SACCADIC VELOCITY MEASUREMENTS IN STRABISMUS

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INTRODUCTION

TRADITIONAL EVALUATION OF PATIENTS WITH STRABISMUS INCLUDES MEASUREment of muscle balance at distance and near fixation as well as an evaluation of ocular rotations in the nine cardinal positions of gaze. Additionally, determination of the deviation in lateral gaze, as well as upward and downward, is useful. An array of sensory tests can sometimes provide interesting information and forced traction testing, under anesthesia in pediatric patients or in the outpatient setting for teenagers and adults, is also of value.

When ocular electromyography became available, it was possible to study the co-firing and reciprocal innervation of various extraocular muscles. Normal muscle firing patterns could be compared with those of paralytic muscles, and recovery could be documented. However, ocular electromyography was available at only a few centers, required sophisticated equipment, and was not applicable to pediatric patients because of some discomfort with the test and their inability to cooperate properly for the study.

An objective test that could provide information about the strength of an extraocular muscle, yet be relatively simple, inexpensive, safe, and able to be performed on patients of all ages would be a useful addition to our evaluation of patients with strabismus. Information would be available to assist in diagnosis and aid in management decisions.

Direct force measurements of muscle force are difficult to perform quantitatively and are usually not possible in the pediatric age group. However, eye movements can be measured relatively easily and accurately with equipment available in many hospitals and medical centers.

Dodge[1] attached the name "saccadic movement" to the rapid changes

TR. AM. OPHTH. SOC. vol. LXXXI, 1983

in eye position typically found between fixational pauses during reading. He also observed that movements constituting the fast phase of nystagmus had similar characteristics. Saccades have a latency of 150 to 200 ms and, once started, cannot be stopped.[2] Electromyographic recordings during a saccade show a large motor unit input.[3]

The velocity of a saccadic eye movement is directly related to the force produced by an extraocular muscle and is thus an indicator of the strength of the muscle. It is not proportional in a simple, linear relationship.[4] A saccade is also a stress test for an extraocular muscle since the muscle must essentially function normally to produce a saccadic movement with rapid velocity.

In 1968 I initiated preliminary studies on saccadic velocity measurements in patients with extraocular muscle palsies, restrictive ocular motility disorders, and various neuro-ophthalmic diseases with strabismus. This thesis is a more conclusive, definitive investigation to evaluate the uses and value of saccadic velocity testing. The sections on superior oblique palsy, paretic vs nonparetic eye fixation, prism calibration of eye movements, saccades in postoperative restrictions, and botulinum toxin injections are areas that had not had preliminary studies.

There are numerous ways to measure eye movement.[5] Direct observation is simple and inexpensive but has the obvious shortcomings of the absence of quantitative data and lack of permanent records. Mechanical transducers are an older method, but interfere with normal eye movements. Direct photographic recording is accurate but requires the head position to be accurately fixed and may consume vast quantities of film. The corneal reflection principle is a special case of direct photographic recording, using the corneal "highlight." There is no interference with normal eye movement, but the linear range is restricted to about 12° and disturbance resulting from lateral head movement is more significant than with other methods. Contact lens reflection is an extremely sensitive technique and has yielded good records of eye movements of less than 10 seconds of arc. However, a foreign object must be placed on the eye. The lens must fit tightly to avoid slipping, which can cause patient discomfort. Even with a tight fit, a small amount of slippage probably occurs with saccadic jumps of greater than 5°. Photoelectric measurements optically detect the position of the limbus and convert the optical information into a voltage signal that may be easily recorded. There is no interference with normal eye movement, but the maximum range depends on the visibility of the limbus. This method is therefore not useful for vertical eye movements; lateral head movements provide a false indication of eye movements, and the head must be carefully fixed.

ELECTRO-OCULOGRAPHY

It has been known since the early 1920s that certain electrical changes are associated with eye movement. Mowrer et al[6] found that by placing electrodes on the skin around the eye, potential differences could be measured between electrodes in the plane of any eye movement. The source is the potential difference between the cornea and retina, resulting in an electrostatic field that rotates with the eye. The cornea is about 1 mV positive with respect to the retina because of the negative potential associated with the higher metabolic rate at the retina. With electrodes placed on both sides of the eye and above and below the eye, potentials indicating both horizontal and vertical eye movements may be recorded without interference to normal eye movements or from head movements. The method is useful and convenient for recording movements in the range of 0.5° to 40°. The method is linear over a small segment of the range, but careful calibration can be used for determination of the extent of large movements.

The technique of electro-oculography (EOG) was chosen for determinations of saccadic velocity since this method could be used in infants and young children without requiring head fixation, and the instrumentation was available and relatively inexpensive. The reduced accuracy, when compared with other techniques of eye movement measurement, was not considered a significant drawback for these clinical determinations.

Both average and peak saccadic velocity measurements were made. Primary attention was given to the eye movement tracing itself, which provided information about average velocity and allowed inspection of the shape of the tracing for comparison with normal recordings.

MATERIALS AND METHODS

Saccadic velocity measurements were made by EOG.[7] For horizontal saccades, Beckmann miniature skin electrodes were placed on the skin just beside the medial and lateral canthi, with the indifferent electrode on the brow (Fig 1, left). Vertical saccades were recorded by placement of the electrodes centrally above the brow and on the lower eyelid, with the indifferent electrode temporal to the lateral canthus (Fig 1, right).

A dynograph with modified rectilinear ink writers, dynograph amplifier, preamplifier, direct nystagmus coupler, and nystagmus velocity coupler were used for EOG recordings. The eye position channel had a band width of 0 to 10 Hz. The gain was 0.6 μV/cm. The velocity channel had a band width of 2.2 to 10 Hz with a gain of 5 μV/cm (Fig 2). The characteristics of this unit are probably responsible for the recording of peak

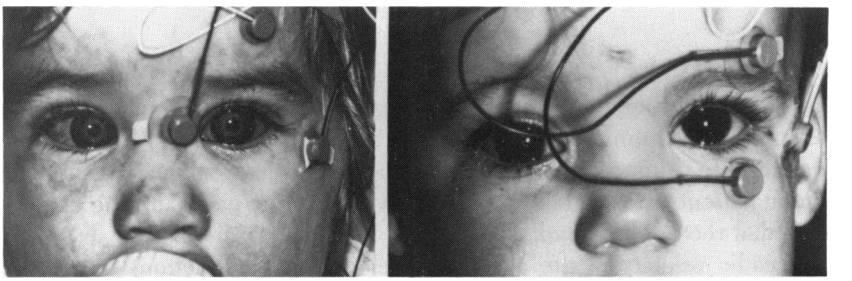

FIGURE 1
Left, Miniature electrode placement for measurement of horizontal saccades. Right, Miniature electrode placement for measurement of vertical saccades.

velocities of 350° to 450°/s in normal subjects, while other recording techniques such as limbal reflectance devices have demonstrated peak velocities of over 600°/s. The tracoustics machine, which is in common use in this country, has a band width of 0 to 30 Hz and a gain of 33 μV/cm.

Saccades were generated by voluntary movements of 20° to 40° across the primary position, either horizontally or vertically. In patients where paralysis or restriction prevented movements of this size, the largest saccades possible were recorded both left and right, or up and down, or both. Calibration was performed by having the patient perform a 20° horizontal or vertical saccade with the eye being studied used for fixation.

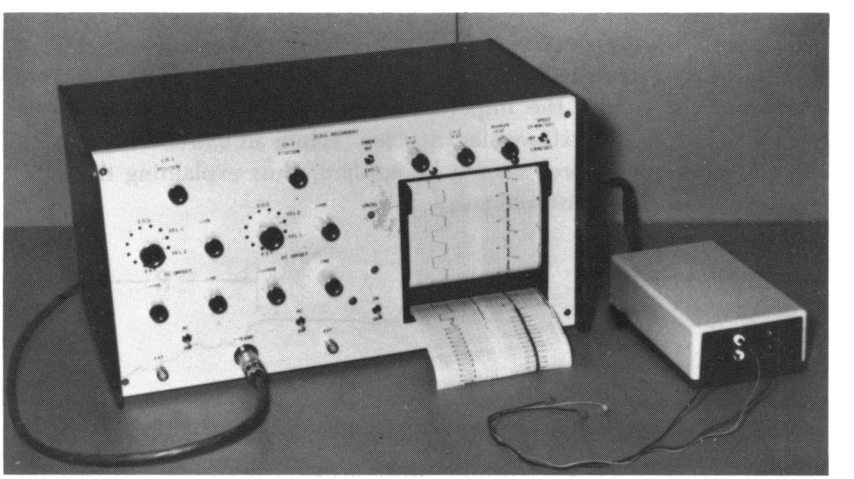

FIGURE 2
Electro-oculography unit.

EXTRAOCULAR MUSCLE PALSY

SIXTH NERVE PALSY

Thirty-seven patients with sixth nerve palsy (30 unilateral and 7 bilateral cases, 44 measurements) were studied. Saccades were made from 20° of adduction to primary gaze or 20° of abduction when possible. This varied among patients, depending upon the severity of the palsy and the extent of medial rectus restriction.

Saccadic velocity of the palsied muscle (toward abduction) was compared with the speed of the movement toward adduction in the same eye (medial rectus function) and with abduction in the opposite eye (contralateral lateral rectus function) when the palsy was unilateral. Average normal saccadic velocity for a movement 20° or greater varied between 200° and 320°/s. Saccades produced by the palsied lateral rectus muscle varied from 40°/s (Fig 3) with complete paralysis[7] to 160°/s (Fig 4) when only mild paresis was present. Recovery of the lateral rectus palsy was documented by increased saccadic velocity with time.

COMMENT AND CONCLUSIONS

In patients with marked or complete lateral rectus paralysis, visual observation of saccades laterally can reveal slowing. This slow movement is caused by relaxation of the antagonist medial rectus muscle without active contraction of the lateral rectus muscle. When only mild to moderate lateral rectus palsy is present, recordings of eye movement are needed to document the reduction in velocity.[8]

Robinson[9] has demonstrated that the system of eyeball and orbital tissue is heavily overdamped. It succeeds in making quick saccadic movements only under the impetus of a large, briefly applied, excess force delivered by the extraocular muscles. When an extraocular muscle is paretic, this excess force cannot be applied, thus explaining the slow movements in cases of muscle palsy.

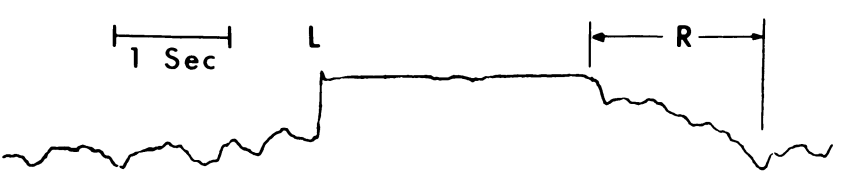

FIGURE 3
Electro-oculographic recording of horizontal saccades in patient with complete lateral rectus paralysis. Upper trace, Abduction saccades (R) are extremely slow, while adduction saccades (L) are rapid.

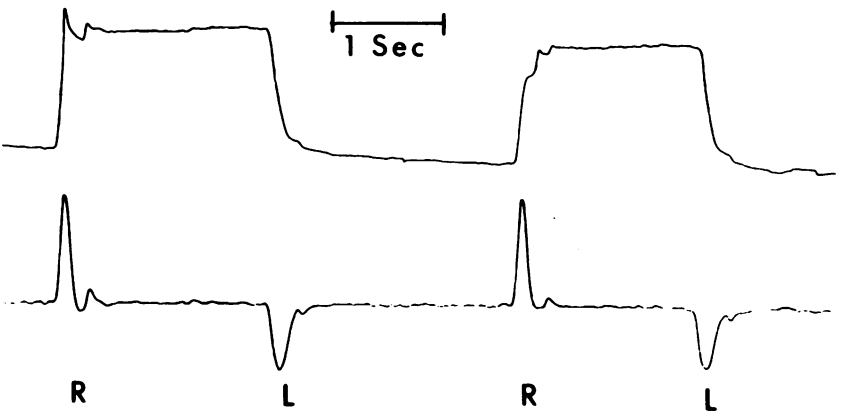

FIGURE 4
Electro-oculographic recording of horizontal saccades in patient with mild lateral rectus palsy. Upper trace, Eye position is shown. Abduction saccades (L) are mildly slowed while adduction saccades (R) are rapid. Lower trace, Peak velocity is demonstrated.

Using high-speed photography, Hyde[10] studied normal saccadic movements and noted subjects were unable to alter saccadic velocity for a fixed-amplitude horizontal movement. Thus, slow saccades could not be made voluntarily. A decrease in saccadic velocity must result from an abnormality in the oculomotor apparatus.

By studying saccadic velocity, it is possible to follow the reinnervation of a paretic muscle[7,8]; in some cases, decrease in velocity may be a more sensitive indicator of abnormality in the oculomotor apparatus than the electromyogram (EMG).[8]

THIRD NERVE PALSY

Twenty-one patients with unilateral partial or complete third nerve palsy were studied. Adduction saccades were compared with the normal saccades produced toward abduction. Vertical saccades were compared with those of the opposite, normal eye. It was found helpful to gently hold the upper lid to avoid the artifact sometimes noted with EOG recordings of vertical saccadic movements.

Adduction saccades in the affected eye varied from an average velocity of 30°/s to 175°/s (Fig 5).[11] Vertical saccades varied from 10°/s to 150°/s (Fig 6), depending upon the extent of the palsy. These slower velocities probably resulted from the fact that some of the vertical saccades were smaller than 20° in amplitude because of significant weakness of both the superior and inferior rectus muscles.

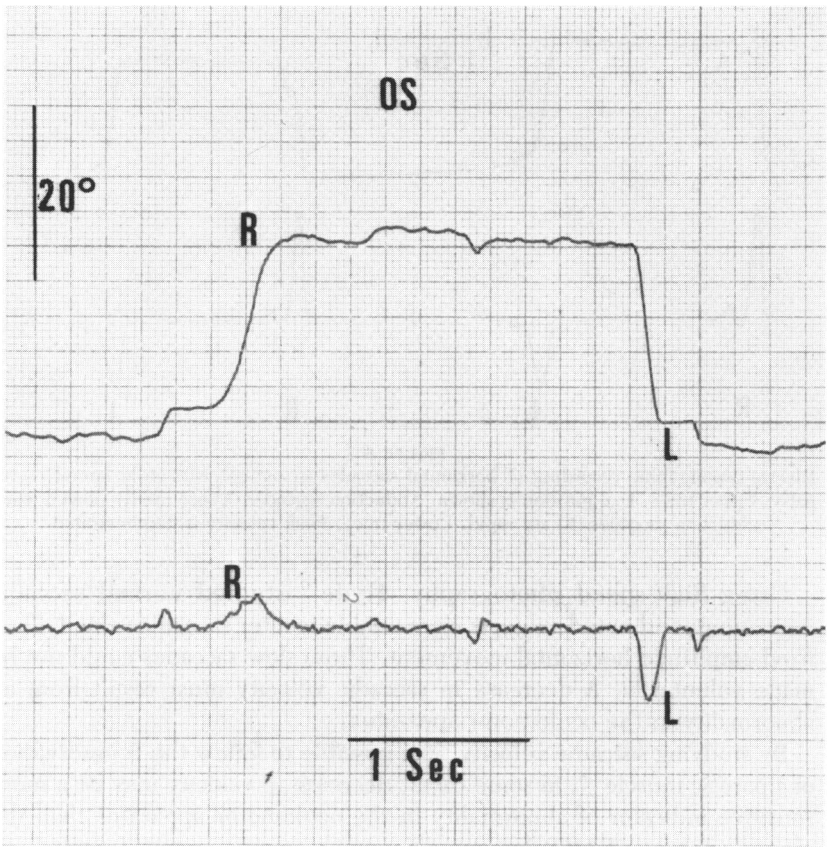

FIGURE 5
Electro-oculographic recording of horizontal saccades in patient with third nerve palsy. Upper trace, Adduction saccades (R) are slowed, while abduction saccades (L) are rapid. Lower trace, Peak velocity is shown.

COMMENT AND CONCLUSIONS

Following complete third nerve paralysis, the eye typically assumes a slightly hypotropic and markedly exotropic position. If this deviation persists, the lateral rectus muscle and adjacent tissues usually become shortened and contractured, and nasal rotation of the globe becomes mechanically limited. If medial rectus muscle function recovers, nasal rotation will remain deficient because of lateral restrictions. Studies of saccadic velocity can reveal the extent of recovery of the medial, superior, and inferior rectus muscles.

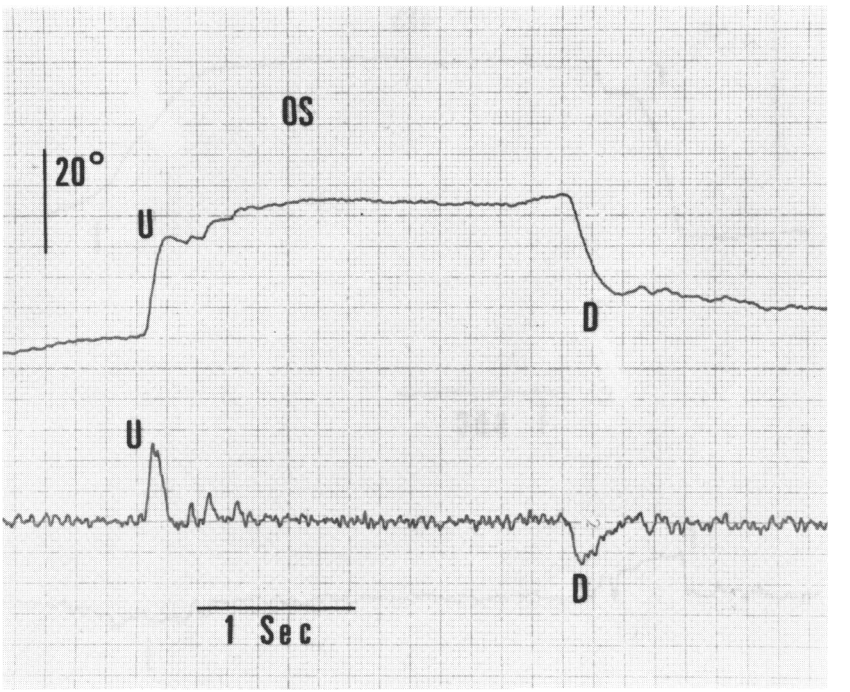

FIGURE 6
Electro-oculographic recording of horizontal saccades in patient with third nerve palsy. Upper trace, Upward (U) and downward (D) saccades are both slow. Lower trace, Peak velocity is demonstrated.

DUANE'S SYNDROME

Huber's classification of Duane's syndrome was used in describing our patients.[12] In type 1 (limited abduction with retraction on adduction), there were 38 patients. There were seven patients in type 2 (exotropia with good abduction but limited adduction and retraction on adduction), and three patients in type 3 (limited adduction and abduction associated with retraction on adduction).

In this group of 48 patients, 67% were women. Fifteen percent had bilateral involvement, 63%, involvement of the left eye (OS) only, and 22%, involvement of the right eye (OD) only. In primary position, there was no deviation in 26% of the patients, esotropia in 60%, and exotropia in 14%.

Type 1[13]—The average abduction saccadic velocity was 90°/s (range 45° to 160°/s). The average adduction velocity was 150°/s (range 70° to 200°/s) (Fig 7).

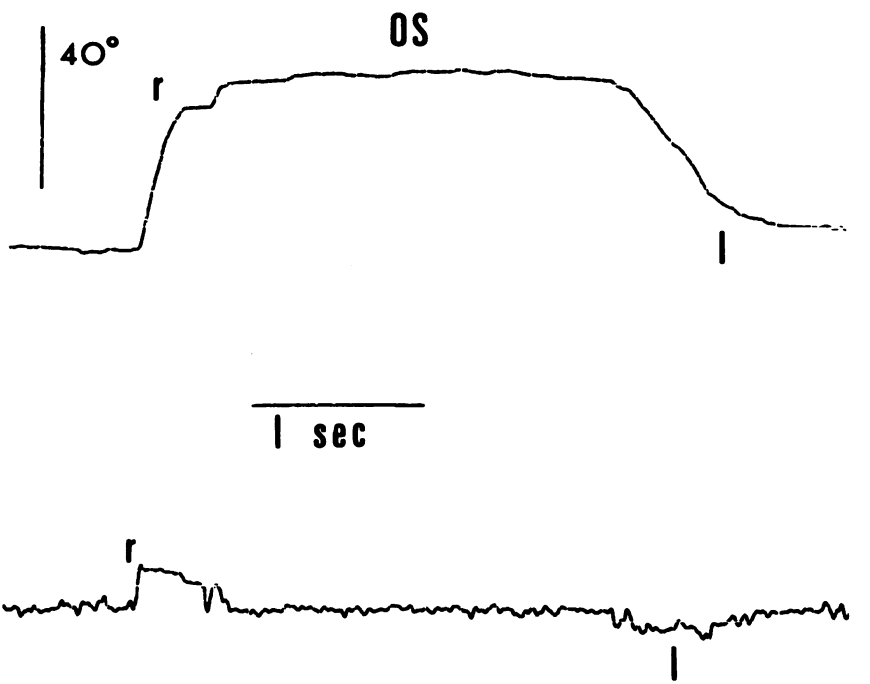

FIGURE 7
Electro-oculographic recording of horizontal saccades in patient with Duane's syndrome, type 1, OS. Upper trace, Abduction saccades (l) are extremely slow (70°/s), while adduction saccades (r) are moderately reduced in velocity. Lower trace, Peak velocity is demonstrated.

Type 2—The average abduction saccadic velocity was 200°/s, while average adduction saccadic velocity was 100°/s (Fig 8).

Type 3—The average abduction velocity was 60°/s, while average adduction velocity was 110°/s (Fig 9).

COMMENT AND CONCLUSIONS

There was marked reduction in abduction saccadic velocity in type 1 Duane's syndrome. This is explained by the ocular electromyographic evidence of lack of muscle firing or recruitment of the lateral rectus muscle on attempted abduction.[14,15] Adduction saccadic velocities were moderately to mildly decreased. Ocular electromyography[14,15] indicates that the medial rectus recruits and inhibits normally in Duane's syndrome. The cause of reduced adduction velocity is probably the paradoxic innervation of the lateral rectus muscle on attempted adduction. This differentiates type 1 Duane's syndrome from lateral rectus palsy.

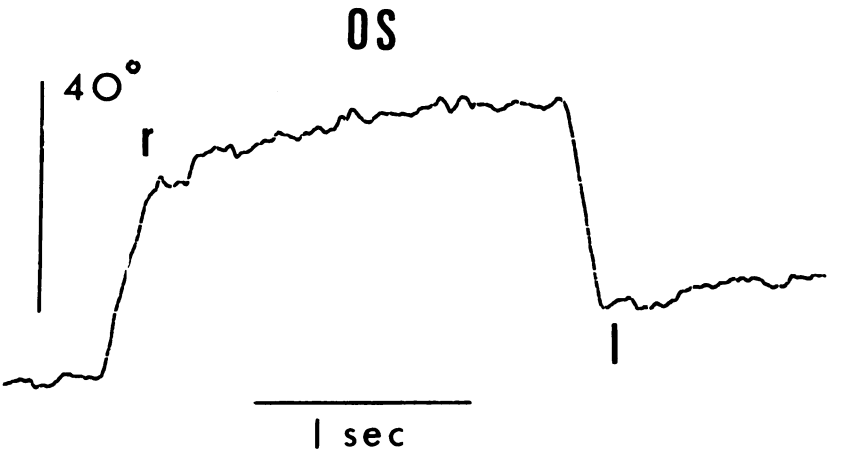

FIGURE 8
Electro-oculographic recording of horizontal saccades in patient with Duane's syndrome, type 2, OS. Abduction saccades (l) are rapid, while adduction saccades (r) are moderately slow.

In one patient with type 1 Duane's syndrome, horizontal saccades were measured before and after lateral rectus recession of the involved eye. Abduction saccades were unchanged, while adduction saccades improved to normal. It is possible that the lateral rectus muscle, which had been co-contracting on adduction, now had less effect following recession, with resultant improvement in the speed of nasal movements.

The importance of identifying anomalous co-contraction of the lateral rectus muscle has been stressed by Blodi et al[16] and by Pabst and Esslen.[17] They suggest that an exact knowledge of the innervational behavior of the horizontal rectus muscles is necessary before planning an operative procedure.

In 18 of our type 1 Duane's syndrome patients, horizontal saccadic velocities were normal in the opposite eye in 17 patients. This is not in agreement with a recently published report by Gourdeau et al[18] in which adduction saccades were found reduced in velocity in the unaffected eye in two of five patients.

SUPERIOR OBLIQUE PALSY

Eighteen patients (21 eyes) with superior oblique palsy were studied. The diagnosis was made by a history of an acquired vertical deviation (with diplopia), a positive Bielschowsky head tilt test, the frequent finding of a V pattern, and overaction of the antagonist inferior oblique muscle; in a

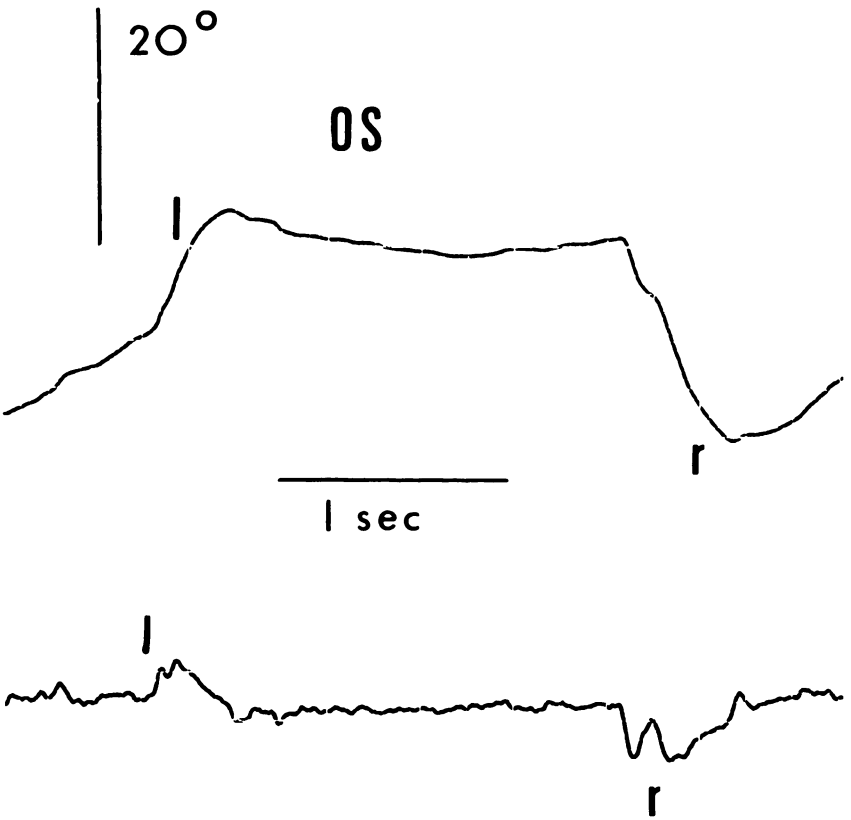

FIGURE 9
Electro-oculographic recording of horizontal saccades in patient with Duane's syndrome, type 3, OS. Upper trace, Both abduction saccades (l) and adduction saccades (r) are extremely slow (45°/s and 60°/s, respectively). Lower trace, Peak velocity is demonstrated.

few cases, underaction in the field of the paretic superior oblique muscle was also present. The group consisted of ten women and eight men. Eight patients had paresis in the right eye; seven, in the left eye; and three cases were bilateral.

Forty-degree saccades were measured from 20° upward to 20° downward in adduction, primary position, and abduction. Vertical saccades were measured in the nonparetic eye for comparison.

In primary position, upward saccades averaged 283°/s, while downward saccades averaged 238°/s. In adduction, upward saccades averaged 317°/s and downward saccades, 267°/s (Fig 10), while in abduction, upward

saccades averaged 332°/s and downward, 279°/s. In all patients where there was a decrease in downward saccadic speed compared with the upward saccade, the opposite, normal eye showed the same, small difference with our technique. It is usual to find upward saccades in normal subjects to be slightly more rapid than downward saccades (about a 15% difference). The range of normal vertical saccades in our laboratory is 200° to 350°/s for 40° movements. All upward saccades were within this range, and only two eyes fell below this range for downward saccades.

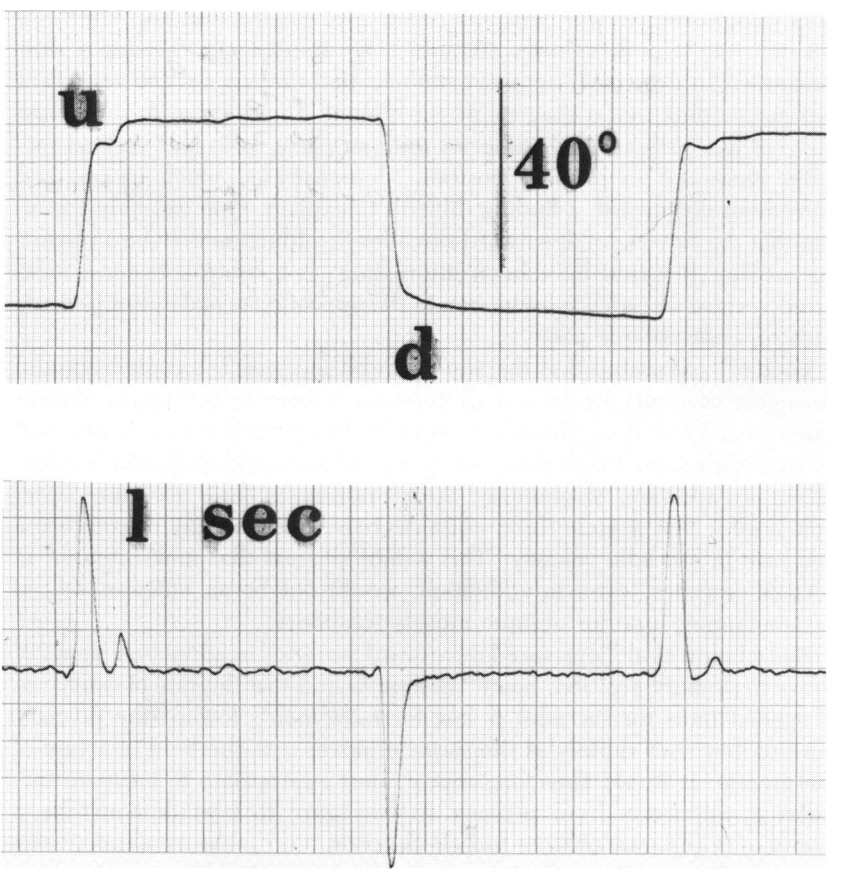

FIGURE 10
Electro-oculogram (EOG) of patient with superior oblique palsy. Upward (u) and downward (d) saccades in adduction are normally rapid. Eye position (upper trace) and peak velocity (lower trace) are shown.

Two patients had vertical saccadic velocity studies before and after bilateral superior oblique tenotomy. Preoperatively, upward and downward saccades averaged 220°/s in primary gaze, with minimal change in adduction and abduction. Postoperatively, no difference in vertical saccadic velocity was measured in either direction.

COMMENT AND CONCLUSIONS

Rosenbaum and associates[19] found that downward peak saccadic velocity was markedly slowed in patients with superior oblique weakness compared with similar measurements in a normal control group. Although peak velocity figures were given in this report, the tracings only show eye position, not a peak velocity channel. The one tracing showing a slow downward saccade after superior oblique tentomy appears to represent two movements, not a slow single movement. In addition, in this series three of five patients with superior oblique palsy, who had vertical saccades measured in primary position, demonstrated much more rapid movement downward, a finding difficult to explain. One can only speculate that the slowing of downward saccades in adduction may be a recording artifact. It would have been interesting, as a control, to have measured vertical saccades in adduction in the opposite normal eye of patients with superior oblique palsy.

Boeder[20] calculated that the superior oblique muscle's contribution to downgaze accounts for 18% of all depression force in adduction. Rosenbaum et al[19] found a reduction in velocity that varied between 54% and 97% in adduction, which does not appear consistent with Boeder's calculations. In addition, Magoon and Scott[4] indicate in their oculinum injection studies that muscle force must decrease substantially to produce a decrease in saccadic velocity. This would suggest that measurement of vertical saccades, even in adduction, would not be an efficient way of demonstrating superior oblique muscle weakness.

Rosenbaum et al[19] measured downward saccades in adduction from 20° above to primary position. Scott[21] demonstrated that the rate of discharge of agonist units for saccades is low for movements toward the primary position but rises rapidly as the saccade moves into the field of action of the muscle. It would therefore appear most appropriate to measure saccades in adduction from primary to downgaze to best demonstrate a decrease in superior oblique muscle function. Our measurements in this area failed to reveal any decrease in vertical saccadic velocity.

Scott[21] has injected procaine hydrochloride (Novocain) with EMG guidance to paralyze individual vertical eye muscles for physiologic investigation. There was a small effect on peak saccadic velocity and isometric

force from oblique paralysis, but a marked reduction from vertical rectus paralysis. Scott[21] remarked that these contributions to force and velocity were similar to his analysis of the percentage participation of oblique and rectus muscles to the amplitude of vertical movements.

FIXATION: PARETIC VS NONPARETIC EYE

There has been some controversy concerning the most appropriate method for measuring eye movements in patients with ocular muscle palsy. The paretic eye or the nonparetic eye may be used for fixation (although the paretic eye must be used for fixation when calibration is performed). The rationale for employment of the nonparetic eye for fixation is that normal innervation will be exerted to drive movement of the paretic eye. The reduction in saccadic velocity resulting from muscle palsy might therefore be more easily recognized than if the paretic eye was used for fixation (since the movement might be driven by supramaximal input by the paretic eye).

Twenty-seven patients with monocular rectus muscle palsy were tested. Diagnoses included lateral rectus muscle palsy, partial third nerve palsy with superior, medial, or inferior rectus muscle weakness, and Duane's syndrome. Some patients had only mild paresis while others had complete paralysis. Voluntary horizontal or vertical saccades of equal size were made with each eye fixating. In all patients, saccadic velocity was equal with either eye fixating (Fig 11). The differences were less than 10%. In some patients, smaller ampltiude movements were noted with the normal eye fixating. In these instances, the uninvolved eye then made larger movements to produce an eye movement in the paretic eye that would be equal to that made when the paretic eye was used for fixation; in this way, equal-amplitude saccades could be compared. In the three patients with Duane's syndrome, the mild slowing of adduction saccades (caused by lateral rectus muscle co-contraction) was also equal with each eye fixating.

COMMENT AND CONCLUSIONS

Lennarson and Scott[22] measured patients with lateral rectus muscle palsy both with the paretic eye and the normal fellow eye fixating. There was little difference in saccadic velocities. Gourdeau et al[18] noted equal slowing of adduction saccades with either eye fixating in patients with Duane's syndrome. These results are consistent with our findings. If there is a difference in velocity with the paretic eye fixating, it appears to be too small to affect the clinical significance of the measurement. It is also important to compare equal-amplitude saccades, especially into the field of action of the paretic muscle.

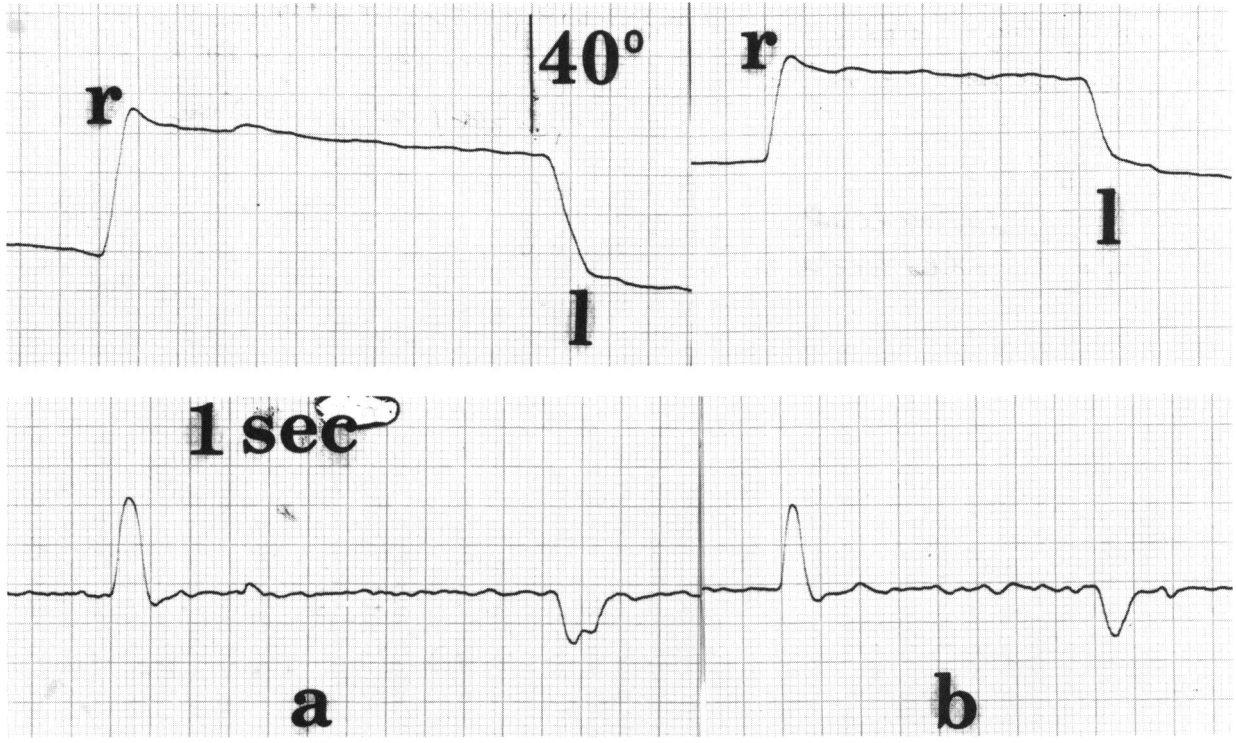

FIGURE 11

Electro-oculogram of horizontal saccades in left lateral rectus palsy with paretic eye (a) and nonparetic eye (b) used for fixation. Upper trace, Eye movement is shown. Lower trace, Peak velocity is demonstrated. There is no difference in velocity with either eye fixating, although amplitude is smaller with fixation of nonparetic eye.

INFANTS AND YOUNG CHILDREN

OPTOKINETIC AND VESTIBULAR NYSTAGMUS

Although voluntary saccades can be elicited in adults, these movements may not be easily obtained from infants and young children. The rapid recovery phase of optokinetic nystagmus (OKN) or vestibular nystagmus is a saccadic movement and can be produced as a reflex eye motion.

For horizontal OKN, the child is seated comfortably on the mother's lap within a large rotating drum containing vertical black-and-white stripes on the inside surface (Fig 12).[23] A variable-speed motor can rotate the drum in either direction and with different velocities. Because of the large size of the rotating striped field, the infant has no other fixation points to block nystagmus, and the reflex eye movement is more easily obtained. A small, rotating drum or OKN tape may also be used. To elicit vertical nystagmus, the child is placed in a supine position upon a parent's lap (Fig 13).

Vestibular nystagmus may be easier to obtain in infants under 1 year of age. After the EOG electrodes are in place, the examiner holds the child at arm's length and spins the child first in one direction, and then in the other. The resultant nystagmus is caused both by stimulation of the vestibular apparatus and an optokinetic stimulus provided by movement of objects in the room across the retina. Children over 1 year of age may be frightened by this and thus may not be as suitable for the technique as the younger group. Vestibular stimulation has been successfully used in a newborn 6 days of age.

Normally rapid, recovery saccades are shown in Fig 14, while Fig 15 depicts the slow recovery saccades produced by the lateral rectus muscle in a patient with sixth nerve palsy.

One example is a 21-month-old infant who suffered head trauma at age 5 months. In addition to both intellectual and motor retardation, the youngster had a large esotropia and bilateral absence of abduction. The diagnosis was bilateral sixth nerve palsy. Sixteen months after injury, muscle balance and ocular rotation were unchanged (Fig 16). Recording of OKN in each eye revealed normal lateral rectus muscle function (Fig 17). During surgery, the forced duction test revealed marked restriction to abduction bilaterally. Each medial rectus muscle was recessed 5 mm, and each lateral rectus muscle resected 7 mm for 75 prism diopters (PD) of esotropia. Postoperatively, the eyes were straight in primary gaze (Fig 18), and abduction was full in each eye.

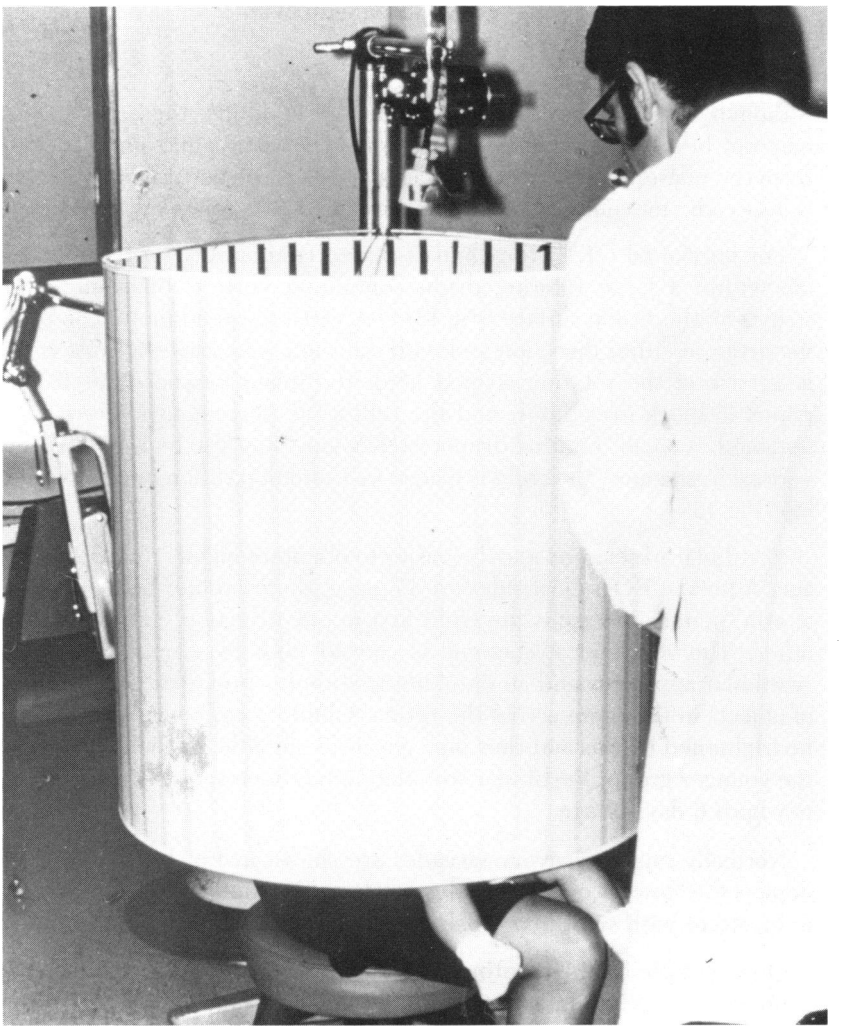

FIGURE 12
Child on mother's lap inside large drum with black-and-white stripes on inner surface.

COMMENT AND CONCLUSIONS

Investigation of saccadic velocity correctly indicated satisfactory recovery of lateral rectus muscle function bilaterally and suggested the proper surgical management necessary to achieve a good result. Had the lateral rectus muscles remained paralytic, greater amounts of recession and

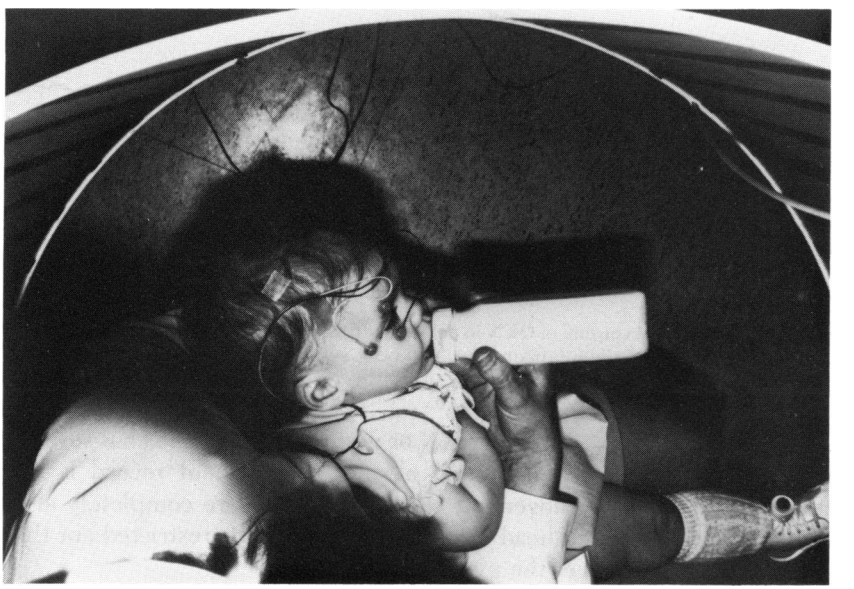

FIGURE 13
Child lying horizontally on mother's lap inside rotating drum to produce vertical nystagmus.

resection would have been required to align the eyes, or transposition surgery would have been necessary to provide some abduction as well as a straight ocular position. Active forces can be measured in adults by use of the Scott active force generation test[24] or by ocular electromyography.[3] These tests cannot readily be done in infants and young children, so other objective recordings are useful.

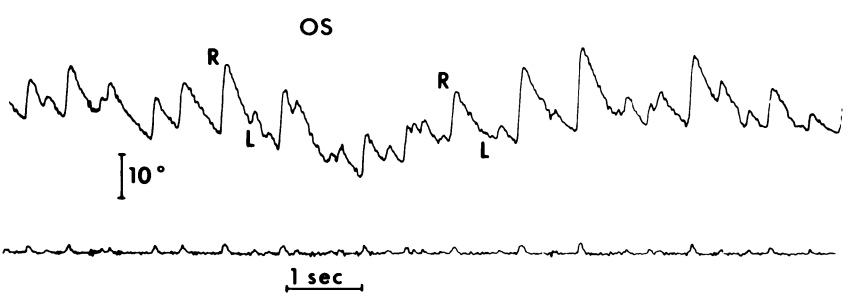

FIGURE 14
Electro-oculographic recording of normal OKN depicting rapid recovery phase of OKN movement. Eye position (upper trace) and peak velocity (lower trace) are shown.

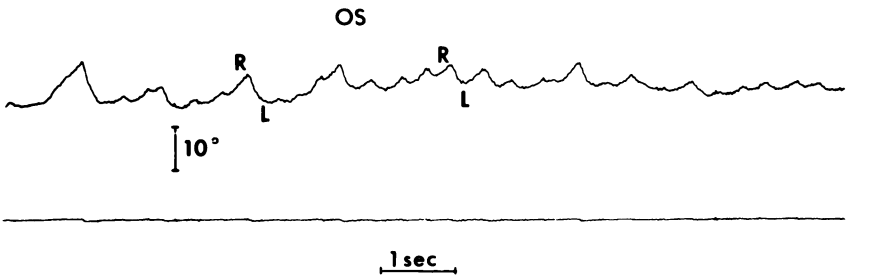

FIGURE 15
Electro-oculographic recording of OKN in patient with sixth nerve palsy. Recovery saccades (L) are slow. Eye position (upper trace) and peak velocity (lower trace) are shown.

Recording eye movements of OKN or vestibular nystagmus has several advantages over visual observation alone. A permanent record is acquired, the pattern of movement can be studied more completely at a later time, and body and head movements need not be restricted nor the lids held apart to inspect the eyes.

PRISM CALIBRATION

To measure velocity of eye movement, it is necessary to know the duration of the movement (time) and the amplitude of the movement. Recordings such as those obtained by EOG reveal the time, but amplitude must be determined by calibration, with the eye being measured changing fixation between two points a known distance apart. This is straightforward in adults, teenagers, and older children. However, in infants and young children this level of cooperation cannot be expected. Attempts at calibration by showing toys of interest a known distance apart invariably results in head movement as well as eye movement, making attempts at accurate measurement of amplitude unsatisfactory.

In an effort to provide reliable calibration in extremely young patients, the child's fixation with one eye was obtained by showing a colorful,

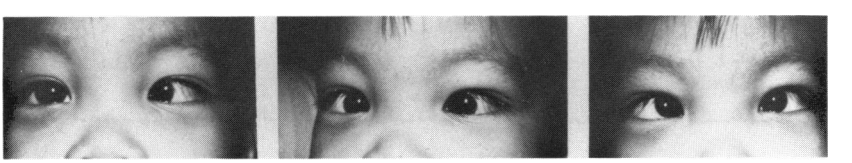

FIGURE 16
Child with large esotropia and bilateral limitation of abduction 16 months after head injury. Right gaze (left), primary gaze (center), and attempted left gaze (right), are shown.

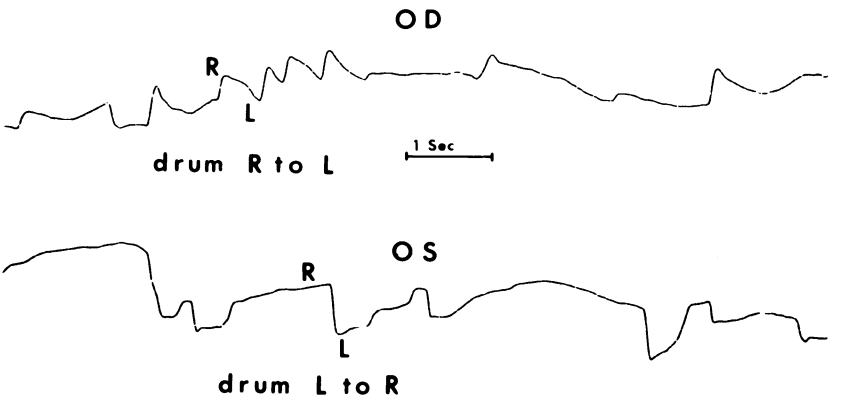

FIGURE 17
Abduction, recovery saccades produced by OKN are rapid in OD (top) and OS (bottom).

moving, noise-producing toy at a distance of one third of a meter. A 10-diopter prism was introduced before the fixating eye, and the resulting horizontal eye movement was recorded by EOG. The infant would usually not move his head but only his eye to effect this small-amplitude movement caused by prism displacement of the image. The recorded eye movement can be used for calibration since the distance the eye moved is known.

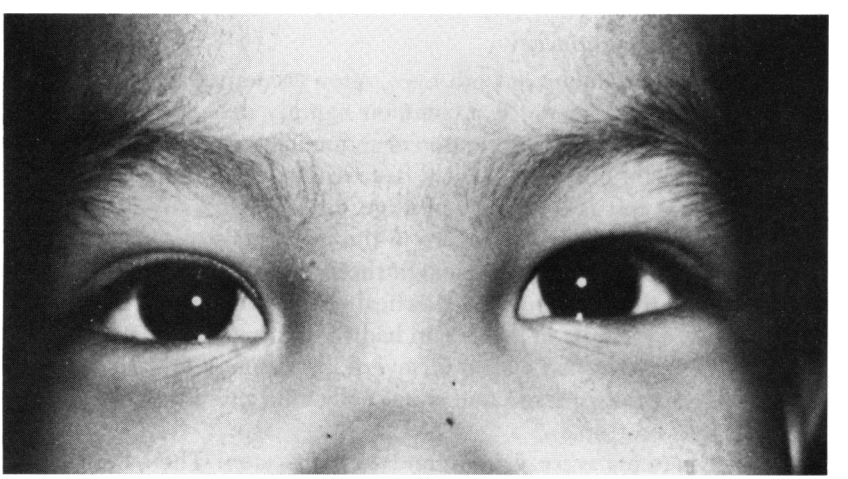

FIGURE 18
Postoperatively, eyes are straight in primary position.

To test the correlation between prism-induced and voluntary eye movements, 12 patients cooperative enough to make accurate voluntary saccades were tested to compare the size of the EOG-recorded eye movement induced by 5° voluntary movements and 10 PD-induced movements. The group consisted of three male and nine female patients aged 10 to 79 years. The eye movement amplitudes were equal with both methods (Fig 19). No difference was noted whether the prism was inserted base-out or base-in.

Ten infants were tested with this technique without significant difficulty. All were too young to compare the prism method with voluntary saccades.

COMMENT AND CONCLUSIONS

Calibration is usually achieved by having the subject fixate on points of known visual angle separation or track a spot moving through a known displacement. With young children, such tasks make more demands on comprehension and cooperation than are ideal. The use of prism-induced image jump before one eye, while the contralateral eye is occluded, to induce the calibration eye movement circumvents this problem. It allows simple fixation of a stationary target and provides an involuntary response.

RESTRICTION

ENDOCRINE OPHTHALMOPATHY

Limited elevation of one or both eyes, often accompanied by exophthalmos and eyelid retraction, is a common motility defect seen in patients with thyroid eye disease. Limitation of abduction, adduction, and depression may also be observed with a decreasing order of frequency. The forced traction test is invariably positive, most notably to upgaze.

Thirty-one patients studied were in the noncongestive, stable stage of endocrine ophthalmopathy. Although three patients had no deviation in primary gaze, the remainder had vertical strabismus ranging from 5 to 75 PD. Fourteen patients in this group had co-existing esotropia (range 4 to 45 PD). All patients had a mild to severe limitation of upgaze in at least one eye. Limitation of abduction was usually mild and was noted in the 14 patients with esotropia.

Twenty patients were women and 11 were men. Their ages ranged from 37 to 71 years. There was one exception with the acute onset of thyroid ophthalmopathy in a 16-year-old girl.[25]

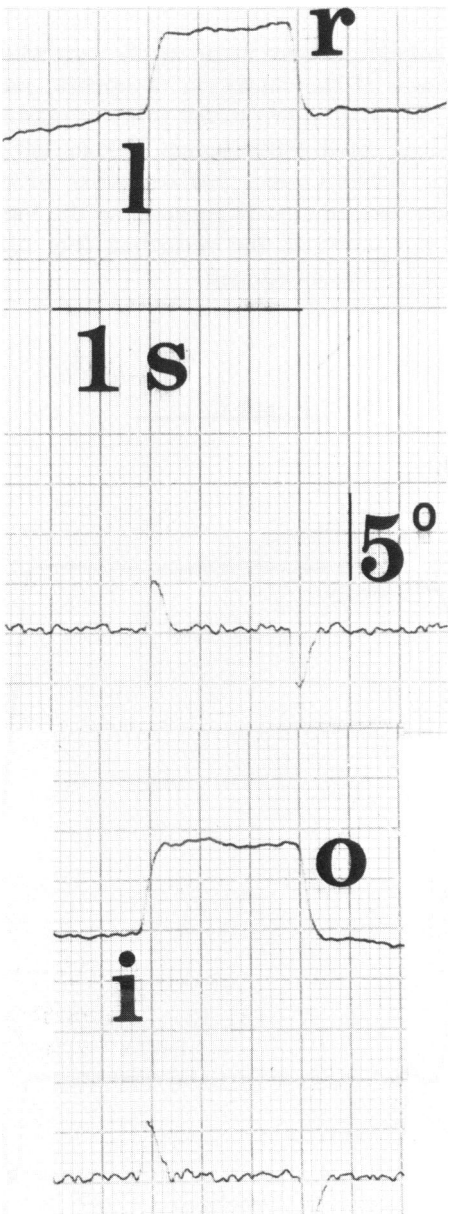

FIGURE 19

Electro-oculographic recording of saccadic eye movements resulting from 5° horizontal voluntary saccades (top) and 10 PD base-out-induced saccades (bottom). Amplitudes are equal. Note movement to right (r) and to left (l). Prism was inserted (at i) and removed (at o).

Saccades of 40° amplitude were made in the area where the eye was free to move. Because of the limitation of upgaze, saccades in some patients were made from 40° down to the primary position.

Upward saccades averaged 260°/s (range 200° to 330°/s) while downward saccades averaged 240°/s (range 180° to 320°/s)[26] (Fig 20). When the extent of the saccade upward closely approached the limit of rotation, a "tailing off" effect was noted in the recording (Fig 21). Velocity was reduced near the end of the movement.

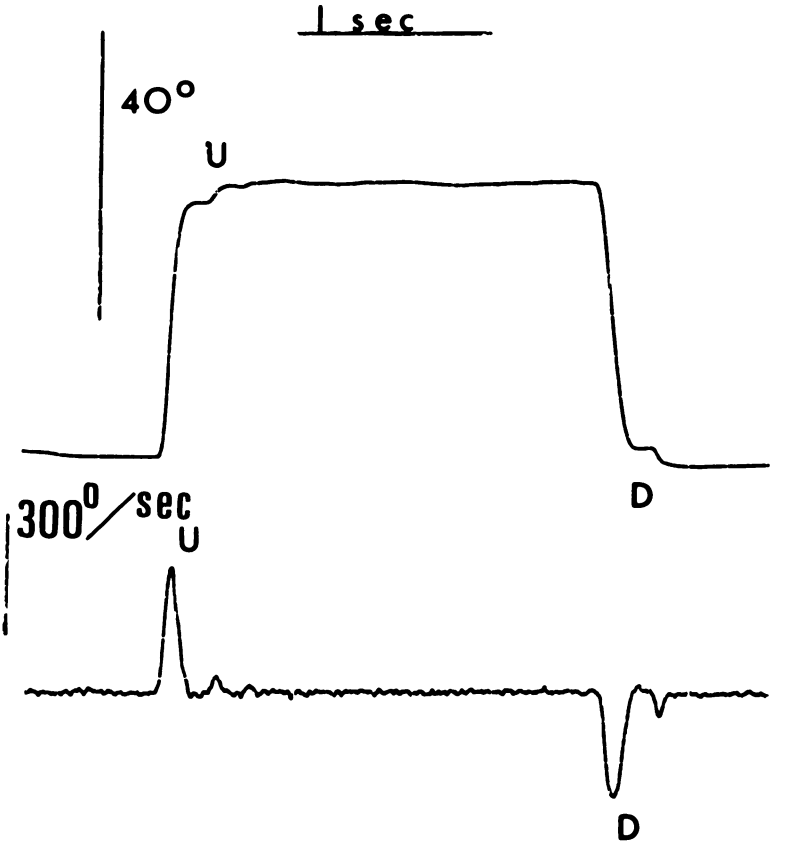

FIGURE 20
Electro-oculographic recording of vertical saccades in patient with endocrine ophthalmopathy. Both upward (U) and downward (D) saccades are rapid and equal. Eye position (upper trace) and peak velocity (lower trace) are shown.

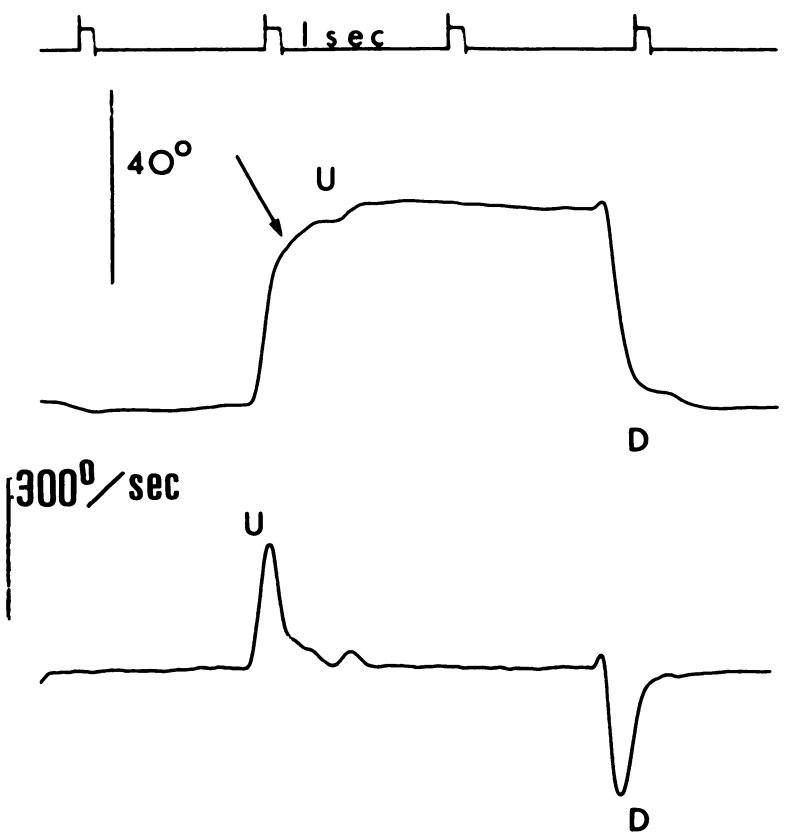

FIGURE 21
Electro-oculographic recording of vertical saccades in endocrine ophthalmopathy. Upward (U) saccades are initially rapid, then considerably slowed (tailing off) at end of movement (arrow). Downward (D) saccades are rapid. Eye position (upper trace) and peak velocity (lower trace) are shown.

Abduction saccades averaged 250°/s (range 180° to 320°/s), while adduction saccades averaged 260°/s (range 200 ° to 330°/s). The values of both vertical and horizontal saccades were within normal limits for our laboratory.

COMMENT AND CONCLUSIONS

Kroll and Kuwabara[27] indicated that extraocular muscles were firm, rubbery, and enlarged in dysthyroid ocular myopathy. Histologically, interstitial edema and round cell infiltration were found. There was marked resistance to passive stretching, most prominently in the inferior and

medial rectus muscles. Pratt-Johnson and Drance[28] indicated that confusion may arise between inferior restriction and superior rectus muscle palsy in dysthyroid ocular disease, particularly when the forced duction test is not obviously positive. Ocular electromyography has generally shown a myopathic pattern in the extraocular muscles with normal recruitment.[15,29]

Our saccadic velocity measurements were within normal limits, suggesting the absence of extraocular muscle paresis in the noncongestive stage of endocrine ophthalmopathy. These findings, combined with a positive forced duction test, implicate mechanical restriction, not weakness, as the cause for limited ocular rotations and strabismus. Release of these restrictions surgically improves the range of rotations and reduces or eliminates strabismus.[30]

Recently a report by Hermann[31] has shown evidence of inferior rectus muscle weakness (with a slowing of the downward saccade) in two patients with dysthyroid eye disease. Judging from our experience, this appears to be an unusual finding.

When saccades were made toward the limit of the range of ocular rotation, a decrease in velocity was noted. This may be caused by restriction of the antagonist muscle. The restriction can probably be stretched out by the force of the saccade, but does not release completely, thus slowing the saccadic movement at its end. This pattern becomes apparent only as the eye movement approaches its limit. It is different from the reduced velocity produced by paresis, where the entire movement is slow.

FRACTURE OF THE ORBITAL FLOOR

In patients with fracture of the orbital floor, it may be difficult to assess the functional status of the inferior rectus muscle solely on the clinical findings. Incarceration of orbital tissue often causes restriction to ocular elevation and depression, discomfort with attempted vertical rotations, and a vertical deviation in primary position. Paresis of the inferior rectus muscle, orbital hemorrhage, and edema may produce similar results. Smith and Regan[32] indicated that diplopia in association with fracture of the orbital floor is usually caused by incarceration of orbital tissues into the fracture site rather than by prolapse of orbital contents into the maxillary antrum alone. These tissues include orbital fat and inferior rectus and inferior oblique muscles and their connective tissue sheaths. The inferior rectus muscle may be paretic or normal in its action, depending upon the degree of injury caused by restriction at the fracture site.

Fifty-nine patients with blow-out fracture of the orbital floor underwent studies of vertical saccadic velocity.[33] The diagnosis was made surgically or roentgenographically or both. In subgroup 1 40 patients had no deviation in the primary position postoperatively; patients in subgroup 2 had residual hyperdeviation on the side of the injury postoperatively after repair of the floor fracture.

Because there was variability among patients in the size of the vertical saccade that could be made on the side of the blow-out fracture, percentage differences between upward and downward velocities were calculated. Patients in subgroup 1 had an average difference of 12% between upward and downward saccadic velocities (0% to 30% range) (Fig 22), and

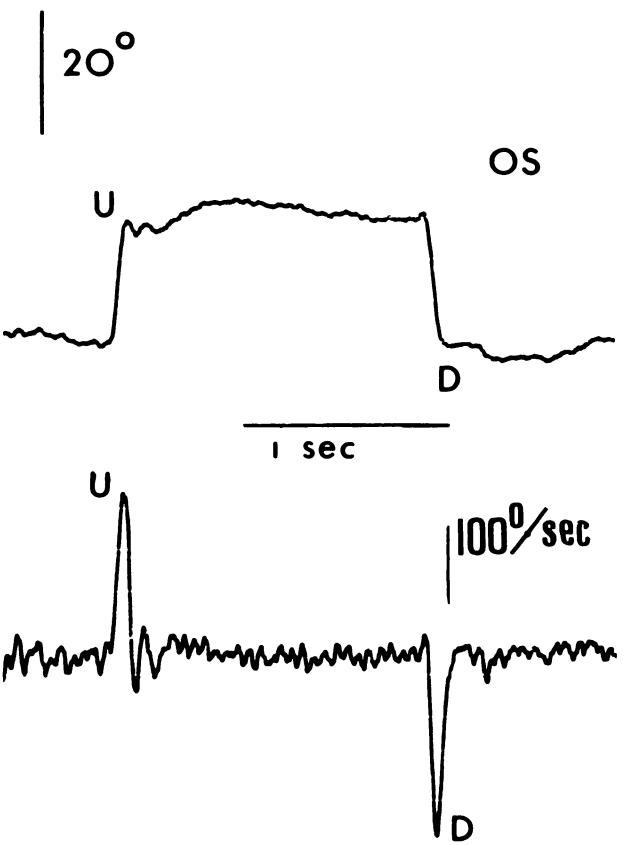

FIGURE 22
Electro-oculographic recording of vertical saccades in patient with fracture of orbital floor. Upward (U) and downward (D) saccades are equal and rapid. Eye position (upper trace) and peak velocity (lower trace) are shown.

the speed of movement was within normal limits. Patients in subgroup 2 demonstrating upward movements were an average of 150% faster than downward saccades (range 50% to 500%) (Fig 23), with downward movement being abnormally slow in all cases. Generally, the greater the percentage difference between upward and downward velocities, the greater the postoperative hyperdeviation.

Active force measurements,[24] both upward and downward, were performed on four patients with slowing of downward saccadic velocities in subgroup 2. There was marked reduction in active force inferiorly in all four patients.

One patient with a medial wall fracture, entrapment of the medial rectus muscle, and restriction of abduction also had normal saccadic velocities both medially and laterally.

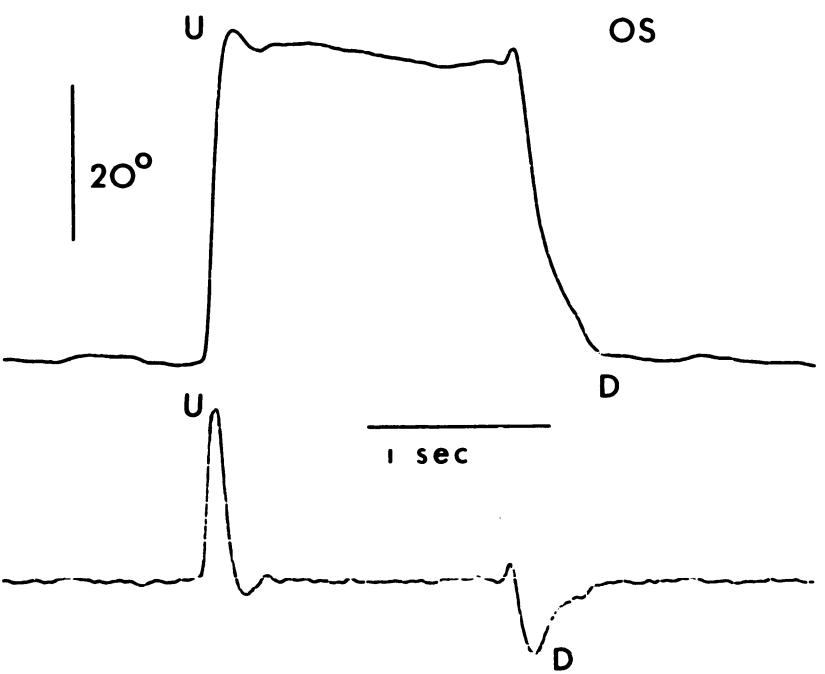

FIGURE 23
Electro-oculographic recording of vertical saccades in patient with fracture of orbital floor. Upward saccades (U) are rapid while downward saccades (D) are slow, indicating inferior rectus muscle weakness. Eye position (upper trace) and peak velocity (lower trace) are shown.

COMMENT AND CONCLUSIONS

When the differnece between upward and downward saccadic velocity was less than 30%, patients were able to fuse in primary position either before or after repair of the floor fracture. Patients with a difference in vertical saccadic velocities of more than 50% had persistent vertical diplopia. The slowing of downward saccades and remaining hyperdeviation after surgery are probably caused by inferior rectus muscle paresis. This is supported by the findings of reduced active force inferiorly measured in several patients.

Emory et al[34] reported that 50% of their patients with blow-out fractures had persistent diplopia despite surgical repair of the fracture, with an average follow-up of more than 3 years. These findings suggest this group of patients might have had inferior rectus muscle paresis. It would be useful to identify these patients preoperatively so that a more accurate prognosis could be given. In addition, when inferior rectus muscle weakness is discovered, saccadic velocity measurements can assist in determining whether the muscle is recovering or remaining paretic.

BROWN'S SYNDROME

Brown's syndrome has the characteristics of diminished elevation in adduction, improved elevation in the primary position, and normal or almost normal elevation in abduction. The forced duction test upward and inward is positive.

Eighteen patients with Brown's syndrome had measurements of vertical saccadic velocity. In 15 cases there was unilateral involvement, and in 3 patients, a bilateral picture (21 eyes). Twelve patients had no vertical deviation in the primary position, while 6 had hypotropia of 5 to 20 PD.

Average vertical saccadic speed in primary position was 300°/s upward and 280°/s downward. In adduction, upward and downward average saccadic velocity was 280°/s and 265°/s, respectively (Fig 24), and in abduction, 290°/s and 275°/s.[35] This slight increased velocity of upward saccades is normal with this technique as used in our laboratory.

Vertical saccades were also measured in the uninvolved, opposite eye (13 eyes). Average velocity measurements were similar to those found in the eye of the patients with Brown's syndrome.

COMMENT AND CONCLUSIONS

The clinical findings in Brown's syndrome may resemble those of inferior oblique muscle palsy. However, Scott and Nankin[36] indicated that elevation in adduction is only mildly reduced and the forced duction test is

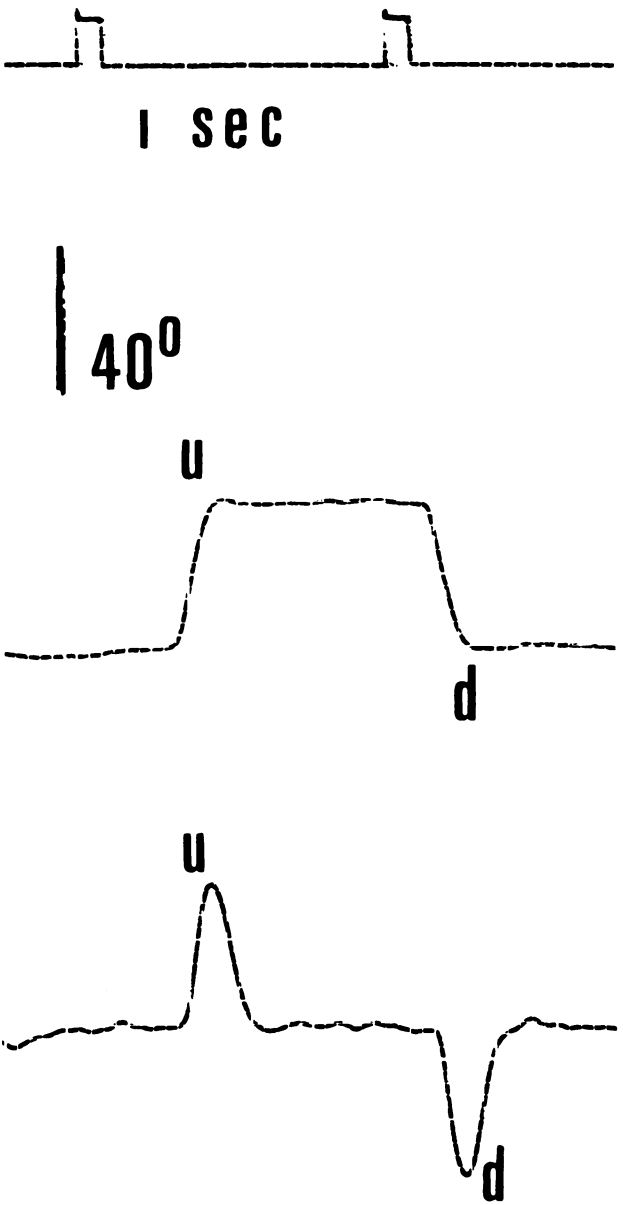

FIGURE 24
Electro-oculographic recording of vertical saccades in adduction in patient with Brown's syndrome. Both upward (u) and downward (d) saccades are rapid and equal. Eye position (upper trace) and peak velocity (lower trace) are shown.

negative. In a patient where the diagnosis is in doubt or in children too young to perform the forced duction test in an outpatient setting, studies of saccadic velocity may be of assistance. Rapid upward saccades would indicate that the cause for limited elevation is restrictive and is not caused by weakness of an elevator muscle.[37]

POSTOPERATIVE RESTRICTIONS

Following surgical treatment for orbital, retinal detachment, and strabismus disorders (especially multiple reoperations), there may be some limitation to full ocular rotation. Is this limitation due to mechanical restriction or weakness (or disinsertion) of an extraocular muscle? Restrictions are probably the most frequent cause of limited motility.[38]

Twenty-eight patients with restricted eye movements were studied. Four patients had previous surgery for orbital disorders, 2 for retinal detachment, 1 for orbital trauma and strabismus, and the remaining 21 patients, for strabismus (one to five previous operations). The forced duction test was positive in all cases, either at surgery or in the outpatient setting. Vertical or horizontal saccadic movements or both were confined to the area where the eye was free to move. Agonist-antagonist saccadic velocities were equal and rapid (Fig 25), indicating no rectus muscle weakness.[36] Similar to the endocrine ophthalmopathy studies, there was a tailing off effect as the eye movement came close to the area of restriction.

COMMENT AND CONCLUSIONS

The forced duction test is a necessary diagnostic step in patients with limitation of ocular movement.[39] The test, however, may be painful and, if the restriction is mild, is difficult to interpret. Although modifications such as the use of a suction cup combined with a force transducer have been developed,[40] the unwieldiness and imprecision of the technique have not been overcome. Differential intraocular pressure (IOP) changes in various gaze directions have also been used as a diagnostic test for adhesions.[39]

Studies of saccadic velocity easily and reliably identify restriction in some patients with limited motility following orbital, retinal detachment, or repeated strabismus operations. In addition, the test can be performed on patients of all ages and is a noninvasive technique.

PARALYSIS VS RESTRICTION

"DOUBLE ELEVATOR" PALSY

Double elevator palsy is a term frequently used to describe unilateral, diminished ocular elevation present in all fields of gaze. Limited elevation

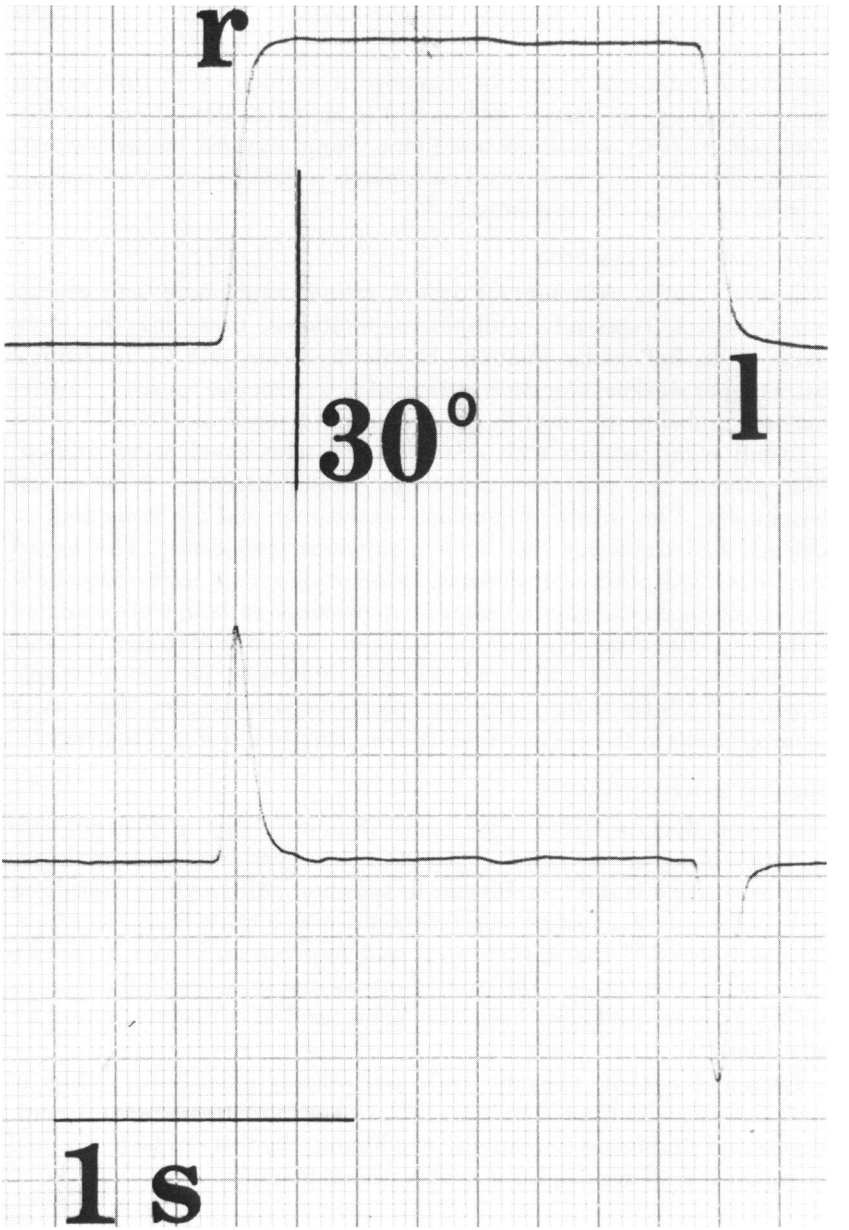

FIGURE 25

Electro-oculographic recording of horizontal saccades in patient with three previous operations for strabismus and abduction limited to 20°. Both adduction (r) and abduction (l) saccades are rapid and equal. Eye position (upper trace) and peak velocity (lower trace) are shown.

may result from innervational causes (supranuclear, nuclear, or infranuclear), restrictive mechanisms in the orbit, or a combination of both. The correct treatment of these problems depends upon the proper identification of restriction or muscle weakness.

To determine the mechanism for limited elevation, 21 consecutive patients with diminished upgaze monocularly had studies of vertical saccadic velocity.[41] The limitation varied from mild to complete absence of elevation above the horizontal plane. Twelve patients had hypotropia of the involved eye when fixating in primary position (range 12 to 35 PD), while 9 had fusion in primary gaze and only demonstrated hypotropia in attempted upgaze. All patients with a history or roentgenographic evidence of orbital trauma or endocrine ophthalmopathy were excluded from this portion of the study.

Nine patients with no vertical deviation in primary gaze had upgaze limitation varying from mild to moderate. The forced duction test revealed restriction upward. Upward saccades averaged 250°/s, while downward saccades averaged 225°/s (Fig 26).

Of the 12 patients with a vertical deviation in primary gaze, 6 had no marked difference in their vertical saccadic velocities. Upward saccades averaged 230°/s and downward saccades, 220°/s. The forced duction test was positive in all six patients. The remaining six patients in this group had definite reduction in upward saccadic speed, averaging 150°/s upward and 270°/s downward (Fig 27). The forced duction test showed free movement upward in five of these six patients. The traction test revealed mild to moderate restriction in the sixth patient, but less than would be expected to explain the limitation of elevation.

COMMENT AND CONCLUSIONS

Of the patients with monocular limitation of elevation who are frequently diagnosed as having a double elevator palsy, 72% had no evidence of elevator weakness. Scott and Jackson[42] reported a high incidence of inferior restriction in double elevator palsies. They noted an accentuated lower lid fold on the hypotropic side in patients with inferior restriction. The fold became more prominent with attempted upgaze.

Some controversy surrounds the cause of double elevator palsy with known elevator weakness. White[43] believed that the inferior oblique muscle was the more involved, while Burian[43] commented that weakness of the superior rectus muscle alone could cause elevator palsy. Jampel and Fells[44] believe that a lesion in the midbrain tectum or pretectum near the oculomotor nucleus or in the nucleus probably accounts for a monocular paresis of elevation. They also think that the superior rectus muscle is

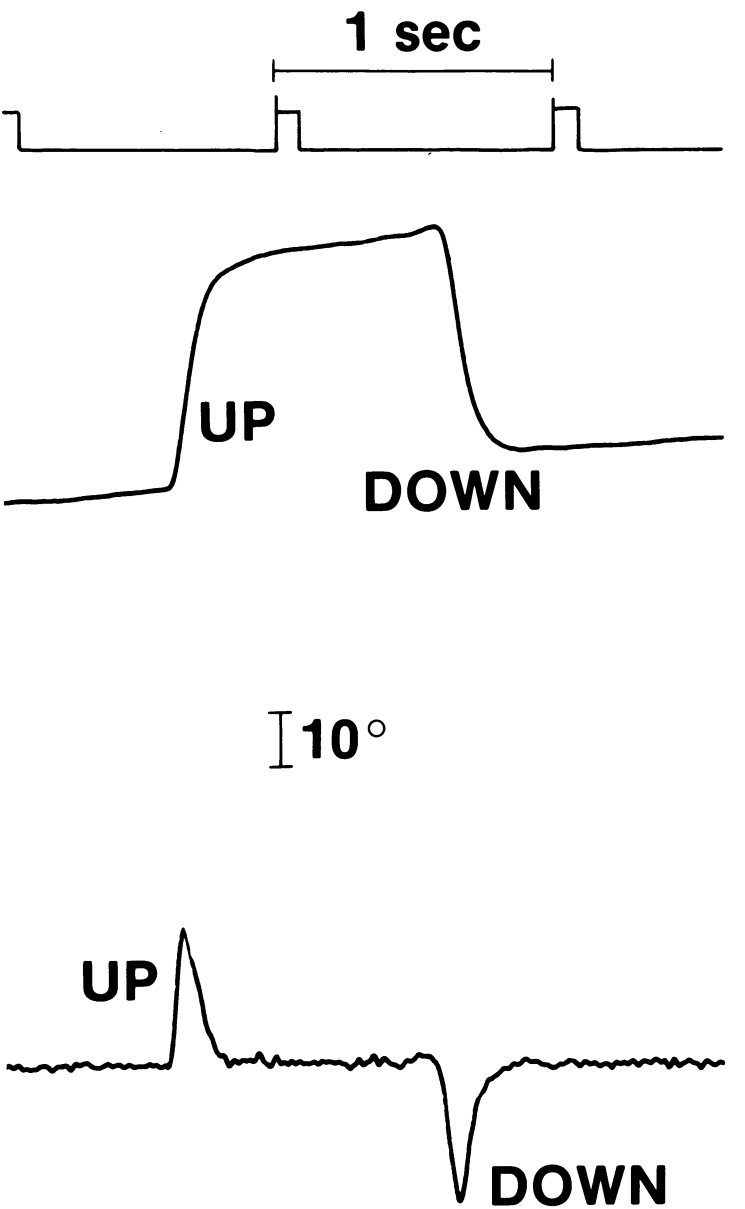

FIGURE 26
Electro-oculographic recording of vertical saccades in patient with mechanical restriction of elevation. Eye position (upper trace) and peak velocity (lower trace) are shown. Upward and downward saccades are rapid and equal in velocity.

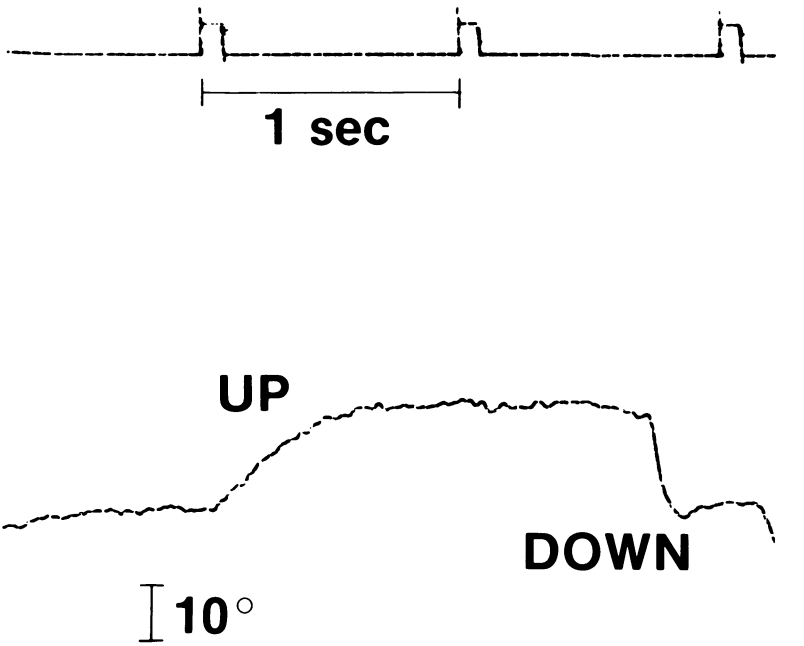

FIGURE 27
Electro-oculographic study of vertical saccades in patient with weakness of elevator muscle. Upward saccades are slow, while downward saccades are normally rapid.

the main elevator of the eye. Thus, monocular paresis of elevation can be caused by a lesion affecting central neural connections of the superior rectus muscle.

Scott[21] found that the effect on peak velocity and isometric force from oblique paralysis was minimal but that a marked reduction resulted from paralysis of the vertical rectus muscle.

Lennarson and Scott[45] have shown that upward saccadic velocity is not altered by a large superior rectus muscle recession (6 to 8 mm). However, with superior rectus muscle disinsertion, vertical saccadic speed is reduced.[46]

In treating double elevator palsy, Dunlap[47] suggested that medial rectus-lateral rectus transposition superiorly should be reserved only for dysfunction of neurogenic origin; dysfunction on a mechanical basis will not be corrected by this technique. It therefore becomes important to perform forced duction testing to identify restrictions and saccadic velocity measurements to assess function of the superior rectus muscle prior to surgery.

Our results indicate that a patient with monocular limitation of elevation and no deviation in primary gaze has about a 25% chance of having weakness of an elevator muscle.[48] Patients with both monocular limitation of elevation and hypotropia in the primary position have about a 50% chance of having an elevator muscle palsy.

LIMITED DOWNWARD GAZE

Although monocular limitations of downward gaze are infrequent, they can be caused by innervational or restrictive factors or both, just as in patients with deficits of elevation. Appropriate management requires knowledge of the forces available to move the globe downward and of the presence of mechanical restrictions that limit downward gaze. The forced duction test provides information about restrictions. Vertical saccadic velocity determinations were used to assess the active force available to move the eye inferiorly in a group of patients with monocular limitation of downgaze.

Eighteen patients with unilateral limitation of downward gaze as the only motility defect were studied. Patients with endocrine eye disease, orbital floor fracture, and complete third nerve palsy were excluded. One normal subject had lidocaine hydrochloride injected into the inferior rectus muscle with an electromyographic needle electrode used for control.

Only one patient did not have a vertical deviation in the primary position. The hypertropia varied from 4 to 50 PD. Limitation of downward gaze was mild in 2 patients (25° to 30° in extent), moderate in 12 patients (10° to 25°), and marked in 4 patients (less than 10°).[47]

The patients could be separated into two groups. In one group, there were six patients with less than a 20% difference between upward and downward saccadic velocities (average 80%) (Fig 28). These cases were not considered to have evidence of inferior rectus muscle weakness. The deviation in primary gaze averaged 16 PD of hypertropia, while downgaze demonstrated mild to moderate limitation.

In the second group of patients, upward saccades were 45% to 275% faster than downward saccades (average 100%) (Fig 29). These patients were all considered to have inferior rectus muscle palsy, varying from moderate to marked. The deviation in primary gaze averaged 25 PD of hypertropia, and downward gaze showed moderate to marked limitation.

The normal subject with a lidocaine-induced palsy of the inferior rectus muscle had upward saccadic velocity 135% faster than downward saccadic velocity.

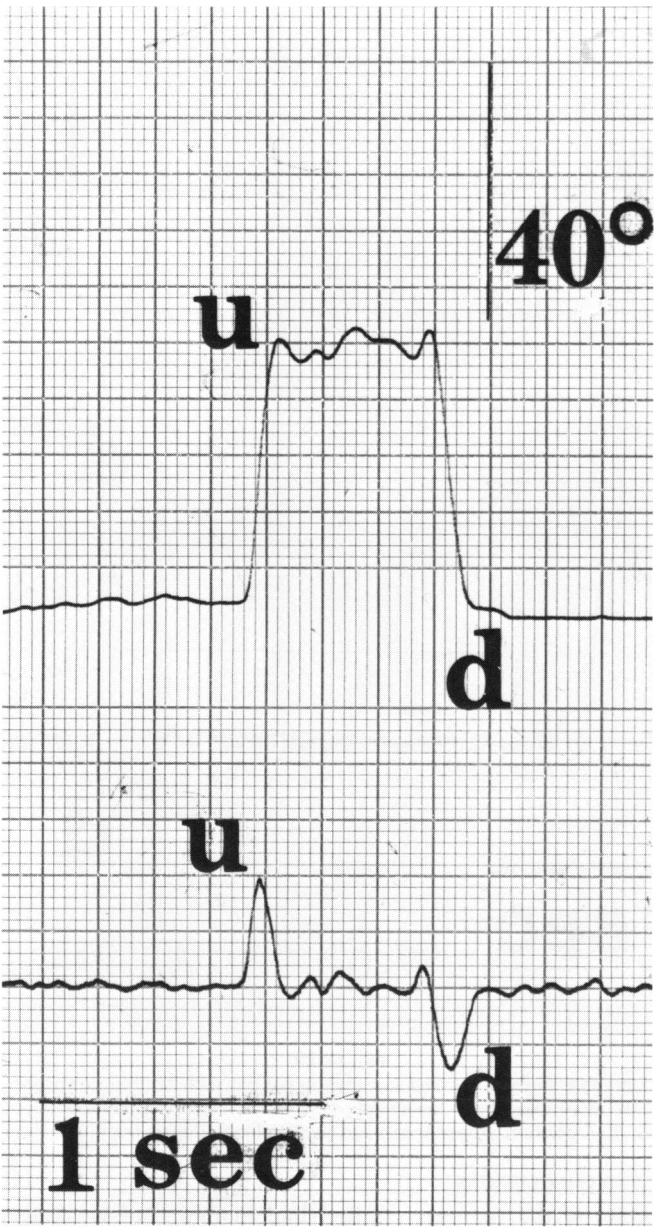

FIGURE 28

Electro-oculographic study of vertical saccades in patient with limited downgaze. Upward (u) and downward (d) saccades are both normally rapid, suggesting normal inferior rectus muscle function. Eye position (upper trace) and peak velocity (lower trace) are shown.

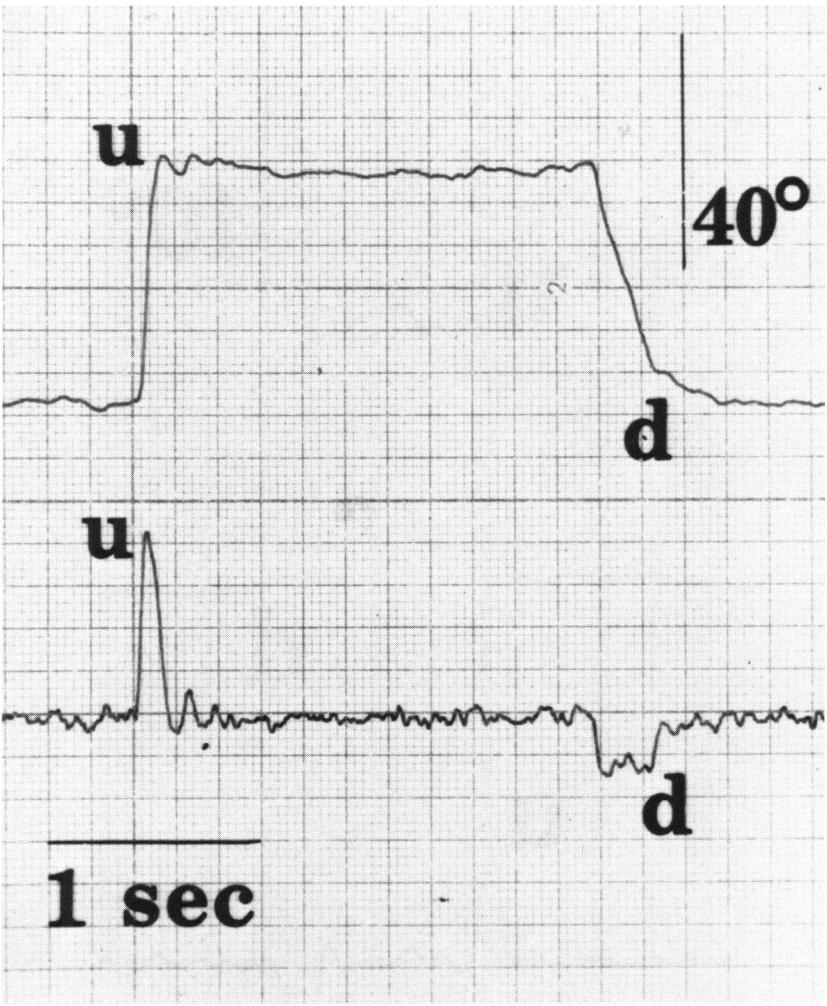

FIGURE 29
Electro-oculographic study of vertical saccades in patient with limited downgaze. Upward (u) saccades are more rapid than downward (d) saccades, suggesting inferior rectus muscle paresis. Eye position (upper trace) and peak velocity (lower trace) are shown.

COMMENT AND CONCLUSIONS

Twelve (67%) of the patients studied had evidence of weakness of the inferior rectus muscle. As in cases of third nerve palsy, inferior rectus muscle paresis, even with intact superior oblique function, is sufficient to cause limitation of downward gaze.

Burian and von Noorden[49] found that paralysis of both depressor muscles of one eye rarely occurs. When seen, it has been of congenital origin. They indicate that mechanical causes that interfere with depression of the eye must be excluded.

Monocular limitation of elevation was infrequently found to be caused by superior rectus muscle palsy.[41] Although monocular limitation of depression may be seen less often, a larger percentage had evidence of rectus muscle paresis. This suggests that restrictions in the superior portion of the orbit are probably less common than those in the inferior orbit.

Roper-Hall and Burde[50] reported monocular, isolated inferior rectus muscle palsy in 19 patients with atypical disease of the third cranial nerve. Cooper and Greenspan[51] found one case of congenital absence of the inferior rectus muscle and reviewed seven similar cases from the literature. The clinical findings were similar to those of inferior rectus muscle palsy.

Chu et al[52] reported on a group of patients with progressive supranuclear palsy (PSP). One of the hallmarks was a downgaze palsy. Saccades made by these patients downward were of a lower peak velocity.

NEURO-OPHTHALMIC DISORDERS

MYASTHENIA GRAVIS

About half of all patients with myasthenia gravis have complaints related to eye movements and lids. Response to intravenously (IV) administered edrophonium chloride (Tensilon) by improved oculomotor function is considered diagnostic. Such improvement may be uncertain or elusive. Tests to demonstrate equivocal degrees of improvement include red-green glass diplopia, tonography, ocular electromyography, and OKN. Since saccadic velocities are susceptible to a mild to moderate degree of paresis, it was of interest to see if analysis of these movements was a consistent, reliable, objective indicator of the effect of Tensilon on the oculomotor function of myasthenic patients.

Eight patients with myasthenia gravis and 20 nonmyasthenic subjects were tested. Any anticholinesterase medication was discontinued prior to testing. Voluntary saccades and optokinetic eye movements were recorded from 10 seconds to 3 minutes after IV administration of Tensilon, and then 10 minutes later.

A comparison of saccadic eye movements was made in all subjects before and after administration of Tensilon. Each of the myasthenic pa-

tients had an increase in velocity after receiving the drug.[53] All nonmyasthenic subjects showed no change following administration of Tensilon. A typical OKN pattern in a myasthenic subject was characterized by low frequency and amplitude that worsened with fatigue (Fig 30). After receiving Tensilon, the amplitude of eye movement increased and did not demonstrate fatigue with continued stimulation. Voluntary saccades in myasthenic patients were slower than normal (Fig 31). After receiving Tensilon, the velocity increased. When the drug had worn off, saccadic speed returned to the initial level.

COMMENT AND CONCLUSIONS

Campbell et al[54] demonstrated an increased amplitude of OKN following administration of Tensilon in patients with myasthenia gravis. Although false-positive effects were not seen, true positive effects were demonstrated in only 50% of their patients. They believe that electronystagmography was of limited value as a diagnostic test in these cases.

Stella,[55] Blomberg and Persson,[56] and Spector et al[57] also demonstrated increased amplitude of OKN following Tensilon injection in myasthenic patients. Spector and associates[57] emphasized that the infrared nystag-

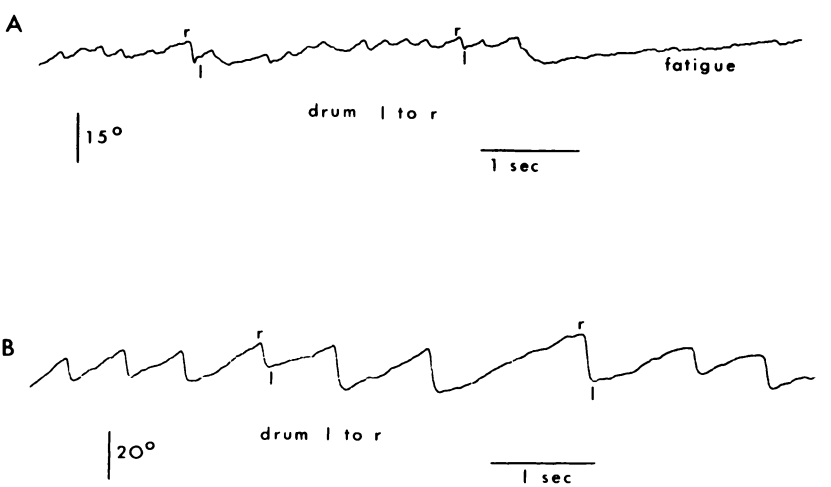

FIGURE 30

Electro-oculographic recording in patient with myasthenia gravis. Upper trace, Optokinetic-induced nystagmus prior to administration of Tensilon, drum rotating left (l) to right (r), is shown. Movements are low-amplitude and have slow recovery phase to left. Lower trace, Following administration of Tensilon, amplitude of nystagmus is increased, and recovery saccades to left are rapid. No fatigue effect is seen.

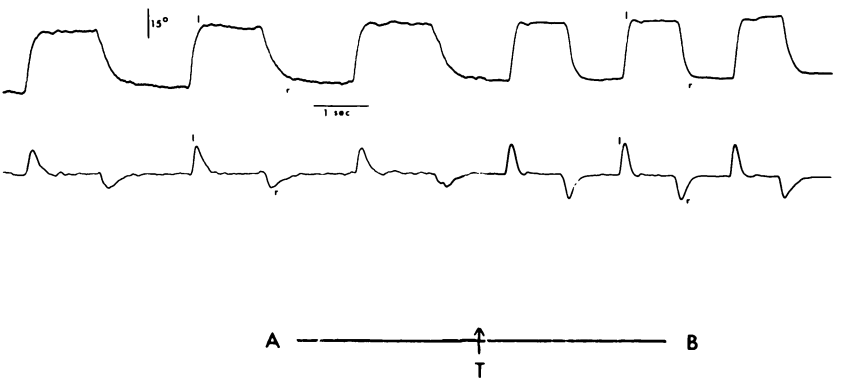

FIGURE 31
Electro-oculogram in patient with myasthenia gravis. Upper trace, Eye position is shown. Lower trace, Peak velocity is demonstrated. Before Tensilon is injected, voluntary saccades at left (l) and right (r) are reduced in speed (at A). Tensilon is administered (at arrow). Within 30 seconds, saccadic velocity has increased both to left and right (at B).

mogram improved the sensitivity of the test. Myasthenia gravis can have various clinical pictures. It has been reported to mimic internuclear ophthalmoplegia,[58,59] a pupil-sparing third nerve palsy,[60] and an isolated inferior rectus muscle paresis.[61] The diagnosis in all these cases was proved pharmacologically. This suggests that even "typical" forms of ophthalmoplegia might be myasthenic in origin, particularly if they are somewhat variable, and that testing with Tensilon with some sort of objective monitoring technique (such as saccadic velocity recordings) can be useful.

Baloh and Keesey[62] found decreased amplitude and velocity of saccades after repetitive refixations. Reversal of this saccadic fatigue by Tensilon injection appeared to be a useful diagnostic sign.

Yee and associates[63] reported that despite the limited range of eye movements in patients with myasthenia, maximum saccadic velocities were not significantly different from those in normal persons, while maximum velocities in patients with other types of ophthalmoplegia were significantly decreased. Their results indicated that maximum saccadic velocity, generated during the initial phase of the saccade, is frequently normal in myasthenic patients since the initial phase of the saccade is spared in this disease. However, during large saccades, a high velocity cannot be sustained. After the rapid, initial movement of the saccades, the velocity decreases rapidly, and the eyes slowly drift toward the target. Intrasaccadic fatigue of extraocular muscle fibers, which normally generate the rapid eye movement in response to pulse innervation from the oculomotor neurons, appears to produce the sudden fall in saccadic veloc-

ity. Clinical estimation of saccadic velocity during the initial phase of saccades can differentiate myasthenia gravis from other types of ophthalmoplegia.

CHRONIC PROGRESSIVE EXTERNAL OPHTHALMOPLEGIA

In its early stages and milder forms, chronic progressive external ophthalmoplegia (CPEO) is often misdiagnosed. Patients frequently have ptosis and ophthalmoplegia. The ophthalmoplegia tends to be symmetric when comparing the two eyes; deficits of both the lid and ocular motility are nonresponsive to administration of Tensilon, which is helpful in differentiating this entity from myasthenia gravis. In addition, no worsening is observed with fatigue.

Seven patients with the diagnosis of CPEO had both horizontal and vertical saccadic velocity measurements in the area where the eye was able to move. Tensilon testing in all patients was negative. Saccadic speed in all patients was reduced in velocity[64] (Fig 32), although the degree of slowing varied noticeably among patients and among the same patient's different eye movements (20° to 150°/s average velocity). The slowing of saccadic velocity appeared symmetric between two eyes of the same patient.

COMMENT AND CONCLUSIONS

The history of patients with CPEO often indicates a long latency period between the onset of symptoms and the eventual substantiation of the diagnosis. These patients may be misdiagnosed as having congenital or

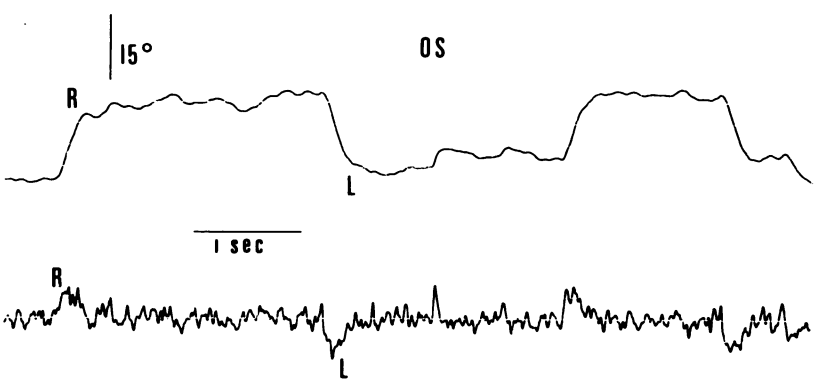

FIGURE 32
Electro-oculographic recording in patient with CPEO. Saccades to right (R) and left (L) are slow. Eye position (upper trace) and peak velocity (lower trace) are shown.

senile ptosis, myasthenia gravis, myotonia congenita, multiple sclerosis, thyroid disease, or orbital tumor.

Both Jampel et al[65] and Koerner and Schlote[66] have described abnormal saccadic velocities with progressive external ophthalmoplegia. Yee et al[63] stated that in CPEO, since muscle fibers are already paretic at the beginning of the movement and there is maximal recruitment of muscle fibers during the saccade, normal saccadic velocities cannot be generated.

In none of our cases was saccadic velocity normal in either eye or in any direction. This may be specific for progressive external opthalmoplegia. The diagnostic value of this finding is that it may obviate the need for unwarranted tests and therapy.

INTERNUCLEAR OPHTHALMOPLEGIA

Lesions of the medial longitudinal fasciculus (MLF) demonstrate deficient adduction and jerk nystagmus of the abducting eye. Patients are often orthotropic in the primary position, and adduction with convergence may be preserved. In some cases of internuclear ophthalmoplegia (INO), the clinical signs are not obvious and clear. The bilateral form is almost always indicative of multiple sclerosis, so that a simple test that could establish the diagnosis would be valuable in patients who otherwise might require more formidable and prolonged studies.

Seven patients with INO, five bilateral and two unilateral, had horizontal saccadic velocities measured in both eyes, from 20° of abduction to 10° of adduction.[67] Abduction saccades were all of normal velocity and averaged 240°/s. In two patients with monocular INO, adduction saccades on the uninvolved side were normal, averaging 250°/s. Adduction saccades in the binocular cases and on the involved side in the monocular cases were slow, averaging 80°/s (Figs 33 and 34). Abduction nystagmus is easily noted on the eye movement tracings. One patient had full adduction in each eye associated with abduction nystagmus. Saccadic velocity in a nasal direction was measurably slow in each eye, confirming early involvement of the MLF.

COMMENT AND CONCLUSIONS

The pontine gaze centers mediate impulses for conjugate, horizontal movements to the subnuclei of the medial recti through the MLF. Convergence not mediated through the MLF is available to cause the medial recti to contract, even when lesions of the MLF are present.

Evinger et al[68] made bilateral cuts through the MLF between the abducens and trochlear nuclei in the monkey. One week later, normal abduction saccades were recorded, but adduction movements did not

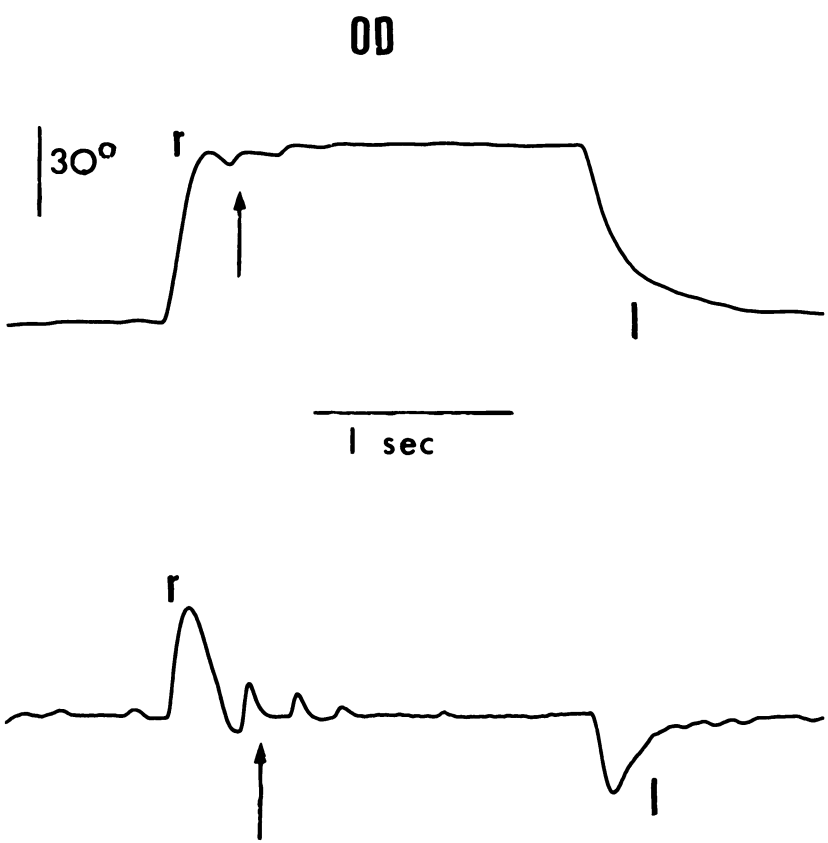

FIGURE 33
Electro-oculogram, OD, in patient with bilateral INO. Abduction saccades to right (r) are rapid, while adduction saccades to left (l) are slow. Note abduction nystagmus (arrow). Eye position (upper trace) and peak velocity (lower trace) are shown.

proceed beyond the midline and adduction saccades were slow. Loeffler et al[69] found excitation of defective medial rectus muscles on attempted lateral gaze, using ocular electromyography. There were no bursts of activity in the medial rectus muscle with attempted nasal saccades.

Smith and David[70] described two tests that are helpful in making the diagnosis of INO. When an optokinetic target is moved in the direction of the involved medial rectus muscle, there is a greater amplitude of response in the opposite eye. The effect is to produce a dissociation of the OKN response. The ocular dysmetria sign was the second finding empha-

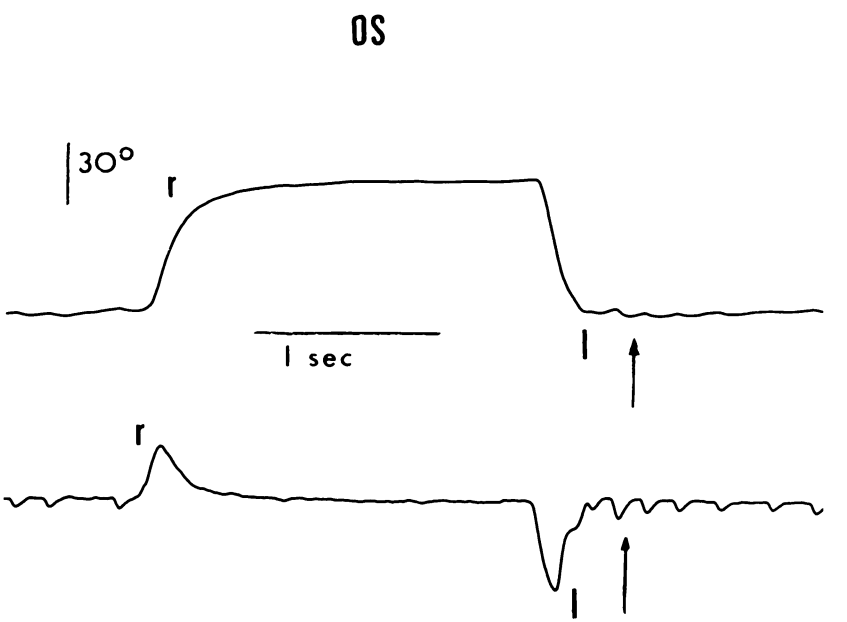

FIGURE 34
Electro-oculogram, OS, in patient with bilateral INO. Abduction saccades to left (l) are rapid, while adduction saccades to right (r) are slow. Note abduction nystagmus (arrow). Eye position (upper trace) and peak velocity (lower trace) are shown.

sized by Smith and David.[70] Ataxic overshoots are seen regularly in the eye opposite that showing impairment of the medial rectus muscle.

Dell'Osso et al[71] also showed that recovery saccades of OKN, produced by the involved medial rectus muscle, are slow. Siroky et al[72] demonstrated slow recovery saccades produced by the medial rectus muscle in postrotary nystagmus in patients with INO. They showed that it was possible to discover the latent form of INO in the early disease state by using eye movement recordings and measuring evoked nystagmus.

Fricker and Sanders[73] also emphasized that INO, in its early stages, may not demonstrate grossly abnormal adduction eye movements. They recorded a decrease in adduction saccadic velocity in definite cases of INO as well as in some subclinical cases.

MÖBIUS' SYNDROME

Möbius' syndrome is characterized by congenital facial displegia associated with restrictions of horizontal eye movements. Vertical rotations are

intact. Other anomalies sometimes seen are lagophthalmos, partial atrophy of the distal tongue, congenital heart defects, and abnormalities of an extremity.

Eight patients with Möbius' syndrome, between the ages of 9 months and 29 years, were studied.[74] Seven of the eight had esotropia, all had facial palsy and absence of abduction beyond the midline; vertical movements were normal. Adduction was also noticeably reduced in all cases.

The amplitude of recorded horizontal eye movements was about 10°. Saccadic velocities were extremely slow both toward abduction and adduction, with a range of 25° to 80°/s (Fig 35). In our laboratory, normal average saccadic velocity for a 10° movement is about 150°/s.

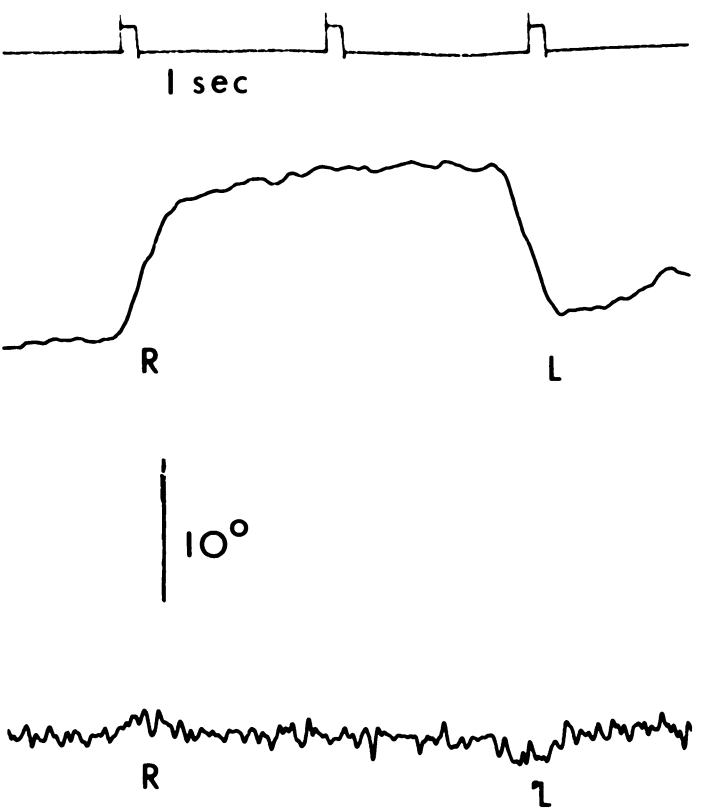

FIGURE 35
Electro-oculogram in Möbius' syndrome. Saccades both right (R) and left (L) are extremely slow. Eye position (upper trace) and peak velocity (lower trace) are shown.

COMMENT AND CONCLUSIONS

Van Allen and Blodi[75] and Merz and Wojtowicz[76] used electromyography to test patients with Möbius' syndrome. Evidence suggested the existence of a supranuclear lesion affecting the horizontal rectus muscles. Involvement of the facial nerve appeared to be primarily nuclear or infranuclear in origin.

Möbius' syndrome has generally been considered a bilateral sixth and seventh nerve palsy associated with other congenital anomalies. Poor adduction has been noted but commented upon infrequently. The slow adduction saccades encountered in all of our patients suggest significant functional weakness of the medial rectus muscle. This disorder does not appear to be part of a third nerve palsy since vertical eye movements, lid position, and pupillary responses were normal. More probably, it is caused by supranuclear or nuclear paralysis of eye movements.

MUSCLE TRANSPOSITION SURGERY

In patients with paralysis of an extraocular muscle, surgical transposition or muscle union procedures may be indicated, not only to straighten the eye but to improve rotation into the field of action of the involved muscle.

The rationale for the effectiveness of muscle transposition has not always been clear. It has been suggested that central nervous system readjustment of oculomotor control can occur.[77] Adler[78] believed that the results achieved by transposing the insertions of the vertically acting muscles to the insertions of the laterally acting muscles resulted from "placing a muscle where it will affect a new moment of force when it contracts." Metz and Scott,[79] using both ocular electromyography and central sixth nerve stimulation, were able to show that no change in the firing pattern of transposed lateral and superior rectus muscles occurred. The eye movements observed after recovery from the tranposition procedure could be explained entirely by the new position of the lateral rectus muscle insertion, with action of the lateral rectus muscle unchanged.

Six patients were studied before and after surgery for muscle transposition.[80,81] One patient had an inferior rectus muscle paralysis, one had Duane's syndrome, one had weakness of the medial rectus muscle, and the other three had lateral rectus muscle palsy (two unilaterally and one bilaterally). The patient with the bilateral sixth nerve palsy had a transposition procedure on one side and a large recession-resection procedure on the other.

In all patients preoperative saccadic velocities in the direction of action of the paralytic muscle were extremely slow (Fig 36). Following transposition surgery, there was a marked increase in saccadic speed (Fig 36).

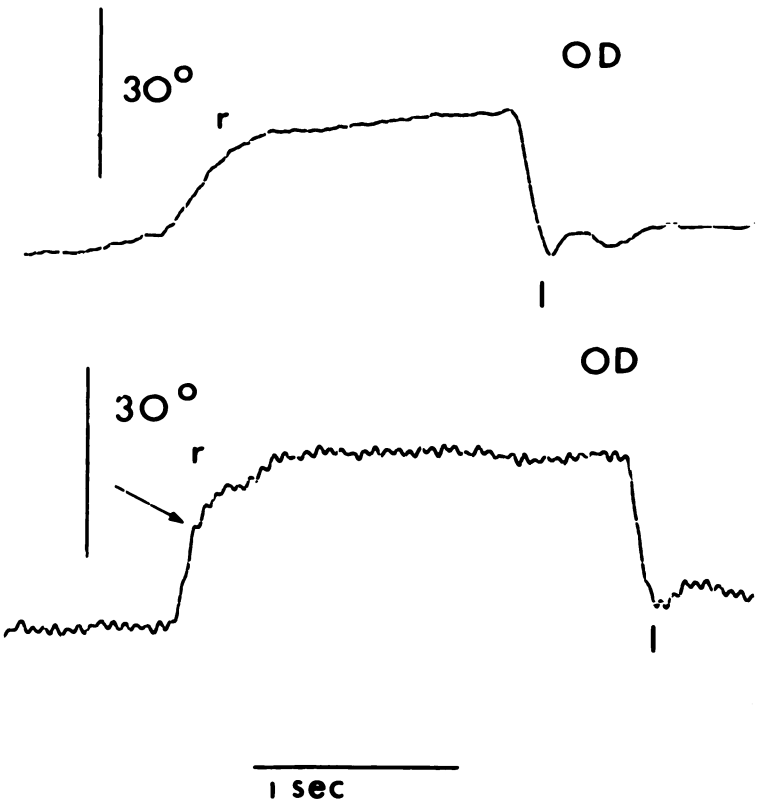

FIGURE 36
Electro-oculogram in patient before and after transposition surgery for paralysis of lateral rectus muscle. Upper trace, Eye position shows slow saccades to right (r) and rapid saccades to left (l). Lower trace, Horizontal saccades after muscle transposition show increase in saccadic velocity to right (arrow).

The patient with bilateral palsy of the sixth nerve had an extensive recession-resection operation on one eye. Saccadic velocities measured before and after surgery (Fig 37) demonstrate no noticeable increase in velocity of the slow saccade measured preoperatively. Abduction past the midline was not seen on this side, while the opposite eye, which had transposition surgery, was able to abduct about 12° lateral to the midline.

COMMENT AND CONCLUSIONS

The results, which indicate that saccadic velocity is improved following transposition surgery of the rectus muscle, are in accord with the clinical

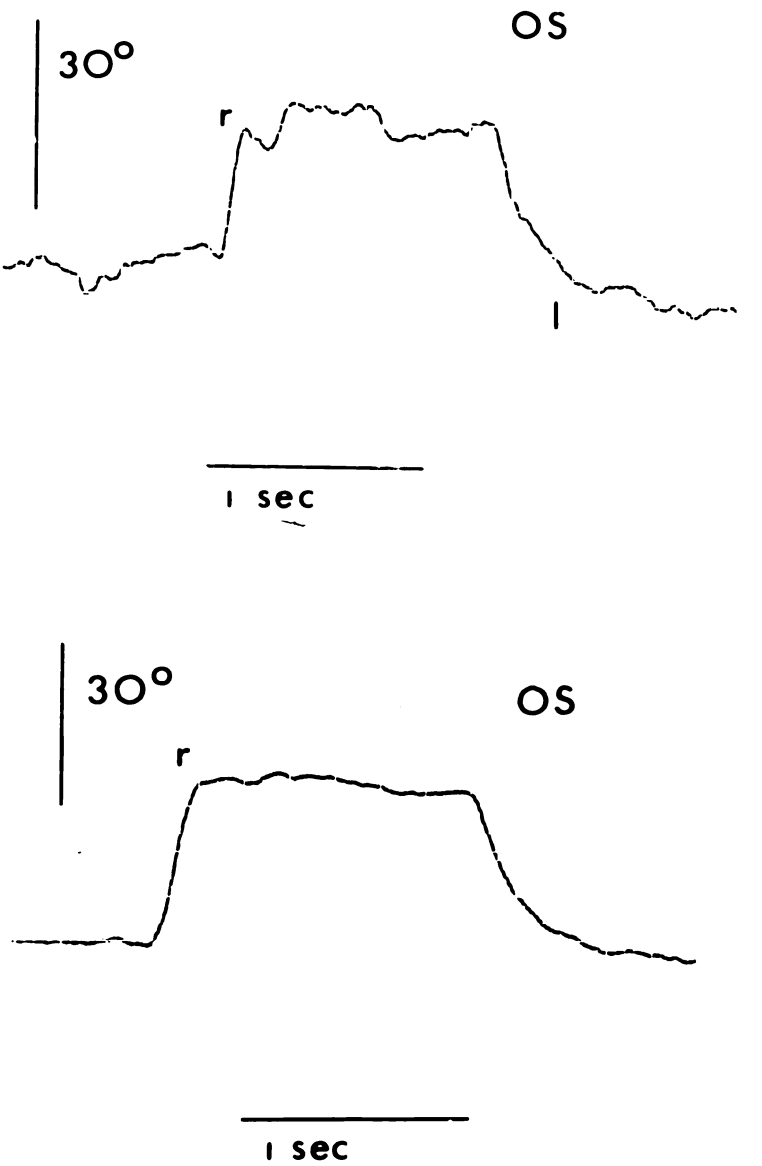

FIGURE 37

Electro-oculogram in patient before and after extensive recession-resection surgery for paralysis of lateral rectus muscle. Upper trace, Saccades to left (l) are slow and saccades to right (r) are rapid. Lower trace, Horizontal saccades after surgery show no change in saccadic velocity to left.

findings of increased rotation into the field of action of the paralytic muscle. By transposing insertions of the rectus muscle to a new position, the vector of forces produced by these muscles is altered slightly to a clinically more useful position. These changes have been measured and take place immediately following transposition surgery. This strongly suggests that a relearning process is not part of the explanation for the changes seen. Lian[82] reported improved adduction 2 hours after transposition surgery for a lost medial rectus muscle. This is consistent with the present studies, which suggest that improved rotation and saccadic velocity result from the mechanical effects of such surgery.

Unpublished studies of Scott,[83] (written communication, 1976), measuring muscle force directly before and after muscle transposition, found an increase in active generated force following the surgical procedure.

DISINSERTED EXTRAOCULAR MUSCLE

A slipped or lost muscle during the immediate postoperative period is a disappointing complication of surgery for strabismus. This diagnosis is suspected if there is an unexpected, large overcorrection or undercorrection of the deviation, especially if accompanied by marked limitation of ocular rotation. Muscle disinsertion may also result from trauma or inappropriate surgery. Prompt recognition and accurate diagnosis are important since early surgical intervention is both technically easier and mechanically advantageous, and secondary muscle contractures may be prevented.

The diagnosis may be confusing shortly after surgery because of pain, edema, and photophobia. A test that could (1) be performed in the immediate postoperative period, (2) be carried out in children, and (3) give relevant, accurate information concerning the diagnosis of a disinserted or slipped muscle would be a valuable adjunct in evaluation of strabismus.

During surgical management of strabismus using only topical anesthesia, information may be obtained about the function of various extraocular muscles and the force a muscle can generate. With a rectus muscle disinserted under topical anesthesia, a good model is created for a lost or slipped muscle after surgery.

A 52-year-old patient with 45 PD of left exotropia was operated on under topical anesthesia. The forced duction test was negative. Horizontal saccades were measured (1) prior to the start of surgery, (2) after the left lateral rectus muscle was disinserted, and (3) after both the left medial and left recti muscles were disinserted.[84]

Prior to surgery, horizontal saccades OS were normal (Fig 38). After disinsertion of the lateral rectus muscle, abduction saccades were reduced to about 40% of the preoperative value (Fig 39). After the left medial rectus muscle was disinserted, adduction saccades were also reduced to approximately 40% of normal (Fig 40).

These results indicate that saccades are slowed, but not eliminated, after disinsertion of a horizontal rectus muscle. The moderate reduction in velocity is probably produced by posterior intermuscular and Tenon's fascial attachments of the disinserted muscle.

To assess the effectiveness of saccadic velocity measurements in diagnosing slipped or lost muscles, the following two groups were studied.[39]

Group 1—Preoperative and postoperative measurements following rectus muscle recession (15 patients). None of the patients had previous surgery for strabismus, and there was no indication of paresis of an extraocular muscle or of restriction.

Group 2—Clinically suspected disinserted or slipped rectus muscles (nine patients). The disinserted position of the muscle insertion was documented at the time of the surgical procedure in each patient. Postoperatively, after the disinserted muscle had been located and reattached to the globe, saccadic velocities were again determined.

Preoperative and postoperative measurements of patients in group 1 showed no significant differences. The usual muscle recessions (3 to 5 mm

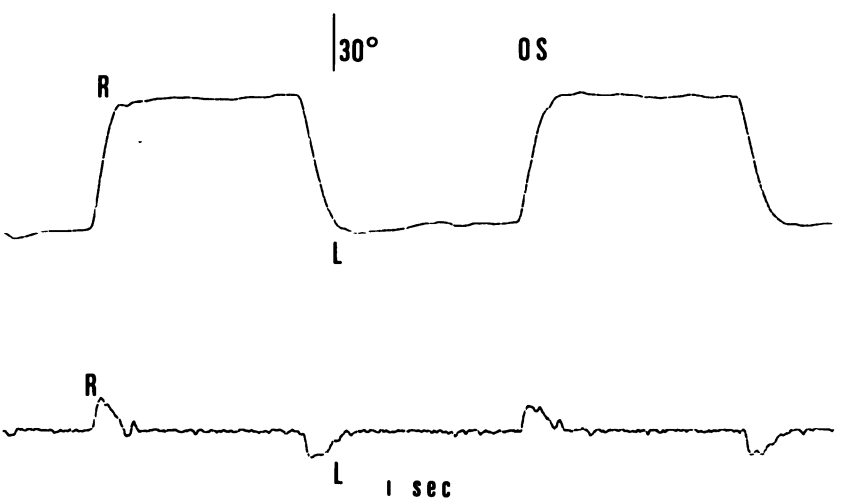

FIGURE 38

Electro-oculogram, OS, of horizontal saccades prior to surgery. Saccades right (R) and left (L) are normal. Eye position (upper trace) and peak velocity (lower trace) are shown.

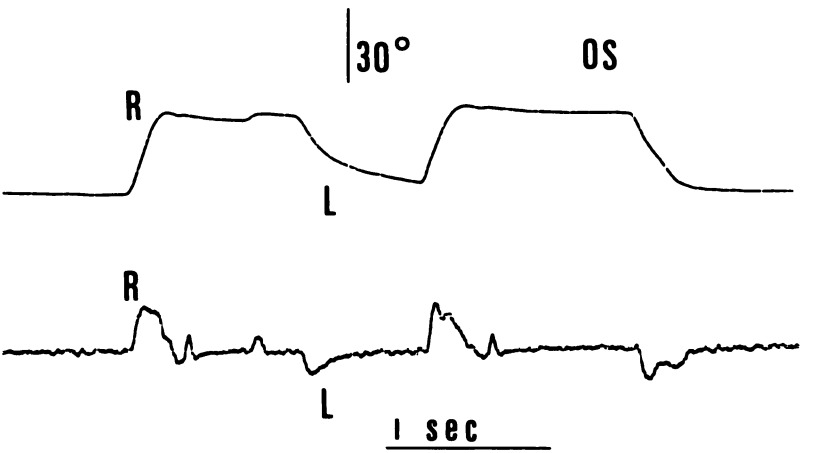

FIGURE 39
Electro-oculogram, OS, of horizontal saccades after left lateral rectus muscle was disinserted under topical anesthesia. Saccades right (R) are rapid and left (L) are reduced to about 40% of normal. Eye position (upper trace) and peak velocity (lower trace) are shown.

for the medial rectus muscle, 5 to 8 mm for the lateral rectus muscle, and 2 to 4 mm for a vertical rectus muscle) do not affect saccadic speed in the immediate postoperative period.

Patients in group 2 demonstrated moderate reduction of saccadic velocity into the field of action of the disinserted muscle. The difference between agonist and antagonist velocities was 48% (Fig 41). In those cases where the muscle was found and reattached to the sclera more anteriorly (seven patients), agonist saccades returned to essentially normal values (Fig 42).

COMMENT AND CONCLUSION

No reduction occurs in saccadic velocities produced after rectus muscle recession. This is true as soon as 1 day after surgery in spite of postoperative pain, irritation, muscle trauma, hemorrhage, and edema. When saccades are measured with rectus muscle disinsertion under topical anesthesia or with inadvertent muscle disinsertion (or tenotomy) following surgery for strabismus, velocity is reduced 40% to 50%. There is definite, although reduced, saccadic speed produced by the slipped muscle, probably because of posterior attachments to the globe and of intermuscular and tenon attachments that persist.

Saccades produced by a paralytic rectus muscle are extremely slow and can usually be observed to "float." Saccades caused by a disinserted

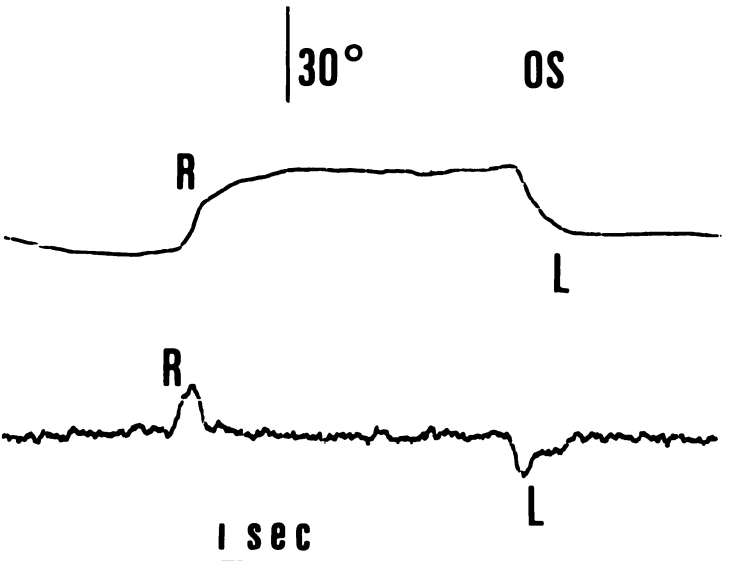

FIGURE 40
Electro-oculogram, OS, of horizontal saccades after both medial and lateral recti muscles were disinserted. Saccades right (R) and left (L) are reduced to 35% to 40% of normal. Eye position (upper trace) and peak velocity (lower trace) are shown.

muscle are somewhat more rapid, and recordings of eye movement are helpful in documenting the reduced velocity. Early diagnosis is helpful, since a slipped muscle is easier to locate within several days following surgery. Often a suture remnant helps identify the muscle, and the muscle may not have retracted far posteriorly. In addition, scarring is usually mild, and tissue dissection and exploration are less difficult. Good alignment with full rotations will be restored only if the disinserted muscle can be found.

INJECTION OF BOTULINUM TOXIN

In the early 1970s, Scott[85] began injecting various toxins into the extraocular muscles to cause a temporary paralysis. He found that a dilute solution of botulinum toxin was the most effective, controllable, and predictable substance. Temporary paralysis of ocular muscles was found useful in treating the antagonist of a paretic muscle.[85] In addition, it was of value in treating many forms of strabismus, causing a temporary change in orbital mechanical factors and ocular position. When the botulinum

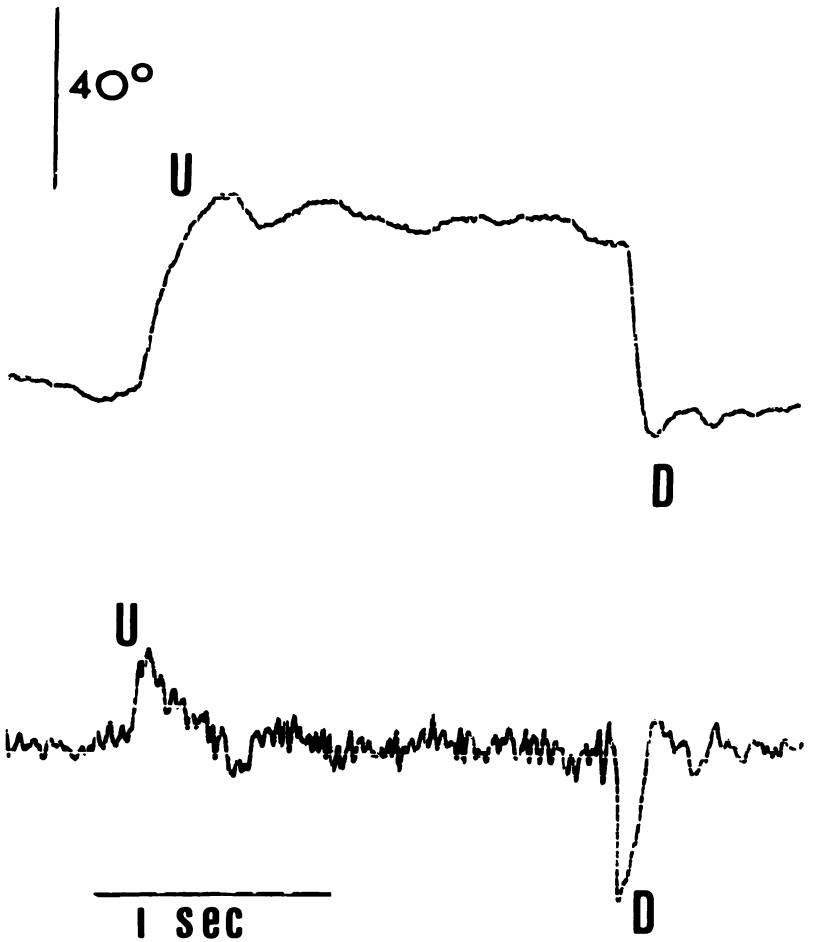

FIGURE 41
Electro-oculogram in patient with unplanned disinsertion of superior rectus muscle. Upward saccades (U) are moderately reduced in speed, while downward saccades (D) are normal. Eye position (upper trace) and peak velocity (lower trace) are shown.

toxin-induced paralysis had worn off, usually in 4 to 6 weeks, there was some return of the eye position to the preinjection alignment. However, the deviation was invariably reduced or eliminated. Large deviations frequently required more than one injection, while small deviations were often straightened with one injection.

Botulinum toxin is thought to interfere with the acetylcholine release from the nerve terminal.[86] It has been suggested that "botulinum toxin in

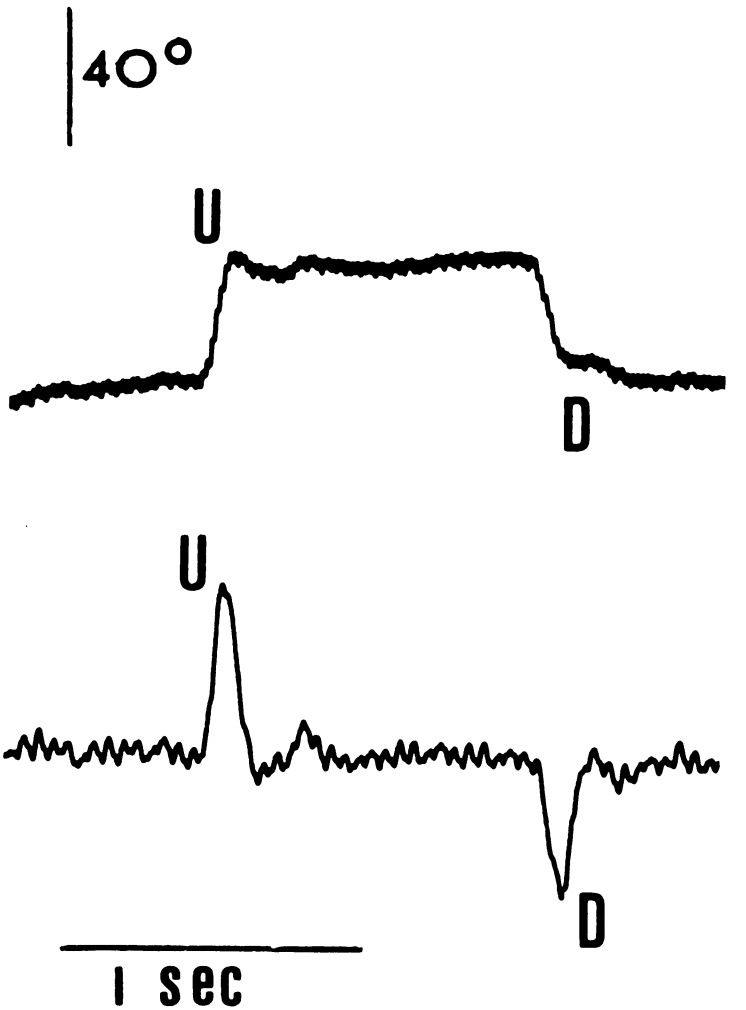

FIGURE 42
Electro-oculogram following location and reinsertion of superior rectus muscle following unplanned tenotomy. Both upward (U) and downward (D) saccades are normally rapid. Eye position (upper trace) and peak velocity (lower trace) are shown.

some ways antagonizes calcium ion transport by serotonin. After depletion of calcium ions, the end plate does not release acetylcholine and the muscle fiber fails to contract."[87]

We treated an adult patient with 50 PD of right esotropia with botulinum toxin injection into the right medial rectus muscle. There was a

history of two previously unsuccessful surgical procedures for strabismus. Adduction OD was full, with a minimal limitation of abduction.

Five days after injection, there was 20 PD of right exotropia and adduction was limited to the midline (Fig 43).

Horizontal saccades OD 5 days after injection demonstrated marked slowing of the nasal saccades, while temporal saccades were of normal velocity (Fig 44).

COMMENT AND CONCLUSIONS

Immediately following toxin injection, there is no change in muscle action or ocular position. It takes 2 to 4 days before an effect is seen. The aim is to cause paralysis of the injected muscle so the eye will move to the primary position or even become temporarily overcorrected. This prevents contracture of the antagonist of a paralytic muscle, if performed soon after onset. In other forms of strabismus, as the eye straightens, the antagonist of the injected muscle picks up the slack and causes an improvement in the ultimate ocular alignment by changing mechanical factors in the muscles and orbital tissues.

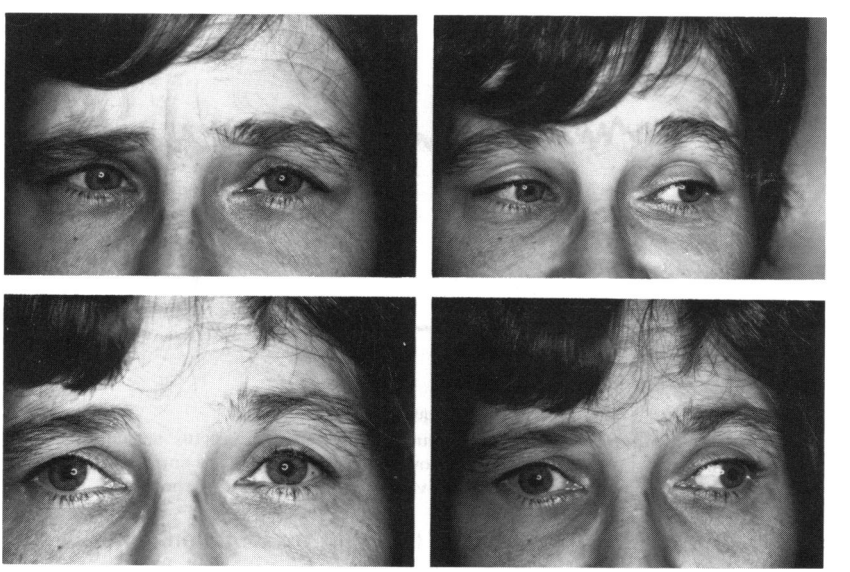

FIGURE 43

Top, Preinjection photographs; bottom, postinjection photographs. Top left, Primary gaze shows 50 PD of right esotropia. Top right, There is full adduction, OD. Bottom left, Primary gaze shows 20 PD of right exotropia. Bottom right, Adduction, OD, is limited to midline.

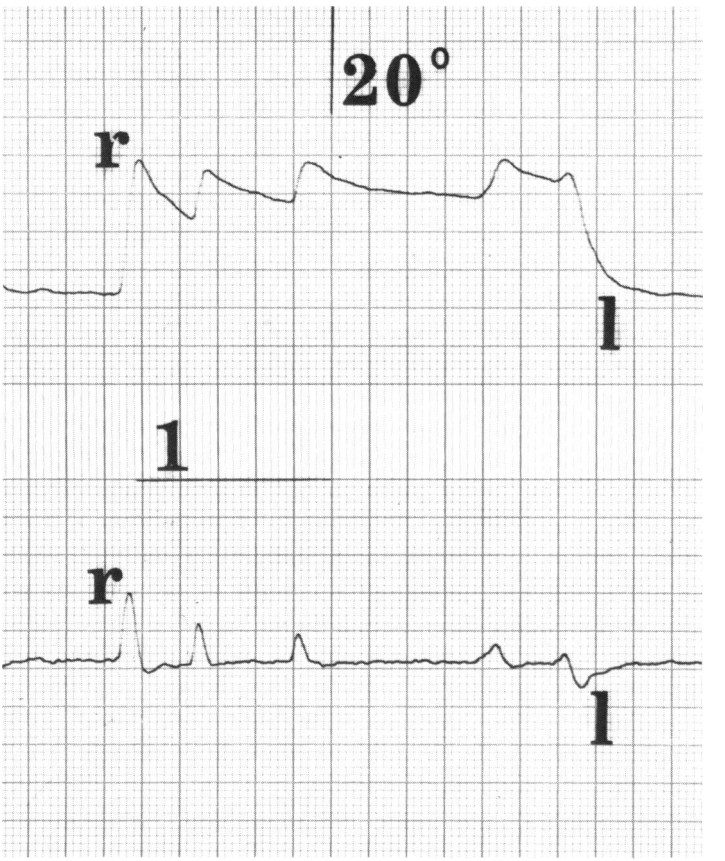

FIGURE 44
Horizontal saccades, OD, 5 days after botulinum injection into right medial rectus muscle. Eye position (top) and peak velocity (bottom) are shown. Lateral saccades to right (r) are rapid, while nasal saccades to left (l) are extremely slow. Calibration was 20°/s.

The marked slowing of saccadic velocity following injection of botulinum toxin is an indicator of the paralysis resulting from this procedure. The results are consistent with those noted after rectus muscle paralysis. This technique can also document muscle recovery as the effect of the toxin wears off in 4 to 6 weeks.

DISCUSSION

Huber[88] concluded that "oculography represents an ideal method for the analysis of pathologic eye motility and furnishes some important measur-

able parameters for the prognostic and therapeutic evaluation of oculomotor disorders of different types."

Yee et al[63] reviewed the concepts of how saccades are performed in normal persons. Single cell recordings from the neurons in the oculomotor nuclei of monkeys[89] and electromyography of human extraocular muscles[90] demonstrate that an increase in innervation in a pulse-step pattern reaches the muscle during a saccade. The pulse represents a sudden, large increase in firing rate of the oculomotor neurons, proportional in amplitude and duration to the size, speed, and duration of the subsequent saccade. Followed by extensive recruitment of muscle fibers, it is responsible for overcoming viscous forces of the globe and orbit and produces the high velocities reached during the saccade.

Collins[91] found that during saccades, the central fibers (fibers closest to the globe) of an extraocular muscle are primarily responsible for overcoming the viscous forces of the globe and orbit and for achieving high velocities in response to the pulse change in innervation. The orbital fibers are more important in maintaining the final position of the eye at the end of a saccade in response to the step change in innervation.

Mims and Treff[92] have measured horizontal saccades in normal subjects. They reported that differences between saccades produced by agonist-antagonist muscles averaged about 5%, while differences between symmetric muscles averaged about 9%. They believe that this type of analysis of the data allows more accurate detection of pathologic factors than comparison of a single saccadic velocity with an average in a normal subject because of intersubject variability.

Kushner[93] reported on a series of patients who underwent saccadic velocity testing after a posterior fixation suture was placed on the medial rectus muscle to correct a high AC/A ratio esotropia. Despite the success of surgery in eliminating the high AC/A ratio, the patients did not exhibit reduced velocities or progressive decrease in saccadic velocity as the eye moved further into adduction. This is consistent with our conclusion that muscle weakness, aberrant innervation, or disinsertion of a rectus muscle can result in reduced saccadic speed, but restrictions do not decrease velocity in the area where the eye is free to move.

Scott[21] pointed out that full paralysis of a horizontal rectus muscle gives a saccadic velocity of about 15% to 20% of normal. This is consistent with our results of saccadic velocities of 30° to 50°/s with complete paralysis, with a range of 200° to 320°/s in normal subjects.

Abel et al[94] studied the effects of age on characteristics of saccadic eye movement. They found that although the input to the fast eye movement

subsystem may deteriorate with time, the functioning of the saccadic generator itself remained unimpaired.

Slow refixation eye movements have been observed in patients with neurologic diseases such as Huntington's chorea,[95] Wilson's disease,[96] progressive supranuclear palsy,[97] spinocerebellar degenerations,[98] and familial ataxia.[99] In two patients with spinocerebellar degeneration, abnormal movements were thought to be slow saccades rather than substituted pursuit movements.

Quere and associates[100] pointed out that at present it is impossible to analyze complex motor impairments, such as strabismus or ocular palsies, and it is even less possible to claim to understand their pathogenic mechanisms without an EOG record. At this time, it does not appear possible to provide high-quality diagnostic and therapeutic care to patients with some forms of strabismus without the availability of recordings of saccadic velocity. The technique is noninvasive, relatively short, simple, and inexpensive, and can be used in patients of any age.[100] In cases of paresis, restriction, neuro-ophthalmic eye movement disorders, and possible slipped muscle, measurements of saccadic velocity are of great assistance in proper diagnosis, and thus in determining appropriate management.

SUMMARY AND CONCLUSIONS

Traditional evaluation of strabismus has included cover test measurements, evaluation of the range of ocular rotations, and an array of subjective sensory tests. These studies could not always differentiate paresis of an extraocular muscle from restrictions and from various neuro-ophthalmic motility disorders.

The measurement of horizontal and vertical saccadic movements can provide an objective test of rectus muscle function. Using EOG, saccades can be recorded easily, inexpensively, and repeatably at any age.

In ocular muscle paresis or paralysis, saccadic speed is reduced mildly to markedly and can be used to monitor recovery. Assessment of saccadic velocity does not appear useful in evaluating superior oblique palsy, although it is valuable in sixth nerve palsy, Duane's syndrome, and third nerve palsy.

When restrictions are the major cause of limited rotation, as in thyroid ophthalmopathy and orbital floor fracture, saccadic speed is unaffected.

The induction of OKN or vestibular nystagmus is helpful in the study of children too young to perform voluntary saccadic movements.

In patients with limitation of elevation or depression, this technique can separate innervational from mechanical causes of diminished rotation.

The specific saccadic velocity pattern in myasthenia gravis, progressive external ophthalmoplegia, internuclear ophthalmoplegia, and Möbius' syndrome is helpful in differentiating these disorders from other neuro-ophthalmic motility problems.

Transposition surgery of the rectus muscle is effective because of an increase in force, seen as an improvement in saccadic velocity and resulting from the change of insertion of the muscles.

Saccadic velocities can also be of assistance in diagnosing a lost or disinserted muscle following surgery for strabismus.

Although analysis of saccadic velocity is not required for the proper evaluation of all problems in strabismus and motility, it can be of inestimable value in the diagnosis of many complex and confusing disorders. Together with forced duction testing, a clinical profile can be obtained concerning muscle force and muscle and orbital restrictions, which are required information for appropriate surgical planning.

REFERENCES

1. Dodge R: Five types of eye movements in the horizontal meridian plane of the field of regard. *Am J Physiol* 1903; 8:307-328.
2. Cook G, Stark L: Dynamics of the saccadic eye movement system. *Commun Behav Biol* 1968; 1:197-204.
3. Tamler E, Marg E, Jampolsky A, et al: Electromyography of human saccadic eye movements. *Arch Ophthalmol* 1959; 62:657-661.
4. Magoon EH, Scott AB: Relationship of muscle force to ocular rotation and saccadic velocity. Abstracted, *Association for Research in Vision and Ophthalmology*, 1982.
5. Young LR: Measuring eye movements. *Am J Med Elec* 1963; 2:300-307.
6. Mowrer OH, Ruch RC, Miller NE: The cornea/retinal potential difference as the basis of the galvanometric method of recording eye movements. *Am J Physiol* 1936; 114: 423-428.
7. Metz HS, Scott AB, O'Meara D, et al: Ocular saccades in lateral rectus palsy. *Arch Ophthalmol* 1970; 84:453-460.
8. Huber A: New techniques in diagnosis of eye muscle palsies: A review. *J R Soc Med* 1980; 73:115-122.
9. Robinson DA: The mechanics of human saccadic eye movement. *J Physiol* 1964; 174:245-264.
10. Hyde JE: Some characteristics of voluntary human ocular movements in the horizontal plane. *Am J Ophthalmol* 1959; 48:85-94.
11. Metz HS: Third nerve palsy: Saccadic velocity studies. *Ann Ophthalmol* 1973; 5:526-528.
12. Huber A: Electrophysiology of the retraction syndromes. *Br J Ophthalmol* 1974; 58:293-309.
13. Metz HS, Scott AB, Scott WE: Horizontal saccadic velocities in Duane's Syndrome. *Am J Ophthalmol* 1975; 80:901-906.
14. Strachan IN, Brown BH: Electromyography of extra-ocular muscles in Duane's Syndrome. *Br J Ophthalmol* 1972; 56:594-599.
15. Jampolsky A: What can electromyography do for the ophthalmologist? *Invest Ophthalmol* 1973; 9:570-599.

16. Blodi F, Van Allen M, Yarbrough JC: Duane's syndrome: An electromyographic study. *Arch Ophthalmol* 1964; 72:171-177.
17. Pabst W, Esslen E: Symptomatology and therapy in ocular motility disturbances. *Am J Ophthalmol* 1964; 58:275-291.
18. Gourdeau A, Miller N, Zee D, et al: Central ocular motor anomalies in Duane's retraction syndrome. *Arch Ophthalmol* 1981; 99:1809-1810.
19. Rosenbaum AL, Carlson MR, Gaffney R: Vertical saccadic velocity determination in superior oblique palsy. *Arch Ophthalmol* 1977; 95:821-823.
20. Boeder P: The cooperation of extraocular muscles. *Am J Ophthalmol* 1961; 51:469-481.
21. Scott AB: Strabismus: Muscle forces and innervations. In Lennestrand G, Bach-y-Rita P (eds): *Basic Mechanisms of Ocular Motility and Their Clinical Implications*. Elmsford, NY, Pergamon Press Inc, 1975, pp 181-191.
22. Lennarson LW, Scott WE: Comparison of saccadic velocities in sixth nerve palsies: Paretic eye fixing vs non-paretic eye fixing. Abstracted, *ARVO*, 1979.
23. Metz HS, Scott AB, O'Meara D: Saccadic velocities in infants and children. *Am J Ophthalmol* 1971; 72:1130-1135.
24. Scott AB: Active force tests in lateral rectus paralysis. *Arch Ophthalmol* 1971; 85:397-404.
25. Metz HS, Woolf PD, Patton ML: Endocrine ophthalmomyopathy in adolescence. *J Pediatr Ophthalmol Strabismus* 1982; 19:58-60.
26. Metz HS: Saccadic velocity studies in patients with endocrine ocular disease. *Am J Ophthalmol* 1977; 84:695-699.
27. Kroll AJ, Kuwabara T: Dysthyroid ocular myopathy. *Arch Ophthalmol* 1966; 76: 244-257.
28. Pratt-Johnson J, Drance SM: Surgical treatment of dysthyroid restriction syndrome. *Can J Ophthalmol* 1972; 7:405-412.
29. Magora A, Chaco J, Zauberman H: An electromyographic investigation of ophthalmoplegia in thyrotoxicosis. *Arch Ophthalmol* 1968; 79:170-173.
30. Schimek RA: Surgical management of ocular complications of Grave's disease. *Arch Ophthalmol* 1972; 87:655-664.
31. Hermann JS: Paretic thyroid myopathy. *Ophthalmology* 1982; 89:473-478.
32. Smith B, Regan WF Jr: Blow-out fractures of the orbit: Mechanism on correction of internal orbital fracture. *Am J Ophthalmol* 1957; 44:733-739.
33. Metz HS, Scott WE, Madson E, et al: Saccadic velocity and active force studies in blow-out fractures of the orbit. *Am J Ophthalmol* 1974; 78:665-670.
34. Emory JN, von Noorden GK, Schlernitzauer DA: Orbital floor fractures: Long-term follow-up of cases with and without surgical repair. *Trans Am Acad Ophthalmol Otolaryngol* 1971; 75:802-812.
35. Metz HS: Saccadic velocity measurement in Brown's syndrome. *Ann Ophthalmol* 1979; 4:636-638.
36. Scott WE, Nankin SJ: Isolated inferior oblique paresis. *Arch Ophthalmol* 1977; 95:1586-1593.
37. Metz HS: Saccadic velocity studies with mechanical restriction of ocular motility. In Reinecke RD (ed): *Strabismus*. New York, Grune & Stratton, 1978, pp 53-60.
38. Knapp P: The surgical treatment of persistent squint. *Doc Ophthalmol* 1978; 34: 221-228.
39. Zaplia RJ, Winkelman JZ, Gay AJ: Intra-ocular pressure changes in normal subjects and the adhesive muscle syndrome. *Am J Ophthalmol* 1971; 71:880-883.
40. Stephens KF, Reinecke RD: Quantitative forced ductions. *Trans Am Acad Ophthalmol Otolaryngol* 1967; 71:324-329.
41. Metz HS: Double elevator palsy. *Arch Ophthalmol* 1979; 97:901-903.
42. Scott W, Jackson OB: Double elevator palsy: The significance of inferior rectus restriction. *Am Orthopt J* 1977; 27:5-10.
43. White JW: Paralysis of the superior rectus and inferior oblique muscles of the same eye. *Arch Ophthalmol* 1942; 27:366-371.

44. Jampel RS, Fells P: Monocular elevation paresis caused by a central nervous system lesion. *Arch Ophthalmol* 1968; 80:45-49.
45. Lennarson L, Scott W: Effects of large superior rectus muscle recession on upgaze saccadic velocities. Reported at *Association for Research in Vision and Ophthalmology*. Sarasota, Florida, April 1977.
46. Rosenbaum AL, Metz HS: Diagnosis of lost or slipped muscles by saccadic velocity measurements. *Am J Ophthalmol* 1974; 77:215-222.
47. Dunlap EA: Vertical displacement of horizontal recti. *Symposium on Strabismus*. St Louis, CV Mosby Co, 1971, pp 307-329.
48. Metz HS: Saccades with limited downward gaze. *Arch Ophthalmol* 1980; 98: 2204-2205.
49. Burian H, von Noorden GK: *Binocular Vision and Ocular Motility*, ed 2. St Louis, CV Mosby Co, 1980, pp 373-374.
50. Roper-Hall G, Burde RM: Inferior rectus palsies as a manifestation of atypical third cranial nerve disease. *Am Orthopt J* 1975; 25:122.
51. Cooper EL, Greenspan JA: Congenital absence of the inferior rectus muscle. *Arch Ophthalmol* 1971; 86:451-454.
52. Chu FC, Reingold DB, Cogan DP, et al: PSP syndrome. *Ophthalmol Times* 1979; 4:2.
53. Metz HS, Scott AB, O'Meara DM: Saccadic eye movements in myasthenia gravis. *Arch Ophthalmol* 1972; 88:9-11.
54. Campbell MJ, Simpson E, Crombie AA, et al: Ocular myasthenia: Evaluation of Tensilon tonography and electromyography as diagnostic tests. *J Neurol Neurosurg Psychiat* 1970; 33:639-644.
55. Stella S: Optokinetic nystagmus in patients with ocular myasthenia. *Invest Ophthalmol* 1967; 6:668.
56. Blomberg LH, Persson T: A new test for myasthenia gravis. *Acta Neurol Scand* [Suppl 13] 1965; 14:363-364.
57. Spector RH, Daroff RB, Birkett JE: Edrophonium infrared optokinetic nystagmography in the diagnosis of myasthenia gravis. *Neurology* 1975; 25:317-321.
58. Glaser JS: Myasthenia pseudo-internuclear ophthalmoplegia. *Arch Ophthalmol* 1966; 75:363-366.
59. Metz HS: Myasthenia gravis presenting as internuclear ophthalmoplegia. *J Pediatr Ophthalmol* 1977; 14:23-24.
60. Osher RH: Myasthenic "oculomotor" palsy. *Ann Ophthalmol* 1979; 11:31-34.
61. Spoor TC, Shippman S: Myasthenia gravis presenting as an isolated inferior rectus paresis. *Am Orthopt J* 1979; 86:2158-2160.
62. Baloh RW, Keesey JC: Saccadic fatigue and response to edrophonium for the diagnosis of myasthenia gravis. *Ann NY Acad Sci* 1976; 274:631-641.
63. Yee RD, Cogan DG, Zee DS, et al: Rapid eye movements in myasthenia gravis. *Arch Ophthalmol* 1976; 94:1465-1472.
64. Metz HS, Meshel L: Ocular saccades in progressive external ophthalmoplegia. *Ann Ophthalmol* 1974; 6:623-628.
65. Jampel RS, Okazaki H, Bernstein H: Ophthalmoplegia and vertical degeneration associated with spinocerebellar ataxia. *Arch Ophthalmol* 1961; 66:247-259.
66. Koerner F, Schlote W: Chronic progressive external ophthalmoplegia. *Arch Ophthalmol* 1972; 88:155-166.
67. Metz HS: Saccadic velocity measurements in internuclear ophthalmoplegia. *Am J Ophthalmol* 1976; 81:296-299.
68. Evinger CL, Fuchs AF, Baker I: Vestibulo-ocular and oculomotor deficits due to MLF lesions in monkeys: Anterior internuclear ophthalmoplegia. Reported at *Association for Research in Vision and Ophthalmology*. Sarasota, Florida, 1974.
69. Loeffler JD, Hoyt WF, Slatt B: Motor excitation and inhibition in internuclear ophthalmoplegia—an EMG study. *Arch Neurol* 1966; 15:664-671.
70. Smith JL, David NJ: Internuclear ophthalmoplegia: Two new clinical signs. *Neurology* 1964; 14:307-309.

71. Dell'Osso LF, Robinson DA, Daroff RB: Optokinetic asymmetry in internuclear ophthalmoplegia. *Arch Neurol* 1974; 31:138-139.
72. Siroky A, Krejcova H, Ymazal J: The early diagnosis of multiple sclerosis by monocular registration of evoked nystagmus. *Acta Neurol Scand* 1973; 49:205-214.
73. Fricker SJ, Sanders JJ: Abnormal timing of horizontal eye motion in early internuclear ophthalmoplegia. Reported at *Association for Research in Vision and Ophthalmology*. Sarasota, Florida, 1974.
74. Abbott RL, Metz HS, Weber AA: Saccadic velocity studies in Möebius syndrome. *Ann Ophthalmol* 1978; 10:619-623.
75. Van Allen MN, Blodi FC: Neurologic aspects of Möebius syndrome. *Neurology* 1960; 10:249-259.
76. Merz M, Wojtowicz S: The Moebius syndrome. *Am J Ophthalmol* 1967; 63:837-841.
77. Olmstead JMD, et al: Adaptation to transposition of eye muscles. *Am J Physiol* 1936; 116:245-251.
78. Adler FH: *Physiology of the Eye*, ed 3. St Louis, CV Mosby Co, 1959.
79. Metz HS, Scott AB: Innervational plasticity of the ocular motor system. *Arch Ophthalmol* 1970; 84:86-91.
80. Metz HS, Jampolsky A: Change in saccadic velocity following rectus muscle transposition. *J Pediatr Ophthalmol Strabismus* 1974; 11:129-134.
81. Metz HS: Changes in saccadic velocity following rectus muscle transposition surgery. Kommerell G (ed): *Symposium der Deutschen Ophthalmologischen Gesellschaft*. Munchen, JF Bergmann Verlag, 1978, pp 101-104.
82. Lian O: Muscle transplantation after complete severance of the medial rectus muscle. *Br J Ophthalmol* 1960; 44:636-637.
83. Scott A: Written communication, 1976.
84. Metz HS, Rice LS: Human eye movements following horizontal rectus muscle disinsertion. *Arch Ophthalmol* 1973; 90:265-267.
85. Scott AB: Botulinum toxin injection of eye muscles to correct strabismus. *Trans Am Ophthalmol Soc* 1981; 79:734-770.
86. Kao I, Drachman DB, Price DL: Botulinum toxin: Mechanism of presynaptic blockade. *Science* 1976; 193:1256-1258.
87. Boroff DA, Das Gupta BR: Botulinum toxin. In Kadis S, Montie TC, Ajl SJ (eds): *Microbiol Toxins*. New York, Academic Press, 1971, vol IIA: Bacterial Protein Toxins.
88. Huber A: New techniques in diagnosis of eye muscle palsies: A review. *J R Soc Med* 1980; 73:115-122.
89. Robinson DA: Oculomotor unit behavior in the monkey. *J Neurophysiol* 1970; 33: 393-404.
90. Miller JE: Electromyographic pattern of saccadic eye movements. *Am J Ophthalmol* 1958; 46:183-186.
91. Collins CC: The human oculomotor control system. In Lennestrand G, Bach-y-Rita P (eds): *Basic Mechanisms of Ocular Motility and Their Clinical Implications*. Oxford, England, Pergamon Press Inc, 1975, pp 145-180.
92. Mims JL, Treff G: Saccadic velocities of horizontal rectus muscles in twenty-five normal humans. *J Pediatr Ophthalmol Strabismus* 1982; 19:129-136.
93. Kushner BJ: The effect of the posterior fixation operation. *Abstract ISA Meeting*. Asilomar, California, 1982.
94. Abel LA, Troost BT, Dell'Osso LF: The effect of age on saccadic eye movement characteristics. Reported at *Association for Research in Vision and Ophthalmology*. Sarasota, Florida, 1981.
95. Starr A: A disorder of rapid eye movements in Huntington's chorea. *Brain* 1967; 90:545-564.
96. Kirkham TH, Kamm DF: Slow saccadic eye movements in Wilson's disease. *J Neurol Neurosurg Psychiat* 1974; 37:191-194.

97. Newman N, Gay AJ, Stroud MH, et al: Defective rapid eye movements in progressive supranuclear palsy: An electromyographic study. *Brain* 1970; 93:775-784.
98. Singh B, Ivamoto H, Strobos RJ: Slow eye movements in spinocerebellar degeneration. *Am J Ophthalmol* 1973; 76:237-240.
99. Murphy MJ, Goldblatt D: Slow eye movements, with absent saccades, in a patient with hereditary ataxia. *Arch Neurol* 1977; 34:191-195.
100. Quere MA, Péchereau A, Lavenant F: Kinetic electro-oculography: Aims, disadvantages and limitations. *Ophthalmologica* 1981; 182:73-80.