What can electromyography do for the ophthalmologist?

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Electromyography is of little use as a diagnostic tool in the usual clinical case of strabismus, and of only limited usefulness in frank neurologic disease. It has been valuable in understanding strabismus mechanisms, and in pointing out new laboratory-clinical correlations, which have led to some new clinical diagnostic techniques.

Key words: strabismus, electromyogram, neurologic disease, exotropia, exophoria, eye movements, convergence, divergence, nystagmus, fusion-vergence mechanism, myopathy, saccade, Duane's syndrome, thyroid disease.

What has ocular electromyography done for the clinician? It has unnecessarily confused him, and made him unnecessarily insecure in not always having access to this very limited clinical tool. I hope to make the clinical ophthalmologist secure and content in the knowledge that he will not be derelict in the clinical management of usual motility problems without this formidable, sometimes painful, and seldom helpful clinical tool.

I shall also attempt to point out some of its practical usefulness in frank neurologic disease, and to especially emphasize its laboratory value in helping to elucidate some fundamental questions in strabismus management.

I have chosen for the text of this presentation the following selection from Job 3:25, "For the thing which I greatly feared is come upon me, and that which I was afraid of is come unto me." To which I might add, relax, my fellow ophthalmologists, ye need not fear this complex technique, since excellent clinical ophthalmology may be practiced without its use.

At this point, I should like to state my conclusions, so that an appropriate overview may be maintained.

1. In the management of the usual clinical strabismus patient, ocular electromyography is more confusing than helpful.

2. In frank neurologic disease, there is a limited practical usefulness.

3. In understanding strabismus mechanisms, and in pointing out new laboratory-clinical correlations, which have led to some new clinical diagnostic techniques, electromyography has been a valuable tool.

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I shall first discuss electromyography as a diagnostic tool per se in the usual clinical strabismus case and in neurologic disease. Then I shall point out its value in helping us to understand strabismus mechanisms.

**Technique**

The technique of ocular electromyography has been thoroughly described. We shall be concerned here only with pointing out some of the common problems in data acquisition and interpretation of electromyograms that hopefully will be helpful to the clinician.

The technique has many practical limitations as far as its clinical usefulness is concerned. The insertion of needle electrodes directly into the oculorotary muscles is seldom possible in young patients, precisely those patients concerning whom one would often like to secure additional information. Some adult patients are poor candidates for this technique.

The equipment necessary for clinical
Fig. 1B. Head band, with jacks for leads from electrodes, in place on subject’s head, showing electrodes inserted into the muscles.

Ocular electromyography (Figs. 1A and 1B) requires careful electrical shielding, since what one wishes to measure is indeed a small electrical muscle potential. Needle electrodes must be inserted directly into the muscle (preferably muscles), and recordings made on tape or paper.

My co-workers and I have previously called attention to the frequent occurrence of electrical artifacts in the recordings and the errors in interpretation that may occur.

It should be noted that general anesthesia obliterates the muscle electrical tonus, thus negating the usefulness of the technique under these surgical conditions. Similarly, local injection anesthesia obliterates the electrical tonus, and the injected anesthetic solution easily filters from the site of injection to adjacent muscles, thus rendering the technique unreliable under these conditions.

I shall discuss (1) some sampling error possibilities, (2) some pitfalls of interpretation, (3) some past inferred, rather than real data, and (4) some points regarding sensitivity of the technique.

Sampling errors. The needle electrode insertion locale determines the amount of electrical recording of the motor units in the muscle. More or less electrical activity will be recorded depending upon whether the recording tip is deep in the muscle belly or more superficial or nearer the tendon end (Fig. 2).

A single motor unit may be recorded by special techniques. The particular motor unit recorded may or may not be a participant in the electrical activity generated by some of its fellow units, at a particular moment, during a particular function (Fig. 3).

The envelope of recorded activity (Fig. 2) does not give useful clinical information as to the relative “strength” of the muscle. Lancaster long ago pointed out that only a fraction of the total strength of the muscle was required for maximum rotation of the globe, and indeed, a muscle’s rotatory defect must be gross in order...
Fig. 2. Three electrodes in the same medial rectus muscle. Note that the activity appears to start in one trace earlier than in the others. When all traces are above threshold, the activity appears proportional in all channels. Hence, the higher threshold units appear to lag behind the lower threshold ones but do not in actuality.

Fig. 3. Patient with 25 prism diopters of esophoria. The right eye was uncovered, allowing it to abduct to fuse. Note the single motor unit in the right medial rectus (RMR) which completely dropped out as the right eye abducted to fuse. This particular single motor unit dropped out at this point. A different motor unit might well not have. Different interpretations might ensue in these two circumstances.

to be reflected in its electromyographic pattern.

An increase or decrease in a given electromyogram does not necessarily indicate that the eye has moved. Alas, one must accept the fact that one may have a change in the electromyographic activity without an eye movement, and vice versa.

Acceptable data with alternative interpretations. Even good electromyographic data fall heir to biased interpretation unless one knows some relevant facts concerning the precise conditions under which the data were acquired. Some of these necessary facts are as follows: (1) whether the recorded eye was fixing and maintained fixation during the recording; (2) whether the fellow eye was occluded, or whether there was bifoveal fusion during the recording; and (3) if fusion was present, was there a significant fusion-vergence operative at the time (i.e., a phoria).

An eye muscle has continuous electrical tonus during the wakeful state, manifested by continuous recordable unit activity. Under ordinary circumstances, when an
Fig. 4. Nasalward rotation from whatever starting point, whatever the cause, will show an increase of electromyographic activity of the medial rectus, and a reciprocal inhibition of the lateral rectus. Temporal rotation, from whatever position, whatever the cause, will show an increased electromyographic activity of the lateral rectus muscle, with reciprocal inhibition of the medial rectus.

In any case, the electromyogram (EMG) of the medial rectus will increase on attempted adduction, whether the eye movement is successful or not, i.e., even in the face of a restriction which prohibits adduction. Similarly, the electromyogram of the lateral rectus will increase with attempted abduction, whether successful or not.

Fig. 5 depicts some electromyographic examples of abduction eye movements occurring under a variety of situations. Note that when the eye rotates, the eye muscles move it, and the electromyographic record of agonist-antagonist muscles shows a reciprocal change in the motor unit activity as expected, regardless of the cause of the abduction movement.

The top trace of Fig. 5 depicts records from an abducting right eye during a version movement of both eyes to the right. Note the increase in recorded activity of the right lateral rectus, and decrease of activity in the right medial rectus. In the middle trace of Fig. 5, again there is an abducting right eye, but this movement is during a recession of convergence. In the bottom trace, there is also an abducting right eye, but now occurring after a break of convergence when the near point of convergence has been exceeded. All three traces reveal an increase in right lateral rectus activity, and decrease in right medial rectus activity.

The factor common to all three recordings is that the recorded eye was seen to
abduct, that is, to turn outward. In each example, the electromyogram is of the same type; increasing lateral rectus activity and reciprocal diminishing activity of the medial rectus of the moving eye. This was the case regardless of the cause of the abduction.

It is therefore apparent that monocular electromyograms recorded from the abducting eye above show the same electromyographic pattern, and therefore tell the clinician very little or nothing about the function or mechanism in play at the time of the recording. The only interpretation possible from such a monocular electromyogram is that the eye was abducting, or, at best, was attempting to abduct. Monocular electromyograms reveal only relative changes in eye position, or attempted changes in eye position, but in no way reveal “strengths or weaknesses” of relative degrees.

Inferred data. Curiously, data obtained from one lateral rectus muscle motor unit activity, in one eye, during a binocular divergence movement have been used to infer that both lateral rectus muscles’ EMG’s are similar. Several competent investigators have curiously followed the pattern of making conclusions relative to the electrical activity of both lateral rectus muscles from evidence based upon the activity of only one lateral rectus muscle, during the important and interesting binocular circumstance of the break from fusion by an intermittent exotrope.

Since Adler, in 1953, first recorded the electromyograph of a lateral rectus muscle during divergence from convergence, it has been clear that the lateral rectus has increasing motor unit activity in this situation. This certainly negates previously held views that this might be a
passive phenomenon, i.e., that the elasticity inherent in the orbital structures played a significant role during this abduction movement.

Others concluded from measurements of one lateral rectus in a patient with intermittent exotropia that "divergence is definitely associated with active innervation of the lateral recti" (italics ours).

Still others appeared to share this view by stating, "the fact that an active innervation of the lateral muscles (italics ours) occurs at the break point (of convergence) could be another proof for an active divergence mechanism."

My co-workers (Tamler, Marg, Scott, Thorson and Nawratski) and I have in no instance found both lateral recti increasing their activity during the break of fusion to exotropia. On the contrary, our binocular electromyograms of intermittent exotropia are similar in every detail to the known instance of diminution of convergence in normal individuals and in exophoria.

This curious inferring of what happens to both lateral rectus muscles, from data acquired from one lateral rectus muscle, has led to some oft-repeated misinterpretations, in our opinion.

**Insufficient sensitivity.** Investigation of the fusion-vergence mechanism by electromyographic techniques has provided conflicting views for the clinician. This is an extremely important area to be resolved by the clinician, for at the heart of this problem is the conflicting clinical view relative to the starting point and causation of ocular deviations. One of the fundamental questions in strabismus—hopefully to be clarified by EMG—was where is the starting point to one's thinking in horizontal strabismus? Should one think in terms of opposing con- and di-vergence (more or less of one or the other) acting to determine a basic or fusion-free and accommodation-free starting point? This view has the natural corollary that one would suppose that strabismus surgery might affect the vergences when one surgically moves ocularrotary muscles around. This is a commonly held view.

The alternative view—beautifully expressed by Walter B. Lancaster, with solid support since the time of Maddox, and one to which I wholeheartedly subscribe—is that there is an accommodation-free and a fusion-free position (basic deviation) from which all horizontal fusion-vergences act as a zero reference point.

Fusional-vergences are used when and if needed to the extent that they are needed, if available, from a reservoir. Con- and di-fusional-vergences do indeed exist, and as Lancaster succinctly pointed out, operate around any fusion-free position or point of fixation. The important difference in clinical outlook here is that a surgical procedure upon the ocularrotary muscles shifts the basic deviation, which is made up of mechanical and tonic influences, and does not directly affect the fusion-vergence mechanisms.

The electromyographic evidence in our laboratory has shown that surgery does indeed alter the basic deviation.

Thus, it is of great clinical importance to examine the electromyographic evidence relative to the fusion-vergence mechanisms, and relative to what indeed is the starting point or zero position (basic deviation) in strabismus.

I shall attempt to show that much of the "evidence" has been negative evidence, i.e., based upon insufficient sensitivity of the electromyographic technique in not always being able to measure a change in real fusion-vergence innervation reflected in motor unit activity.

I shall attempt to show that binocular myograms during fusion-vergence processes, especially in exophoria and intermittent exotropia, provide firm evidence in support of Lancaster's notion of a "basic-deviation" starting point (accommodation-free and fusion-free) as elucidated above. I shall emphasize that the important point in providing electromyographic evidence is that binocular electromyograms must be recorded, and that the fusion-vergence
must be at least 15 prism diopters in magnitude, with good recordings, for there to be sufficient sensitivity to show the appropriate changes necessary to support this view. Negative evidence based upon insufficient sensitivity due to sampling, or insufficient fusion-vergence magnitude, is inconsistent with the psychophysical eye movement data since the time of Hering and the binocular electromyograms from our laboratory group.

**Exophoria.** In exophoria there is general agreement that *fusional-convergence* is the fusion innervation required to keep the deviation latent. There is diplopia whenever the eyes are divergent. This we have noted to be the main differential diagnostic criterion distinguishing exophoria from intermittent exotropia.*

**Intermittent exotropia.** Intermittent exotropia poses "the problem" of whether it is active convergence which keeps the deviation latent or active divergence which produces the exotropia. Certainly it is not a passive phenomenon when an intermittent exotrope breaks from fusion into manifest exotropia.

It appears logical and consistent to state that, since it is generally accepted that fusional-convergence is the fusion innervation which keeps an exophoria latent, in the same way convergence keeps an intermittent exotropia latent. This author has previously emphasized that the two entities differ primarily in the presence or absence of suppression during any exotropia phase. Indeed, the very purpose of orthoptic management is to eradicate the suppression, thus converting intermittent exotropia into exophoria.

Another paradox is posed for those who propose that active divergence, rather than a diminution of convergence, is somehow associated with the manifest exotropia phase. This inexplicable and inconsistent switch in reasoning must have firm support if it is to be accepted.

Observation of the circumstance surrounding the break of fusion in intermittent exotropia reveals that it is often triggered by inattention. Alertness and attention are necessary for the maintenance of the psycho-optical fusion vergence reflex. Which vergence? Convergence, of course! Inattention causes the fusional-convergence to wane and the intermittently exotropic eye may be observed to return slowly, surely, and steadily to the fusion-free exodeviation. Would one seriously believe the paradox that inattention activated divergence?

All psychophysical data since the time of Hering support the concept that there is a peripheral mixture of version and vergence in an asymmetric vergence.* All the known characteristics of vergence innervations favor this view. Intermittent exotropia is no different.

It would have been supposed that human ocular electromyography, as an electrophysiologic counterpart to the psychophysical experiments, would have corroborated and complemented the previous conclusions. Rather, the electromyograms from several laboratories, as we have indicated, have been interpreted (on the basis of what we would consider negative evidence) as supporting the "active divergence" notion of intermittent exotropia breaking from fusion into manifest exotropia.

Fusion-vergence assessment by binocular electromyograms must have the following minimal criteria in order to avoid unwarranted conclusions from negative evidence: (1) binocular simultaneous electromyograms to record both lateral rectus muscles, and preferably all four horizontal rectus muscles; (2) a minimal asymmetric vergence of 15 prism diopters (in order to obviate negative evidence based upon insufficient sensitivity of measurement); and (3) fixation of the dominant eye to be unchanged, i.e., on the same target, at an unchanged fixation distance, in order to assess

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*Asymmetric vergence, i.e., fixation target stationary on the axis of one eye, is a much used laboratory circumstance by which to study vergence, since one eye remains stationary, and one eye does all the moving during the vergence, thus simplifying the analysis. Of course, for an intermittent exotrope, asymmetric vergence is his real way of life.
the circumstance of a pure fusion-vergence disjunctive movement.

When these criteria are followed, the lateral rectus of the fixing eye during the break of fusion does not increase its activity (as erroneously inferred from monocular electromyograms); rather, it decreases its activity, as it should in any creditable diminution of convergence, or shows no change at all. This may be repeatedly demonstrated for any unchanged fixation distance wherein the latent intermittent exotropia is made manifestly exotropic.

The following electromyograms show that in patients with exophoria and in patients with intermittent exotropia, there is never an increase in both lateral rectus muscles during the break of fusion following occlusion of one eye and the resulting manifest exodeviation. We shall show that this is in contrast to the situation where real fusional-divergence is in fact used (as in esophoric patients), where there is always an increase in both lateral rectus muscles when obtaining fusion and parallel alignment, from the fusion-free esodeviation position. Similarly, both lateral recti increase in the circumstance where a normal patient is subjected to base-in prisms and demonstrates a real divergence fusion movement. These latter two instances are indeed demonstrations of an actively invoked divergence mechanism, and in this circumstance one does record both lateral rectus muscles simultaneously increasing their motor unit activity.
Fig. 7. Top and bottom: Normal patient during slow asymmetric convergence from distance to near. Arrow represents break point of convergence followed by abduction of left eye. Note decrease in activity of stationary right lateral rectus as left eye abducts.

In Fig. 6, the arrow indicates covering of the right eye in a patient with intermittent exotropia of 25 prism diopters. The occlusion is followed by a break of fusion and manifest exotropia. As the right eye goes out, the right lateral rectus activity increases, and the right medial rectus activity decreases, as expected. But observe that the left lateral rectus decreases in activity at the same time. The fixation is unchanged throughout. Note also that the right lateral rectus does not "pull against" its antagonist medial rectus.

Active contraction of the lateral rectus muscle, after the break point of convergence in the eye which abducts (Fig. 7), is not evidence for the binocular function of active divergence. Again, when all four horizontal rectus muscles are recorded simultaneously around the break point of convergence it will be seen that the lateral rectus muscle of the "stationary" eye decreases in activity as the other eye abducts.

In both electromyograms of Fig. 7 the arrow represents the break point of convergence in two normal subjects, for comparative purposes. In each, the left eye abducts when fusion is lost, and the horizontal rectus muscles of the moving left eye show the expected reciprocal innervation. But note that, in each subject, the activity of the lateral rectus of the "stationary" right eye decreases after the break of convergence.

Fig. 8 shows a detailed analysis of the break of fusion (arrow 1) when the right eye is covered in a patient with intermittent exotropia. Note the gradual increase in activity of the abducting right lateral rectus, and the gradual reciprocal decrease in the right medial rectus, as this eye very slowly abducts to its fusion-free position of exotropia. This gradual motor unit reciprocity in the abducting eye is consonant with what is seen clinically, namely, the gradual "release" from fusion with a gradual dissipation of the long-maintained vergence. Note again that there is no surge of activity in the right lateral rectus which has been said to "pull against" a resisting medial rectus, when the recording is free of base-line artifacts.

One can observe a single motor unit in the left lateral rectus of the stationary fixing eye which reflects the diminution in activity as a decrease in frequency following the break of fusion (the left medial rectus recording is unsatisfactory).
Fig. 8. A higher recording paper speed (spread out) episode of covering the right eye (arrow 1), in a patient with intermittent exotropia.

Fig. 9 shows all of the aspects of refusion of 40 prism diopters of intermittent exotropia. The reciprocity in the moving right eye is clearly seen during its fast race toward foveal engagement during the refusion vergence. The initial disengagement of fixation of the "stationary" left eye is noted as a brief saccadic electromyographic pattern, a version in both eyes, of very short duration with saccadic complete inhibition of antagonist muscles. Once the briefly disengaged (and moved) left eye has regained fixation, and fusion is obtained, there is a clear demonstration of maintained co-contraction of both horizontal muscles of the fixing left eye (compare before and after arrow 1). The left lateral rectus recording shows a single motor unit which reflects its increase in activity as an increase in frequency. The left medial rectus recording, of many motor units, reflects its increase in activity by a similarly obvious maintained increase in discharge of many units.

Fig. 9 alone should cause doubters to accept the view expressed above which has been almost solely supported by our EMG laboratory group. A critical examination of this figure allows no other reasonable alternative interpretation of the innervational (motor units) events occurring in the eye muscles in the refusion of intermittent exotropia. The version-vergence mechanisms are reflected at the muscle level.

It is desirable, but not always practically possible, to document an eye movement by recording with a high-fidelity eye-movement device (such as a limbal-sensing device) simultaneously with electromyograms of all four horizontal muscles. Fig. 10 shows cover of the left eye (arrow 1) and the subsequent time course of the left eye movement (EM) after the break of fusion in intermittent exotropia and gradual diminution of the convergence.

Thus it can be seen that in a patient with intermittent exotropia or exophoria, when breaking from fusion into exotropia, there is the expected increase in electromyographic activity of the moving eye's lateral rectus as it abducts, but in every instance, there is a decrease in the activity of the stationary fixing eye's lateral rec-
Fig. 9. Arrow 1 shows uncover of the exotropic right eye, followed by a convergence refusion of 40 prism diopters of intermittent exotropia. It clearly shows both the reciprocity of the moving eye, and the maintained co-contraction of the fixing eye. Note the fixation disengagement of the fixing left eye in this brief but “powerful” refusion vergence. This electromyogram clearly shows all the details of refusion in intermittent exotropia.

Fig. 10. An eye movement (EM) recording channel has been added (limbal light-sensing device). Arrow 2 shows the beginning abduction of the left eye when covered, in the break of fusion (same patient as shown in Fig. 9).
Fig. 11A. Orthophoria, balanced electrical tonus compared with convergence. With an activated vergence, there is co-contraction (increased unit activity in both medial and lateral rectus muscles of stationary fixing eye), and reciprocity in the moving eye.

Fig. 11B. Esophoria, co-contraction in the fixing unmoved left eye during active divergence (to keep the phoria latent during fusion).
tus activity (a co-decrease in lateral and medial activity; with refusion, a co-increase).

When there is sufficient sensitivity of the testing circumstances, recorded data (as opposed to inferred data) from both lateral rectus muscles consistently show these changes. This is entirely compatible with the notion of diminution of convergence in this circumstance, rather than an active divergence.

We have shown similar changes in patients with esophoria. The common denominator is that whenever there is a circumstance of obtaining bifoveal fusion via a fusion-vergence mechanism, there is

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Fig. 11C. Intermittent exotropia, co-relaxation of fixing unmoved left eye during diminution of a real fusion vergence—in this case, diminished convergence after break of fusion by cover.

Fig. 12. Eye movements recorded during the break and make of fusion in a patient with a diagnosis of exophoria (10 prism diopters). In the upper tracing the dominant right eye is covered at arrow 1 and a very slow gradual dissipation of fusion-convergence occurs. At arrow 2 the right eye is uncovered and the initial slow movement (convergence) is followed, or masked by a fast version noted by arrow 3. The same maneuver was then repeated (right hand part of the diagram), this time with only a fast version re-fusion noted in the moving right eye. Note at arrow 3 that the fixing left eye is momentarily disengaged by the fast-moving right eye. This occurred to a greater extent during the second maneuver when there was a greater velocity of the moving right eye, during the re-fusion vergence process.
co-contraction of the stationary fixing eye’s medial and lateral rectus muscles in the asymmetric vergence circumstance (which allows one eye to remain stationary and unchanged in its fixation).

Fig. 11 diagrammatically shows the circumstances of pure fusion-vergences under a variety of conditions, with the appropriate diagrammatic electromyographic changes that are recorded.

Fig. 12 shows a binocular eye movement recording (electronic limbal movement sensing devices) during a cover-uncover (dissipation of vergence and obtaining of fusion-convergence) which shows the fast re-fusion eye movement of the moving right eye during asymmetric vergence, and also clearly shows the momentary dislodgment from fixation engagement of the usually firmly fixed stationary left eye. Eye movement recordings have long been shown to support the notion that there is a peripheral (muscular) manifestation of the mixture of fusion-vergence and the version movements, even in the circumstance of asymmetric vergence during such a re-fusion mechanism. It is of especial importance to note that the mixture of vergence-version is not to be looked upon as a neatly packaged centrally rearranged innervation flow to the peripheral muscles.

In conclusion, it is to be emphasized that the evidence completely supports Lancaster’s notion of the basic deviation, that fusion-free, accommodation-free position which is clinically determined by having the usually fixing eye fix a distant object, with accommodation neutralized completely, and fusion-vergence neutralized completely. Under these controls, with whatever tonic influences remain, this basic deviation should be the starting point of our strabismus thinking and concepts. Surgery can modify and shift the basic deviation.

One need not postulate, and indeed no evidence that I know of supports the contention that some mysterious supranuclear non-retinal tonic divergence. It is time to put this notion to rest. In my opinion, electromyography has helped to clearly establish this point, consistent with all the previous historical psychophysical and eye movement evidence.

**Blinks—as an artifact, and as a visual mechanism.** At the 1967 National Meeting of the Association for Research in Ophthalmology I presented evidence that a blink may play more than an incidental role in the visual processes. A blink is a curious and almost constant phenomenon associated with a re-fusion movement in intermittent exotropia. This is a blink which under usual seeing conditions invariably precedes the re-fusion movement. In fact, some intermittent exotropes cannot re-fuse without a blink, even when conscious attempts are made to re-fuse.

This might be supposed to be purely an incidental finding, but a more detailed examination will reveal that, once fusion is broken, and exotropia ensues, a refusion is often difficult if not sometimes impossible without a blink.

If the usually deviating eye is forced to fix by covering the dominant eye, the following sequence of events usually occurs when occlusion is removed in this nonusual situation: a blink occurs prior to refixation by the habitually dominant eye (a completed conjugated version), which is then followed by another blink which precedes the refusion convergence movement. In other words, the eyes appear to have to “get set” with the habitually fixing eye fixing; which is followed by the usual blink which in turn usually precedes or indeed may initiate the refusion. In some instances, a refusion cannot take place unless this precise sequence of events occurs. It is probable that a blink plays a significant role, therefore, in the refusion of intermittent exotropia.

What role does a blink play under normal circumstances in normal patients? Davson7 states that “the normal blinking rate is apparently determined by the ac-
Fig. 13. Patient with intermittent right exotropia of 70 prism diopters. Very high-speed photography during normal blink (frames 5 to 9) which initiated the re-fusion.

Curiously, a divergent movement occurs during the blink in patients with exophoria, as might be expected.
Fig. 14A. Same patient as shown in Fig. 13, with 70 prism diopters of intermittent right exotropia. An intentional blink of approximately 0.7 second elicits a bilateral convergence movement. (The normal duration of a blink is approximately 0.2 to 0.5 seconds.) This figure shows a series of three blinks, each one "pumping" the right eye in a convergent direction to join with the re-fixated left eye, so that fusion is obtained after the third blink (arrows 3, 4, and 5). The left eye was uncovered at arrow 2. Re-fusion was co-mingled with a blink.

Fig. 14B. Same patient as shown in Fig. 14A. Note the slow dissipation of the fusion vergence at the point of covering the left eye (arrow 1). Note also that when the left eye is uncovered (arrow 2), a partial convergence movement is considerably augmented by a blink, which resulted in immediate re-fusion.

Fig. 14C. Same patient as shown in Fig. 14B. This 70 prism diopter intermittent exotrope was then asked to attempt to align the eyes even in the absence of a fusional stimulus. Arrow 2 indicates the plateau area where the eyes were "aligned," and quite accurately so, despite the complete occlusion of the left eye. Arrow 3 marks the exotropic position attained after the subject "let" the eyes diverge to the fusion-free position of exotropia.
prism diopters of intermittent right exotropia. Frames 1 to 4 show occlusion manifestation of the exotropia. Frames 5 to 9 show normal blink. It will be noted that just prior to lid closure during the blink, the eyes appear to be in their full 70 prism diopters of exodeviation (frames 5 and 6) and that just subsequent to lid closure in the blink, when the eyes are barely opening (frame 7), the eyes appear to be aligned. Thus, during this short period of time, 70 prism diopters of movement of the exotropic eye occurs, with little apparent movement of the fellow fixing eye (a vergence).

Figs. 14A through 14E present a series of five eye movement recordings showing what would seem to be an essential role of a blink in the refusion process of intermittent exotropia. The role of a blink appears to be more than incidental.

The electrophysiologic investigation of eye movements invariably is associated with blink artifacts. The investigator comes to recognize the occurrence of a blink which often takes place at the precise moment of importance in studying the eye movement. This is especially true when fixation is changed from one position to another, or when an eye moves without refixation (during suppression) as in the fusion break of intermittent exotropia. The blink "artifact" which occurs at the precise "moment of truth" often obscures the information sought. The baseline electrical recorded artifact induced by the blinks

Fig. 14D. Same patient as shown in Fig. 14C. The left exotropic eye is uncovered at first arrow followed by a convergence movement which plateaus at a second, still insufficient, level of convergence. An initial blink is followed by another blink (second arrow) which is associated with the converging eyes and fusion is obtained. The third arrow is a blink during fusion.

Fig. 14E. Same patient as shown in Fig. 14D. Manifest exotropic phase (unfused exophoria) showing convergence movement occurring with, or initiated by, a blink, without re-fusion.
(which usually occur in all recorded channels) has sometimes led to important misinterpretations. We have noted previously, in discussing electromyograms, that such a misinterpretation of a blink as oculorotary muscle unit activity has led to the misconception that the lateral rectus "pulls against" the medial rectus, when an intermittent exotrope breaks from fusion into exotropia. As we have noted, when the blink artifact does not confuse this episode, smooth physiologic reciprocity takes place, as might be expected. Blink artifacts may be eliminated by electronic circuitry, or separately detected either by lid electromyography or lid movement light sensor.

On the other hand, asking the subject not to blink, or preventing a blink, by a speculum between the lids, or by other laboratory interference, may similarly produce an artifact. If a blink is a necessary part of the program of change of fixation in normals, or of re-fusion in intermittent exotropia, then not allowing a usual blink to occur might considerably alter the usual reaction time and speed of eye movement. Asking the patient not to blink, in order to get more stable recordings, may give spurious time relationships.

We have already speculated that the blink in re-fusion of intermittent exotropia does not serve the purpose of disengaging fixation of the exotropic eye, since it is already disengaged. In this instance, a plausible explanation of the blink "function" is that it may facilitate the receipt and delivery of information in the visual sensory-motor system during a switching episode. It is important to know that a blink in normal subjects may be accompanied by a slight vergence movement. It is probable that this vergence relationship is exaggerated in intermittent exotropia as demonstrated by the above figures, and serves a useful purpose.

A blink may represent a necessary visual pause while messages of one sort are received and of another sort are transmitted. Similar blinks have been noted in studying the reception of conflicting auditory messages.

For the present, we might speculate that a blink may be an important pre-programming step in the re-fusion mechanism, or aids the "switching mechanism."

In conclusion, some patients with intermittent exotropia must blink in order to initiate the re-fusion. Others cannot blink while exotropic without re-fusing. Less commonly, a blink must precede the break of fusion into exotropia. It appears that the blink in intermittent exotropia occupies a position of some importance, and we are further investigating this aspect of the problem.

**Summary.** The summary points of note at this juncture are as follows:

The electromyogram does not discern relative strengths and weaknesses of muscles, except for gross defects.

EMG changes reflect relative changes in eye position, or attempted changes in eye position. The electromyogram does not reveal what specific function the muscle may or may not be performing.

EMG's of fusion-vergences should have a minimal fusion-vergence amplitude of 15 prism diopters, and be accompanied by other relevant binocular eye movement data for adequate interpretation, such as: Was the recorded eye fixing, and did it maintain constant fixation? Was there fusion, or was the fellow eye covered? If fusion, what type of fusion-vergence was utilized to mask the latent deviation (phoria)?

Electromyography, insofar as helping the clinician is concerned in establishing a diagnosis, or in management of the usual strabismus patient, is of no practical usefulness (a comment also made about Bluebeard's seventh wife).

In a 1959 paper entitled "Artifacts and normal variations in human ocular electromyography" my colleagues and I quoted Sir Francis Walsh's discussion in "The Future of Neurology" at a 1955 meeting of the Royal Society of Medicine. He stated his conviction that "a fruitful
future for neurology lies rather with experimental pathology than with electrophysiological studies. In the case of the latter, it seems that many of the studies reveal more about the properties of the apparatus used than of the functions of the nervous system.12

**Use of electromyography in frank neurologic disease**

If electromyography is of little help to the clinical ophthalmologist in managing the usual strabismus patient, how does electromyography fare in patients with frank neurologic disease?

Electromyography does have some clinical relevance in helping to establish the diagnosis of myasthenia gravis, and in some myopathies.

**Myasthenia gravis.** This is ordinarily diagnosed by clinically observing gross improvement in eye movements immediately following test medical therapy. Sometimes, however, one might have a subclinical improvement, i.e., an increase in electromyographic response of the muscle, but insufficient to elicit observable improvement in eye movement. In this circumstance, an immediate increase in electromyographic activity during constant fixation (without eye movement) following the intravenous test dose may establish the diagnosis which would otherwise be undetected by ordinary clinical means. It is important to be aware of this differential diagnostic capability, since it may alter other diagnostic procedures which might be dangerous.

My admitted biased presentation becomes evident again, however, by taking note of the observation made by Stella13 in our laboratory.

Fig. 15 reveals that the far simpler test of optokinetic nystagmus stimulation and skin electrode oculographic eye movement tracings (ordinary nystagmogram) shows an increase of nystagmus frequency (as well as amplitude and velocity changes) immediately following intravenous test medication dose. This far simpler and easily available nystagmogram procedure, during optokinetic nystagmus, promises to enhance the sensitivity of the usual diagnostic procedure for myasthenia gravis, namely, that of simple clinical observation of improvement in eye movement.

**Myopathies.** In some myopathies, there may be poor ocular movement, and yet a fairly good electromyographic recording. This combination is almost pathognomonic of myopathy, and is a helpful diagnostic procedure in these otherwise confusing patients. There is poor mechanical muscle capability, despite apparently adequate electrical capability.

**Use of electromyography in understanding strabismus mechanisms**

Now let us discuss how electromyography, as a laboratory tool, has helped

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![Figure 15](image_url)

**Fig. 15.** This is an electronystagmogram of a subject with ocular myasthenia showing positive response to tensilon. Note increase in frequency of nystagmus (as well as amplitude and velocity), during unchanged fixations (after S. Stella).
understanding of strabismus mechanisms, and has opened new vistas for new and simple, yet quantifiable, strabismus diagnostic procedures.

If electromyography is of little clinical help in the common strabismus diagnostic situation, how does a clinician resolve the clinical problem of electrical versus mechanical events. That is, when an eye does not move in a given direction, how does one determine whether there is a weak agonist or a restriction of the antagonist (or surrounding tissues)?

What are the simple clinical measures that help resolve the important and common clinical problem of weakness versus restriction? We shall attempt to point out how electromyography has helped illuminate the problem so that new simpler clinical means, well correlated with electromyography, allow insights into this critical problem not heretofore appreciated.

**Electric versus mechanical events.** These may be differentiated by three clinical means: (1) observation of eye movements; (2) feeling the presence or absence of rotation restriction, by manual passive and active forced duction tests; and (3) measuring saccadic velocity differences in agonist-antagonist in the involved eye, or in comparing binocular yoke muscle velocities.

1. **Observation of eye movements.** The observation of limitations of eye movements during the usual examination of ocular rotations is a simple, valuable, traditional method. Two common examples are congenital so-called "double elevator palsy" and abduction weaknesses.

Traditionally a monocular limitation observed in the fixing eye's extreme gaze position has been termed a paresis of weakness. This is so, in my opinion, only if the patient's volitional limited rotation can be easily augmented by the examiner's manual forced passive duction rotation. If the examiner can easily rotate the eye past the point of the patient's volitional rotation limit during continued extreme gaze attempt, then and only then may one term this a weakness.

2. **Feeling for rotation restriction.** On the other hand, if the examiner's manual forced duction rotation is also limited, either at the same point as the patient's or at some further rotation point, then restriction in the antagonist muscle or its surrounding tissue is a cause of the limitation. This is usually found to be the case, rather than a weakness or a paresis (Fig. 16).

Dr. Alan B. Scott and his co-workers in our laboratory have pointed out that a contracture or tissue restriction has a "leash effect," i.e., there may be normal manual rotation to a point where the tissue resistance or "mud" impedes the globe's normal movement. Rotations past this "slow-down point" may be possible either volitionally or manually, but with care and practice this point may be determined manually and the type and degree of restriction evaluated. The notion of a "leash effect" in restrictions of motility has a
Fig. 17. Diagrammatic representation of left lateral rectus paralysis with secondary contracture of the medial rectus which limits abduction. A represents straight-ahead axis. B represents limit of volitional abduction, and also limit of manual forced abduction traction. Shaded area is area of no movement.

Fig. 18. Left lateral rectus paralysis with limit of volitional abduction at line C. In this instance, however, the manual forced abduction can be easily augmented from C to B before resistance and restriction to abduction is felt. Any degree of manual forced traction past the voluntary limit, diagnoses a gross weakness or paresis.

The most important clinical concept derived from combined laboratory electromyographic and mechanical muscle-pull (tension) studies by Scott and his associates. 14

Fig. 17 diagrammatically represents a situation where the patient's volitional rotation limit is the same as the examiner's manual forced duction limit (or leash effect, "orbital mud"), thus indicating a limitation primarily due to restrictive forces.

In other words, the eye cannot be easily manually rotated any degree beyond the point of volitional rotation limits. In this circumstance it is not possible to say more than that limitation is due to the restriction. There still might be a paresis. I have previously pointed out that this additional diagnosis may be made in suspected lateral rectus palsy by detaching the antagonist medial rectus muscle under topical anesthesia to see whether now abduction is volitionally normal. If it is still not possible, then a weakness or paresis can be diagnosed in addition to the restriction.

Fig. 18 depicts a condition in which the examiner's manual rotation can abduct the eye by traction some degree beyond the patient's volitional limit. Any degree of manual forced traction past the voluntary limit diagnoses a gross weakness or paresis. It is not necessary to be able to manually rotate the eye completely, since there may be only a partial restriction. Therefore, any degree of easy manual augmentation of the volitional limit signifies a gross weakness. We have used this clinical notion to advantage.

The restrictive forces may be due to operative procedures, prolonged eye position in large degree of strabismus, or following restrictive contractures which may follow an actual and real paresis, which may partially or totally recover. In the latter instance, immediately following a paresis there may be no contracture or restriction, in which case the manual forced augmentation of rotation may be to the extreme canthal limits, past the point of volitional restriction, signifying gross weakness.

As the contracture develops, manual augmentation of rotation past the volitional
limits to *any degree* still indicates a gross paresis, but complete manual rotation may not be possible (leash effect) if *some degree* of contracture has developed. Thus, both the gross weakness diagnosis, and the degree of contracture may be diagnosed by the nuances of the passive forced traction test.

It is only when the degree of contracture is such that it limits manual rotation at the same point of volitional restriction, that one is unable to say anything about a possible residual or complete weakness, and can only make comments relative to the presence or absence of contracture. Fortunately, however, this dilemma has been cleverly clarified by the development of the Scott active force-generation test.14

**The Scott “Active” Force-Generation Test.** Referring again to Fig. 18 (but as a different example, as cited here), suppose that the eye volitionally abducts to “B” by agonist action, or simply by relaxation of the antagonistic group of muscles. The important point is that the eye is observed to abduct, however it does so, at least to “B”. There is freedom of globe rotation without restriction within points “C” to “B.”

Now if within this area of unrestricted rotation (C-B), the globe is *manually held steady* (in contradistinction to manual passive rotation), the actual force generated by the agonist activity may be manually felt against the forceps steadying influence, when the patient is asked to voluntarily move the eyes in the direction of limited movement. Under normal circumstances, the considerable agonist force is easily felt. If there is any significant paresis, this considerably diminished force generated by the agonist may be manually detected. This notion, derived from laboratory evidence of mechanical eye-pull (tension) investigations, muscle force determinations, and electromyographic studies, has evolved into a simple direct and most useful clinical test—the Scott active force-generation test. It is an important extension of the passive manual forced traction test, only in this instance it is a manual *steadying* maneuver in an area where additional unrestricted maneuver has been observed to be possible, and the *active* force of that agonist rotation is manually assessed.

To recapitulate, the observation of limitation of volitional eye movement is an important traditional motility test. The manual forced traction test has a *passive* rotation aspect which may define the leash effect of a restrictive limitation to movement, and additionally may define a gross weakness. The *active* aspect of the manual rotation test steadies and prevents rotation within an area where rotation is observed to be possible and unrestricted, and further defines the problem of weakness versus restriction.

The nuances of these tests are so well correlated with laboratory techniques that these simply performed clinical maneuvers very adequately obviate the necessity of electromyography in most of these instances.

Now we shall discuss the third clinical method of resolving the problem of weakness versus strength.

3. Saccadic velocity comparative measurements. The velocity of a saccade is a reliably repeatable and recordable event in a given individual. A decrement to this velocity (for a given saccadic amplitude) compared with a previously recorded similar finding, or compared with the velocity of its antagonist, or the velocity of its yoke muscle in a version saccade, is an excellent index of a quantitative decrement in muscle function, far more sensitive than other clinical tests. Similarly, the recovery of this velocity is a sensitive index of quantitative muscle recovery. Elaboration of this simpler technique is more sensitive than electromyography.

Direct clinical observation of the decreased saccadic velocity was first pointed out by Sherrington. When the agonist lateral rectus is completely paralyzed, nor-

*A saccade is a fast eye movement. A voluntary saccade quickly changes yoked fixations from one point to another.*
mal saccadic velocities occur in adduction (medial rectus agonist), but in an attempted volitional saccade from an adducted position to an attempted temporalward saccade, there is a slow "floating" eye movement, due primarily to the antagonist medial rectus inhibition and relaxation rather than agonist lateral rectus activity. This slow floating movement toward midline in the direction of the paralyzed agonist may be observed clinically in man and is pathognomonic of complete paralysis.

We have previously reported such a simulated paralysis after procaine injection (and electromyographic silencing) of one of the four horizontal rectus muscles. Fig. 19 shows electromyographic patterns of saccades from extreme left to extreme right gaze before and after the left medial rectus has been completely paralyzed by direct procaine injection. Note that during the saccade to the right (left medial rectus agonist fixation) there is a nice demonstration of Hering's law, with electromyographic overaction of the yoke right lateral rectus (compare with normal A). Additionally, during the volitional attempted saccade to the right (B), the left fixing eye was noted to "float" from abduction (left gaze) toward the midline (attempted right gaze saccadic adduction), primarily due to relaxation of the antagonist left lateral rectus. Nevertheless the innervation exaggerated attempt by the agonist (procainized) left medial rectus was reflected in the right lateral rectus yoke overaction.

More subtle decreases in saccadic velocity, which as previously mentioned is an index of decreased muscle function, may be assessed by using as a comparative control either (1) the direct antagonist velocity or (2) the yoke muscle velocity during an equal version saccade.15, 17

Such voluntary elicited saccades are simply clinically elicited by commanding a quick voluntary right and left saccade in the direction opposite the affected muscle (monocular comparative observation), or, alternatively, commanding a voluntary saccade in the direction of the affected muscle (binocular comparative velocity of the equally yoked version). Metz, Scott, and co-workers in our laboratory are thoroughly exploring this valuable clinically applicable tool of comparative saccadic velocities and have reported their preliminary findings.

Additionally, involuntary yoked saccades are easily elicited clinically by optokinetic nystagmus stimulation, and each eye movement is separately monitored by simple skin electrode oculograms (nystagmograms). Clear focus on velocity compar-

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**Fig. 19.** A shows electromyograms recorded from all four horizontal rectus muscles during extreme right to left gaze and return saccadic movements. B shows the same after procaine electrical silencing of the left medial rectus muscle. Note that when the left eye was the fixing eye in attempted right gaze saccade, there was a yoke overaction exhibited in the right lateral rectus (compare right lateral rectus activity in right gaze B versus right gaze A). The left eye was noted to float from left to right gaze midline during what ordinarily would have been a fast saccadic velocity, during saccadic fixation from left to right.
sons may be made by available velocity channels as depicted in Figs. 20 A, 20 B, and 21 (after Metz, Scott et al.).

It can be seen from these figures that voluntary saccades over a large amplitude of eye movement reveal any significant difference in what should be equal right-left saccadic velocities, either by observing the difference in slopes of the velocity curves (see Fig. 20) of voluntary right versus left gaze, or by comparing the special velocity channels (compare the height of the velocity channel right eye, left eye in Fig. 20).

Shorter saccadic velocity amplitudes can be elicited by optokinetic nystagmus stimulation with recorded eye movements, so that frequently repeated involuntary nystagmus saccades may be compared by velocity channels (Fig. 21). Additionally, yoked (binocular) velocity comparisons may be made.

It is my opinion that this technique of comparing saccadic velocities is a most practical clinical and sensitive measurement of impaired muscle function, (i.e., weakness) and I would hazard a prediction that this quantifiable clinical assessment will be one of the most fruitful new diagnostic aids in strabismus diagnosis. Its derivation history is of interest.

**Summary.** In summary, ocular electromyography as a laboratory tool has served well in helping illuminate and define simpler clinical tests (such as oculography saccadic velocity comparisons, passive forced traction, and active forced-generation stabilization tests), rendering unnecessary the technique of electromyography per se.

For the usual strabismus patient, (1) **observation** of eye movements, (2) **feeling** areas of restriction and weakness by passive and active forced traction and forced stabilization tests, and (3) **measurement** of saccadic velocity differences by skin electrode eye movement tracings provide a strong and useful armamentarium for the clinician in strabismus management.
Use of electromyography in other clinical conditions

Finally, there are two clinical conditions—Duane's syndrome and thyroid motility defects—where electromyography again has helped unravel the problem in such a way that new insights into clinical diagnoses by ordinary means have been facilitated.

**Duane's syndrome.** This has been shown to be sometimes associated with an active co-contraction (EMG) of agonist and antagonist muscles (medial and lateral recti) on attempted adduction. In such cases there may be co-inhibition of both muscles during attempted abduction. It is my clinical impression that, when significant enophthalmos occurs with attempted adduction, this is correlated with active co-contraction. Minimal or absent adduction enophthalmos clinically infers minimal or lack of co-contraction in adduction.

It is of clinical importance to make this distinction in Duane's syndrome, since the surgical effect of recessing the medial rectus will be considerably augmented if there is co-contraction, i.e., the lateral rectus activation during adduction considerably augments the weakening effect of the medial rectus recession. On the other hand, if there is simply a dead or inactive lateral rectus, a medial rectus recession will have considerably less effect when opposed by such an inactive muscle.

These clinical predictions are useful in planning surgical management of Duane's syndrome. Naturally, the forced traction test for both medial and lateral rectus restrictions, and inspection of the lateral rectus muscle, are often helpful in conjunction with noting the presence or absence of significant enophthalmos on attempted adduction.

**Thyroid defects.** Thyroid disease of the ocularotary muscles is another area where electromyography has helped elucidate for us the often perplexing problem of retraction of the upper lids.

In Fig. 16 we noted that mechanical restriction to upgaze globe rotation may prevent the eye from actually rotating upward with volitional attempts, as indicated by increasing electrical activity of the attempting superior rectus muscle. The more the (unsuccessful) attempt, the more retraction of the upper lid during the attempt. This is true regardless of the cause of the restriction to upgaze.

Some time ago we theorized that in thyroid restrictions to upgaze (due to restriction of the inferior rectus) it was likely that some or all of the upper lid retraction could be due to excessive innervation to the levator associated with

![DRUM LEFT](image1)

**Fig. 21.** Electrooculogram (LE) with optokinetic nystagmus (OKN). Left lateral rectus palsy. Upper trace, drum left (fast phase of OKN to the right), involuntary saccades right (R) are fast and sharp. Lower trace, drum right (fast phase of OKN to the left), saccades left (L) are slow and drifting.
upgaze restriction, producing the levator overaction-retraction. The levator overaction-retraction from this cause may be present even in primary position gaze, because the upper lid has the freedom to overact if the upgaze globe restriction was stressful even to primary position fixation.

Although it is a common belief that inspection of the muscles in thyroid disease reveals them to be enlarged and edematous, this may be true only during the wet stages of the disease. In my experience, it is more common to find hypertonic muscles (all muscles) which appear to be normal by direct inspection but retract very markedly indeed when severed from the globe. Recession of as much as 10 mm. for such a muscle may be necessary in order for the dynamic forced duction test during surgery to allow unrestricted manual duction in the opposite direction. The hypertonicity persists during general anesthesia.

In some patients, the cosmetically disfiguring upper lid retraction, is due almost entirely to the restriction of upward globe rotation caused by inferior rectus restriction. In such instances, recession of the inferior rectus, sufficient to allow unrestricted upgaze rotation, eliminates the overaction of the levator with attempted upgaze, thus correcting the levator overaction-lid retraction.

Fig. 22 shows a patient with retraction of the upper lid in primary position fixation. Note that in downgaze there is no lid retraction, with no lidlag on the left and minimal on the right.

Figs. 23A and 23B show bilateral 7 mm. recessions of the inferior recti, which appeared to be normal in all respects except for the marked hypertonicity, i.e., spastic type of retraction when severed from the globe.

Fig. 24 shows the postoperative result, with absence of retraction of the upper lid in primary position gaze, restored upward rotation of the globe, and much...
reduced retraction of the upper lids even in far upgaze.

This very preliminary report is given here only as another example of the question—posing and answering value of electromyograms in all restrictive rotations, by helping to point out that the mechanism of lid retraction in such a patient is simply an overaction of the levator due to restriction of the inferior rectus. Such a surgical approach is obviously not a panacea for all patients with lid retraction, since the causes are many.

However, EMG and traction tests have made us aware that when the following triad is present, the upper lid retraction may indeed be due to a levator overaction-lid retraction, and be related primarily to the restriction of the inferior rectus as a result of the thyroid disease. The diagnostic triad is as follows: (1) little or no lidlag in downgaze, (2) upgaze globe rotation restriction, and (3) upper lid retraction which starts at the point where upgaze restriction begins.

When this triad is present, one might anticipate a marked amelioration of the lid retraction even in the primary position, upon sufficient freeing of the inferior rectus restriction. Thus, two goals are accomplished by inferior rectus recession: (1)
improved upgaze globe motility and (2) improvement of that part of the lid retraction due to the levator overaction-retraction mechanism.

If there is significant lid lag with lid retraction in downgaze it is obvious that causes other than simple levator overaction-retraction exists.

When indicated, the inferior rectus recession procedure must be sufficient so that there is no limitation to upgaze by forced traction or volition, if the cause is to be eradicated.

Conclusions

1. Electromyography is of no practical usefulness in management of the usual strabismus patient.

2. Electromyography has proved to be a very valuable laboratory and investigative tool insofar as providing understanding of the electrical-mechanical events of ocular rotations. This has been most helpful in leading to new concepts relative to some extraocular muscle clinical pictures, in defining the basic deviation, and especially insofar as re-focusing attention upon and expanding the immense value of passive and active forced traction and stabilization tests.

The technique has also aided the development of the far simpler and more sensitive practical clinically useful eye movement quantified measurements (nystagogram equipment) for comparison of saccadic velocities as an index of impaired muscle function (weakness).

3. Electromyography has limited usefulness and application in some neurologic diseases affecting the eye muscles.

Eleven years ago my co-workers and I published the following statement concerning the clinical value of ocular electromyography. “Human extraocular electromyography is an important research tool but is of limited clinical value. If there is, at present, a practical usefulness of the technique, it lies in neuro-ophthalmological diagnosis and prognosis.” . . . “Usually, limitation of eye movement by paralysis, paresis, or mechanical restriction can be diagnosed by clinical means such as observation of eye movements and the forcedduction test under general anesthesia. Electromyography only confirms these findings. Therefore, electromyograms are, generally speaking, neither necessary nor helpful in usual practical motility problems.”

I find no compelling reason to significantly alter that 1959 statement, except to add that electromyography has been an important laboratory tool which has helped understand some specific strabismus problems, and to better define some fundamental strabismus concepts.

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