

AN ELECTROMYOGRAPHIC STUDY OF ASYMMETRIC CONVERGENCE*

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Asymmetric convergence refers to convergence occurring in any other direction than along the median plane. In this paper, we are concerned only with a special type of asymmetric convergence in which the fixation points are restricted to the stationary visual axis of one eye. In the past few years, there have been several reports by various investigators using different techniques to determine what occurs in the apparently stationary eye during this type of asymmetric convergence, and how their findings correlate with Hering's law of equal innervation to the extraocular muscles.¹⁻³

The problem is this: In Figure 1, as the right eye converges from A to B, two points along the axis of the left eye, the left eye appears to remain stationary. Yet, according to Hering's law, the left lateral rectus should receive a stimulus for levoversion, and the left medial rectus for convergence. What happens, if anything, to these innervations to the left eye to enable that eye to remain apparently stationary?

Hering⁴ himself simply explained that the two innervations, one for version and one for

vergence, acting in opposite directions, counteract each other, resulting in no movement of the eye on whose axis the target lies. Hering felt that both innervations actually reached the antagonistic muscles of the stationary eye rather than balancing out centrally in the brain. He even suggested that this might be proved by testing for an increase in intraocular pressure in the stationary eye. In addition, he was able to hear an increase in muscle noise in the stationary eye by means of a "noise funnel" (Schalltrichter).⁹ He also described a slight quiver of the apparently stationary eye, suggesting that this oscillation resulted from different speeds of the two antagonistic innervations in reaching the eye.

Breinin¹ had observed by electromyography that whenever an eye turned, be it during a version or vergence movement, there was an increase in the electrical activity of the agonist and reciprocal inhibition of the antagonist. When he noted no change in activity in either horizontal rectus of the stationary eye during asymmetric convergence, he concluded that the antagonistic innervations for a version and vergence movement cancelled each other in the brain and never reached the muscles. He says that it must be so, for if the innervation of that eye were to change, it would have to move, but fixation precludes such movement. The

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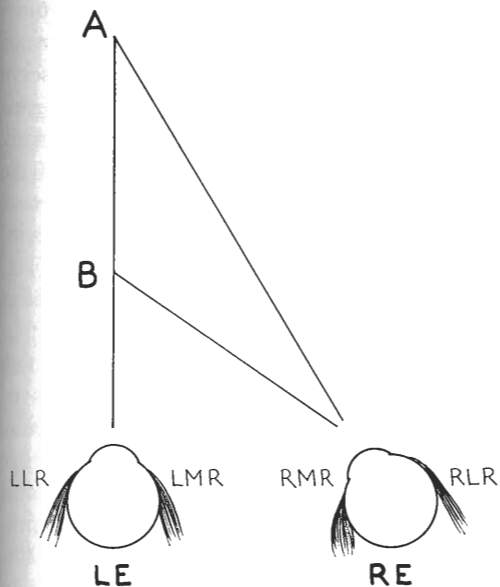


Fig. 1 (Tamler, et al.). Diagram of asymmetric convergence in which fixation points A and B lie on the stationary visual axis of the left eye.

alternative, he goes on, of an equal increase in innervation of medial and lateral rectus, does not occur. In view of the fact that Hering did not believe in central neutralization of opposing innervations and, indeed, noted slight movement of the apparently stationary eye, it appears to us that Breinin's electromyographic data do not agree with Hering's interpretation of this phenomenon. In a more recent statement on this problem, Breinin does not significantly alter his point of view.^{5, 6}

Shortly after this work of Breinin, two other reports appeared utilizing other techniques, which conflicted with Breinin's ideas. Westheimer and Mitchell² recorded the movements of both eyes during rapid asymmetric convergence by imaging the reflections of a light source in each of the subject's corneas on a continuously moving photographic film. They noted two separate movements of both eyes. First, there is a saccadic or rapid version movement of the two eyes. This is quickly followed by a convergence movement of both eyes, bringing the visual axes in position to intersect at the new fixation point.

Thus, even the apparently stationary eye swings out in unison with the version movement of the other eye, and then is brought in again during the binocular convergence response, so that it finally assumes the same direction it had at the outset. The same observations were made by Dodge⁷ in 1902. Such movement indicates that the innervations reach the muscles of the "stationary" eye and that there is a peripheral adjustment rather than a central neutralization.

Alpern and Ellen³ confirmed the findings of Westheimer and Mitchell using electro-oculography.* In binocular asymmetric convergence, they, too, found that the eye on the axis of the target makes a small rapid movement at first, but then quickly returns to its original position. They also noted similar movements in the fixating "stationary" eye when a small horizontal prism (five to 15 prism diopters) is suddenly introduced before the other eye. Their findings also indicate a peripherally manifested adjustment between vergence and version. They offer an explanation for the electromyographic failure to record change in activities in the involved muscles of the "stationary" eye by suggesting that the needle electrodes did not pick up enough electrical activity during these relatively small movements to give an observable change on the tracing.

An electromyographic study by Blodi and Van Allen⁵ agrees with Breinin that there is no change in electrical potential of the horizontal muscles of the apparently stationary eye during asymmetric convergence. It is interesting to note that they do report one instance of an increase from 60 microvolts to 80 microvolts in activity of the external rectus of the stationary eye just before break of convergence. This is a change of at least 20 percent.

So, to summarize the recent literature thus

* Electro-oculography is a technique discussed by one of us (E. M., Arch. Ophth., 45:169, 1951) for measuring direction and amount of eye movement by the use of surface electrodes on the skin around the orbit.

far on this subject, we find Hering's view of peripheral adjustment of opposing version and vergence innervations confirmed by the nonelectromyographic work of Westheimer and Mitchell and Alpern and Ellen. The electromyographic data to date, that of Breinin and Blodi and Van Allen, appear to be in conflict with this.

It is the purpose of this paper to present electromyographic data which support Hering's original thesis as confirmed by Westheimer and Mitchell and Alpern and Ellen.

Our technique of multiple channel electromyography has been described in detail in another paper.⁸ Using this approach, we have tested asymmetric convergence by the following two stimulus methods:

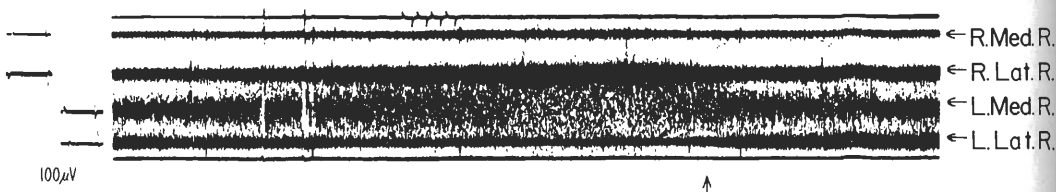
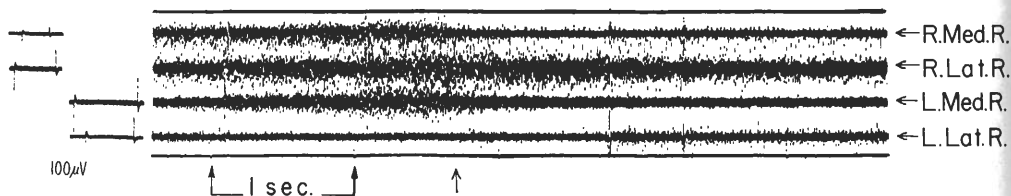
1. Slowly and smoothly bringing a fixation object (light source) in along the axis of one eye until the near point of convergence (break point) was exceeded. Both eyes were open and fusing up to the "break point."

2. Covering and uncovering one eye while the other eye maintains fixation on a stationary target.

The sensitivity of the apparatus is such

that the change in electric activity of a muscle corresponding to a slow movement of less than eight degrees is difficult to observe on the electromyographic tracing. Fortunately, the near point of convergence in many subjects comes to within a few inches of the eye and, therefore, the angular movement induced was large enough so that yoke innervation to the ipsilateral, apparently stationary, eye could be recognized on the electromyogram.

Figure 2 is the electromyogram of smooth asymmetric convergence along the axis of the right eye. In the first part of the tracing, as convergence proceeds and the left eye turns inward, there is increase in electrical activity of the left medial rectus and reciprocal decreased activity in the left lateral rectus. The arrow indicates the near point of convergence after which the left eye rotated outward. Therefore, after the "break point," there is an increase in activity of the left lateral rectus and a reciprocal decrease in the left medial rectus. Now, all during the time that the left eye was converging and diverging, the right eye remained stationary.



Figs. 2 and 3 (Tamler, et al.). (Above, fig. 2). Binocular smooth asymmetric convergence along the axis of the right eye. Arrow indicates "break-point" of convergence. The right eye is stationary throughout. Note simultaneous increase in activity of horizontal rectus muscles of the right eye before "break-point" and simultaneous decrease after it. (Below, fig. 3). Another example of smooth asymmetric binocular convergence along the axis of the right eye. Arrow indicates "break-point" of convergence. The right eye is stationary throughout. Note increase before and decrease after "break-point" of horizontal rectus muscles of the right eye.

Note that there is a simultaneous increase in activity of both horizontal rectus muscles of the stationary right eye as the left eye converged. Note further that when fusion is broken and the left eye diverges, there is a simultaneous decrease in activity of both horizontal recti of the stationary fixing right eye.

Figure 3 is the electromyogram of another patient, again following a target in along the axis of the right eye. Again as the left eye converges, there is simultaneous increase in electrical activity of the horizontal recti of the right eye, up to the "break point" (arrow). After that, there is again a simultaneous decrease in activity of the horizontal recti of the stationary eye as the left eye diverges.

If one covers an eye of a subject with an exo deviation during near binocular fixation, that eye will deviate outward under cover. If, while the unoccluded eye maintains fixation on the near target, one removes the cover from the deviating eye, the latter will make a fusional convergent movement while the former will appear to remain stationary. This, too, is a type of asymmetric convergence. It is necessary to select subjects with high exophoria in order to get observable electromyographic changes in the fixing eye.

In the first half of Figure 4, the left eye of a patient with 15 degrees of intermittent exotropia is under cover and turned outward while the right eye fixes a near target. The arrow indicates uncovering of the left eye. The left eye then makes a fusional convergent movement represented on the tracing by increase in activity of the left medial rectus and reciprocal decrease in activity of the left lateral rectus. The stationary right eye, continuously maintaining fixation on the target, shows simultaneous increase in activity of both its medial and lateral rectus muscles as the left eye converges to fuse. This increase is small since we are working near the limits of instrument sensitivity, but significant.

In Figure 5, we again have a fusional convergent movement of one eye while the

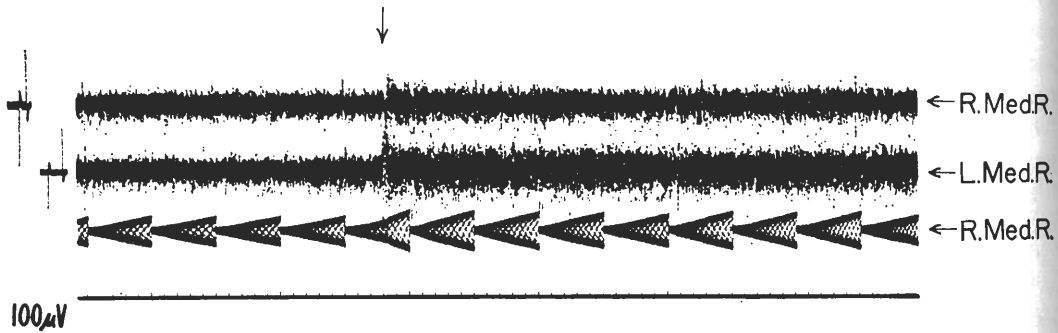
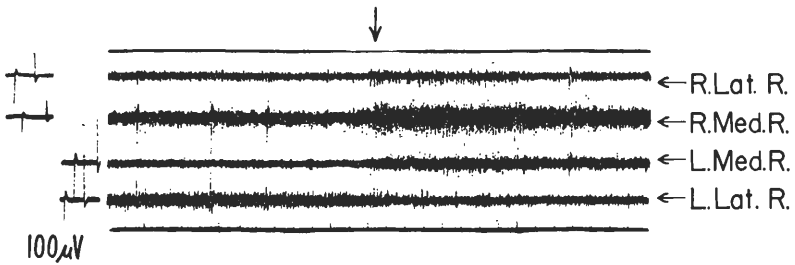
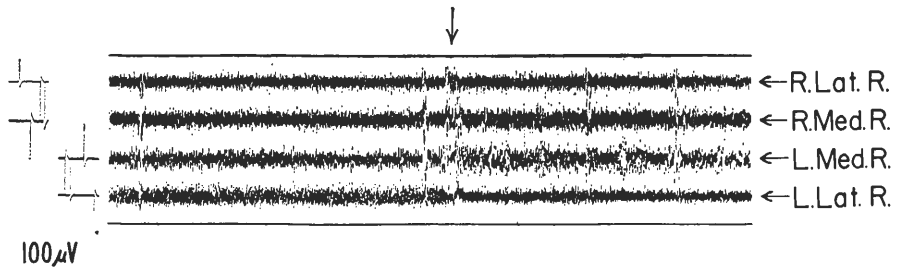
other eye maintains its fixation on a near target. This was in a patient with 18 degrees of intermittent exotropia who could voluntarily let one eye diverge and converge to fuse. In the first half of this figure, the left eye is diverging. The arrow indicates the start of a voluntary fusional convergent movement of the left eye with subsequent reciprocal innervation of the appropriate horizontal muscles of that moving eye. As the left eye converges, the stationary fixing right eye shows simultaneous increase in innervation of its horizontal recti.

In Figure 6, we are recording both medial recti of another patient with approximately 15 degrees of intermittent exotropia. The bottom channel is the integrator display* of the activity of the first channel, the right medial rectus. The arrow indicates the point of uncovering the divergent left eye as the right eye continues to steadily fixate a light at near. As the left eye now converges to fuse, the stationary fixating right eye shows simultaneous increase in activity of its medial rectus, both on the direct trace and the integrator trace.

If a patient with an exo deviation is binocularly fixing at near, and one eye is then covered so that fusion is broken, the fixing stationary eye will then often show a simultaneous *decrease* in activity of both its horizontal muscles. In Figure 7, the arrow indicates the movement of covering the left eye of a subject with 15 degrees of intermittent exotropia. The left eye then diverges and the horizontal recti of the left eye are reciprocally innervated. When the left eye diverges, there is a simultaneous decrease in activity of both horizontal recti of the stationary fixing right eye. The following example, utilizing the integrator, shows this change more clearly.

In Figure 8, there is a decrease in activity of the medial rectus of the stationary fixing

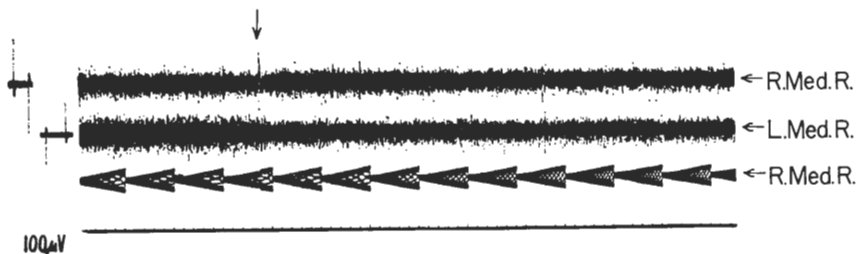
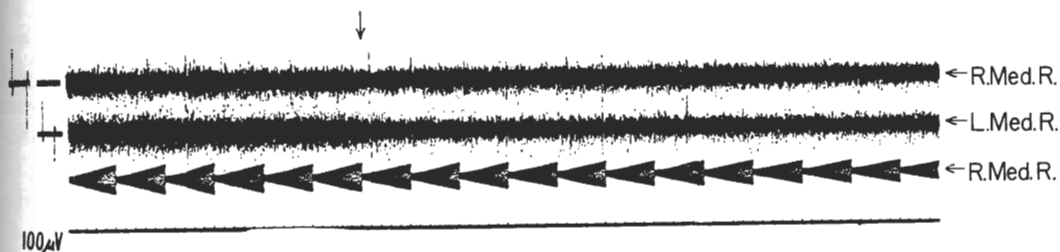
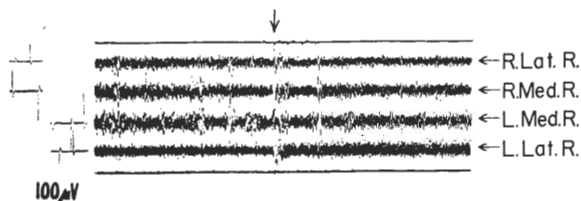
* The integrator is an electronic device for taking the integral (sum) of the electromyographic potentials with respect to time. The vertical height of the step is proportional to the average value of the EMG electric potential without regard to polarity for the time interval between steps.



Figs. 4, 5, and 6 (Tamler, et al.). (Above, fig. 4). An asymmetric convergent movement induced by uncovering (arrow) an exodeviated left eye. The right eye is fixing and stationary. Note increased activity of horizontal rectus muscle of the right eye after uncovering. (Center, fig. 5). A voluntary asymmetric convergent movement in a patient with intermittent exotropia. Arrow indicates start of refusion movement of the left eye. The right eye is fixing and stationary. Note the increased activity of the horizontal rectus muscles of the stationary eye with refusion. (Below, fig. 6). Uncovering (arrow) of the left eye in intermittent exotropia. Note the increase in the medial rectus activity of the fixing, stationary right eye both on the direct trace and the integrator trace (lowest channel) with asymmetric fusional convergent movement of the left eye.

right eye as shown by both the direct trace and integrator trace, as the left eye of a 15-degree intermittent exotropia is covered (arrow). This simultaneous decrease in horizontal recti activity of the stationary eye with break in fusion is not found as consistently or seen as readily as the simultaneous increase in

horizontal recti activity with refusion, even in the same patient. In Figure 9, for example, in the same patient as Figure 8, the left eye diverges after being covered (arrow). There is no obvious change in the trace of the right medial rectus. Yet in the same subject, a fusional convergent movement of the left



Figs. 7, 8, and 9 (Tamler, et al.). (Above, fig. 7). Covering (arrow) of the left eye in a subject with intermittent exotropia. The right eye fixing throughout. Note the decrease in activity of the horizontal rectus muscles of the right eye when fusion is broken. (Center, fig. 8). Covering (arrow) of the left eye in another subject with intermittent exotropia. The lowest channel is an integrator display of activity of the right medial rectus. Note the decreased activity of the medial rectus of the fixing right eye both on the direct trace and the integrator trace when fusion is broken. (Below, fig. 9). Same as in (center) Figure 8. No significant change in right medial rectus as fusion is broken.

eye never failed to give an observable increase in electric activity of the right medial rectus. The explanation for this apparent difference in refusion as compared to break in fusion is not clear to us at present.

The quick movement made by the "stationary" eye at the start of rapid asymmetric convergence, noted by Hering,⁴ Westheimer and Mitchell,² and Alpern and Ellen,³ can be seen sometimes by electromyography. Not all subjects show it for, as Hering⁴ noted, it appears that trained observers show it less frequently than naive subjects.

In Figure 10, when the divergent left eye was uncovered (arrow), the fixing right eye was observed to make a quick conjugate

movement to the right before returning to its previous fixing position. The trace of the right medial rectus shows a momentary inhibition

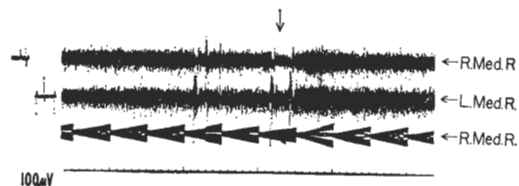


Fig. 10 (Tamler, et al.). When the left eye is uncovered (arrow), a rapid momentary conjugate movement of the right eye to the right was observed, followed by return of the right eye to its previous fixing position. Note the momentary inhibition of the right medial rectus, corresponding to this initial dextroversion.

bition accompanying the quick dextroversion movement which is confirmed by the integrator trace. This represents the peripheral manifestation of the initial rapid version innervation component.

DISCUSSION

Our electromyographic data support Hering's concept of peripheral receipt and adjustment of opposing stimuli to the apparently stationary eye during asymmetric convergence. The reason the eye remains stationary is due to cocontraction of opposing horizontal recti, that is, there is a simultaneous increase in innervation of the lateral and medial rectus muscles. One possible reason why other investigators did not find this by electromyography was, perhaps, a failure to induce a sufficient angular amount of convergence in the moving eye in order to register observable changes on the electromyogram of the stationary eye.

It is interesting that Hering's law is supported by both test methods of asymmetric convergence utilized in his study, that of smooth, binocular convergence along the axis of one eye, and that of uncovering one eye to force a fusional convergent movement while the other eye continues to fixate.

It is of further interest that in breaking fusion we often find simultaneous decrease in innervation of the horizontal recti of the stationary eye. The necessity for such a decrease of innervation is easy to explain. If it did not occur, then continued refusion movements with repeated covering and uncovering of an eye would cause greater and greater build-up of electrical activity in the horizontal muscles of the stationary eye ad infinitum.

Our electromyograms not only appear to show evidence of the quiver of the stationary eye at onset of rapid asymmetric convergence, but, following this, reveal further peripheral changes in innervation despite the

fact that the same eye subsequently remains stationary. This demonstrates that one may have a change in the electromyogram without necessarily having an associated eye movement.

SUMMARY

1. Our electromyograms of asymmetric convergence reveal that the apparent lack of movement of the stationary eye is associated with a simultaneous increase of electrical activity in both its medial and lateral rectus muscles. This agrees with Hering's theoretical prediction of a peripheral balancing of opposing vergence and version stimuli to the stationary eye.

2. Our electromyograms of reciprocally acting horizontal muscles show evidence of a small, rapid initial movement of the stationary eye in asymmetric convergence which occurs with some subjects. This agrees with the work of other investigators who found a quick movement by nonelectromyographic techniques.

3. There is a peripheral manifestation of vergence and version in the horizontal recti of the stationary fixing eye as evidenced by two stimulus methods: (a) smooth binocular convergence along the axis of one eye; (b) the refusion movement which occurs when uncovering a previously covered abducted eye of an intermittent exotrope.

4. The evidence presented in this paper supports Hering's law and differs from other electromyographic investigators, both in interpretation of Hering's law and supporting electromyographic data.

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DISCUSSION

FREDERICK C. BLODI (Iowa City, Iowa): This is an extremely interesting and provocative piece of work. It is interesting because it concerns itself with a problem of basic oculomotor physiology and it is provocative because its results seem to be diametrically opposed to the conclusions reached by all previous authors working on this subject.

Before offering my questions and criticism may I congratulate the authors on their excellent technical result. I am especially impressed by the simultaneous recording from four muscles. This is not an easy feat as anybody can testify who has tried to test more than two muscles at the same time.

The author's slide 2 is one of the crucial electromyograms. It is supposed to show cocontraction of the horizontal antagonists during asymmetric convergence. I have two technical questions on this chart: (1) Why this unusual difference in level at which the various muscles are recorded? This makes the interpretation somewhat difficult; and (2) why this time-lag of more than one second after the breakpoint of convergence before the lateral rectus picks up in electrical activity?

The principal question, however, is "Does this eye remain really stationary when these electrical changes occur?" From the records available it seems that these cocontractions occur only just the moment before the breakpoint. It is possible that at this time the stationary eye begins to show some small movements. These may account for some of the increments in the electrical activity of the muscles concerned. In addition some subjects, especially untrained ones, may experience a defense-mechanism when an object comes too close to their eyes. This might lead to cocontraction, but has nothing to do with asymmetric convergence.

In the instances of intermittent exotropia the increase in electrical activity, especially visible in the medial rectus of the fixing, presumably stationary, eye is a phenomenon connected more with the fusional movement (which may induce a convergent

movement in the fixing eye) than with any sustained effort we would expect in asymmetric convergence. This fusional vergence may be preceded by a lateroversion as seen in Figure 10. That this is more a fusional vergence movement than a true cocontraction is also seen in Figure 9 in which no change occurs in the fixing eye as the nontested eye deviates.

It all boils down to the fact that changes in the electric potentials of the stationary eye are caused by small movements of adjustment at or near the break of asymmetric convergence. These changes have nothing to do with the steady state which exists in the stationary eye during this vergence. We have found this steady state to exist through a wide angle of convergence up to the break of fusion.

The authors were most kind to send me the manuscript months ahead of the meeting so that Dr. Van Allen and I had a chance to do some additional experiments on this problem.

The fusional component of asymmetric convergence can be studied on the haploscope which has the advantage that no object comes so close to the eye that a defense mechanism would be elicited. Our results show quite convincingly that there is no increase in the electrical activity of the horizontal muscles of the stationary eye. There may be a slight increment just before or after the breakpoint reflecting a slight quiver of the eye. This fact has been mentioned by us before.

We have repeatedly done asymmetric convergence while approximating an object in the visual axis of the stationary eye. The results were identical with those obtained at the haploscope. The fact that the horizontal muscles of the stationary eye do not recruit during asymmetric convergence becomes especially obvious when the patient first makes a lateroversion out of the range of the tested muscle. If then an object is approached along the visual axis of the stationary eye, the electric activity of

the tested muscle is and remains small. Even a slight increment could easily be picked up here. But none occurred. This experiment can of course also be done while the patient looks into the range of the tested muscle. Here the baseline of electric activity is high to begin with and changes may be more difficult to detect. None could be observed.

It seems to me therefore that on the basis of available evidence we must favor the theory of a central adjustment in the innervation of opposing forces and accept the fact that this adjustment is more subtle and efficient than a simple tug-of-war between the horizontal recti of the stationary eye. Cocontraction of antagonists occurs only in unusual or pathologic circumstances and the changes which have been shown to occur just before the breakpoint of fusion in asymmetric convergence in the authors' subjects is such a circumstance.

DR. EDWARD TAMLER (San Francisco): Dr. Blodi finds our paper provocative because our evidence is quite different from the previous electromyographic evidence on this subject. He feels we are probably wrong.

We would like to dissent very strongly from this feeling, for the following various reasons: In Dr. Blodi's own paper he shows one instance in asymmetric convergence where a lateral rectus muscle increases its activity by at least 20 percent in the stationary eye during the same type of asymmetric convergence.

Dr. Blodi says that perhaps this cocontraction that we see before the break of convergence is due to small movements of the eye. We pointed out that we cannot record small movements below eight or 10 degrees. The innervations we have recorded are rather large; they are equivalent to movements of at least 10 degrees, probably more.

The other point is that if we got a movement of the eye of any appreciable magnitude, we would see reciprocal innervation, not cocontraction of the horizontal recti muscles.

Then Dr. Blodi points out that perhaps these changes in the horizontal rectus muscles are part of a defense mechanism, a protective reaction of the patient to an object approaching close to the eye. We feel this is certainly not so, for the following reasons:

If you put an electrode into the auxiliary muscles, such as the superior rectus and inferior rectus and inferior oblique, and perform asymmetric con-

vergence (this is convergence along the axis of the right eye), the right lateral rectus will show some increase in activity, but the other muscles, the inferior oblique or the vertical recti will show no change. If this were a defense mechanism one would expect every muscle to tighten up at the same time.

Furthermore, if one does asymmetric convergence with the moving eye occluded so that the only stimulus the stationary eye gets is an accommodative one, then there is no change in the horizontal recti muscles of the stationary eye. We have done this and find no change. Again, if this were a defense or protective mechanism one would expect some changes.

Furthermore, we had a couple of intermittently exotropic patients who, when fixing on a near target, showed unsteadiness of their fixation. One eye went in and out. The target remained at the same distance, but every time one eye changed fixation the other fixing, stationary eye had a simultaneous change in innervation of both horizontal recti. The target did not move, and yet these changes occurred. Again, we think this is evidence against a defense mechanism.

Furthermore, among the first few slides I showed you of smooth asymmetric convergence (figs. 2 and 3), after fusion was broken, the innervation of both horizontal recti decreased. The target was still coming in and yet the innervation was decreasing. Again, it is certainly evidence against this idea of a defense mechanism.

We have no doubt that cocontraction of the horizontal recti of the stationary eye does occur in asymmetric convergence. We have found it consistently and frequently enough to believe that our results are reliable.

Incidentally, the answer to Dr. Blodi's questions regarding slide 2 is that the difference in level of muscle activity and time lag is simply due to differences in muscle insertions. This matter is taken up in detail in our paper on normal variations and artefacts in electromyography, soon to be published.

I might mention also that the haploscope only goes in to 30 degrees and is probably not an adequate stimulus for these tests. We are grateful to Dr. Blodi for discussing our paper and for raising these interesting points. Dr. Alpern's comments are also appreciated.