text{ — } 12 \text{ gms.}}{1.2 \text{ gm/degree}} = 1.0°$). As both eyes are equally affected, there is no binocular difficulty.

In 30° of upward gaze the vertical torque of the slipped horizontal rectus muscles has increased to about 15%. In this position the effect of both horizontal rectus muscles is approximately 3.0 degrees $\frac{(2 \text{ — } 12 \text{ gm x } .15}{1.2 \text{ gm/degree}} = 3.0 \text{ degrees}$).

As the eye moves into oblique elevation-adduction, the rotation in Listings Plane moves the medial rectus insertion to the horizontal plane. Thus, the high tension (50 gms), on this muscle in 30° of adduction, has no force whatsoever for elevation. In this position the lateral rectus, if able to slide up over the globe, has a significant vertical effect: 15 grams (the lateral rectus tension is increased slightly from the primary position due to passive stretching from the medial rectus) x 67% (the much increased vertical upgaze vector) divided by 1.2 gms/degree = 8° of supraduction tendency from the lateral rectus. It is important that the lateral rectus is normally held down and is not normally able to slip upward over



the globe. When it does, this is one cause of upshoot and downshoot in oblique adduction positions. (Fig. 1,A)

The tension is abnormally increased in these gaze positions due to co-contraction of the medial and lateral rectus muscle together or due to a lateral restriction, we have measured forces as high as 80 gms. in both of these muscles. In this position the supraduction effect of the lateral rectus is very great: $\frac{80 \text{ gms} \times .67\%}{1.2 \text{ gms/degree}} = 45^{\circ}$ of supraduction!

At surgery we have identified the lack of intermuscular membrane tissue in such cases holding the lateral rectus (and the medial rectus in some cases) in the normal pathway. This absence allows the muscle to slide upward. In some Duane's syndromes this occurs only in adduction and elevation, not in depression. We suppose this is caused by abnormal increase in innervation to the superior rectus and the inferior oblique creating slight elevation with adduction which allows the horizontal rectus muscles to begin their abnormal contribution. In the normal situation forces increa-

singly tend to restore the muscle back into horizontal line; in this abnormal situation the further the eye elevates or depresses the worse is vertical tendency of the horizontal rectus muscles. If this force occurs only for a vertical gaze upward constantly, the intermuscular membranes are stretched is this one direction, allowing vertical contribution by the horizontal rectus muscle as it slips over the globe.

An additional factor in Duane's syndrome operating with vertical gaze is that co-contraction of the medial and lateral rectus may occur with gaze upward or downward. [2, 5] Thus, if the lateral rectus increases during upward gaze this sets the stage for the high horizontal rectus tensions necessary to produce the overshoots. In such cases, the lateral rectus typically diminishes innervation in downgaze thus reducing the horizontal rectus force. Thus, the combination of "V" pattern with overshoot in elevation but not overshoot in depression is common. Less frequently one sees the reverse: "A" pattern Duane's syndrome with downshoot, but without upshoot. In the majority of cases, however, both upshoot and downshoot exist. It is not necessary that abnormal innervation occur. For example, most Duane's syndrome cases are physically tight to passive adduction (even without abnormal innervation) due to structural tightness of the lateral rectus muscle or lack of its relaxation during attemptted adduction. In the tight lateral rectus syndrome with scar tissue or a tight lateral rectus from extensive and prolonged exotropia, the force is simply one of high tension produced by contraction of the medial rectus against unyielding lateral tissue. These concepts were mathematically modelled by Robinson [6] and substantially support this description.

TREATMENT

Treatment may be directed in 3 avenues. The first is to reduce the abnormal forces by recessing the lateral rectus and medial rectus and lateral tissues posteriorly. This suffices in the tight lateral rectus syndrome and in large angle exotropia to reduce the vertical upshoot and downshoot. A substantial recession of these tissues to a point so the effective insertion will be approximately at the equator when the eye is in adduction, is appropriate. In such cases, an "X" pattern (greater exotropia in upgaze and in downgaze that exists in the primary position) may have existed prior to surgery. [7] The abduction in upward and downward gaze in such cases is probably the consequence of structural contracture of the oblique

muscles. In the manner similar to that which occurs in Brown's syndrome, the eye is prevented from moving into full adduction-supraduction or adduction-infraduction by the tight oblique muscles, and is thus turned into abduction during vertical upgaze movements. These "X" patterns gradually disappear as these oblique muscles stretch over the months following horizontal alignment. Recession of the lateral rectus alone in exotropia Duane's syndrome is effective on the overshoots.

Second, one may move the insertions of the horizontal recti to a point near the equator to prevent the upward sliding effect. Of course, this mechanism is also a component of any recession operation (See Souza-Dias, this volume).

Third, in Duane's syindrome where the abnormal forces occur Without strabismus deviations, it is possible to make a Faden operation [8] fixation suture posterior to the equator on the horizontal recti. This prevents the upward slipping of the muscle(s) over the globe and maintains it in a horizontal pathway so that the vertical upshoot cannot occur. (Fig. 1, B). This has dramatically abolished the upshoot in the cases presented here.

ABSTRACT

Abnormal vertical forces and abnormal vertical slip over the globe of horizontal rectus muscles cause upshoot and downshoot in some Duane's syndromes, the tight lateral rectus syndrome, and in large angle exotropia. Prevention of vertical slip by recession of the lateral rectus insertion to the equator, or by creation of a positive insertion suturing the muscles to the sclera at the equator abolished the upshoots and downshoots.

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