OCULAR DIVERGENCE MECHANISMS*

BY *Arthur Jampolsky*, MD, FACS†

IT IS THE PURPOSE OF THIS PRESENTATION to examine and compare the characteristics of various mechanisms of ocular divergence; i.e., where relative divergence of the visual axes occurs. We shall use the tools of psychophysics and electrophysiology as comparative criteria, in normal subjects and in patients with latent and manifest strabismus, and shall focus our attention on an area of maximum controversy; namely, intermittent exotropia.

OCULOROTARY MUSCLE TONUS

In order to understand ocular divergence mechanisms, it is necessary to differentiate clearly between tonic divergences of (1) nonretinal origin and (2) retinal origin (optically elicited fusional divergence). The former, some feel, is part of the ongoing tonic substrate which is opposed by a similar ongoing tonic convergence substrate, both of nonretinal origin. The latter is an active optomotor mechanism which may be clinically demonstrated and manipulated. Since all vergences are said to be tonic in nature, we must also consider the characteristics of tonic innervations.

One of the central problems in strabismus management, especially in the pathophysiology of exodeviations, has been the failure to always differentiate between these two types of divergences, that of nonretinal tonic substrate, and that of retinal fusion. The problem is of essential importance when speaking of palsies, voluntary control, excesses, and insufficiencies, of either convergence or divergence.

Duane's term of "excessive divergence" was used[6] in a descriptive sense, with reference to a characteristic syndrome. He described

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†Smith-Kettlewell Institute of Visual Sciences, Presbyterian Medical Center, San Francisco, California.

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(retinal) *fusional*-divergence since he defined the amount of prism base-in; i.e., the amount of prism fusional-divergence which characterizes this group. Unfortunately, others have used the term "divergence excess" to mean an excessive amount of tonic nonretinal divergence. Indeed, Bielschowsky[7] in speaking of "divergence palsy" described the test for prism-induced (retinal) fusional-divergence which is apparently absent in this syndrome. Posner[8] clearly makes this distinction. When one speaks of a "divergence palsy" one must clearly have either a retinal or nonretinal or final common path type of lesion in mind.

Thus, when one speaks of an "active" divergence mechanism, one must have clearly in mind which of these two, (*a*) nonretinal or (*b*) retinal, types of divergence is meant.

Since both are tonic in character, this descriptive term does not aid in the important differentiation. Therefore, we shall use the terms (*a*) nonretinal (tonic) divergence or convergence, considered by some to be part of the ongoing continuous tonic sources from both cortical and subcortical areas, which exist as a total neural tonic substrate acting upon the mechanical oculorotary apparatus, and (*b*) retinal (tonic) divergence or convergence (optically elicited – optomotor), for example, the commonly known fusional-divergence.

Another problem has been the failure clearly to designate the "starting point" or zero reference point in the different circumstances of (1) sensory fusion, (2) movements, and (3) fusional-vergences.

(1) From the normal sensory *fusion* aspect, the zero reference point is the relative position of the foveas, or the nasal-temporal vertical raphe (under normal circumstances). This zero reference point is distinguished by the fact that in this position bifoveal fusion may occur; i.e., "the gears may mesh." There are also nasal-temporal retinal differences at this reference point (the foveal line) which are important relative to strabismic suppression, fusional convergence versus divergence differences, and other adaptive anomalies.

(2) From a *motor* aspect, we will see that electromyographically there is nothing whatever unique about the monocular zero reference point of the primary position. The primary position is merely a point in the continuum between far right and far left gaze and there is no electromyographic zero reference point that is of importance.

(3) We will see that the zero reference point, or "starting point" for the assessment of either fusional-convergence or fusional-divergence (in normal individuals) clearly must be the fusion-free position

(the basic fusion-free, or any other fusion-free position, with appropriate accommodation for a particular fixation distance). In other words, the normal fusional-vergences are the fine tuning mechanisms (horizontal, vertical and torsional) which secure fusion around any point of regard, acting from the fusion-free status at this point of regard. It is of some considerable importance to differentiate this latter fusion-free zero "starting point" for normal fusional-vergences, from the zero reference point of ortho (parallelism) around which there are supposedly opposing con- versus di- tonic nonretinal vergences.

We will also see that in some patients with a manifest strabismus (esotropia) there may be a real vergence movement elicited by prisms from the zero reference point of the primary position basic deviation; i.e., with manifest strabismus. Normal fusional-vergences operate around a zero reference point of any fusion-free position. Fusion-vergences in strabismus may operate around a zero reference point of esotropia in the primary position as it exists.

These points are important relative to the crucial problem to be considered in intermittent exotropia: to determine whether the starting point is referable to parallelism, from which there is an active divergence mechanism carrying the eyes to exotropia and maintaining them there, or whether the starting point (basic fusion-free position) is the exotropic position, from which a convergence mechanism operates to keep the eyes fused at parallelism or ortho.

We shall now turn to the analysis of the components of the *basic deviation*, which are mechanical-anatomic oculorotary factors and neural factors, nonretinal in origin. We shall follow this by an analysis of the neuromuscular mechanisms (vergences), which act upon the basic deviation and which are of retinal origin.

THE MECHANICAL-ANATOMIC COMPONENT OF THE BASIC DEVIATION

One frequently notes reference to the concept that exodeviation in adult life is caused by a continually increasing anatomical divergence of the boney orbits. It is well known that the orbits go from a position of divergence toward one directed more straight ahead during the early developmental years, and then remain unchanged.

Also, contrary to common belief, the eyes do not assume a divergence of the visual axes when all the muscles lose their tone as after death. Cogan,[3] in speaking of the absolute or anatomic position of

rest, states, "the eyes are directed straight ahead or slightly divergent and are said to be in the anatomic position of rest."

In this section we propose to discuss the anatomic elements of muscles and nonmuscular surrounding tissue in the orbit which constitute the atonic state, and which participates in the composition of the basic deviation.

First, we would like to point out that all the oculorotary muscles do *not* exert equal opposite forces, in keeping the globe stationary or in causing rotations. Clinicians generally agree that surgical strengthening or weakening of the medial rectus muscles produces greater surgical effect than equal units of strengthening or weakening of the lateral rectus muscles.

We hold to the notion that the medial rectus muscles have greater mechanical reactivity to equal increments or decrements of tonic inflow than do the lateral rectus muscles. Any change in *level* of tonic inflow to the orbits will produce a greater reactive increment or decrement of the medial rectus muscles than of the lateral rectus muscles, with a corresponding disjunctive movement.

There is ample evidence for this notion to be derived from (*a*) results of surgical studies upon the medial and lateral rectus muscles, and (*b*) the effects of alteration of the *level* of tonic inflow to the muscles.

(*a*) Surgical evidence. Surgical procedures, either strengthening or weakening, of the medial rectus muscles produce a greater effect upon the basic deviation than similar surgical procedures on the lateral rectus muscles.

Additionally, if under topical anesthesia (i.e., conjunctival anesthesia only) one completely severs both the medial and lateral rectus muscles of both eyes, there will be an increase toward exodeviation in an alert, attentive patient fixing in the primary position. In the latter instance the medial rectus muscles "lose more" mechanically than do the lateral rectus muscles.

(*b*) Alteration of the level of tonic inflow to the muscles. What happens to the relative position of the visual axes in those circumstances where the level of tonic inflow to both orbits is sharply increased, or conversely, sharply and totally shut off?

The syndrome of retraction nystagmus is representative of a massive tonic inflow to all muscles. In this instance there is a characteristic convergent movement coincident with the nystagmus and the retraction. At one time this was believed to represent a burst of

convergence impulses to the medial rectus muscles, without a corresponding relaxation of the lateral rectus muscles.[9] However, Breinin[10] and others, have shown that both the medial and lateral rectus muscles receive simultaneous innervation and show simultaneous co-contraction.

We believe this circumstance to be evidence in support of the notion that, when a burst of tonic inflow occurs which raises the general level of tonus of all of the eye muscles, convergence ensues along with retraction because the medial rectus muscles "win out" over the lateral rectus muscles as all the muscles co-contract to produce the enophthalmos. There is no need to postulate specific convergence and divergence innervations around a zero reference point of parallelism. It is simply explicable on the basis of the different mechanical response of the medial rectus versus the lateral rectus muscles to a burst of neural inflow.

On the other hand, the effect of shutting off all the tonus to all the muscles, such as occurs after death, is to produce a greater decrement in the mechanical effect of the medial rectus muscles than of the lateral rectus muscles, with a resultant slight to moderate exodeviation. This is simply explicable upon the basis of the medial rectus "losing more" than the lateral rectus muscles. There is no need to postulate that a specific mechanism of nonretinal tonic convergence relaxes more than divergence.

While it is evident from the above that the relative strengths of the medial and lateral rectus muscles are not equal, either in keeping the globe stationary or in causing its rotation, we have no real evidence as to whether the average neural inflow is equally distributed to all the ocular muscles. All of the evidence is consistent with the notion of a level of tonus (not specific convergent or divergent nonretinal tonus) which is a continuous ongoing substrate of innervation to the oculorotary muscles, which results in a different mechanical effect of the medial and lateral rectus muscles.

In this connection certain observations on patients with cerebral palsy are pertinent.

It is generally accepted that the cortex has an inhibitory influence upon the subcortical mechanisms. In patients with congenital neurological deficits, such as cerebral palsy, there may be diminished inhibitory cortical control of the ongoing tonic substrate, with the net effect of increased level of tonic inputs to the eye muscles. As we have noted, such a general increase to the muscles does not elicit equal mechanical responses, and the medial rectus usually

"win" over the lateral rectus muscles; hence, there is an expected increase in esodeviation in cerebral palsy and related conditions.

Additionally, the patients usually show an "unexpectedly" marked decrease in the esodeviation under anesthesia or deep sleep, which should not be surprising since the medial rectus muscles lose more mechanically than the lateral rectus muscles, when the generally increased level of tonus to all the muscles subsides.

One need not invoke any more complicated notion than the fact that the medial rectus muscles have greater gain or loss in mechanical effect than the lateral rectus muscles when the general level of ocular tonus is increased or decreased. There is abundant evidence for this notion, and one avoids the insupportable position of postulating excesses or insufficiencies of specific neuro-muscular convergences and divergences of nonretinal origin. We shall return to this point later.

THE EFFECTS OF ALTERATIONS OF THE MECHANICAL FACTORS

Mechanical changes in the oculorotary muscles and surrounding tissues alter the basic deviation. It is the purpose of this section to point out the consequences of anatomic changes in the muscles and tissues secondary to a gradual natural (nonsurgical) change in deviation, and also secondary to surgical procedures upon the ocular muscles; to point out how a change in the tonic inflow to one muscle is likely to affect the tonic inflow to all of the other muscles; and to discredit the notion that surgical procedures upon the oculorotary muscles affect specific functions of convergence or divergence. This point is essential in our later discussion of what surgery accomplishes in the management of exodeviations.

Among the factors affecting ocular muscle tonus, and therefore the basic deviation, are anatomical and mechanical influences. Permanent mechanical changes secondary to eye position are well known; therefore, only a few examples will be presented.

A constant esotropia of marked degree and long duration will usually be associated with a mechanical contracture of the medial rectus muscle, and associated restrictions of motility of the surrounding tissues.

A similar effect is seen with a 6th nerve paresis, which often develops a secondary contracture of the medial rectus muscle, which persists even when resolution of the lateral rectus paresis occurs. Other secondary changes in the surrounding tissue, apart from the muscles per se, may occur, which similarly contribute their mechanical com-

ponent to the makeup of the basic deviation. Patients with a marked degree of esotropia, who are operated upon under topical (conjunctival) anesthesia as the only medication, will still reveal a restriction to voluntary abduction even after both medial and lateral rectus muscles have been completely severed from the globe and their intermuscular membranous attachments severed so that the muscles retract well. In this circumstance there may be abduction rotation 10 to 15 degrees past the primary position, with still full adduction. In the same way, if a similar procedure of severence of both medial and lateral rectus muscles is carried out in a patient with a longstanding marked degree of exotropia, the patient will exhibit almost full abduction, but nasalward rotation still will be restricted to slightly past the primary position. It appears that the longstanding eso- or exodeviation produces secondary changes in the surrounding tissues, apart from whatever changes might occur in the muscles per se.

It should also be recognized that muscles other than the horizontal ones may become involved, especially in a longstanding constant exotropia of marked degree. A divergent position of the visual axis of one eye, relative to its fellow fixing eye, allows the development of overaction-contracture of *both* oblique muscles in the divergent eye, as we previously have pointed out.[11]

The underaction of one extraocular muscle is usually associated with overaction of its direct antagonist. Overaction of both agonist and antagonist in the same eye during examination of its motility, is not an expected or seemingly physiologic process, yet in the majority of patients with an exodeviation this phenomenon of the oblique muscles does occur.

The overaction of one or both obliques in the divergent eye, described for unilateral exodeviations, may be found as a secondary bilateral anomaly consequent to an alternating exotropia. The overaction of the obliques may not be equal in an eye, or between the two eyes (Figure 1).

Although it may at first seem self evident that anatomical changes, by definition, affect the basic deviation, it is necessary to emphasize this point in relation to our later discussion of the effect of surgery upon the mechanical element of the basic deviation; namely, a shift of the basic deviation; i.e.; the starting point; rather than, as some propose, a surgical effect upon vergences. It does not matter how the anatomic components were derived, whether by natural occurrence, or secondary to strabimus, or consequent to a surgical procedure.

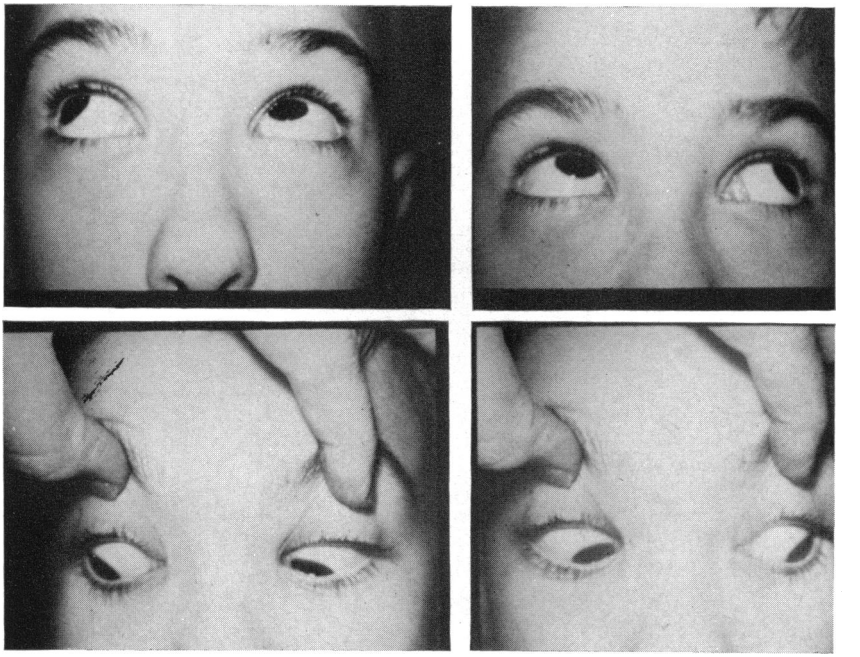


FIGURE 1
All four oblique muscles may be overacting in alternating exotropia.

It may be generally stated that tonic alterations which primarily affect one muscle have some influence on all muscles.

A single example will be given to illustrate the far-reaching effect of a paresis or weakness of just one horizontal muscle.

Figure 2 depicts the right eye fixing alone in the primary position before and after procaine has been injected into the body of the right medial rectus muscle (fellow eye occluded). Note in B, that procaine silenced the motor unit activity in the right medial rectus while fixing in the primary position. (The right inferior oblique electrode was not recording.) Apparently there was an increased effort by the medial rectus to maintain fixation after the procaine block, which resulted in a reciprocal inhibition of the antagonist lateral rectus muscle. Note also some decrement in the superior rectus activity.

It is apparent that when one muscle in the fixing eye alters its tonic or relative mechanical contribution to fixation, the other muscles are affected, and if it is the fixing eye which dictates the innervational pattern of both eyes, there are far-reaching consequences in the tonus

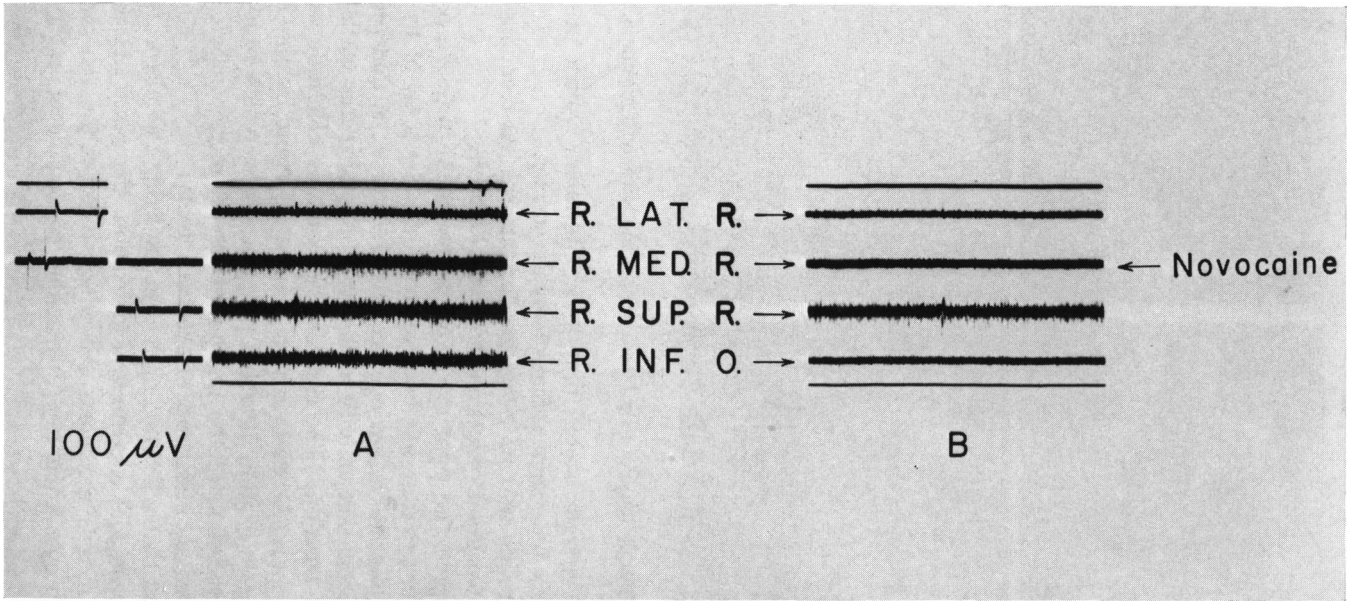


FIGURE 2

A is an electromyogram of the indicated muscles of the right eye while fixing in the primary position (fellow eye occluded). B, the same circumstance after the right medial rectus has received a novocaine block in the body of the muscle.

of both eyes. When this eye was caused to move to an extreme position of right gaze, the inactive medial rectus muscle reached its minimal action potential range. Procaine block could not cause further inactivation of the muscle, and therefore the lateral rectus, the prime mover (antagonist of the paretic muscle), did not differ from normal in its motor unit response in this position. There would be minimal or no past-pointing in this position, and maximal past-pointing in left gaze. In right gaze it apparently does not matter "how" the medial rectus loses its motor unit activity.

It is of clinical interest to note here that surgical alteration of one muscle in the fixing or dominant eye has much more far-reaching consequences relative to the basic deviation of both eyes (through alteration of the tonic inflow to both eyes), than does a similar procedure performed on the nondominant, nonfixing eye.

It must be noted that any alteration of *either* mechanical anatomy or tonic input to one muscle affects other muscles. We may dismiss the naive view that *one* agonist muscle alone moves an eye. Whenever an eye moves, all of the muscles, be they agonist, antagonist, assisting, or whatever, have a change in their recordable activity.

It is appropriate at this point to consider the effect of surgically altering muscle positions. We shall note that a mechanical surgical procedure upon the oculorotary muscles does indeed alter the basic deviation, or "starting point." This concept is contrary to that held by some, that one operates upon "convergence" or "divergence."

The surgical effect of altering the basic deviation is nowhere better seen than in the transfer of the amplitudes of fusional vergences from the old to the new basic deviation.

A patient with 15 prism diopters of exophoria was found to have 20 prism diopters of fusional-convergence amplitude (a larger amount than normally found), and 5 prism diopters of divergence fusional amplitude. He was able to mask and hold latent his basic exodeviation of 15 prism diopters easily and consistently. A recession of the lateral rectus and resection of the medial rectus resulted in a basic deviation of orthophoria. During the postoperative weeks he was found to still have 20 prism diopters of fusional-convergence from the new ortho position, and 5 prism diopters of divergence. Fusional-vergences, as will be discussed later, operate around any fusion-free position. Surgical alteration of the basic deviation merely shifts the starting point for these fusional-vergence mechanisms.

Similarly, another patient with an amazing 30 prism diopters of vertical fusional amplitude was able to overcome and keep latent 20 prism

diopters of right hyper(phoria). Surgical correction resulted in a residual right hyperphoria of 5 prism diopters. From the new basic deviation starting point, there could be measured a shift of the same large degree of vertical fusional amplitudes, now re-positioned around 5 prism diopters of right hyper.

An interesting example of a surgical alteration of the basic deviation in intermittent exotropia, studied by electromyography, has been reported by Blodi and Van Allen.[12] These authors were proponents of the divergence-excess nature of intermittent exotropia. Their electromyographic studies during different stages of surgical management revealed that the electromyograms did not confirm the concept of divergence excess. They reported that their findings threw "grave doubt on the role of central control in 'divergence excess' in the pathogenesis of intermittent exotropia." We agree entirely with their electromyographic findings, and their conclusions. Curiously, they did not make the final step to join our ranks in considering this to be excellent confirmatory evidence of the fact that surgery upon the oculorotary muscles merely alters the basic deviation. Our electromyographic recordings pre- and postoperatively are consistent with those reported by these authors. We welcome their confirmatory evidence and the forthright presentation of their data. We are only surprised at their surprise – "our investigation did not yield the expected results."

It appears clear that any local mechanical change in the oculorotary muscles or their surrounding tissue, which may occur naturally or surgically, results in a change in the basic deviation, or starting point, from which any specific vergence mechanism may begin to act.

It should be noted however that surgery may alter not only the basic deviation (distance fixation), but preferentially the near deviation, by appropriate recession of both medial rectus muscles. We look upon this as a mechanical alteration of the medial recti response to a perhaps unchanged neural accommodative-convergence input. Thus, the accommodative-convergence relationship as clinically determined can be altered by mechanical surgical means as was first suggested by the author in 1956.[13]

We shall return later to this point that the effect of surgery is a mechanical shift of the basic deviation from which all retinal tonic influences operate; from a new "starting point," the altered basic deviation.

Let us now turn from the mechanical-anatomic elements of the basic deviation to an examination of the neural origins of the continuous tonic substrate to all of the oculorotary muscles.

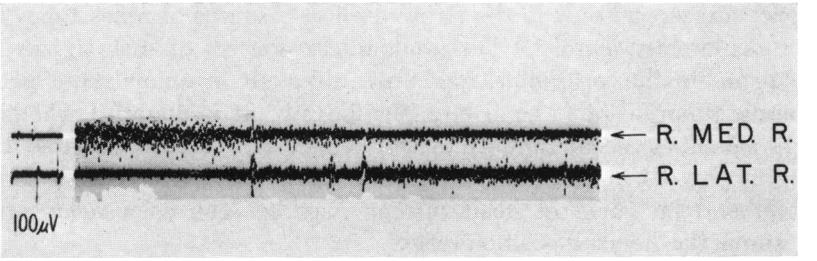


FIGURE 3
Electromyogram of right medial and lateral rectus muscles from extreme left gaze to extreme right gaze. Note that there is nothing unique about the primary position.

NEURAL (TONIC) NONRETINAL COMPONENTS OF THE BASIC DEVIATION

The oculorotary muscles have continuous electrical tonus in the normal waking state. The muscles are electrically silent in deep sleep or anesthesia. The motor unit activity of the medial and lateral rectus muscles recorded during a following movement from extreme left to right gaze reveals nothing unique about the primary position (Figure 3).

What is the evidence for the prevalent concept of a specific nonretinal tonic convergence, alone or opposed by a specific nonretinal tonic divergence?

First, let us examine the known sources of nonretinal tonus, and then the evidence for specificity of convergence and divergence innervation of this type.

There is a continuous ongoing neural substrate serving to provide tonus to the oculorotary muscles as well as the other muscles of the body. As Lashley[14] noted, "The analyses of postural tonus made by Sherrington and by Magnus have shown that the tonic activity of each spinal motor center is influenced by almost every sensory stimulus to which the organism is subjected. ..." With reference to the ocular system, Lashley continued: "In cortical activity there must be postulated a persistent substratum of tonic innervation upon which are superimposed the fluctuating patterns resulting from current stimulation in the same way that the innervation of voluntary movement is superimposed upon the spinal pattern of the postural tonus."

Oculorotary muscle tonus derives from such neural nonretinal sources as the labyrinths and semicircular canals; all the other sense organs; the reticular formation of the brain stem; and the cortex.

The postural sense organs signalling the position of the head in

space are second only to the retina itself as a source of reflex tonus to the oculorotary muscles.[4] The predominant sources of such signals in man are in the vestibular apparatus, although in animals the neck muscle proprioceptors contribute significantly. It is doubtful whether appreciable tonic alterations to the ocular muscles are initiated by proprioceptors in the neck muscles in adult man, although in early childhood an effect of head turning may be seen as a reflex arm position, the "fencing position reflex".

The turning of the eyes toward the source of a sudden noise, or a sharp pain, indicates the effect of other senses upon the oculomotor system.

Manipulation or alteration of subcortical nonretinal tonic sources elicits only conjugate deviations.

The "doll's eye" reflex elicits a conjugate movement with sudden head movement. This reflects a tonic change in all of the muscles of both eyes and may be elicited with eyes open or closed since it is entirely nonretinal in origin. Another example is the commonly used "forced head-tilt" test. This vestibulo-ocular reflex is mediated subcortically (nonretinal origin) and elicits a tonic change in the ocular muscles. This not only involves the vertical torters, which help restore vertical orientation for the retina, but all of the muscles. The physiologic basis for this is well described in text books and will not be repeated here. Figure 4 is an electromyographic example of this nonretinal, subcortical reflex which shows simultaneous motor unit activity in both the superior rectus (an elevator muscle), and the superior oblique (a depressor muscle) when the head of a normal subject is tilted to the left shoulder. To my knowledge, this is the first electromyographic documentation of the clinical explanation of this circumstance (unpublished: Alan B. Scott).

It may be said that nonretinally elicited alterations of the cortical or subcortical sources of tonus either affect the general *level* of tonus going to both eyes (with, as we have seen, unequal responses for the medial versus lateral rectus muscles), *or* elicit *conjugate* eye movements.

Alteration of the tonic activity of any one muscle at a more peripheral level may have some influence on all muscles.

A variety of impulses impinging on the brain stem reticular formation ultimately reach the eye motor nuclei to alter muscle tonus. All postulated supranuclear centers for conjugate gaze are in parts of the reticular substance, which receives inputs from the cortex as well as from all sense organs.

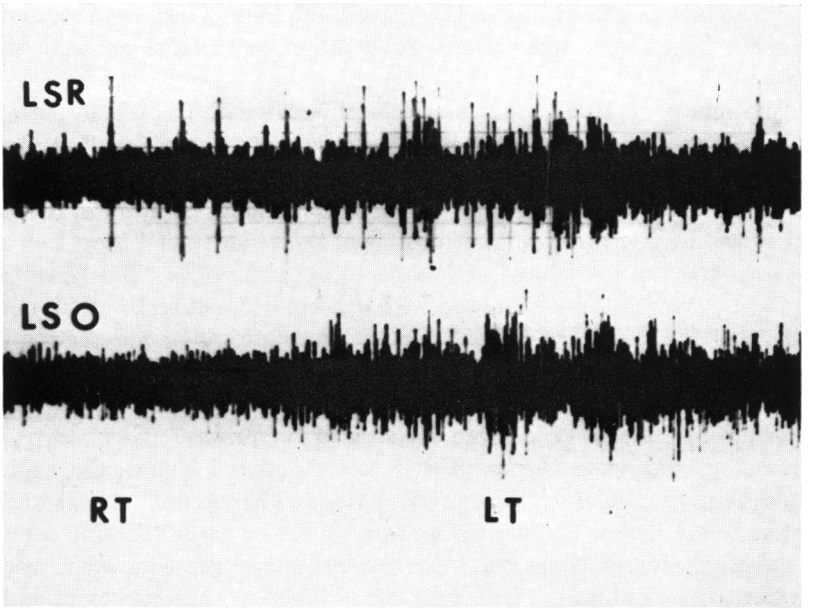


FIGURE 4

Upper channel: electromyogram of the left superior rectus; lower channel: electromyogram of the left superior oblique. From the upright position, the head is first tilted toward the right, and then toward the left. There is increased co-contraction when the head is tilted to the left. This documents the well-known clinical observations, but electromyographic documentation, especially of the superior oblique (which is difficult to record) has not heretofore been achieved (recordings by Dr Alan B. Scott).

The level of attention, intimately related to the reticular activating system, affects oculorotary muscle tonus. There is a corresponding loss in oculorotary muscle tonus (electrical activity) and in reticular activity during sleep or anesthesia.[15]

From the cerebral cortex a variety of impulses serves to alter tonus of the oculorotary muscles. Thus, the occipital cortex not only includes the primary visual receiving areas, but other areas which mediate visually evoked reflexes of following, fixation, and fusion. Further, voluntary eye movements are of frontal cortical origin. In addition to these specific effects, cortical impulses are considered to exert a general inhibition on the tonus of the oculorotary muscles.[3,4]

Of considerable significance in strabismus management and to our problem of divergence and exodeviations is the state of attention, and the emotional level. It is well known that hyper-excitability will pro-

duce an enhanced esotonus (we avoid the term "increased convergence") which may decrease an exodeviation, or increase an esodeviation.

The status of attention is a psychophysical variable which affects not only psychophysical but electrophysiologic assessments[16] and is difficult to predict or evaluate and control. Its importance has been known since before the time of Helmholtz, and alterations of the attention level have been known to affect the security of fusion lock as well as the level of tonus of the oculorotary muscles. The "level of arousal",[17] which may be altered by a normal emotional flux or with drugs or anesthesia, considerably alters the patterns of innervation to the muscles. Such alteration can be reflected in terms of evoked visual cortical potentials recorded on the electroencephalograph.

There appears to be a basic skeleton of "centrality" when different locales up and down the brain stem are stimulated electrically, under conditions of minimal or reduced alertness (Nembutal, partial anesthesia, etc.), when there is no fixation object or target. Under conditions of greater alertness, the same locales, whose previous stimulation caused eye "centering", will now cause discrete movements of both eyes; either right, left, up or down.

Some individuals are "habitually over-alerted" as a usual status. This is not infrequently seen in the examination of patients with an esodeviation which may become exaggerated during the examination procedure. This may be especially evident if a surgical procedure is immediately pending, and in some instances, an increased esotonus may mask a previously well documented exodeviation.

In contrast, it should be noted that other less alerted states, such as anoxia and intoxication, may also produce an increase in esotonus with a shift in the basic deviation. Thus, increased alertness or excitability, as well as anoxia and intoxication, may produce an increase in esotonus, so that one may select either instance to fit one's theoretical concept. It must be admitted that we do not know the exact effect of excitation or inhibition of the higher centers upon the tonic inflow to the muscles which affects the basic deviation. It would seem to be more appropriate to state that the "net effect" of the tonic inflow is more eso- or less exodeviation (or vice versa). We cannot, and should not, state that it is a specific stimulation of convergence, or a decrease of divergence, as if these are separate specific nonretinal tonic sources capable of known manipulative alterations at a cortical level.

The concept of specific cortical opposing influences of convergence

and divergence has long clouded the basic laboratory and clinical approach to strabismus management, and we must further examine the basis and consequences of such notions.

SPECIFIC TONIC CONVERGENCE VERSUS DIVERGENCE

What is the experimental evidence for separate centers or mechanisms of convergence and divergence tonus (nonretinal)?

There is an abundance of experimental evidence from animal investigations to show that stimulation of a cerebral locale will elicit a convergent movement or a divergent movement, either alone or in conjunction with pupillary changes. However, after years of discussing "centers" for such elicited functions, it is now considered inappropriate to designate these locales as such, since there may be several areas exhibiting similar phenomena.

Jampel,[18] by electrical stimulation in the brains of Macaque monkeys, notes that although stimulation of the frontal and occipital eye fields could elicit convergence, as well as pupillary miosis and accommodation changes, it is possible to obtain only convergence by discrete stimulation. It is his opinion that the convergence alone might be a "fusion reflex" while the complex of convergence, pupillary constriction and accommodation might be the "near reflex." Pathways for convergence would appear to be limited to the anterior region of the midbrain; namely, the anterior brachium and superior colliculus and from there to the oculomotor nuclei. The precise area in the central nervous system wherein the near reflex is organized is unknown. Jampel further feels that although convergence, miosis and accommodation are associated in the near reflex, each is independent of the other. Thus, lesions placed in the superior colliculus usually result in defective vertical gaze as well as absence of convergence, while lesions in the pineal body usually result in pupillary abnormalities in addition to impaired convergence.[4]

It is appropriate here to point out the general criticism of central nervous system stimulation relative to the locale of specific oculomotor functions; namely, that one is not certain whether one is stimulating a tract going to a "center," or a pool of cells, or a tract coming from such an association center, or such a center itself. Jampel's fundamental and thorough studies did relate interesting differences between convergence movements and divergence movements insofar as the frontal and occipital eye fields are concerned.

It must be pointed out, however, that in man a center of convergence has not been confirmed, nor have the fiber tracts in man

definitely been localized. One must be cautious in the extrapolation and inferences relative to the mechanisms of nonretinal versus retinal sources of convergence and divergence.

Admittedly, central nervous system stimulation may elicit an esotropia or an exotropia. For those who champion the view of nonretinal tonic convergence versus divergence, one cannot say whether these result in increases or decreases in convergence or divergence tonus of the nonretinal type. A convergence movement may be held to be an increase in convergence tonus, *or* a decrease in divergence tonus. Although a stimulating electrode might be placed in a discrete locale which would separate convergence from accommodation and miosis, such a laboratory circumstance cannot be taken as evidence for an ongoing tonic nonretinal convergence that opposes an ongoing nonretinal tonic divergence.

These conclusions are mentioned at this point in the hope that the reader will become more receptive to the following decidedly nonclassic descriptive analysis of the clinical entities of so-called "divergence palsy," as well as other clinical anomalies of pure convergence and pure divergence.

We shall conclude this section with some quotations which demonstrate the clinical "clinging" to the theoretical excesses and deficiencies of opposing convergence and divergence, in spite of admitted paucity of evidence in support of these notions.

Cogan,[3] in pointing out our limitations in knowledge regarding higher centers for the tonic innervation of the disjunctive movements, convergence and divergence, states that "It is nonetheless certain that these functions are tonically innervated since removal of either results in an unopposed action of the other."

As we shall soon note in some detail, so-called "divergence palsy" results in an esotropia. But should there not be an exotropia as a result of so-called "convergence palsy"? This does not occur. Something is wrong.

The divergence of a blind eye in an adult is held to be "a greater loss of convergence tonus than divergence tonus." Additionally, "if both eyes are blind, both convergence and divergence decrease together and the eyes remain approximately straight as in death."[3]

Adler[4] has proposed the predominating influence of variations in convergence tonus, over that of divergence, in terms of cortical inhibition of convergence. In his schema he states, "Tonic convergence is present during the waking hours from birth on and is

generally excessive in early childhood and diminishes with age." Further, that cortical inhibition of convergence is not great at birth, but increases with age.

Yet, as Cogan[3] remarks in another vein, children with congenital complete blindness have relative orthotropia. The author agrees with this observation, and would suggest further that this single fact overwhelmingly negates the concept that infants, in the absence of fusional stimuli, have an excessive continues convergence which causes the esotropia. As we shall suggest later, it is the difference between the retinal imagery in two functioning eyes that is of paramount importance in the causation of strabismus.

It must be emphasized that there is no real evidence for postulating excesses or deficiencies of either convergence or divergence of a tonic nonretinal nature from the higher centers which oppose each other. Adler[4] ably summarizes this when he states, "there is no anatomic justification, therefore, for the assumption of any so-called convergence center that relays impulses for binocular adduction to both medial recti (as opposed to the impulses to one medial rectus in a versional movement to the opposite side). And yet, as we will see, the concept is useful in any discussion of strabismus."

We have already pointed out some of the paradoxes and consequences of these concepts, and shall suggest that there is neither the evidence nor indeed the need for such views. Let us now turn from the admitted paucity of evidence to justify an anatomical basis for this type of nonretinal tonic convergence and divergence, and turn our attention to an analysis of the clinical evidence which consists of pathological conditions apparently exhibiting anomalies of convergence or divergence that are (1) pareses; (2) spasms; or (3) voluntary control of these two mechanisms.

Since the anomaly of so-called "divergence palsy" has long been considered to be of critical importance as clinical evidence, we shall first turn our attention to this.

ANOMALIES OF DIVERGENCE AND CONVERGENCE

In this section we propose to present evidence that so-called "divergence palsy" is, in fact, an anomaly of the 6th nerves, and that critical tests will demonstrate that a function or mechanism of divergence (fusional) is always present.

Further, we shall attempt to show that apparent anomalies of "pure" convergence such as palsy, spasm, and "voluntary" conver-

gence, are primarily anomalies of accommodation and only secondarily of accommodative-convergence, as revealed by critical tests with adequate monitoring of accommodation.

Since anomalies of convergence and divergence have been considered to provide crucial evidence for a theory of convergence and divergence mechanisms within the tonic ongoing nonretinal substrate, it is necessary to examine them in some detail. Further, it would appear of benefit to suggest clinical measures and criteria to aid in the more critical diagnosis of apparent anomalies of these two functions or mechanisms.

So-called "divergence palsy"

First we shall discuss what we consider to be the main point in the diagnosis of divergence palsy; namely, the presence or absence of a demonstrable function or mechanism of divergence. We shall point out that this, in fact, should be the main diagnostic point for such a label, and shall describe a simple but critical clinical test which establishes or rejects this diagnostic notion.

We shall then consider the usual diagnostic signs and symptoms and discuss their basis, and point out some neurological problems that have served to confuse the issue. We shall, expectedly, emphasize the point that so-called divergence palsy is synonymous with a bilateral 6th nerve palsy; does not exhibit a paresis of the function or mechanism of fusional-divergence; and is not evidence, as often purported, for a mechanism of nonretinal tonic divergence.

Of all the anomalies of vergences, so-called divergence palsy is of primary importance since it has been considered to provide evidence for "active divergence" or a "divergence center." We have already committed ourselves to the thesis that there *is* an active divergence function or mechanism of *fusional*-divergence. This may be demonstrated easily in any normal individual with base-in prisms, or in an esophore who compensates with fusional-divergence to mask his deviation. We choose to divorce ourselves from the notion of a divergence "center" and speak instead of a mechanism or function of fusional-divergence.

Surprisingly, little or no mention is made in the literature of test criteria specifically directed at the presumptive diagnosis of paralysis of the function or mechanism of divergence. When critical tests for a mechanism of divergence are employed in such cases, no such absence of a divergence function has ever been found by us, as will be shown. We shall first turn our attention to whether there is

a presence or absence of a function or mechanism of divergence, which curiously is not often included in the diagnostic criteria, and which we feel is absolutely essential since, indeed, it is "the name of the thing."

We shall now attempt to show that whatever the cause of a sudden onset of esotropia for distance during adult life, with consequent loss of fusion at distance, the divergence fusion amplitude (as well as other fusion amplitudes) will decrease in magnitude through disuse, but that its presence is readily demonstrable with appropriate adequate fusion tests.

Only three such examples (out of 12) which we have examined will be presented here.

Example 1. A male, age 68, complained of sudden onset of diplopia. "Comitant" esotropia 16 prism diopters at distance fixation. In actuality there was a very slight incomitancy (plus or minus 4 prism diopters) detected by prism cover test. The patient exhibited twenty prism diopters of esotropia in 40 degrees left gaze, and 12 prism diopters of esotropia in 40 degrees of right gaze. All of the other clinical signs and symptoms of so-called divergence palsy were present (see later).

There was inability to fuse a light at 6 meters, or a large 20/200 E.

When a panorama of many 20/200 Es was presented at the same fixation distance, and the deviation precisely corrected with 16 prisms of base-out prisms, fusion was easily obtained. From this starting point, base-in prisms of 8 prism diopters could be introduced before diplopia again occurred. This was considered a perfectly normal range of demonstrable divergence amplitude.

Twelve prism diopters of convergence was similarly demonstrable.

With a projection apparatus for very large fusionable targets on a screen at the same distance, and a stereoscopic fusion lock, slightly greater values for the divergence fusional amplitude were obtained.

Example 2. A female, age 57, had a sudden onset of transient diplopia in straight ahead gaze for one week, which was worse in left gaze.

She exhibited three prism diopters of esophoria in the primary position, which increased to 10 prism diopters of esotropia with diplopia in left gaze, and 6 prism diopters of esotropia with diplopia in right gaze. Note here that again there was *slight* incomitance if accurately measured. The fusion apparatus was capable of keeping the deviation latent in the primary position, but less so in left gaze and still less so in right gaze.

From the primary position (where she was already employing 3 prism diopters of demonstrable divergence fusional amplitude, in order to keep the 3 prism diopters of deviation latent) there could be demonstrated an additional 2 prism diopters of fusional divergence (as measured from

the zero reference point of parallel alignment and fusion). Thus, the patient's total divergence fusional amplitude was 5 prism diopters.

Following one hour of usual orthoptic training of both convergence and divergence fusional amplitudes on the major amblyoscope, the total divergence fusional amplitude increased to 8 prism diopters. The divergence fusional amplitude at near fixation increased from an initial 10 prism diopters to 14 prism diopters.

Example 3. A male, age 74, complained of sudden onset of double vision. He exhibited 18 prism diopters of esotropia with diplopia in the primary position at distance, and was described as "comitant." Prism cover measurements were 22 prism diopters of esotropia at 40 degrees left gaze, and 20 prism diopters esotropia at 40 degrees right gaze.

There was no fusion even for large targets at 6 meters. There was even difficulty in superimposition and blending of the images, because of a very slight vertical defect of 1 to 2 prism diopters.

However, when a large fusionable target was binocularly presented with the projection apparatus at 6 meters, and both vertical and horizontal deviations were precisely corrected with prisms, and under the stimulation of slight concomitant motion of the projected images for the right and left eye, a firm fusion lock was obtained. From this starting point, 2 to 4 prism diopters of divergence fusional amplitude could be elicited. However, as we shall see, this small amount could be a sensory (nasal retina) "stretching" of the fusion amplitude, without the expected divergent movements (unless carefully monitored separately).

Orthoptic training of fusional amplitudes was instituted three hours a day for three days. Following this, the deviation in primary position was measured as 15 prism diopters of esotropia. This slight change was interpreted as the persistence of the divergence fusional innervation which may transiently, but not permanently, shift the basic deviation. The divergence fusional amplitude now measured 10 prism diopters. Thus, even in this age group, the dormant divergence fusional amplitude could be increased even to an excess of its normal value with intensive training. A divergence mechanism (fusional) was present.

Appropriate prisms were prescribed as a clip-on procedure in order to maintain a firm fusion lock. This was successfully accomplished with demonstrable maintenance, even under usual clinical testing procedures of a respectably normal divergence fusional amplitude without continued training.

These examples serve to illustrate the fact that before one can hope to demonstrate the presence of a divergence fusional amplitude, one must first obtain a firm fusion lock. This is an essential first step, and is not always accomplished in the usual clinical situation.

Simply expressed, one must present optimal conditions for obtaining fusion, and for demonstrating a divergence fusional amplitude.

When the degree of esotropia is insurmountable for the usual amount of divergence fusional amplitude, the classical signs and symptoms of so-called divergence palsy may be present, with diplopia for distance, but perhaps fusion at some nearer fixation distance. It should be noted, however, that if the sudden esodeviation at distance is of minimal amount, the subject may be fortunate in having a fusion mechanism capable of overcoming this small amount of esodeviation. Such patients may demonstrate an admirable divergence mechanism, not only in overcoming the initial latent deviation at distance, but in continuing to keep the deviation latent by continued habitual use in increasing the divergence fusional amplitude.

The author recalls that two of his memorable teachers, Walter B. Lancaster and Conrad Berens, each had significant vertical oculorotary defects which slowly increased during adult life. Their fusional mechanism adaptation, in terms of a slow rate of change, increased several fold, and maintained the deviation latent. (Whenever they wished, they could allow it to become manifest, much to the consternation of examining students.) The author has one patient who demonstrated 40 prism diopters of vertical fusional amplitude consequent to a unilateral superior oblique paresis, who maintained fusion with slight head tilt over a period of years.

In another example, the author has seen a 20-year-old man with frank bilateral 6th nerve palsy following a head injury, with consequent 20 prism diopters of esotropia and diplopia. During his prolonged stay in a military hospital, he was shown how to give himself orthoptic training on a major amblyoscope. Over a period of weeks, he successfully increased his divergence fusional amplitude to 40 prism diopters! He not only was able to keep 20 prism diopters of esotropia latent, but had 20 prism diopters additional divergence fusional amplitude in reserve.

Many orthoptists believe that it is difficult, if not impossible, to increase the vertical or divergence fusional amplitudes. It is quite apparent that it is only more difficult to do with these fusional amplitudes than with convergence fusional amplitude.

It may be taken as axiomatic that whenever a manifest tropia is acquired in an adult patient, the fusional amplitudes will be considerably diminished with disuse. To be rekindled, one must first secure a fusion engagement, and then, and only then, assess the

presence or absence of a divergence fusional amplitude by the ability or inability to maintain fusion under the stress of base-in prisms.

These statements hold regardless of the cause of the manifest tropia, or whether it is vertical, or horizontal, or both. Patients with bilateral superior oblique palsy commonly have an esotropia in downgaze, and not infrequently only a slight esodeviation in the primary position. The same statements hold true in this instance. Because of the minimal esodeviation in the primary position consequent to this defect (obvious bilateral superior oblique palsy), the question of presence or absence of a divergence palsy seldom enters the picture. However, one might facetiously suggest that patients with obvious bilateral superior oblique palsy have a so-called "divergence palsy" in downgaze.

In summary, therefore, one may say that whenever insurmountable stress is made upon the fusion demands at distance, to overcome a recent onset of an esodeviation in an adult patient, diplopia results which may persist for a period of time. One may expect that the measurable function of fusional divergence, or of convergence, or of vertical vergences, will appreciably diminish purely from disuse. However, when an adequate stimulus situation for fusion exists, a fusion lock may be obtained with slight effort and practice at the zero reference point or fusion-free position. Orthoptic procedures readily demonstrate that the fusional amplitudes are merely dormant and may be easily rekindled not only in patients with so-called divergence palsy, but in other subjects who lose their fusion engagement. The presence of the divergence mechanism may always be demonstrated.

The following simply performed clinical test is considered essential and critical for a diagnosis of "paralysis of divergence," or indeed of convergence (to be discussed later).

The central point we wish to emphasize here is that in order to assess the presence or absence of a divergence mechanism, consequent to an acquired manifest esotropia, one must first fulfill Mueller's principle of an adequate stimulus. No one has proposed, regardless of their notions on this presently discussed entity, that there is a defect in the "fusion faculty" of blending, or whatever the fusion process is. Thus, one should first be able to engage the eyes in secure fusion by proper optical means such as with prisms or major amblyoscope or projection apparatus. The security of the fusion lock is enhanced if a stereoscopic fusion lock is obtained. Once a

binocular fusion lock is secured in this fashion, with bold fusionable large peripheral as well as central contours, then the gradual addition of base-in prisms for divergence (or base-out prisms for convergence) should reflexly initiate the appropriate vergence, even though it be of small initial amount.

We find that fusional divergence cannot always be elicited upon initial examination. However, it can be rekindled or reawakened with orthoptics. Repeated attempts at one session may be sufficient, even during the course of 30 minutes, to rekindle a dormant amplitude of fusion. Thus, prior to orthoptic training, divergence is weak or diminished from disuse, as are vertical vergences. But over a period of time, once the fusion lock has been obtained with prisms and orthoptics, the fusional amplitudes increase. Indeed, there "always" is a function or mechanism of divergence that can be elicited; therefore there is no paralysis of divergence. "Divergence palsy" really represents a crutch, in both theory and practice.

Preferably the eye movements should be monitored by an objective means, such as an electro-oculogram, or corneal optical sensing device, to determine objectively whether or not a fusion movement occurred during maintained monopia in response to the prisms.

Training increases the fusional amplitude. It does not really matter whether the training is done at distance fixation or near, or by what method, just so long as accommodation remains fixed. The result is a qualitative and quantitative reawakening of the fusion sensory "blending" process which is manifested as an increased fusional motor amplitude. Once the fusion sensory-motor process is improved, this functional mechanism, acting around any fusion-free position, will be like a reservoir, available for tapping when adequate fusional stimuli engage the mechanism, whether it be at distance fixation or at near fixation.

What is the effect upon the basic deviation (in the primary position) of increases or decreases in the magnitude of a horizontal fusion vergence? One can build up the divergence fusional amplitude and it will not affect the basic deviation. One can build up the convergence fusion amplitude and it will not affect the basic deviation. Convergence and divergence fusional amplitudes do not pull against each other, as opposing forces. If one does not use his fusional-vergences, they decrease to practically zero. This is what happens in any acquired squint where the fusion powers are not used.

In order to make a diagnosis of an absence of a mechanism of divergence, the following simple clinical test becomes critical: (1)

obtain a fusion lock with adequate stimuli at the fusion-free position (basic deviation). Then (2) from this starting point, one must be able to demonstrate an absence of the divergence mechanism, adequately stimulating the divergence mechanism over a reasonable period of time.

If then an absence of the divergence mechanism is demonstrated, then – and only then – can one state that there is a divergence palsy (or, in the opposite situation, a convergence palsy). As obvious as this criterion may seem, it is not included in the usual diagnostic characteristics of so-called divergence palsy.

Let us now turn to an examination of the clinical characteristics of so-called divergence palsy as they are usually described.

They are usually listed as follows: (1) an acute onset of diplopia at distance, with esotropia; (2) comitancy in right versus left gaze; (3) no restriction in motility; (4) fusion at near, with a range of fusion; i.e., the "recession phenomenon," namely a maintenance of fusion at near while receding the target an appreciable amount toward distance.

According to Duke-Elder,[19] the syndrome of divergence paralysis is "characterized by the appearance of convergent strabismus with homonymous diplopia of the concomitant type when the eyes view a distant object, together with the absence of any limitation of movement of either eye in ductions or in versions of the field."

Cogan[3] comments that: "Divergence paralysis may be differentiated from bilateral 6th nerve paralysis by the normal excursions of the eyes on lateral gaze, and from spasm of convergence by its constancy, by the absence of other spastic phenomena of the near reflex, and by the normal or diminished amplitude of the convergence. In any case this is a diagnosis to be made only with considerable caution, since a latent esotropia (divergence insufficiency) may produce the same syndrome without evidence of neurologic disease."

The majority of authors agree that the primary criterion for differentiating a divergence paralysis from a bilateral 6th nerve palsy is comitancy in the former, and incomitancy in the latter. This often becomes the sole differential diagnostic point, about which much has been written, and it therefore requires critical analysis.

Comitancy may not be as valid a criterion for differentiation as was formerly believed. While crude rotation tests indicate a gross comitancy in so-called divergence palsy, refined quantitative use of the prism and cover test reveals some measurable incomitance in all cases which are mild or moderate in degree. In point of fact, in

our experience, patients compatible with the clinically described picture of so-called divergence palsy have not exhibited true comitancy when measured by the prism and cover test at distance for right gaze – primary position – left gaze.

Bielschowsky[7] discusses the issue of comitancy versus incomitancy in the differential diagnosis of so-called divergence palsy and bilateral 6th nerve as follows:

"I am convinced that in many cases a diagnosis of divergence paralysis is wrongly made. I have observed many patients with paralysis of one or both abducens nerves in whom typical symptoms were present at first, but the characteristic symptoms were gradually lost and a concomitant type of deviation developed so that it no longer increased, or, on the other hand, decreased when looking to the right and left. Sometimes this transformation may even occur within a few days. If such a paralysis is seen only in the later atypical stage, it is difficult to distinguish it from the divergence paralysis. ...

"Although it must be admitted that the differential diagnosis in many cases is extremely difficult, sometimes even impossible, I am sure that divergence innervation exists and that I have seen instances of a true divergence paralysis which not only presented the typical manifestations of the anomaly but, and this is of decisive importance, also changed rather suddenly into an equally typical case of abducens-nerve paralysis. Such development is proof of an organic lesion, localized at first near the intact abducens nucleus but later extending to and finally injuring the nucleus itself."

Thus, there is general agreement that divergence paralysis often merges into bilateral 6th nerve paralysis. Since the issue of comitancy versus incomitancy is the critical differential diagnostic point, it behooves us to determine whether indeed these two syndromes are one and the same entity with one and the same nuroanatomical basis.

In this connection it is of interest that the underlying etiology of divergence palsy is noted in the literature[20–22] to be remarkably similar to that of bilateral 6th nerve palsy. Thus, the most frequent causations of *both* are (1) brain stem involvement (pontine) in the area of the posterior medial longitudinal fasciculus and abducens nuclei, as in multiple sclerosis; (2) actual nerve involvement by inflammatory processes at the base of the brain, as in syphilis; (3) toxic processes such as diphtheria and encephalitis.

It is our contention that the two clinical entities are inseparable. We feel that it would be more fruitful to consider cases of so-called "divergence palsy" to be in reality cases of bilateral 6th nerve palsy.

Additional links in this chain of reasoning for a common neuroanatomical basis and clinical course have been suggested in a most plausible fashion by my research colleague, Dr Norman Fisher, as follows: A "center" for divergence has been proposed to exist in the pons, adjacent to the abducens nuclei.[4] Indeed, Adler suggests that a location near the midline in this area would explain why, late in the course of divergence palsy, "associated paralysis of one lateral rectus develops as the lesion spreads from the midline towards one or the other side."

The pertinent question is whether a lesion in the floor of the fourth ventricle, near the abducens nuclei and adjacent medial longitudinal fasciculus, can account for the signs and symptoms of divergence paralysis.

The main point to be made here is that involvement of the medial longitudinal fasciculus very amply accounts for the relative comitancy in so-called divergence paralysis. The medial longitudinal fasciculus is known to coordinate the nuclei for the oculorotary muscles with each other and with other cranial nuclei. From the occipital areas for conjugate lateral gaze, fibers have been traced to the pons as well as other areas of the brain stem. The medial longitudinal fasciculus serves to "mediate impulses for ocular adduction as part of the act of horizontal conjugate ocular deviations."[20]

A lesion in the medial longitudinal fasciculus would necessarily interrupt impulses passing from the pons to the third nuclei. Indeed, lesions of this tract produce a characteristic clinical picture, known as internuclear ophthalmoplegia, in which there is paresis of the medial rectus on attempted lateral gaze, with preservation of the medial rectus function during convergence.

In such an instance, along with some involvement of the lateral rectus, when the defective lateral rectus is caused to fix in abduction, there is a bilateral "gaze" involvement, and the expected overaction of the yoke medial rectus does not occur because this innervation is blocked by the medial longitudinal fasciculus involvement. Therefore, the esotropia in so-called divergence palsy might easily be relatively comitant in lateral gazes.

Thus Dr Norman Fisher suggests the following course of events in so-called divergence paralysis.

A lesion develops in the floor of the fourth ventricle, which affects the medial longitudinal fasciculus and encroaches on the abducens nuclei (see Figure 5). This results in some limitation of abduction in one or both eyes. Slight weakness may be evidenced by nystag-

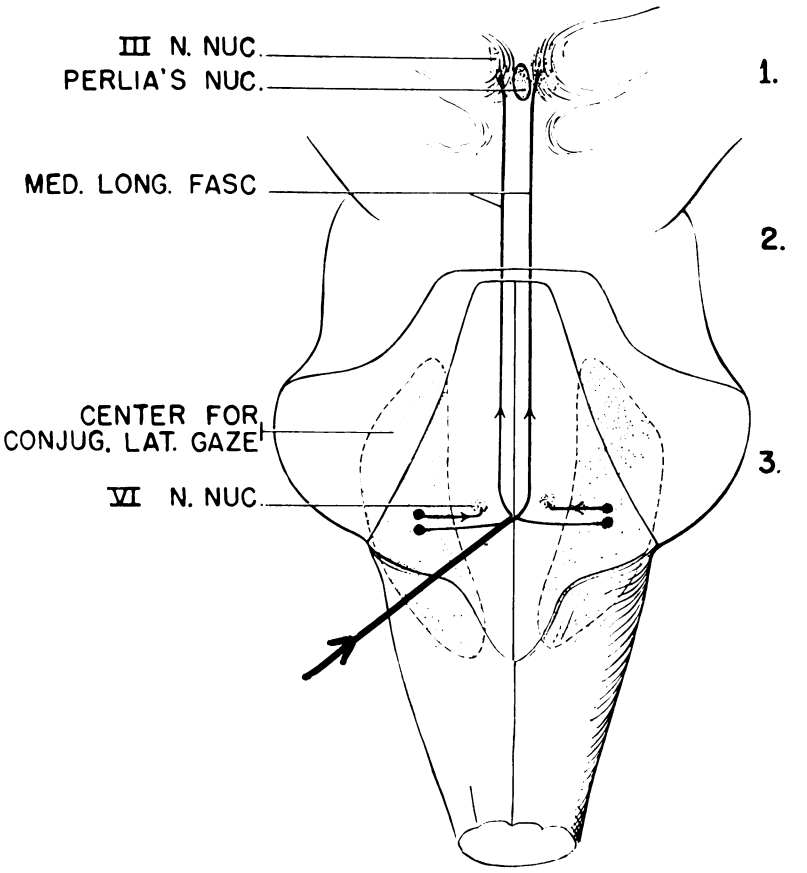


FIGURE 5

The arrow marks the site of a lesion involving the medial longitudinal fasciculus and the abducens nuclei, which is compatible with the clinical course of bilateral 6th nerve palsy – often called "divergence palsy." (From *Neurology of the Ocular Muscles*, Cogan, 2nd ed., page 85, Thomas, 1956.)

moid movements on attempted far gaze. The relative comitancy in lateral gaze may be explained by a blocking of the expected overaction of the yoke medial rectus through involvement of the medial longitudinal fasciculus. As the lesion progresses, there is more pathological involvement of the 6th nucleus or nerve, and a more marked 6th nerve palsy may be seen.

Thus from a clinical point of view, we suggest that the signs and symptoms of bilateral 6th nerve involvement are dependent upon

the degree of esotropia in the primary position, and the degree of weakness of the lateral rectus, both of which are dependent upon the severity of the defect. For instance, the esodeviation in the primary position may at first be only very mild, following paresis of the lateral rectus muscles.

This last fact was well recognized by Maddox[23] who stated "therefore, for this reason (remaining elastic tension of the palsied muscle and the tension in the muscle capsule) paralysis of a muscle only produces a slight effect at first, while the healthy eye is in the primary position. ..."

Indeed, if the bilateral 6th nerve involvement is very mild, such as to allow the always present or dormant divergence fusion apparatus to compensate for the new existing deviation, the deviation may be completely masked and kept latent by the fusion apparatus. There may be no diplopia, and almost perfect comitancy in right and left gazes.

With more marked bilateral 6th nerve involvement, not only does the fusion apparatus fail to compensate for the greater degree of esodeviation, but there may be, with time, secondary contracture of the direct antagonist, the ipsilateral medial rectus. At this stage, the medial rectus contractures, as well as the greater degree of involvement of the lateral rectus muscles, give a picture of frank incomitancy with gross deficiency of abduction in each eye.

Although it is often said that there is no restriction to motility in so-called divergence palsy, we have usually been able to demonstrate some nystagmoid movements on attempted extreme abduction. We take this to be a reflection of a mild lateral rectus paresis. It is well known that a muscle need utilize only a percentage of its full strength in order to turn the eye to an extreme position of gaze. Mild paretic involvement still does not limit full abduction, but may only be reflected in a mild nystagmoid movement.

One sees a similar type of nystagmus when the medial longitudinal fasciculus is involved in "internuclear ophthalmoplegia." We interpret this apparent unilateral nystagmus in the same way; namely, an effort or gaze nystagmus in attempted extreme abduction. The apparent lack of nystagmus of the fellow eye is due to its limited adduction. This is true we believe not only in internuclear ophthalmoplegia, but in bilateral 6th nerve involvement.

The author has seen precisely the same picture in some patients with previously operated strabismus where there is a tight or inelastic lateral rectus muscle which limits adduction in one eye. When this

is combined with deficient abduction in the fellow eye, there may be nystagmus on attempted though restricted abduction of one eye (when the lateral rectus is maximally utilized). The fellow eye, having reached its limit of restricted adduction (being restricted by the inelastic lateral rectus) has its "nose against the wall." Thus, it does not show the rhythmic nystagmus oscillations visible in the fixing eye during its attempted fixation with maximal use of the deficient lateral rectus. This type of "unilateral nystagmus" is not uncommonly seen in strabismus management, especially after multiple muscle surgery, and is of the same character as that seen in internuclear ophthalmoplegia and in bilateral 6th nerve paresis. Our interpretation of the electromyographic evidence in patients with a clear diagnosis of internuclear ophthalmoplegia is consistent with the views stated above.

The common denominator for "unilateral nystagmus" in abduction is a mild neurogenic or mechanical limitation of abduction of one (or both) eyes with a neurogenic or mechanical limitation of adduction in the fellow eye. This circumstance may reveal a unilateral nystagmus in attempted abduction, and relative comitancy of any deviation because of the restricted adduction in the fellow eye.

On the basis of the preceding analysis, we shall suggest that alternative views of so-called divergence paralysis and bilateral 6th nerve palsy can be reconciled, if we consider a sequential involvement of the 6th nerve and medial longitudinal fasciculus, which presents different clinical aspects depending upon the severity (mild or severe) and duration (early or late).

Now let us turn to the other usually described clinical signs and symptoms of so-called divergence palsy; namely, the sudden onset of diplopia and esotropia at distance, and the fusion at near with a range of near fusion, the so-called "recession phenomenon."

The esodeviation in the primary position is simply a consequence of the weak lateral rectus muscles and the expected mild effect this has initially upon the basic deviation. We have noted that if the involvement is mild, there may be only very slight esodeviation in the primary position, capable of being entirely masked and held latent by the adequate divergence fusion apparatus. If it is more marked, there may be a frank esotropia with diplopia. The differential diagnosis of this circumstance from a long-standing well compensated esophoria, which has now become manifest through debilitation but not through specific bilateral involvement of the 6th nerves, has been previously discussed.[24] Whether or not a patient has diplopia there-

fore, depends upon whether or not he has "given up the struggle," either because of an insufficient fusion mechanism effort, or due to an insurmountable degree of esodeviation.

The fact that the esodeviation in the primary position may at first be only very mild, even with significant involvement of the lateral rectus muscles, is an interesting point. Descriptions of so-called divergence palsy in the literature have often hedged relative to the deviation in the primary position and its comparison to the esodeviation in right and left gaze. The esodeviation in extreme gazes may be equal, but there is usually slightly less deviation in the primary position, for reasons which are obvious from the application of Hering's law.

In this connection my research colleague, Dr Paul Bach-y-Rita, states that preliminary observations lead him to suspect that, in cats, the eyes do not appreciably deviate in the primary position following a complete sectioning of the 6th nerve. Further, nystagmus induced by rotation produces outward movement beyond the midline on the side with the complete nerve section.

The onset of diplopia following a surgically induced esotropia at distance (consequent to injudicious surgery in patients with strabismus other than the cases being discussed here) leads one to similar conclusions relative to the relationship of the magnitude of esodeviation, the ability of the divergence fusion powers to overcome it, and the presence or absence of diplopia. If a perfectly normal orthophoric patient has a surgical recession of one lateral rectus muscle, there is a shift in the basic deviation in an eso direction simply by mechanical manipulation of the lateral rectus anatomy. If the esodeviation is of insurmountable amount, there is diplopia, with consequent diminution of the divergence fusional amplitude through disuse which, however, may be rekindled with appropriate testing and training conditions.

Clinicians generally agree that surgical alteration of the position of tension of the lateral rectus muscles affects the distance deviation more than the near deviation. This empirically determined surgical observation is pertinent, since bilateral 6th nerve defects of mild degree also affect the distance deviation more than the near deviation.

In either instance, the patient with such an esodeviation has some finite near distance where the visual axes cross in their fusion-free status. Thus, he may hold an object, usually reading material, at this position and have good fusion with no stress upon either the divergence or convergence fusional amplitudes. This, in fact, is one of

the clinical characteristics of so-called divergence palsy; namely, that fusion is obtained at some near distance. Interestingly, under perfectly natural viewing conditions there is an adequate stimulus situation at this near position, which is not present at distance fixation. At near, with the additional cues of larger, bolder contours capable of being presented at the "objective angle of strabismus," it should not be surprising that when fusion is engaged, it may be maintained over a range. This is reflected in the ability of the patient to recede the near targets over a reasonable range while still maintaining fusion. This has been described as the "recession phenomenon" in so-called divergence palsy. We take this to be only a demonstration of the divergence fusional amplitude at near, under conditions where more optimal psychophysical parameters for the stimulus situation exist, and where it is usually possible to fulfill our previously stated criteria of first engaging fusion and then demonstrating a divergence fusion amplitude. The near range of monopia is usually great enough to be practically useful. The clinically described "recession phenomenon" which is one of the characteristics of so-called divergence palsy, is, in fact, a negation of the presumptive diagnosis of a divergence palsy, since in itself it demonstrates a divergence function to be present. It is simply fortuitous that an adequate stimulus exists at near, which is not present for the distance fixation circumstance. However, as we have already indicated, if an adequate stimulus is made available for distance fixation, a creditable divergence fusional amplitude may be demonstrated. Indeed, this is a confirmation of the concept that fusional amplitudes are the tuning mechanism that operate around any fusion-free position. Divergence should not be considered an inhabitant of distant ranges, and convergence one of near ranges. There is no more basis for this than to consider sursumvergence an inhabitant of distant ranges (where occlusion hyperdeviations are elicited to a greater degree), and deorsumvergence one of near ranges (where hyperdeviations are less easily demonstrated).

Thus, it will be seen that all of the clinical signs and symptoms of so-called divergence palsy are perfectly consistent with the diagnosis of mild or marked degrees of bilateral 6th nerve paresis, with involvement of the medial longitudinal fasciculus. The fusion, or esotropia at distance or near, is dependent upon the relationship between the magnitude of the deviation and the competency of the divergence fusional mechanism to keep it latent. It is quite certain that, in any instance, so-called divergence palsy is a misnomer, since

it is always possible to demonstrate a mechanism or function of fusional divergence. We propose to drop the term "divergence paresis" and to consider such cases henceforth as degrees of bilateral 6th nerve paresis.*

A very brief discussion of the management of bilateral 6th nerve paresis is pertinent here, since the prognosis and results are evidence in support of our thesis.

The prognosis for fusion is excellent with either nonsurgical or surgical methods. Treatment is directed at (1) obtaining a fusion lock by prisms, orthoptics, or surgery, and (2) preventing or correcting the contracture of the medial rectus muscle(s).

The forced duction (traction) test is a useful office procedure to determine the presence or absence of a medial rectus contracture in adults. However, the demonstrable presence of a medial rectus contracture still does not provide information as to whether the lateral rectus is still paretic or has returned to normal. It is only in the absence of a demonstrable contracture of the medial rectus muscle, and with the patient still unable to abduct the eye voluntarily under usual circumstances, that one may presume there is a weakness of the agonist lateral rectus. The latter question can be confirmed in selected instances at the time of surgery under topical anesthesia, only after a severance of the contracted medial rectus muscle. If, in this circumstance, attempted voluntary abduction still shows a deficiency, then one may reasonably infer that the lateral rectus is paretic. Of course, electromyography, which may demonstrate aberrant motor unit responses, is diagnostically helpful when practicable.

In general it may be said, in this and other strabismic problems that a "weak" muscle may only be diagnosed when a contracture of the direct antagonist has been ruled out or released.

The problem is even more difficult in infants with marked degree of bilateral esotropia in whom the above tests are not applicable. It is inappropriate to detail here the clinical management of these strabismus problems, except to state that one of the most difficult problems in strabismus surgery is their gross surgical undercorrection. This sometimes results even after very significant amounts of bilateral recess-resect procedures, in infants who probably have an undiagnosed paresis of the lateral rectus muscles. The latter cannot always be differentiated adequately from similar cases with normal lateral rectus

*Much of the orthoptic work demonstrating the presence of a divergence fusional mechanism in the above described cases was performed by Dr Ellen Takahashi and Miss Margaret Fitton to whom grateful acknowledgment is extended.

muscles, but with a similar degree of bilateral esotropia.* It is well known that the amount of surgical resection that must be performed on a paretic muscle is considerably greater than on a normal muscle, even when the antagonist medial rectus muscle has been sufficiently weakened. The central problem here is that the medial rectus is contracted in either instance; i.e., with or without persistent lateral rectus paresis. At present we know of no practical way to make the differential diagnosis in infants, and we suggest that bilateral 6th nerve paresis is a masked etiology in some of the failures described above.

It has been remarked[3] that although "old 6th nerve paralyses are apt to be accompanied by progressive contractures of the medial recti so that the convergence increases with time, this is not true, curiously, with congenital palsies." Also,[25] "It should be noted that even in cases of congenital abducens paralysis, the eyes are very frequently straight in the primary position."

We would offer the alternative suggestion that so-called "congenital abducens palsies" do not represent cases of simple 6th nerve involvement. Rather, the whole group of "congenital abduction weaknesses," which includes Duane's syndrome and others, characteristically involves both medial rectus (3rd nerve) and lateral rectus (6th nerve), with aberrant innervations to each. There is no experimental documentation that such congenital abduction weaknesses are indeed simple 6th nerve palsies, and thus, consideration of these congenital anomalies is not necessarily helpful here.

The observation has been made by my co-workers (Bach-y-Rita, Scott) and myself, that the lateral rectus muscles appear grossly normal during surgical inspection after apparent long-standing documented 6th nerve palsy. Pursuing this, my research colleague, Dr Alan B. Scott, has made intracranial section of the 3rd nerve (because of its easier approach) in two dogs and has found no evidence of histological changes in the 3rd nerve innervated muscles after one year's duration. Dr Scott has kindly contributed the following statement:

"Denervation of oculorotary muscles causes no definable loss of muscle substance for many months. For example, intracranial removal of 45 mm of the 3rd nerve in two dogs resulted in typical and persistent mydriasis, exodeviation, and ptosis. However, one year later, gross and histological examination of the oculorotary muscles supplied by the 3rd cranial nerve showed no identifiable change as compared to muscle supplied by the normal 6th nerve on that same side. Also, the presence of nerve fibers to the muscle supplied by the 3rd nerve was documented by histological

*The saccadic velocity of the paralyzed lateral rectus may be observed (or recorded by oculograms) to be subnormal.[72]

examination of the motor nerves. (Scott, unpublished data.) These findings differ from those generally reported for innervation of skeletal muscle. Wide variations in the rate and degree of muscle atrophy following the innervation occur depending upon the species and the particular muscle group involved.

"It is not known to what degree passive motion of the innervated muscle may retard or accelerate atrophy. After cutting the 3rd nerve in dogs, in any case, motility is greatly restricted and no large amount of passive movement of these muscles could occur.

"One can speculate upon the population of multi-innervated fibers within the oculorotary muscles, differentiating them from other skeletal muscles, as possibly contributing to this lack of atrophy over lengthy periods of time."

It appears that we have much to learn about the innervation of the 6th nerve to the ocular muscles in humans and the consequences of its clinical involvement. There is urgent need for more detailed studies of the neuroanatomy of the 6th nerve, nucleus, and surrounding region, with particular reference to the possibility of bilateral clinical effects.

The diagnosis of so-called divergence paresis is not to be considered as evidence for a tonic nonretinal source of divergence; in fact divergence paresis, as such, does not even exist!

It was considered germane to plod the arduous route toward this last statement since divergence palsy has been cornerstone "evidence."

We shall now very briefly consider "voluntary divergence," and "divergence excess."

Voluntary divergence

Adler[4] states that "divergence from parallelism is entirely reflex; there is no possible control by the will, except in the relaxtion from voluntary convergence." We agree entirely that there is no such function as "voluntary divergence." Further, there is no negative accommodative counterpart to the usual positive accommodation that is controlled by the will or learned. The willful part of convergence is the learned control of accommodation, as we shall see. There is no counterpart, therefore, in divergence. So-called voluntary control over divergence is merely "letting go" of a fusional-convergence which masks the basic fusion-free exo position and keeps it latent. (See later figures of eye movement and electromyographic recordings of this circumstance.)

The misleading terms of "voluntary" and "involuntary" should be dropped as they apply to the term "tonus."

"Divergence excess" will be discussed under the consideration of intermittent exotropia. The only situation wherein, indeed, an "excess"

of divergence may be said to exist is exemplified by the patient discussed previously, who was taught to give himself orthoptic training several hours a day over many weeks, with a resulting fusional-divergence amplitude of 40 prism diopters. This amount of fusional-divergence is excessive only in the sense that it was considerably above the normal limits. Such an excess, however, is not continually active, but is only tapped from the reservoir if available, when necessary. Patients with esophoria ordinarily tap such a divergence reservoir and successfully accomplish the fusion task. Normal persons may develop with training such a reservoir, but since it is not needed it will be untapped, and will soon diminish to within normal limits.

Thus, on the basis of clinical and neuroanatomical evidence, we must conclude that the concepts and terms of "divergence palsy", "voluntary divergence", and "divergence excess", are not helpful crutches, and should be relegated to their historical places.

Now let us turn our attention to anomalies of convergence in pursuing further a differentiation between the characteristics of specific retinal fusional-divergence and convergence mechanisms, which we accept, and those of *specific* tonic *nonretinal* divergence and convergence, a concept which we reject.

ANOMALIES OF CONVERGENCE

We propose that all anomalies of convergence can be considered as anomalies of *accommodation*, with consequent or subsequent over- or under-stimulation of accommodative-convergence. Such anomalies may be organic excitations or inhibitions of accommodation; they may be voluntary or feigned. In the latter case they represent functional control of accommodation and therefore only apparent anomalies of pure convergence.

Almost all authors who write about anomalies of convergence caution of the extreme difficulty in differential diagnosis from functional disease. The critical test in our opinion is the accurate monitoring of accommodation (as by retinoscopy, or visual acuity assessment with the aid of plus and minus lenses) when changing fixation from distance to near while reading. Simple demonstration of the ability, or inability, to read at near does not provide sufficient evidence. Proper diagnostic tests for so-called anomalies of convergence require (*a*) control of accommodation during testing, (*b*) control of fixation, and (*c*) control of the fusion vergence, by monitoring observable vergence reflexes under monocular and binocular viewing conditions during the tests.

In the present section we shall first present our concept of how

convergence anomalies such as (a) voluntary convergence, (b) spasm of convergence, and (c) palsy of convergence, may all be explained on the basis of anomalies of accommodation by proper monitoring during the test conditions. We shall then compare these hypotheses with reports in the literature, and discuss the differing views.

Voluntary convergence

So-called voluntary or willed control over convergence is the "trick" of crossing one's eyes at will, a "trick" learned by many children to impress or confound their friends and parents. This is always accomplished by means of accommodation and its associated convergence. It may be bilateral, or one may control fixation and move either eye nasalward. The task can be accomplished with real or imaginary stimuli. This has been interpreted as "voluntary convergence."

The author, in many years of examining such subjects, has never found a single instance of deliberate "crossing of the eyes" that was not associated with accommodation, if accommodation was accurately monitored by noting refractive error changes by retinoscopy and visual acuity changes (with or without the aid of plus and minus lenses).

Accommodation is mediated by the autonomic nervous system. But there is ample precedent for voluntary control of a function so mediated. Indeed, as Gellhorn[26] states, "the term autonomic is somewhat misleading for, although the function of these nerves is largely independent of voluntary control, the degree of autonomy with which the system operates is much less than its name implies."

Adler[4] has stated that, "Even in the adult convergence as a purely voluntary act is seldom found without some training." We agree entirely with this. It will be noted that our explanation of the mechanism for apparent voluntary convergence differs mainly in degree from opinions in current literature. Adler further states,[4] "Convergence is always associated with accommodation and miosis, the collective phenomena being called the near-point reaction."

There is one instance in which apparent voluntary convergence is unassociated with accommodation. An esophoric patient may voluntarily "let go" of his divergence fusion mechanism which holds the deviation latent. In such an instance, the esophoria or intermitten esotropia becomes manifest without accommodation changes, since there is willed control for the invoking or dissipation of the fusional-divergence. This so-called "voluntary convergence" is similar to the apparent "voluntary divergence" previously described. In either instance it is control over or release from the fusional vergence mechanism.

In all other circumstances so-called "voluntary convergence" is not voluntary control of pure convergence, but rather the voluntary control of accommodation and its associated convergence.

Some children with a constant exotropia (not intermittent by definition) may confuse and confound the orthoptist and ophthalmologist by invoking accommodative-convergence to fuse the two blurred images when the visual axes reach parallel alignment where fusion is possible. Sometimes a child will do this to "please" the orthoptist during training. This is a far different circumstance from the mechanism involved in intermittent exotropia.

Spasms of convergence

Whenever convergence spasm is mentioned in the literature, hysteria and malingering are mentioned in the same instance, and references are made to cycloplegia which sometimes causes the spasm to disappear. For example, Walsh[27] states, "I am inclined to agree with Bielschowsky that such cases (convergence spasm) should be considered with suspicion and that malingering is probably nearer the truth than hysteria." In this connection it should be noted that children can easily accommodate, and it is not impossible that this voluntary control over accommodative-convergence may eventually, if of long duration (spasm), result in a secondary anatomical contracture of the medial rectus muscle with a real esotropia.

In addition to such "voluntary" spasms there may be "organic" spasms of convergence. There is general agreement that "organic" lesions resulting in convergence spasm usually involve the whole near reflex. Cogan[3] states, "Spasm of convergence occurs along with spasm of accommodation and miosis, and is therefore more comprehensively designated 'spasm of the near reflex'."

Tests for spasm of convergence should meet the same criteria as tests for other anomalies of convergence: control of accommodation, control of fixation, and control of fusion vergence. One must be aware, in testing a patient for (voluntary) accommodative-convergence "spasm", that he may momentarily relax the voluntary accommodative spasm in order to catch a momentary clear glimpse of the visual acuity chart and then resume his "spasm." This possibility should be permitted to occur, since it is diagnostically helpful to observe the momentary straightening of the eyes and pupillary changes, with the brief relaxation of accommodation. This possibility may then be curtailed by random changing of the acuity letters. One must carefully control the random or nonrandum presentation of preferably single acuity

symbols at near and distance fixations; carefully observe pupillary changes and vergence movements, as well as monitor accommodation during the test by retinoscopy, in order to differentiate between an organic or a functional accommodative "spasm."

Convergence palsy

In the author's opinion, convergence palsy occurs only consequent to an involvement of accommodation, and the associated accommodative-convergence. I know of no concrete evidence that there is a paresis solely of convergence in the presence of intact accommodative and pupillary mechanisms. The lack of convergence in cases of so-called convergence paralysis reflects the fact that the patient either, (1) cannot accommodate (as in toxicity from diphtheria, alcohol, influenza, or ptomaine poisoning[27] or (2) will not accommodate (as in functional disorders), which is more common.

As with other anomalies of convergence, it is our contention that tests must meet rigid criteria. There must be strict adherence to the policy of control of (1) accommodation monitoring, (2) fixation, and (3) fusion vergence eye movements. It has long been known that one must inspire the effort to accommodate in the patient, since it is the accommodative effort that elicits the accommodative-convergence. One must observe the pupillary action, and eye movements (with one eye occluded), while the patient demonstrates the ability or inability to read randomly presented individual letters at near fixation distance. The observer may document the change or lack of change of accommodation by retinoscopy during the test as well as by the patient's ability or inability to read small print.

In order to diagnose a paresis of convergence, as separate from accommodative-convergence, one must demonstrate not only the ability to accommodate, but its actual performance. Further, one must demonstrate that there is no associated convergence mechanism during the accommodative act. Otherwise, it simply must be construed as an anomaly of accommodative-convergence. This is usually sufficient. However, one may further invoke the same test criteria for demonstrating the presence or absence of fusional-convergence that were discussed under divergence paresis. The criteria of this test are as follows: (1) First obtain a fusion lock at the fusion-free position, and (2) From this position demonstrate the presence or absence of convergence in response to gradually increasing base-out prisms.

One can further document the fact that a convergence movement has taken place in response to the prism by covering one eye during the

prism vergence test and noting if the covered eye resumes its fusion-free position. The convergence response to base-out prisms is reflex in nature, but only if an adequate stimulus (size and contour) is appropriately presented to a cooperative patient.

Paralysis of convergence is said to be present commonly during oculogyric crises and also in severely debilitated and obtunded patients. This is indeed a difficult time during which to judge a convergence paresis according to our stated criteria. The lack of ability, desire, or effort to accommodate is the more important trigger of the function to be assessed.

A number of authors urge caution in making a diagnosis of paralysis of convergence. Adler[4] mentions the importance of such factors as attention value of the target, the patient's cooperation in making an effort to converge, the vision of the eye and the refractive error. Walsh[27] cautions that "paralysis of convergence should not be accepted as certain unless with attempts at convergence there is pupillary constriction and failure of convergence." Bielschowsky[28] urged that one must take extreme efforts to induce the patient to accommodate appropriately on near targets and then monitor the refractive state and the pupil. In addition, Bielschowsky states, "Many of the cases published (convergence paralysis) are no doubt instances of functional disturbances that are not always easy to distinguish from true organic paralyses." One of the conditions posed by Bielschowsky for diagnosis of convergence paralysis of organic origin is that "the accommodation and the convergence reaction of the pupils must be producible without the corresponding convergence." The present author would add to this statement "... and fusion is prevented by covering one eye, since a secure fusion lock may mask the convergence with fusional vergence even though the images are blurred with accommodation." Indeed, this author can voluntarily accommodate and thus converge, while maintaining fixation with either eye, or a secure fusion lock can also be maintained during willful accommodative-convergence with observable miosis without converging.

In our experience, none of the patients who have been diagnosed as convergence paralysis have demonstrated that the other components of the near reflex, namely, accommodation and miosis, can be elicited without eliciting accommodative-convergence. It is our feeling, therefore, that all cases of so-called convergence paralysis may be interpreted as anomalies of accommodation, with its secondarily associated accommodative-convergence.

For those who champion the notion that there is a nonretinal tonic

specific mechanism of convergence opposing divergence, there is a paradox here which must be explained. A "convergence spasm" results in an esodeviation at distance fixation. "Convergence paralysis" ought to result in an exodeviation for distance fixation. This does not occur simply because there are no specific opposing mechanisms of nonretinal convergence versus divergence, operating around a zero reference point of parallelism, which can be paralyzed.

CONCLUSIONS

(1) We reject the notion that there is such an entity as pure "voluntary convergence" despite its time honored place in the literature. "Voluntary" control over accommodation can be learned, even in the absence of appropriate accommodative stimuli, and many school children utilize this "trick" in crossing their eyes at will.

(2) Anomalies of convergence, apparently unassociated with other components of the near reflex, must always be held suspect. Voluntary convergence, and spasm or paresis of convergence, will always be found to be in association with some aspect of the accommodation (and miosis), if accommodation is critically monitored.

(3) Spasms of convergence invariably involve the entire near reflex; thus the term "spasm of convergence" is a misnomer. Although we must admit the possibility that a discrete lesion could "tease" pure convergence apart from the other components of the near reflex, we have yet to see, or know of, a well documented instance of this.

(4) The critical tests are (*a*) to monitor the change in refractive error, or lack of it, via accommodation by retinoscopy during the test, with or without visual acuity assessment (with plus and minus lenses); by controlling fixation; and by observing vergence eye movements while one eye is occluded, and (*b*) to demonstrate the presence or absence of the convergence mechanism by first securing fusion with adequate fusion stimuli, and secondly, from this fusion-free position, to demonstrate the presence or absence of a vergence movement under the stress of base-out prisms.

CONCLUSIONS RELATIVE TO SPECIFIC MECHANISMS OF NONRETINAL TONIC CONVERGENCE AND DIVERGENCE

The concept of nonretinal tonic convergence and divergence has become incorporated in the literature largely as a result of anomalies of these vergences. The concept of a tonic (nonretinal) convergence, as a continuous tonic substrate force opposing a tonic (nonretinal) divergence, has been a central theme in the causation of strabismus

for generations. Excesses and insufficiencies of each "tonic" vergence are postulated to explain almost any type of horizontal comitant strabismus.

It is our contention that anomalies of divergence are in reality a problem of bilateral 6th nerve or nuclear damage, and thus are peripheral in origin. Also, anomalies of convergence are in actuality secondary to anomalies of accommodation, and thus are retinal in origin. We propose to discontinue the use of the term "tonic" as applied to convergence or divergence, since all vergences, regardless of what might be their origin (retinal or nonretinal) might be so classed.

Further, we feel that there is neither clinical nor laboratory evidence for specific nonretinal origins of convergence and divergence as opposing mechanisms around a zero reference point of parallelism of the visual axes. Nor, do we feel, is there a need for this confusing clinical notion in strabismus management.

We consider all the tonic innervational inputs to the oculorotary muscles from the diverse nonretinal sources as a complex neural matrix acting on the mechanical ocular structures whose net effect is such as to produce a basic deviation which is either ortho, eso, or exo.

The sum total of all the continuous tonic neural factors does not act upon an equal oculorotary muscle apparatus, inasmuch as the medial rectus muscles are "stronger" in reacting to a given increase or decrease of tonus than are the lateral rectus muscles, thus producing a disjunctive movement through changes in the general level of tonic inputs.

We must, in our ignorance, term the product of all the tonic nonretinal innervations acting on any existing mechanical apparatus of muscles and tissues, simply as a "net effect." However, it is possible to determine clinically the sum total of these neuromechanical factors, which we term the "basic deviation." As we have seen, and will discuss again subsequently, the neural and mechanical factors comprising the basic deviation may be altered.

All of this is consonant with the view, for strabismus management, that there is a basic deviation for distance fixation (as defined), with an accommodative-convergence mechanism which then determines the near fusion-free position.

All fusion vergences have a zero reference point of the fusion-free position (at any fixation distance). There is a mechanism of fusional-divergence (and fusional-convergence) which may be enhanced with use and training, or decreased with disuse, without permanent effect

upon the basic deviation. Fusion vergences are used when and if needed, to the extent that they are needed, if available from a reservoir. The noninvoked fraction of a given fusion vergence may properly be called the "reserve fusion vergence amplitude."

This writer is not so naive as to expect that the time honored concepts of (nonretinal) specific continuing tonic convergence and tonic divergence, as opposing forces in strabismus diagnosis and management, will be easily placed on the shelf in their historical perspective, despite the foregoing detailed analysis of laboratory and clinical evidence. Nevertheless, we shall conclude this section by stating that specific neuromuscular mechanisms for convergence and divergence are all retinal in origin. There are no specific con- or divergence mechanisms from nonretinal origins. There are only different mechanical responses of the medial and lateral rectus muscles, to different levels of the continuous neural tonic substrate which goes to the muscular apparatus.

Before discussing the retinal vergences (fusional, accommodative, proximal), there is one additional possible source of nonretinal tonic innervation that must be discussed; namely, that mysterious mechanism or function of ocular proprioception.

Ocular proprioception

One cannot conclude a discussion of the nonretinal sources of tonus without a study of the possible functional role of an ocular proprioceptive mechanism and its possible contribution to ocular tonus.

Proprioception has been defined as "appreciation of position, balance, and changes in equilibrium on the part of the muscular system, especially during locomotion."[29]

The retina, and especially the macula, represents the final visual sensor for the visual apparatus, and is, for the visual apparatus, what the finger kinesthetic sense is for the hand, in sensing when it has reached its final directed goal. Cyberneticists delight in the example of the finger "touching" the object sought, whence the feedback error is reduced to zero, and the hand and finger movement stops. Similarly, for the visual apparatus, the eye "feels" with its macula, and stops when the object of regard is securely "felt" by the visual macula.

The consequence of an eye with a developmentally poor sensor (macular lesion or blurred image) is an exaggeration of the ever present normal physiologic nystagmus which, among other things, represents a visual "feeling" averaging process. This "feeling" must be done with the nearest functional retina available, adjacent to the

insufficiently functioning foveal area. The result is an observable so-called optical or fixation nystagmus, an exaggeration of normal.

Further, with complete bilateral congenital blindness (never any visual input) the subject never "learns" a successful visual fixation completion which is associated with an oculorotary muscle innervation. The macula never "sees" the target. No association between vision and eye movement or innervation is "learned" and hence there are no voluntary eye movements in the congenitally blind. One must clearly differentiate between complete and total blindness at birth, and crude visual localization and rudimentary vision which gradually may lead to early blindness. This is a difference which is often difficult to document.

All one observes in the congenitally blind are wild, roving, random jerky movements. The congenitally blind subject does not know where the eye is, and has no sense as to if and where the eyes move, although the conjunctival and lid sensations are intact. Similarly, in the adult who has had normal vision, it has been amply shown that when there is an absence of visual input (complete blindness), the subject is unaware that the eye is being moved when it is moved by forceps applied to the anesthetized conjunctiva. There are no nonvisual cues to eye position under these circumstances. One must conclude that there is no "conscious proprioception" for the visual system, as there is for the skeletal muscle system of the limbs. The wild, random movements of the congenitally blind subside completely during sleep and deep anesthesia, as does the normal tonic motor unit activity in normal individuals.

The wandering "nystagmus" of the congenitally blind is an open-loop system without rhythm, and is not made up of distinct horizontal or vertical movements, but rather is random. These movements represent the net effect of the nonretinal tonic inputs from all other sources, unharnessed by retinal influences of any sort, including even a fixation reflex.

It will be recalled from the discussion of the "basic deviation" that this, too, represents the "net effect" of all the nonretinal tonic inputs to the oculorotary muscles, but in this instance it is harnessed by a monocular fixation (retinal) reflex.

It is most important to note that the basic deviation of the congenitally blind is not only free of fusion, accommodation and proximal retinal influences, but it is also free of the retinal fixation influence. It is of especial importance to note that under these conditions the eyes of the congenitally blind are relatively orthotropic, and move con-

jugately during the wild unharnessed excursions. There is no evidence whatever of a persistent esotropia, which might be expected if there were an unharnessed tonic convergence mechanism under these conditions, as some propose.

The fact that congenitally blind eyes are relatively orthotropic, and not esotropic, during wakefullness or sleep, is an almost overwhelming negating impact to those who hold that during childhood there is an excessive tonic convergence which causes an esotropia when fusion is not possible.

On the other hand, an adult with normal vision, who acquires sudden and complete blindness, continues to have the ability to move his eyes voluntarily and "fixate" in any direction of gaze. He maintains his "learned" innervation sense, even after the complete loss of vision.

We have observed a remarkable demonstration of the implications of the "innervation sense" during strabismus surgery under topical anesthesia (i.e., no anesthetic agent administered either systemically, or locally into the muscles). Following surgical exposure of the medial and lateral rectus muscles of an eye, an intense vertical after-image light source is foveally fixated by an eye with normal vision. A three-foot-wide cardboard screen is placed over the patient such that the concave screen surface faces the patient's eye, which is approximately at the center of curvature of the concave screen surface. Large bold numbers along a horizontal screen meridian are easily observable by the surgical subject. The perceived after-image, as might be expected, is localized wherever the eye is directed by the subject, at different numbers along the screen arc. This, of course, is perfectly normal. The fellow eye remains occluded.

The medial and lateral rectus muscles are then completely severed from the globe, and their intermuscular attachments are severed, so that the muscles retract well. The eye is then immobilized in the primary position with forceps on the insertional stumps of the medial and lateral rectus muscles. The patient is then asked to direct his attention so as to "look" at a screen number to the right or left of the zero straight ahead position. Under these conditions, the after-image is perceived wherever the patient "thinks" he is "looking", although the eye is held rigidly in the primary position, and the muscles are completely detached from the globe and well retracted.

Thus, the perceived position of the after-image is consonant with wherever he "wills" his vision to be. The subject's "innervation sense" determines the localization of the after-image.[30]

It is apparent, therefore, that an "innervation sense" is acquired

only with developmental visual input, and that the innervation sense is capable of determining the localization in space. Further, the innervation sense remains intact even after complete loss of all vision in adult life.

If there is no "conscious proprioception" for the visual system, is there then "subconscious proprioception?" What, if any, is the function of ocular proprioception?

Is there a "stretch reflex" of the ocular muscles? In the cat and monkey, action potentials were recorded in the brain stem after stretch of the oculorotary muscles.[31] However, it has been our experience that pulling on the unanesthetized eye muscles of man, during surgery under topical anesthesia, elicits only the sensation of pain. One questions whether recorded brain stem activity upon stretch of the muscle is proprioception or pain. In a similar connection, it has recently been shown that the so-called oculocardiac reflex may be elicited when all of the ocular muscles have been extirpated, and even the globe enucleated. Mere traction on the posterior orbital tissue, presumably by traction on the 5th nerve fibers, may elicit the oculocardiac reflex. One must conclude that, in animals or in man, there is yet to be conclusive evidence that an active stretch reflex exists in the ocular muscles.

However, release of tension in the ocular muscles may be another story. Breinin[10] initially showed that normal ocular muscle tonus diminished considerably following the severence of the muscle from the globe. Our own electromyographic evidence completely supports this. However, in contrast to the studies of Breinin and our own group, others[32,33] did not observe a change in electrical activity when an oculorotary muscle was cut from the globe, unless the cut muscle was markedly retracted.

Conflicting electromyographic evidence exists with respect to stretch of intact muscles.[34,35] Our own repeated electromyographic results in this regard, showed that detachment of the muscle from the globe usually resulted in a diminution of electrical activity, but never to complete electrical silence. Further, my co-workers (Tamler, Marg, Scott, Thorson) and I have found that if the completely severed and retracted (electrical silent) muscle was gradually pulled back toward its insertion, its recorded electrical activity increased to that level seen prior to its severence from the globe. There was approximately the same level of activity as under normal circumstances, regardless of the position of gaze in which the procedure was performed. However, further stretching, beyond this normal insertion position, did not

elicit further recorded increase in electrical activity. These observations confirm those of Breinin[10] who concluded that "These data were considered to support the concept of a proprioceptive tension recording mechanism in extraocular muscle of man. It appeared clear that phasic stretch (tendon jerk) did not occur, but that a basic static stretch mechanism which could not be augmented beyond a limiting point was present."

We feel that the evidence justifies what we might term an "unstretch reflex" in the oculorotary muscles, which possess some information as to the position of insertion. This would seem apparent since the motor unit activity of the cut muscle gradually returns until the insertion end of the cut muscle is at the position of its actual globe insertion, but no further electrical activity increases beyond that point. There would appear to be a signalling of decreased length or tension in the oculorotary muscles, rather than the signalling of increased length or tension such as is observed in skeletal muscle.

So-called "abrupt recruitment" has been held to be evidence for the existence of ocular proprioception.[10] Abrupt recruitment describes the loss of a finely graded reciprocal innervation between agonist and antagonist after the muscles have been severed from the globe. This was believed to indicate that the proprioceptive spindle system acted as a feedback mechanism under normal conditions and now was disrupted with the abnormal relaxed tension of the muscles.

However, in our experience, abrupt recruitment and decruitment occurs only if the involved muscles are in the fixing eye, which is required to fix and follow a slowly moving target. If the fellow normal eye is required to fix and follow, with occlusion of the operated eye, the previously noted abrupt reciprocity in the eye with the cut muscles now changes to a normal smooth reciprocity, despite the fact that the muscles are still completely severed from the globe (Figure 6).

Apparently now the cut muscles receive their smooth reciprocal innervation from the normally fixing eye (according to Hering's Law).

The left side of Figure 6 shows relatively smooth recruitment and decruitment during the reciprocal innervation these severed-retracted muscles received when the opposite normal right eye was fixing and following a lighted target, while the left eye was occluded. The reciprocal innervation from these muscles appeared to be relatively normal despite their retracted status, as long as innervations were dictated (according to Hering's Law) by the dominant normal right fixing eye.

The right side of the figure shows so-called "abrupt recruitment"

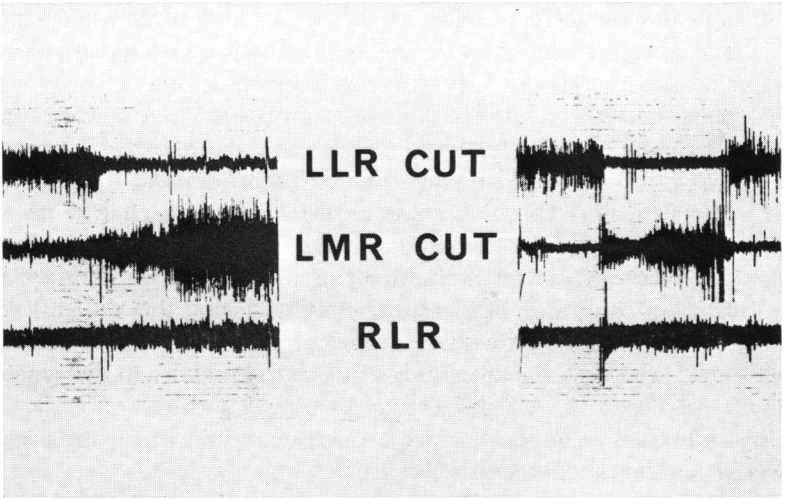


FIGURE 6

"Abrupt recruitment." Diagnosis: left exotropia. During surgical procedure with only topical anesthesia, both horizontal muscles of the left eye were completely severed from the globe and allowed to retract approximately 10 to 15 millimeters by virtue of cutting their intermuscular membranous attachments. The electromyographic electrodes were inserted prior to the severance and stabilized in place with sutures at some distance from the recording tip (see text).

in the severed muscles when the involved left eye was made to fix and follow the same moving target, while the normal right eye was occluded. The left eye, of course, has some difficulty in fixing and following because of the severed horizontal muscles, but it could abduct and adduct within a range of approximately 20 prism diopters.

We would offer the alternative interpretation that abrupt recruitment and decruitment represent reciprocity under duress; these should be considered a manifestation of what might be expected if the eye with the severed muscles is required to fix during attempted following movements. The "agonist" would then be expected to recruit very rapidly, once it failed in its initial attempt to move the globe. There would be an expected equally abrupt reciprocal inhibition or decruitment in the severed antagonist. The abrupt recruitment-decruitment reciprocity is merely a reflection of the attempt to adjust quickly to the task of moving the globe when the muscles are incapable of doing so. The observation that relatively smooth reciprocity persists when the fellow normal eye fixes and follows (despite the physically relaxed

tension in the retracted muscles of the occluded recorded eye) indicates that abrupt recruitment-decruitment cannot be used as evidence for, or against, a functional proprioceptive mechanism.

We have observed a similar "abrupt recruitment" in a normal superior rectus muscle upon attempted upgaze, in a patient with a fracture of the floor of the orbit and an inferior rectus muscle incarcerated in the fracture site. The globe was mechanically restricted in its upward rotation and the attempted upward following movements resulted in relatively abrupt recruitment of the normal superior rectus. The recorded amplitude of electrical activity during this attempt was considerably greater than would have occurred under perfectly normal conditions. Similarly, the amplitude of electrical activity in the severed agonist, in Figure 6, reached extraordinary proportions.

An additional example of abrupt recruitment, occurring in a non-severed muscle, is depicted in Figure 7.

In this instance, a partially anesthetized, drowsy patient exhibited very abrupt recruitment of the agonist left lateral rectus (which was in normal unoperated position on the globe) upon being aroused from his depressed status by a command to gaze left. Note the relatively smooth recruitment and decruitment in following to left and right gaze after partial arousal.

In another vein, we shall cite briefly an instance of what we believe might be additional evidence for a functioning mechanism of ocular proprioception. (This concept will be discussed subsequently in further detail, in the section on fusional vergences.)

This demonstration pertains to patients with intermittent exotropia during assessment of the divergence fusional amplitude consequent to increasing base-in prisms. During the gradual increase of prism, and maintained monopia, a point is soon reached (far before the fusion-free exotropic position) when the eyes suddenly become disengaged and quickly go from parallel alignment to full exotropia. During this maneuver, the sensory fusion stimuli inputs are unchanged. Yet at some point of divergence (under no stress since, as we shall see, this represents relaxation of convergence), there apparently is a signal, possibly from the muscles, which we hypothesize triggers suppression; thus, disengaging the fusion lock. This has been termed (Dr Ellen Takahashi) "fall-apart" fusional-divergence, and is characteristically found in intermittent exotropia. While we have not yet fully documented the fact that a divergence eye movement actually occurs in response to the prisms in this condition, it appears that a myosensory signal of eye position might operate as a trigger mechanism.

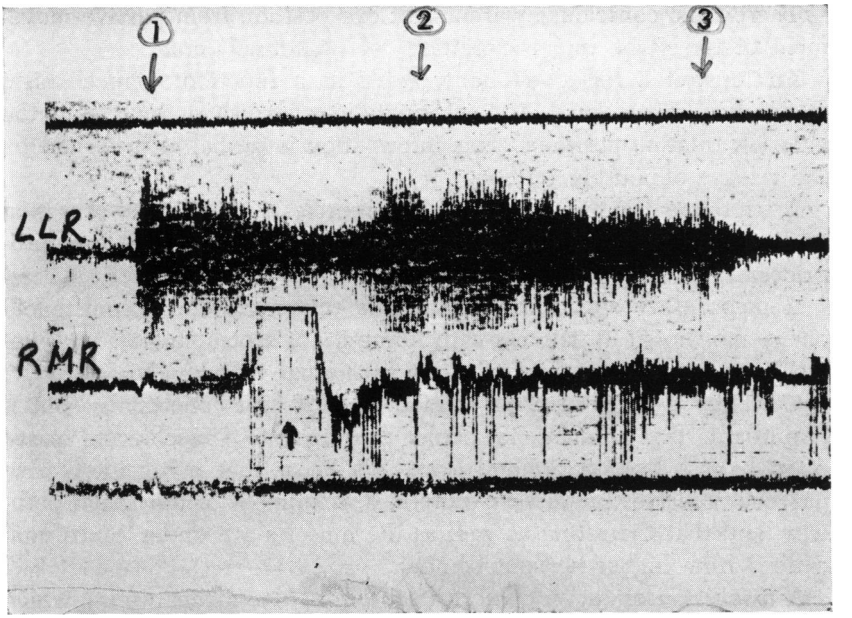


FIGURE 7

Electromyogram of the left lateral rectus and the right medial rectus recorded when a partially anesthetized, drowsy patient was asked to gaze to the left upon arousal (medication: 3 grains barbiturate, 50 milligrams Demerol, 1/200 grains Scopolamine). Note the abrupt recruitment in this patient's left lateral rectus (arrow 1) upon arousal from the depressed status. Note the relatively smooth recruitment and decruitment in following to the left (arrow 2), and to right gaze (arrow 3), after continued partial arousal.

A similar circumstance has been observed by Ogle[36] who, referring to asymmetric convergence at near fixation, suggests that "a compensatory change in the magnification of the images is apparently associated with the turning of the eyes in an asymmetric convergent position, especially when eye movements are involved in this discrimination. No optical stimuli for such changes arise from the pattern themselves. This fact is of importance in studying the nature of the phenomenon and it certainly suggests a myosensory influence of the ocular muscles."

In summary, it is apparent that normal visual adults "learn" an "innervation sense" or an "awareness" of innervation. This learning is dependent upon successful accomplishment of visual tasks during the visual developmental period. Further, this innervation sense persists later, even after complete blindness.

There is no conscious awareness of eye position from passive movement of the globe under conditions of nonretinal input.

An "unstretch reflex" probably exists as a functioning mechanism at a subconscious level, and information is furnished relative to the muscle's rotation positions. This information is probably furnished by the release of muscle tension.

A stretch of the human oculorotary muscles results in the sensation of pain, and recorded activity may, or may not, be related to a proprioceptive mechanism.

A myosensory signal possibly occurs under certain circumstances of eye movement in patients with intermittent exotropia, and in asymmetric convergence at near fixation in normal individuals.

Certainly there is insufficient evidence for firm conclusions. But it is probable that a functional ocular proprioceptive mechanism exists, possibly at a local peripheral level. We know that many integrative nervous processes begin very early in development of the visual pathway, and that the retina as well as the muscles are under centrifugal control from higher nervous centers.

A discussion of the *vergences* (neuromuscular mechanisms) which are *retinal* in origin, has been deleted here because of space limitations. This will be published subsequently. Only the following summary is given here.

There is no neat centrally integrated programmed process which results in a precisely executed "pure" fusion vergence in which *no changes* occur in the apparently stationary fixing eye. Either there is an eye movement or, if the eye is stationary, there is co-contraction; if the latter, this is maintained so long as the disjunctive movement is maintained.

One easily observes, and records both by eye movement tracings and electromyographs, a peripherally manifested competition for fixation and refixation. The components of this depend upon the direction in which the moving eye is going, its speed, and if (or how well) one or the other eye is fixed or becomes engaged in fixation.

Both exophoria and intermittent exotropia start from a zero point of exotropia. *Convergence* fusion innervation is required to obtain and maintain fusion. The eye movement recordings and electromyograms are similar, with the possible exception that the dissipation of fusional convergence in intermittent exotropia *can* be very rapid.

Fusional-*divergence* is the mechanism required to maintain fusion under the stress of base-in prisms from any fusion-free position. It is the mechanism used by an esophoric subject to maintain fusion at parallel alignment of the axes. It is our impression that the dissipation

of fusional-divergence is characteristically slower than the dissipation of fusional-convergence.

It may be concluded that whenever a fusion vergence movement is required to obtain or maintain fusion, in normal patients or in patients with heterophoria, and in most patients with intermittent exotropia, there is a *simultaneous increase* of the motor unit activity in both the medial and lateral rectus muscles in the "stationary" fixing eye during asymmetric vergence. Whenever there is a break from an active vergence status (a disruption of fusion), there is a simultaneous decrease in electrical activity of both medial and lateral rectus muscles of the "stationary" fixing eye. This statement holds regardless of whether the fusion vergence considered is convergence or divergence, or how it is brought about.

The above electrophysiological measurements also confirm the concept that fusion vergences operate around a zero reference of the fusion at *any* fusion-free position. It is no different for exophoria, esophoria, or intermittent exotropia. The zero point or starting point is that found by the rules of determining the *basic fusion-free deviation* (for distance) or any other fusion-free position. Then, and only then, does one have the starting point from which one may rationally manage strabismus problems.

One must entertain an alternative hypothesis to explain the different speeds and time courses characteristic of the "fusion-vergence mechanisms." This would be based simply on fixation and refixation mechanisms (version-conjugated) with different degrees of impedance and damping, depending upon (1) if, and how well, one eye obtains and maintains secure fixation, and (2) the velocity (mainly dependent upon the distance it has to travel) of the disengaged moving eye.

"Fusional-vergence" might be "fusional-bifixation". Theoretical confirmation of this interpretation of fast and slow components of a fusional "vergence" is to be found in the concepts of Linksz, quoted above.

The difference between convergence and divergence, which sometimes appears puzzling, we feel may be explained on the basis of nasal-temporal retinal differences. The nasal retina may sensorially usurp some slack when base-in prisms activate fusional-divergence, without necessarily invoking a corresponding eye movement.

It is interesting to speculate as to the possible correlation between the not infrequently observed *symptoms* and the prolonged maintenance of a fusion vergence to keep a deviation latent (heterophoria). One observes a "struggle" between each eye in gaining and regaining fixation during fusional vergence. This is reflected in the maintained

co-contraction as long as the fusion vergence is maintained. It is interesting to note that both the maintained co-contraction and symptomatology may slowly subside with fusion held in abeyance.

We have noted that some patients with intermittent exotropia, even as much as 70 prism diopters, are completely asymptomatic in maintaining fusion over very prolonged periods of time. It is interesting to speculate, although we have no correlative evidence, that these might be the patients with intermittent exotropia who do *not* show a maintained co-contraction.

Some patients with intermittent exotropia have some degree of "control" or "awareness" of eye position and can align their eyes without the sensory stimulus to fusion. It is a frequent clinical observation that occlusion of one eye frequently does *not* result in the dissipation of the vergence. Rather, the eyes remain aligned while only one eye is seeing. The patient is often unaware of this. This accounts for the masked diagnosis of intermittent exotropia in many subjects, a pitfall known to every ophthalmologist.

Some of the patients with large degrees of intermittent exotropia, who manifest some of the unique characteristics we have just noted, may even have some degree of greater independence between the two eyes, with some imagined uncoupling within even the simple mechanical black box model. The full flowering of what might be considered an atavistic type of vision is seen in the alternating exotropic patient with equal vision.

It is to be noted that the hypothesis which we have entertained as an alternative applies only to the fusion-vergences. This is not to say that the eyes are necessarily independent, but rather that there is not a finely integrated central program during a disjunctive movement as there is a finely integrated program in a voluntary saccade involving both eyes disengaging fixation and conjugately refixing.

Accommodative-convergence, which may be exhibited when one eye is completely occluded, is a different problem. This may be related to the autonomic "near reflex" with integration of accommodation, miosis, and convergence.

ADVANTAGES AND PITFALLS OF ELECTROPHYSIOLOGIC AND PSYCHOPHYSICAL MEASUREMENT TECHNIQUES

Sir Francis Walshe,[48] in discussing "The Future of Neurology" at a 1955 meeting of the Royal Society of Medicine, stated his conviction that "a fruitful future for neurology lay rather with experimental pathology than with electrophysiological studies. In the case of the

latter, it seemed that many studies revealed more about the properties of the apparatus used than of the functions of the nervous system."

My co-workers and I began our paper on artifacts in electromyography with the above statement. We closed this 1959 article with the comment, "We can understand Sir Francis Walshe's pessimism regarding electrophysiology, quoted in the opening paragraph, but we cannot agree with his conclusions. With strong scepticism and painstaking care, artifacts may be largely, if not entirely, exposed and eliminated or rejected."

As we have seen, electromyographic techniques for assessing both eye movements and the pattern of electrical activity from the ocular muscles, and the interpretations of these, have differed widely in different laboratories. It is our opinion that most of these differences arise from either insufficient sensitivity of the recording equipment, or insufficient evidence. Interpretations are sometimes based upon negative results, which may not be in agreement with the historical flow and context of previous research, and which therefore demand even stronger evidence.

Additionally, whenever a clinical situation for the assessment of the visual apparatus is reduced to a laboratory status, one runs the risk of eliminating seemingly small parameters that later prove to be of great importance. One "throws the baby out with the bath."

For the proper interpretation of eye movements (EM) or electromyographic (EMG) information, one should, whenever possible, provide the following additional information:

(1) Which eye was fixing,

(2) Which eye was the habitually dominant eye,

(3) Whether fusion was present or not, and when it was regained or disrupted,

(4) Whether the eye was seen to move or not,

(5) Whether both eyes were monitored,

(6) What was the character of the fixation target and its surround,

(7) Were normal blinks prevented from occurring.

The following statements appear to be warranted: When an eye is seen to move, eye muscles move it, and the appropriate motor unit activity of the agonist and antagonist in reciprocal patterns may be electromyographically recorded.

One of the unique characteristics of a true saccadic (fast) movement is the complete inhibition of the antagonist. Thus, it is readily identifiable as a true saccade in electromyography in a superior fashion.

When an eye is seen to remain stationary, it may do so with no

change in the motor unit activity of the horizontal muscles, or equal and opposite activity in the horizontal muscles, or an unequal change in the horizontal muscles if it is struggling to maintain fixation against the sweeping current of its fellow fast-moving disengaged eye.

It is desirable, although not always practicable, to have some monitoring system for eye movements along with the electromyogram. Or, in place of this, to have multiple insertions in the eye muscles of both eyes so that observations of the electromyograms of the fellow eye will indicate whether or not a version took place, which might not have ordinarily been detected by observation of just the monocular electromyogram.

What type of electrical activity is recorded with present EMG techniques? Does one record differences between different types of muscle fiber systems, one for fast (saccadic) and one for slow (tonic) movements?

According to recent information there are at least three types of muscle fibers in the oculorotary muscles. These appear to be (1) large, very fast twitch fibers, (2) slower twitch fibers, somewhat comparable to the twitch fibers found in limb muscles, and (3) fibers with all the characteristics of amphibian slow multi-innervated muscle fibers.

The slow twitch, and slow multi-innervated amphibian type, are undoubtedly active in tonic eye movements. Electrical activity which is not propagated (slow "tonic") probably can only be recorded intracellularly. If one assumes that fusional vergences are tonic in nature, and mediated only by the slow fiber system, one should not be able to record them electromyographically with an extracellular electrode such as is commonly used. But we can and do record fusional vergence activity with this type of electrode.

Whatever the explanation may be, the fact remains that co-contraction can be recorded electromyographically, especially the maintained co-contraction during an obvious fusional "vergence." The maintained aspect probably means that the co-contraction is not merely an "assist" from the fast fiber system as has been suggested.[49] There may be a slow fiber system of nonpropagated intracellular nature which we do not record, in addition to the activity which we do record.

If the electromyographic pattern is recorded in a moving eye during a vergence, one can make no conclusions regarding the character of the recorded activity (fast or slow – tonic systems) since the usually recorded fast fiber system shows reciprocity whenever the eye moves, and one might merely be recording this type of activity. However,

asymmetric fusional vergence allows the opportunity to record not only the transient changes in motor unit activity of the "stationary eye" as refixation adjustments take place, but what happens *after* bifixation is secured. Reference to a previously published example[72] shows unmistakable evidence of maintained co-contraction in a stationary eye, reflected in one muscle's single motor unit as a maintained increased frequency, and also reflected as a maintained general level of activity in its antagonist muscle. In this instance, one might conclude that since there is maintained increase of activity which parallels the maintenance of the fusional vergence, and a dissipation of the motor unit's activity with dissipation of the observable fusional vergence, that the electromyogram is therefore recording the tonic type of activity; i.e., a vergence. This is not an unreasonable interpretation.

However, an alternative interpretation must be considered which relates the explanation of this recorded activity to the suggestion I have made regarding the nature and character of fast and "slow" eye movements during a fusional "vergence." The recorded co-contraction may be the usual recording of the fast fiber system, reflected in only conjugated system(s) operating to obtain fixation (with or without its maintenance) for each eye in a non "fusion-free" state (heterophoria). That is to suggest that the fusion vergence mechanism may not represent a "slow tonic" mechanism. Rather the "slow" eye movements in fusional vergences may be "slowed-down fast movements."

In another vein, we have recorded what appear to be two patterns of activity from a single unit during strong oculorotary muscle contraction. These two patterns of activity suggest that the second group of fibers described above (slow twitch) may have "two gears" capable of participating in both fast (phasic), and slow (tonic) types of muscle activity.

Figure 8 shows single unit responses recorded even during strong contraction by means of a concentric electrode with a 10 to 25-micron diameter core. During a progressively increasing contraction which took eight seconds, a single unit was followed and its amplitude and frequency (or period) were plotted (Marg). Two patterns of activity were distinguished. A tonic activity is seen, starting at less than 10 pulses per second. Superimposed on this tonic response the same unit exhibits brief bursts of one to four pulses, with frequencies as high as 270 pulses per second, which may occur as often as at half-second intervals. In another subject, a unit reached 350 pulses per second. It would appear from this that tonic and phasic innervation can be

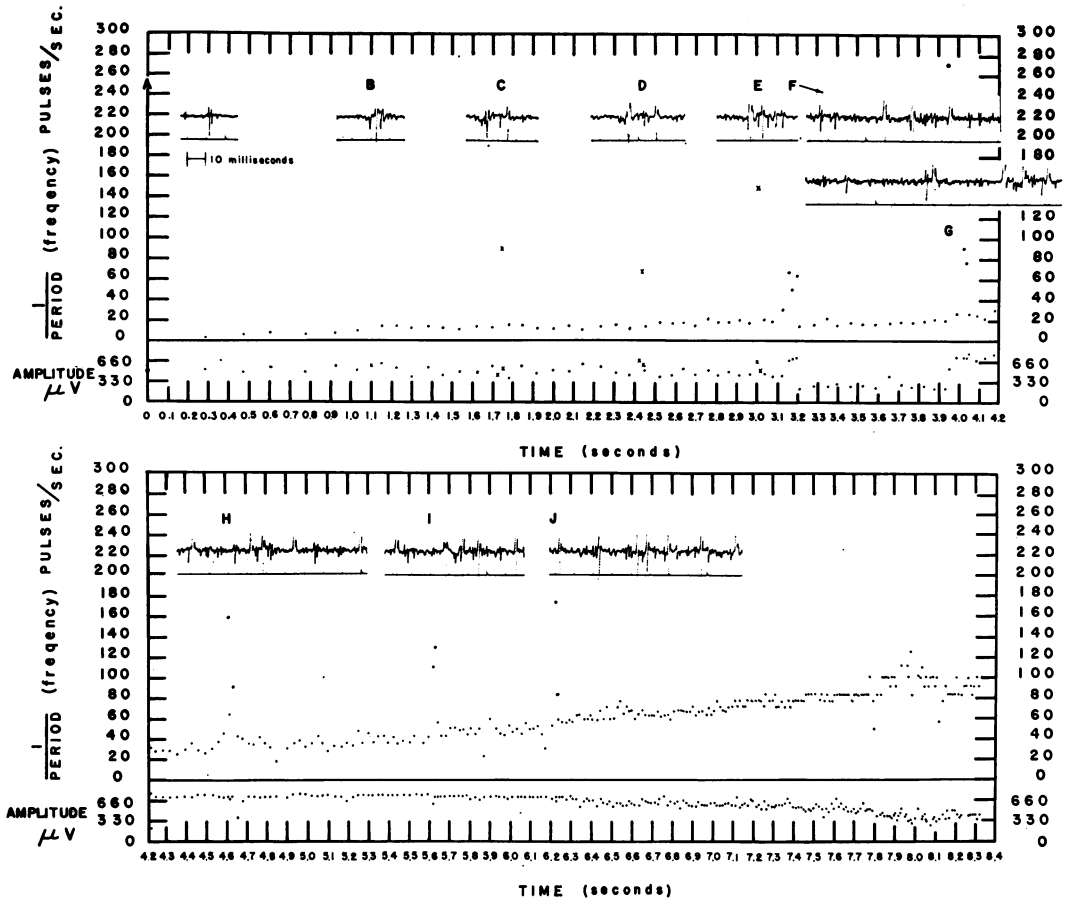


FIGURE 8

A unit from the lateral rectus during a following movement of the eye from extreme adduction (relaxation) to extreme abduction (contraction). The two graphs are continuous. The movement started four or five seconds before the observed unit first appeared at time 0.0 seconds on the abscissa. The unit was followed for 8.3 seconds. Insets A to J with expanded time scale show the action potentials at specific points of interest, the position of the letter on the abscissa indicating the time of occurrence of the inset. The amplitude is measured from the trough of the negative wave to the crest of the following positive wave. The crosses signify some doubt that the datum point is based on the same single unit. Note that the maximum frequency (or minimum period) displayed is 270/sec at time 3.94 seconds. The amplitude indicates the relative position or movements of the electrode tip.

two inputs to the same single effector unit.[50] Thus, a single motor unit might be capable of "shifting gears" to perform both fast and slow functions.

Sir Francis Walshe's admonition, quoted above, is indeed appropriate today. Shrewd and accurate clinical observations are not necessarily inferior to sophisticated laboratory technical devices. Maddox,[23] in 1882, assessed the rate of relaxation of convergence by timing from a pendulum and stated, "It begins with me in less than half a second and goes on with decreasing speed which becomes inappreciable after ½ a minute to a minute and a half." These observations are still valid 85 years later.

Optimally, in order to monitor each eye's movement simultaneously with the electromyogram, the recording of eye movements should be made with all electromyograms. However, this is not always possible or practical.

THE IMPORTANT ROLE OF BLINKS IN THE REFUSION OF INTERMITTENT EXOTROPIA

We have noted a curious and almost constant phenomenon associated with the refusion movement in intermittent exotropia. This is a blink, which under usual seeing conditions, invariably precedes the refusion movement. In fact, some intermittent exotropes cannot fuse without the blink, even with conscious attempts to refuse. Others, when aware of this association, may consciously "try" to inhibit the blink during refusion.

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

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; this is followed by the normal 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. Since this "missing link" entity of intermittent exotropia is one of the

main targets of investigation in this presentation, we shall not leave this intriguing stone unturned.

What role does a blink play under circumstances in normal patients?

Davson[51] states that "the normal blinking rate is apparently determined by the activity of 'a blinking center' in the globus pallidus of the caudate nucleus." However, the blink rate may be changed by the emotional state or fatigue. "When any given route for sensory impulses is blocked, for example by cocainization of the cornea, the blink rate is, on the average, unchanged; i.e., characteristic of the individual." Davson goes on to remark that a movement of the eyes is generally accompanied by a blink; this, he feels, aids the eyes in changing their fixation point. Thus, he concludes that "blinking in the normal individual is an aid, although not a necessary one, in inhibiting the fixation reflex preparatory to adoption of a new point of fixation."

Cogan,[52] in discussing congenital oculomotor apraxia, states that "when a person with normal control of eye movements is asked to look at an object to one side, he first turns his eyes (usually associated with a blink of the lids) and then his head. ..."

That the eyes move during a blink was studied subjectively by Ginsborg,[53] who found that the eyes converge slightly during the blink. Stewart,[42] on the other hand, found that the eyes may slightly converge or slightly diverge during a blink.

Our own observations and studies of the blink, which appears to initiate the refusion in intermittent exotropia, reveals that the blink sometimes initiates a considerable convergent movement which occurs during the normally appearing blink. It should be noted that the disengaged exotropic eye need not be further disengaged by a blink, when it is sent on its way to refusion, since it is suppressed. In this instance the blink does not appear to serve the facilitation of fixation disengagement.

Figure 9 is a high speed motion picture study of a blink in a patient with 70 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.

This series of six recordings show what would seem to be an

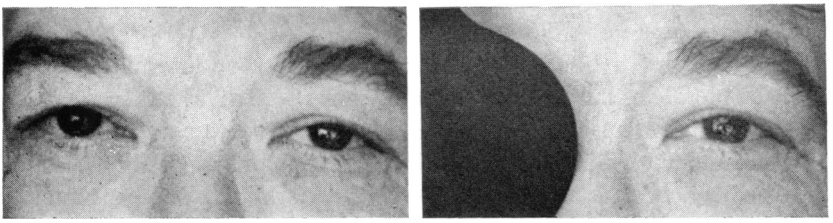
essential role of a blink in the refusion process of intermittent exotropia. The role of a blink appears to be more than incidental.

The electrophysiologic investigation of eye movements invariably is associated with blink artifacts. The investigator comes to recognize the occurrence of a blink which often occurs 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 artifact induced by the blinks (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.

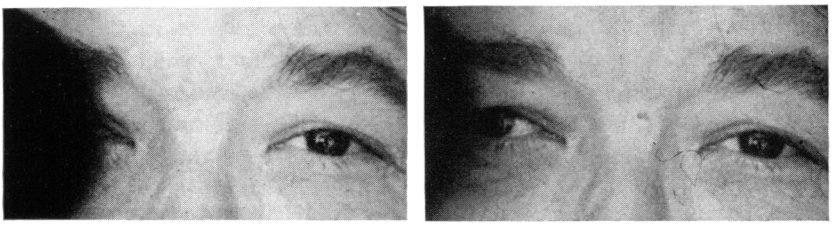
On the other hand, asking the subject not to blink, or preventing a blink, by a speculum between the lids, or by other laboratory interference, may similarly produce an artifact. If a blink is a necessary part of the program of change of fixation in normals, or of refusion in intermittent exotropia, then not allowing a normal 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 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 convergence movement. It is probable that this convergence relationship is exaggerated in intermittent exotropia as demonstrated by the previous Figures, and serves a useful purpose.

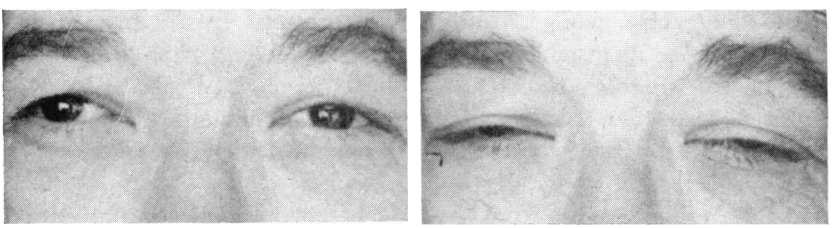
A blink may represent a necessary visual pause while messages of one sort are received and another type is transmitted. Similar blinks



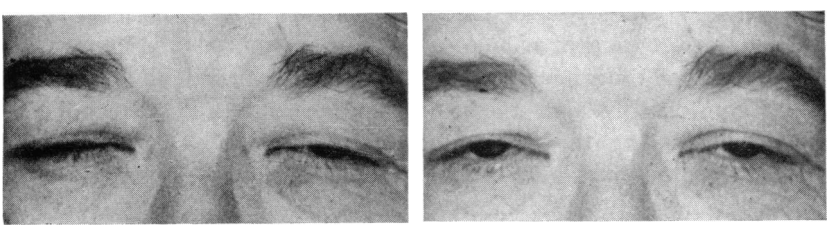
Frame 1 Frame 2



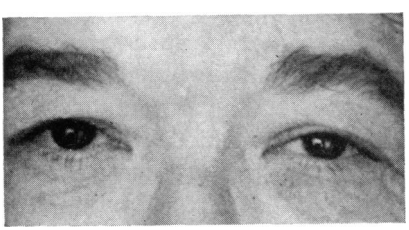
Frame 3 Frame 4



Frame 5 Frame 6



Frame 7 Frame 8



Frame 9

FIGURE 9

Patient with intermittent right exotropia of 70 prism diopters. Very high speed photography during normal blink (frames 5 to 9) which initiated the refusion.

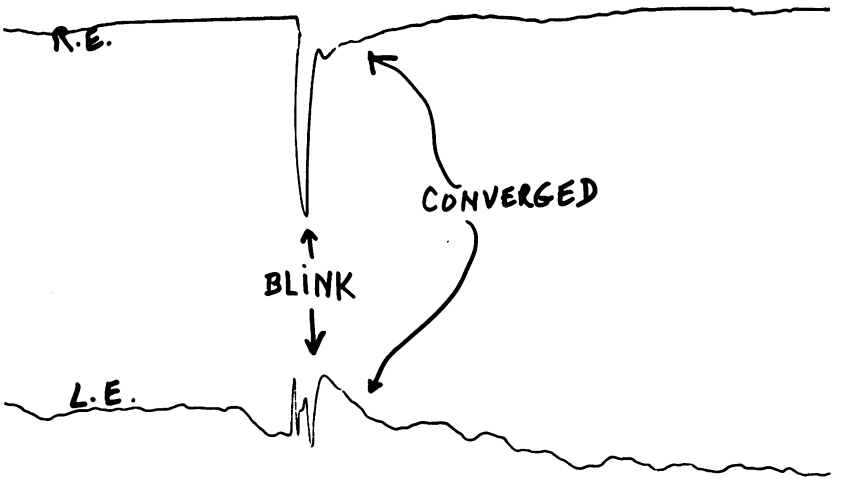


FIGURE 10

Same patient as in Figure 9, as are following 5 figures. 70 prism diopters of intermittent right exotropia. A 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.

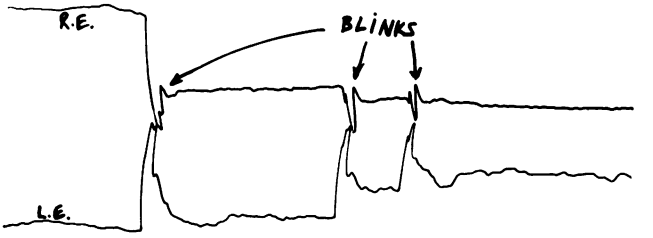


FIGURE 11

Shows a series of three blinks, each one "pumping" the left eye in a convergent direction to join with the refixated right eye, so that fusion is obtained after the third blink.

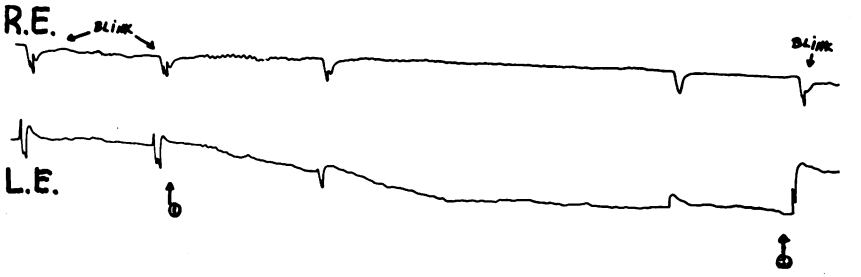


FIGURE 12

(Calibration different than previous fiigure.) The left eye was uncovered at arrow 2. Refusion was co-mingled with a blink and refusion of 70° took 0.2 seconds.

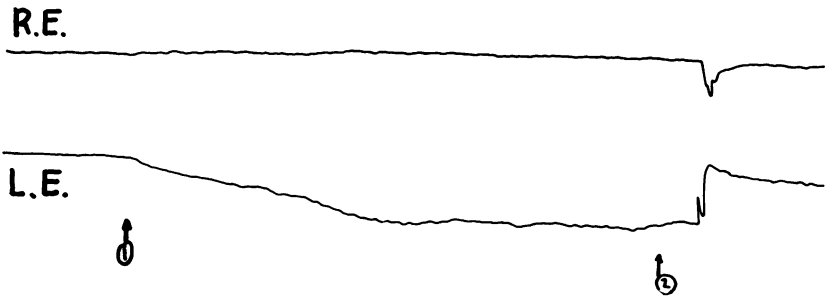


FIGURE 13

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 refusion.

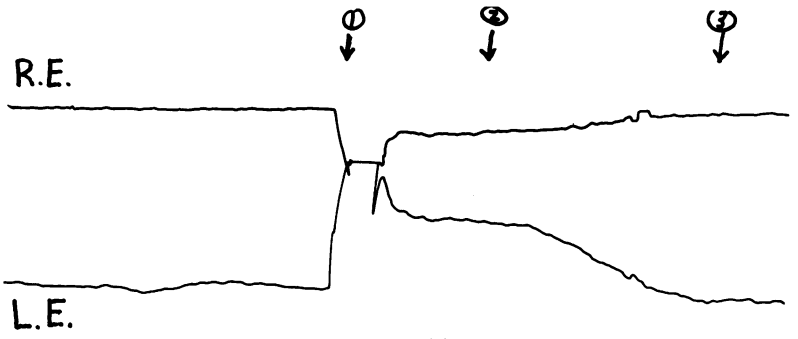


FIGURE 14

Arrow 1 marks a prolonged, purposeful blink, during which time the left eye was covered and remained covered throughout the rest of the figure. 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.

have been noted in studying the reception of conflicting auditory messages. It is not within our scope to detail such an analysis, but it is our belief that the application of theories in terms of panel capacity, redundancy, storage, and programming mechanisms, will give valuable insight insofar as the mechanisms of intermittent exotropia and similar conditions are concerned.

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

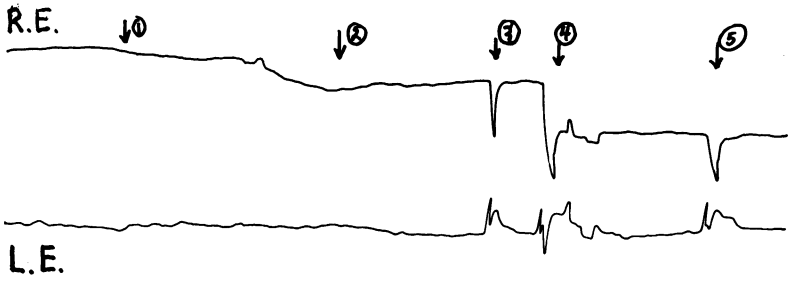


FIGURE 15

The left exotropic eye is uncovered at arrow 1, followed by only a slight convergence movement which plateaus at a second, still insufficient, level of convergence (arrow 2). An initial blink (arrow 3) is of little help in refusion, but it is followed by another blink (arrow 4) which is associated with the converging eyes and fusion is obtained. Arrow 5 is a blink during fusion.

In conclusion, some patients with intermittent exotropia must blink in order to initiate the refusion. Others cannot blink while exotropic without refusing. 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.

ACCOMMODATIVE-VERGENCE AND ASSOCIATED PHENOMENA

It is our purpose at this time to examine only those aspects of accommodative-convergence which are germane to our problem of exodeviations.

We shall consider the following problems:

(*a*) Is accommodation used as the trigger for the convergence which invokes the refusion of intermittent exotropia? Is there possibly a negative accommodation mechanism, with associated divergence, which plays a role opposite to that of positive accommodative-convergence?

(*b*) Is the accommodative-convergence ratio normal or abnormally high in intermittent exotropia? What accounts for the test measurement differences among the different methods of assessing the A-C/A in this condition? What are the methods of altering the A-C/A in intermittent exotropia? Relative to this, we shall consider the role of minus lenses and of surgery.

(*c*) Is there a "remote-divergence" as the counterpart to "proximal-convergence"?

(*d*) We shall assess the time course characteristics of eye move-

ments during "pure" accommodative-convergence, and the mixture with fusional-convergence, under the normal circumstances of changing fixation from distance to near and vice versa. This has special relevance to our problem in intermittent exotropia, since one would suppose that if there were any instance in which the mixture of version-vergences would be centrally integrated and well planned, it would be appropriately programmed in this often used usual circumstance.

(*a*) That accommodation per se is not the trigger mechanism, which invokes accommodative-convergence as an essential part of the refusion mechanism in intermittent exotropia, may be determined by critical monitoring of the accommodation status. Retinoscopy during refusion and break of fusion in intermittent exotropia reveals that there is essentially no change in the accommodative status in either of these instances.

The possibility of a negative accommodative mechanism must be considered. Is there a sympathetically innervated divergence mechanism which counterbalances the parasympathetically innervated accommodation-convergence mechanism? Cogan[54] proposed that accommodation for near is parasympathetically innervated, involving the circular fibers of the ciliary body and that accommodation for distant objects involves the sympathetic nervous system control of the radial fibers of the ciliary body.

Although it is customary to conceive of accommodative-convergence starting from a zero accommodation point in a positive direction, and to consider that the relaxation of accommodation and relaxation of convergence takes place in going from near to distance fixation, it is conceivable that the latter is more than a passive process of relaxation of accommodative-convergence.

A number of studies have shown that direct stimulation of the cervical sympathetic chain causes a further relaxation of accommodation beyond the usual zero relaxed accommodative state. It has been held[55] that the refractive changes with sympathetic stimulation are due to transient vascular changes with consequent volume alterations affecting the lens zonule, thus resulting in modest refractive error changes.

There is no significant evidence that a negative accommodative-divergence mechanism exists during life, in normal or in pathologic conditions. However, it is intriguing to recall that Jampel[18] reported that appropriate brain stimulation in the occipital eye field in a

monkey always elicited a dilatation of the pupils with any divergence movement.

(*b*) The accommodative-convergence ratio (A-C/A) appears to become increased in exodeviation. It has been our observation that the distance basic exodeviation appears to become greater in degree at a greater rate than does the near exodeviation. Is this the question of an increase of the A-C/A by mechanical or neurogenic factors?

In normal individuals, the A-C/A ratio is relatively linear within physiologic limits, and is stable throughout life.

In the clinical measurement of the A-C/A ratio, one must be aware that the accommodation stimulus does not always invoke a corresponding accommodation response.

Further, it makes a difference whether the stimulus is invoked by alterations of real fixation distances, as from distance fixation to near fixation (phoria method), or whether the stimulus is invoked by manipulating the accommodative status through induced plus or minus lenses at a fixed distance (gradient method). In either instance, the assessment is made by changes in the deviation under fusion-free conditions, in order to evaluate the "pure" accommodative-convergence mechanism.

The essential difference between the distance versus near deviation comparison method, and the lens induced accommodation-convergence method, is that in the former there is a "proximal-convergence" element. This implies that mere awareness of the object's proximity invokes some associated convergence, independent of any accommodative stimulus. This is probably psychic in origin and "learned."

We have found that in exodeviations of significant degree, there may be a real difference between the method of comparing distance deviation with near deviation, and the lens induced method of manipulating accommodation carried out at a fixed finite distance. This dichotomy we interpret to represent an alteration of the proximal-convergence factor in intermittent exotropia and constant exotropia. This may be related to the fact that at distance fixation, both the magnitude of deviation increases, and the fusion coverage diminishes, to a greater extent and earlier than at near fixation distance. The near fusion status is the last to undergo a decrement, sometimes exhibiting orthophoria and a firm fusion lock at near, with degraded constant exotropia at distance. We shall return to this point again in discussing the possible alteration of the A-C/A ratio with minus lens prescriptions.

It is our contention that assessing the accommodative-convergence relationship in terms of distance deviation versus near deviation is an assessment under the most usual every day visual habits. We prefer to utilize this method for the clinical determination in strabismus management. After all, this is the way people use their eyes, and in strabismus management we are concerned with any alteration of this usual way either during the natural course of events or subsequent to alteration by orthoptic or surgical means. We therefore consider the distance to near comparison, for the assessment of the accommodative-convergence relationship, superior for clinical purposes. This method includes the natural proximal-vergence factors which, we have indicated, may possibly be altered in intermittent exotropia.

It might be supposed that the usual lesser degree of exodeviation at near is in reality more exo than one initially measures, and that this may be unmasked by prolonged occlusion. Such is not the case in our experience.

In this regard, Morgan[56] has shown that in normal individuals, the mean value of the proximal A-C/A phoria measurement method was significantly larger than by the lens induced assessment method (gradient method) and was 9 diopters.

The phoria measurement is larger probably because it takes into consideration the proximal factors. Thus, in the clinical management of intermittent exotropia, the phoria method (comparison of distance and near deviation) may allow one to better assess the very factors one seeks to manipulate, namely the proximal factor.

Is it possible to alter the accommodative-convergence ratio in a natural way which includes the proximal factor, by (1) orthoptics and (2) surgery? It is a common custom to manipulate the accommodative-convergence relationship by the prescription of added minus lens power to the basic refractive error correction, in order to mask an exodeviation and keep it latent over a longer period of time. This is a most useful clinical maneuver with practical benefits in augmenting and maintaining fusion, as well as in deriving cosmetic improvement during the time the lenses are worn. Children will tolerate a considerable amount of such excessive accommodative stimulus (commonly 3 diopters, and not infrequently 5 diopters).

One may predict an enhanced value from minus lenses when the A-C/A ratio is high, since it does not matter how one manipulates the accommodation, whether naturally or by lenses, drugs, or other methods. Whenever one manipulates the accommodation in a patient

with a high ratio, one will get a greater change in convergence. The high ratio is the predictor of the clinical effect.

Thus, minus lenses have maximum benefits in the management of the commonly observed high A-C/A ratio in intermittent exotropia. Several years ago, it became apparent to the writer that although the distance deviation is masked while the lenses are worn, the esodeviation at near (which replaced the usual near orthophoria), soon becomes readjusted so that the near deviation with minus lenses readjusted to near ortho within a matter of weeks. One could consider this to be either a lessened accommodative effort and response or a transient decrease in the A-C/A ratio (which proved to be the case). When the minus lenses are removed after being worn many months, the previously near ortho near deviation is significantly more exo (without the lenses). If the minus lenses are removed for a matter of weeks or months, the A-C/A returns to its previous pre-treatment values. However, it is most helpful to realize that one can manipulate the A-C/A ratio and transiently change it with a prescription of minus lenses, especially when there is a fusion backstop of initial ortho at near.

This maneuver is especially applicable to those instances where the distance deviation is a large constant exotropia, and the near deviation is almost ortho. This is one of the most difficult problems in strabismus management. The prescription of minus lenses therefore, in this high A-C/A ratio circumstance, transiently increases the near exodeviation toward a value more consonant with the distance deviation, after removal of the lenses. This makes immediate surgical management simpler if it is not long delayed (since near and distance deviations are more equal).

Alpern[37] suggests that one might change the accommodative-convergence relationship if one could restrain accommodative-convergence while changing accommodation. Essentially this is what the above-described maneuver accomplishes when there is a high A-C/A ratio and almost ortho at near which limits and locks convergence.

Others[57] suggest that stimulating more than the usual amount of accommodation, for a particular fixation distance, may in some way dissipate the proximal-convergence factor. This may be an additional factor for the effect of minus lens prescription described above.

Similarly, uncorrected myopes were considered to have their A-C/A ratio "adaptively modified."[58]

In summary, therefore, it appears that the A-C/A ratio is character-

istically higher in patients with intermittent exotropria (especially adults) than in normals. This may be related to the proximal-remote factors in the accommodative-convergence relationship. It appears possible to modify the accommodative-convergence relationship by orthoptics, especially by the prescription of minus lenses to stimulate accommodation, while holding accommodative-convergence fixed, because of the usually characteristic relative ortho at near in these patients. The alteration in the accommodative-convergence relationship is transient but it is a practical aid in preparing patients for surgery and making the near deviation more consonant with the distance deviation at the time of surgery.

We have previously called attention[13] to the surgical mechanical alteration of the accommodative-convergence relationship. An example is the recession of both medial rectus muscles for an esotropia with distance orthophoric fusion and near esotropia. In this example, there is a distance fusion lock at ortho, and a mechanically reduced A-C/A.

(*c*) Is there a "remote-divergence"? In a previous section we discussed the secondary anatomic changes in the abducting group of muscles, especially both oblique muscles in the usually exotropic eye. These purely mechanical secondary impediments to adduction may considerably alter the measured A-C/A, even though the neural factors may or may not be changed. An alteration of the A-C/A, it should be noted, may come about by either mechanical or neural changes, or both.

Since, as we have just seen, it is possible that there is an alteration in the proximal-vergence phenomenon, and in a possible remote-vergence phenomenon, a few further comments are pertient.

Laboratory experimental conditions often dissipate the very factors that bring about proximal-vergence.[59]

It is probable that the real life-learned proximal factors consist of such parameters as attention, bolder fusionable contours, larger imagery, better structured peripheral field, stereoscopic fusion, hand-eye augmentation, etc. It is also probable that these naturally occurring proximal factors account for the difference in A-C/A ratio by the phoria versus gradient methods.

Proximal-convergence may be defined as convergence induced by apparent (not actual) nearness of the fixation object. Awareness of nearness can be taken to serve as both a facilitating stimulus, and as a de-facilitating stimulus, if the stimulus is identified inversely as awareness of distance rather than of nearness.

The above mentioned factors operable for proximal-convergence are diminished with distance fixation. This is especially so if the distance fixation involves not 20 feet but 200 feet, as by the usual clinical circumstance of observing a telephone pole through a window, in which case the object of regard is surrounded by an highly illuminated structureless field.

It is a common clinical observation that an exodeviation may be found to be greater at 200 feet under such circumstances, than it is under conditions of 20 feet fixation within the office confines.

We would suggest the real possibility of a remote-divergence, based upon concepts of perceived distance, and perhaps more importantly the unstructured high intensity peripheral surround (even for the fixing eye) that accompanies truly remote viewing circumstances.

The intermittent exotrope has a unique need for a richly structured field surround in order to firmly fuse. Frequently this is seen when attempting to assess either the subjective or objective angles with the major amblyoscope. This clinical instrument limits the peripheral fusionable field to approximately 15 degrees. Under these circumstances there is a great instability and variability in quantitative assessments. There is much more difficulty in attempting to gain fusion than is observed for normal individuals. Patients with intermittent exotropia often require a rich structured surround in order to fuse.

The amount and intensity of light per se, as we shall see, is a most important optically elicited source of oculorotary tonic innervation. The finding that sometimes there is a significantly greater exodeviation for truly remote targets than for those at 20 feet, is supportive clinical evidence that there is a remote divergence. This has been remarked upon previously.[60]

We suggest that the remote-proximal factors may undergo considerable alteration in intermittent exotropia, and that these factors; i.e., an altered proximal-convergence, with or without an altered remote-divergence, are the factors responsible for the clinically measured high A-C/A in many exodeviations.

It should not be surprising that clinical tests by the gradient method might produce different results than clinical tests by the proximal-remote method especially in this circumstance. Our stated preference for the latter method is particularly germane in exodeviations where the proximal-remote factors and their alterations may play a more important role than usual.

(*d*) Is the mixture of accommodative-convergence and fusional-

convergence, which is employed so frequently under normal circumstances in changing from distance to near, peripherally manifested in the eye movements? Or is this mixture centrally integrated and programmed? The reader will recognize this "problem"; it is mentioned here since it is felt that if ever there were a situation in which a mixture were centrally neutralized and programmed, it might be in this frequently repeated circumstance. This will help illuminate and, as we will see, further support the view we have presented previously, relative to the always peripherally manifested mixture seen in pure fusional-vergences.

First, let us examine the time course characteristics of "pure" accommodative-convergence. In real life this probably only occurs when one eye is deeply amblyopic or blind and strabismic, since in this situation there is an absence of any fusion-vergence stimuli.

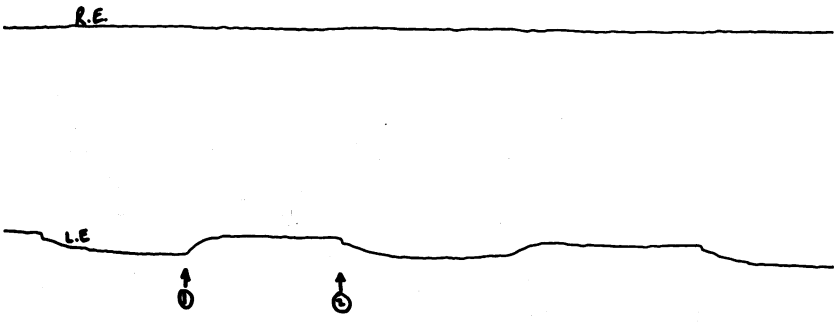


FIGURE 16

Normal subject during a sudden change of right eye fixation from distance to near, the left eye completely and continuously occluded. Both distance and near objects of regard on the axis of the right eye.

This is an example of a normal subject exhibiting *pure* accommodative-convergence (asymmetric) in suddenly changing right eye fixation from distance to near (left eye completely occluded throughout) (Figure 16). Note that the relaxation of accommodation-convergence (arrow 2), in changing fixation from near to distance, is considerably slower than is the invoking of the accommodation-convergence, by changing fixation from distance to near (arrow 1). This has been shown previously.

Note the stable fixation of the right eye. It will be seen that in the circumstance of pure asymmetric accommodative-vergence in a normal individual with one eye completely occluded, the fixing eye does not, indeed need not, become disengaged when changing fixation

from distance to near, or from near to distance. There is no second image that suddenly appears which might or might not shift localization, and there are no fast "fusion" movements with which to contend.

It is to be noted that the speed of accommodative-convergence alone would be much too slow to keep up with the demands for changing fixation between distance and near targets, if it were the sole vergence available. Fortunately, under real life visual conditions, the more rapid fusion stimulated movement is available to reach the goal of fusion. As we shall see, this rapid movement (fixation) overrides the slower accommodative-convergence, which tags along to take over as a holding operation.

This is another example (Figure 17) of pure accommodative-convergence (left eye occluded), in a patient who had orthophoria at both distance and near. Arrow 3 marks a distance to near fixation. Note the very rapid convergence associated with this accommodation, and the slow dissipation of this same vergence (arrow 4) when changing fixation from near to distance.

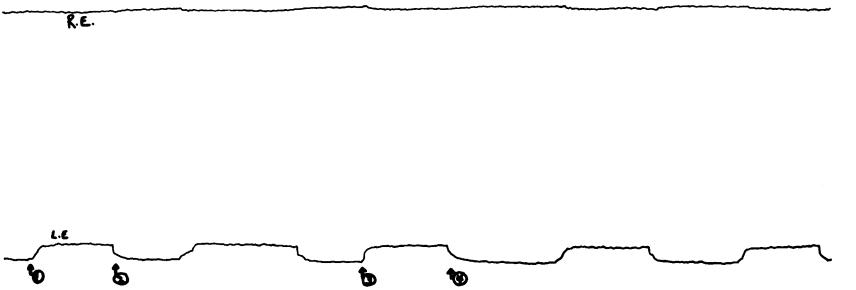


FIGURE 17

Another normal subject with an accommodative-convergence relationship such as to produce orthophoria at near fixation. The left eye is occluded throughout while the right eye changes fixation from distance to near (arrow 1) and from near to distance (arrow 2). This is repeated several times.

This is the same subject as in Figure 17, now with both eyes open, changing fixation from distance to near, and from near to distance (Figure 18). This individual had orthophoria at both distance and at near. Thus, his accommodative-convergence was quantitatively sufficient for bifixation at near. It might be supposed that his accommodation-convergence alone would suffice, and that since there is no phoria at near, a fusional-convergence would not be elicited. However, note the very fast converging movement of the left eye at arrows 1 and 3, when changing fixation from distance to near. The rapid saccadic

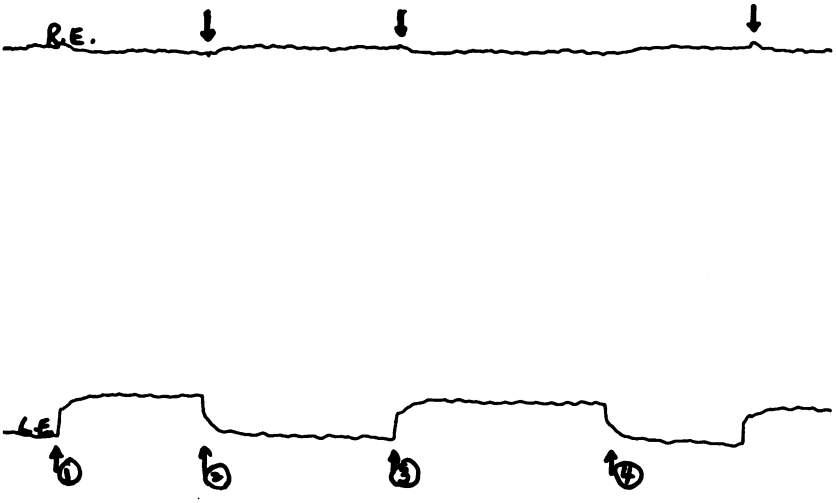


FIGURE 18

Same patient as in Figure 17. A normal patient with orthophoria for both distance and near, changing fixation from distance to near (arrows 1 and 3), and from near to distance (arrows 2 and 4). Both eyes open.

movement overrides the previously seen slower pure accommodative-convergence (Figure 17).

There is an initial large, fast saccadic movement (arrow 1) followed by a slow vergence movement. Note that when the fixation was changed from near to distance (arrows 2 and 4), the stimulus to fuse the distance target now initiates a fast saccadic movement of shorter duration, followed by a longer, slow vergence movement. The entire episode of near to distance fixation and fusion is longer than from distance to near.

The three arrows in the upper tracing indicate a slight fixation disengagement now of the fixing right eye.

Thus again, we see a peripheral manifestation and mixture of the version-vergence as observed in other asymmetric vergences.

A normal subject with dominant left eye (Figure 19). A normal change of fixation from distance to near, with the usual and normal mixture of accommodative-convergence and fusional movements ("vergences" or refixation) (both eyes open). Note that the fixing right eye became disengaged in each circumstance before refusion was obtained (arrows 1 and 2). Further, it should be noted that the dominant left eye rapidly moved from near to distance to gain fixation, "dragging" the right eye off fixation, followed by a slow refix-

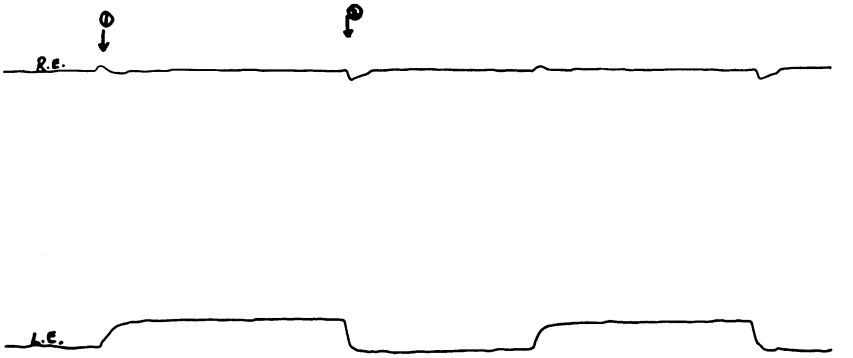


FIGURE 19

Both eyes are open during this normal patient's change of fixation from distance to near (arrow 1), and from near to distance (arrow 2).

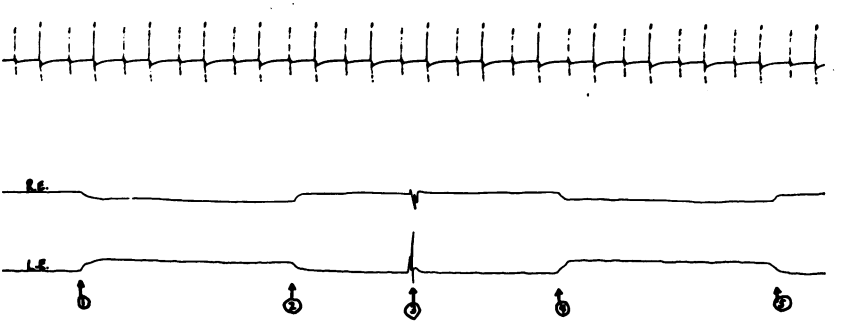


FIGURE 20

Another normal patient during symmetric convergence; i.e., changing fixation from midline distance to near (arrows 1 and 4), and from near to distance (arrows 2 and 5).

ation of the right eye to join the already fixated dominant left eye. It is important to note the overriding consideration of dominance in assessing the results of such investigation.

It is of especial interest to note that there is an initial fast movement of the left eye (at arrow 1) followed by a slow movement during this symmetric vergence (Figure 20). Others have remarked that in the "natural" circumstance of symmetric vergence each eye converges slowly, and that there is absence of saccadic movements in this circumstance. The recording here of the short saccadic movement may be related to the discussion of measurement methods.

In symmetric vergence there is disengagement of both eyes, which then travel in opposing directions.

From the above series of eye movement tracings during symmetric and asymmetric vergence, where there is a mixture of "slow" accommodative-convergenc, and fusional-convergence, it must be concluded that there is a peripherally manifested mixture of fast and slow (version-vergence) eye movements under usual conditions of sudden fixation changes between distance and near. The fast component overrides the slower accommodative-vergence, and as may be noted in the electromyograms of the same circumstance, the fast movements for fusion-bifixation ("fusional-vergence") come in to assist the slower accommodative-convergence. This is manifested in electromyograms by the surges of electrical activity that are seen in the fixing eye. After the necessary "assist", the slower accommodative-convergence is left on its own if it is adequate to maintain the task.

The eye muscles and eye movements do what they must, in order to rapidly secure bifoveal engagement, and then to maintain this engagement. There are many peripheral adjustments that are easily observed and recorded. There is no unified "decision" in the central nervous system, based on an integration of all the relevant factors, which would be reflected in an innervation pattern to the oculorotary muscles for a fusional vergence, or bifixation.

As we have noted, a conjugate saccadic refixation eye movement, on the other hand, indeed shows unmistakable evidence of a complex computer program which delivers a unified and integrated package of innervation to the eye muscles. Once this type of well calculated charge of innervation is sent to the eye muscles of both eyes it cannot be altered until the "program" is completed. We find no such evidence of a finely programmed and centrally integrated control mechanism for either symmetric or asymmetric vergences, in normal individuals, patients with eso- or exophoria, or patients with intermittent exotropia. There is always a peripheral manifestation of the mixture of types of eye movements with considerable local readjustments taking place.

We do not agree with the prevalent notion[61] that there is a regular sequence of events when a normal person changes fixation from distance to near in asymmetric convergence. It is held by many that an initial conjugate lateral movement occurs without a change of convergence so that the visual axes bisect the now nearer object of regard, and that this is subsequently followed by an equal and

symmetric convergence movement of each eye. In other words, the vergence aspect does not occur until the eyes "get set" with a version, so that a "clean" symmetrical convergence may take place. We would suggest, in contrast, that the major factors are the security of fixation of the engaged eye, and the variables previously noted for asymmetric vergence. A neat sequence of events, such as has been described, would suggest a central programming and calculation of the version positioning in order to make the symmetric vergence possible. We reject this notion for this circumstance.

We believe that such a neat scheme of things is needlessly and erroneously applied to asymmetric and symmetric vergences under any conditions. It is like the Vieth-Müller Circle; nice on paper, but it does not work out in practice.

OTHER RETINAL SOURCES OF TONUS

We have previously discussed the nonretinal sources of tonus and have seen that there may be specific conjugate sources or higher or lower *levels* of this total type of continuous tonus. On the other hand, specific neuromuscular mechanisms or functions of vergences are retinal in origin.

The adequate retinal stimuli were (1) fusion, (2) accommodation, (3) proximal cues, and possibly (4) remote cues. The motor response with each of these appropriate stimulus situations was a vergence. In the case of fusion the motor response may be fast (with or without slow) eye movements for fusional bifixation. In the case of the "near reflex" (accommodation and proximal-remote cues), the coordinated motor response is a slower vergence.

We now propose a slow divergence motor response for a relatively unrecognized, but we believe important, retinal (inter-retinal) stimulus. This divergence is maintained as long as the retinal stimulus situation is adequate and appropriate.

In this section we will describe and detail the adequate stimulus for this interretinally elicited (optomotor) tonus, which we believe to be of paramount importance in the etiology and pathogenesis of exodeviations. This vergence, a divergence, is the motor response to the stimulus where there is an interretinal diffusion. The appropriate stimulus is an overall diffusion of the retinal contours of one eye, compared with the fellow eye (interretinal competitive diffusion). As we will see it is not an adequate stimulus situation if there is simply a lack of fusion, or complete unilateral blindness. There must

be a competition between the two eyes, and a competition which is defined by the criterion of an overall blurred contour of the macular and peripheral imagery in one eye compared with the fellow eye.

In proposing this hypothesis for a retinal origin of tonus which is reflected as a divergence, we shall find its wide applicability to clinical problems. These include (*a*) unilateral exotropia secondary to acquired poor vision in one eye; (*b*) nonsurgical consecutive exotropia (which is found only with high hyperopia and amblyopia), (*c*) the phenomenon of shutting one eye in bright lights, and (*d*), most important, the significance in strabismus as a cause of strabismus in general, and specifically in the actively increasing degree of basic deviation in the exodeviations, especially intermittent and constant exotropia.

We shall begin with a commonly observed clinical fact, namely, the increasing degree of unilateral exotropia that so frequently occurs secondary to an acquired unilateral cataract in adults, or to some other ocular pathology which diffuses the image in one eye relative to the other. It should be pointed out that this exotropia, which may reach 80 prism diopters or more of basic exodeviation, may occur in patients who formerly had a basic deviation of ortho. This alteration can hardly be explained by anatomical divergent changes of the bony orbit as is often held, since the exotropia occurs in adult life long after bone factors have ceased to change. Therefore, there must be a functional basis for the shift in the basic deviation; that is, a change of the tonic input to the muscles which is nonaccommodative, nonfusional, and yet retinal in origin.

Such a significant increase in basic exodeviation does not occur when there is complete and sudden unilateral blindness. There is a very significant difference in the situation where an eye has gradually become blind over a period of time, as opposed to the situation where one eye has suddenly become totally blind.

Two other commonly observed clinical facts are pertinent here. Exotropia of significant degree does not occur when only the peripheral visual field imagery of one eye is blurred, as in late glaucoma in which a central macular island may remain functional. Nor is there a significant exodeviation following a unilateral macular disease which leaves a well functioning peripheral retina. The essential ingredient is an overall diffusion, or decrement, in contours of both the macula and periphery in one retina relative to the fellow eye.

The macular fusion lock, in the case of glaucoma just cited, is sufficiently secure to prevent a manifest deviation, and the basic

deviation is not altered. Similarly, in the case of macular disease just cited, which wipes out the function of one macula but completely spares the peripheral retina, the clear peripheral contours are sufficient to prevent any significant change in the basic deviation.

However, when both peripheral and macular images of one eye are blurred relative to the fellow eye, as is commonly seen with unilateral dense cataract, vitreous opacities, or corneal scars, there is the adequate stimulus situation for an interretinal competition (induction phenomenon) which results in an "active" (tonic) motor divergence response. As long as this adequate stimulus situation exists, we suggest that the divergence response exists. This results in increasing degrees of exodeviation which, as we will see, may be augmented by secondary anatomical affects.

The fact that this represents a real shift in the basic deviation deserves some amplification. In sudden complete unilateral blindness, such as occurs with a severed optic nerve, surgical removal of a section of an optic nerve with glioma, and similar circumstances, there is relative orthotropia, sometimes resulting in about 15 diopters of exotropia, but not the 60 to 80 prism diopters not uncommonly seen when one eye differs from the other as we have described. That a totally blind eye may be markedly exotropic, of course, is a common clinical observation. In these circumstances the eye became blind with time, and during this time, which was essential for the active divergence, the adequate stimulus situation of interretinal competition existed. Complete constant occlusion of an eye does not result in large degrees of exodeviation.

We have already mentioned the observation that in a person with 80 prism diopters of exotropia during life, consequent to acquired, unilateral cataract, the position of the eyes after death represents a basic deviation of 80 prism diopters. The same individual had he not had a unilateral cataract during life, would have had relative ortho or only a very slight exodeviation after death. Let us attempt to analyze the component sensory defects that comprise this interretinal diffusion-divergence.

MONOCULAR INTERACTIONS (NASAL-TEMPORAL RETINA)

Mislocalization is characteristic of *any* eye with an overall diffused image (macular and peripheral amblyopia).

It has long been know that patients with dense unilateral cataracts have "faulty nasal projection." That is, these patients, when asked to localize a light in front of the afflicted eye, are able to localize

it easily and correctly when it is in the temporal field, but not when it is in the nasal field. This common clinical corollary of a dense cataract does not indicate retinal pathology, as the clinician has learned since the admonition of von Graefe.[62]

The same phenomenon of "faulty nasal projection" may be demonstrated easily in any normal naive subject merely by diffusing the image in one eye by a diffusion lens or, even better, simply by closing the lids while the fellow eye is completely occluded (no light whatever reaches even the closed lids).

Under these conditions the subject will correctly localize the light in the temporal field, but usually he will mislocalize a light placed in the nasal field and describe it "as if" it were coming from the temporal field (Figure 21). Or, if localization of the light in the nasal field is correct, it will be much less definite than in the temporal field. This represents a significant difference in subjective localization between the nasal and temporal fields.

We have described a hemiretinal difference in amblyopia in an earlier publication.[41] Patients with congenital amblyopia as a result of a diffused image, will show sensitivity to light (photophobia) and a marked mislocalization; (a difference in the nasal-temporal fields of that eye), if they have only one eye. That is, they will localize a light in the temporal field correctly, and mislocalize a light in the nasal field. Indeed, it is most difficult to examine the fundus of such an eye in children, since they turn their nasal retinas to the light; that is, adduct the eye to "see" with the temporal field. It is as if the temporal retina (nasal field) is washed away. Not infrequently they fixate in this same way when attempting to read letters. Parenthetically, strabismic eccentric fixation is very much more commonly seen on the nasal-retina than on the temporal-retina (percentagewise). The superiority of the nasal-retina in this circumstance of congenital amblyopia is comparable to the situation in the adult who acquires a dense cataract, or who sees through the closed lid, where diffusion of the retinal image apparently precipitates a marked nasal-temporal difference.

In children with one amblyopic eye and one normal eye, it should not be surprising that the amblyopia and nasal-temporal difference alone might precipitate an esotropia. Thus, an amblyopic eye might "turn the nasal-retina to the world", since the temporal-retina is relatively washed away. It is not just an instance of persistance of the nonoverlapping temporal field conus.

The esotropia is especially evident in cases augmented by child-

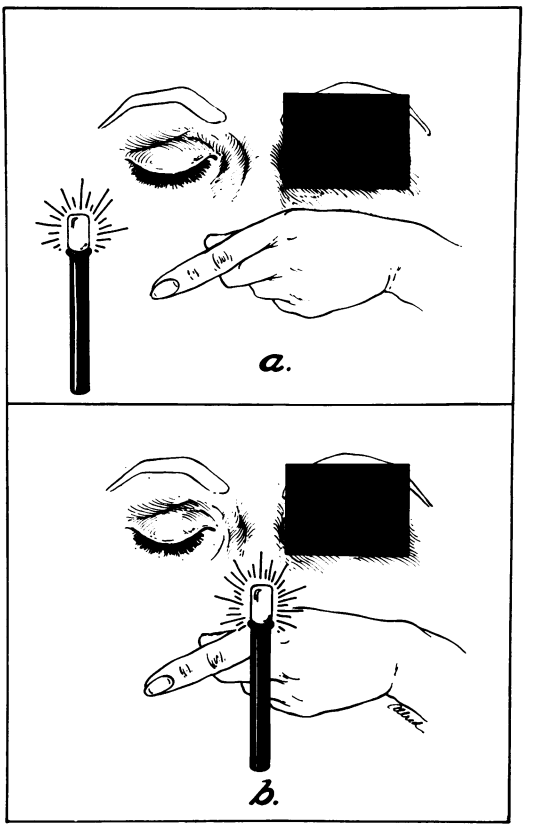


FIGURE 21

A normal individual with the left eye completely occluded, and the right eye occluded by lid closure. Under photopic conditions of normal room illumination, a small light will be localized correctly in the temporal field, even though it is diffusely perceived (*a*). When the light is placed in the nasal field of a naive observer (*b*), the light is usually perceived as if it is in the temporal field. This is a simulation of the well known old clinical term of "faulty nasal projection" observed in patients with a dense unilateral cataract, but without other intraocular pathology. Gross diffusion of the light source entering the eye by the closed lids precipitates a nasal-temporal perceptual field difference. Patients with congenitally grossly diffused retinal imagery in one pathologic eye may show this phenomenon without the diffusion induced by lid closure.

hood hyperopia or hypo-accommodation or both. Such patients must exert increased efforts of accommodation with resultant increased accommodative-convergence. Indeed they may remain esotropic if there is a secondary medial rectus contracture. Worth[47] at the end of the last century, stated: "When one eye only is blind its behaviour will depend, to a great extent, on the refraction of the seeing eye. If this is normal or myopic the blind eye will as a rule diverge. If it is markedly hypermetropic it will usually converge."

In this regard it is important to note that unilateral high myopia in infants does not ordinarily result in an esotropia. Rather it results in an intermittent exotropia or constant exotropia. This exception is worthy of note since it is evidence in support of Worth's early statement, with which we agree entirely.

A patient with a unilaterally diffused image will exhibit the nasal-temporal field differences to a light under photopic conditions in such a fashion that one may plot the physiological vertical raphe.[63] If there is a fairly well illuminated surround, a light in the nasal field may be so poorly localized that the patient is not entirely sure that he sees it. If he "sees", he may remark that he feels he is seeing it with his completely occluded good eye. If the light is moved across the physiological vertical raphe, there is a sudden and remarkable increase in the awareness of the presence of the light, and of its locale. It becomes "brighter". The light appears to be normal, including its localization, in the temporal field. This is characteristic of the entire nasal versus temporal fields, but, and this is especially important to note, we have found it to be most noticeable in the lower fields.

Do perfectly normal individuals have some subclinical evidence of this type of nasal-temporal inferior field difference to light under photopic conditions, and subsequently have an exaggeration of this difference under the clinical conditions noted above? It has long been known that many visual abnormalities in development are exaggerations or distortions of normal physiology.[64]

A similar difference between the nasal and temporal fields to a small light under photopic surround conditions can be demonstrated in normal people without diffusion. We have found that a naive subject, tested monocularly, commonly notices a difference in "brightness" of a light on either side of the vertical raphe in the inferior fields. An ordinary ophthalmoscope bulb as a test light in a normally illuminated room, testing in the inferior nasal versus temporal field,

is often sufficient to demonstrate the phenomenon. Similar differences are not noted with the ordinary visual field targets, either on a white background or dark background. The phenomenon appears to be easily demonstrated with the clinical test of a light under photopic conditions. We therefore suggest that this is a normal physiologic nasal-temporal difference which is exaggerated when the retinal image is grossly diffused. It is reminiscent of the exaggeration of normal physiologic nystagmus into grossly observable pathologic nystagmus as a developmental abnormality under certain conditions.

We choose to look upon this nasal-temporal retinal difference, elicited by overall diffusion in one eye, in terms of Bender's extinction phenomenon.[65] One might say that the "good half" of the retina inhibits the "poor half" and produces a further decrement. This is true in the half visual fields of one eye, and, as we shall see, is also true for one eye versus the fellow.

BINOCULAR INTERACTIONS (RIGHT VERSUS LEFT EYE)

One of the characteristics of patients with a diffused retinal image in one eye (whether it is from ocular pathology, amblyopia, or a large difference in dominance) is that they shut one eye in bright lights. The shutting of one eye in bright lights; that is, when going from a lesser to a greater general illumination, is very characteristic, indeed almost pathognomic, of intermittent exotropia. This subject has been previously reviewed by us.[41]

However, shutting one eye in bright lights is not confined to intermittent exotropia. It is not uncommonly seen in normal individuals in whom there is a significant difference in dominance between the two eyes. Individuals who have a significant diffusion of the retinal contours in one eye, consequent to optical aberrations of any sort, also shut one eye when going from a lesser to a greater illumination. Thus, it is not a characteristic confined to exodeviations. Rather, as we have previously pointed out, it is a characteristic of the diffusion.

Linksz[39] has used the term, "differential cerebral impressiveness". This term is most appropriate since it implies that it is the perceived difference between the two eyes that is of importance. This concept is more widely applicable to those instances of strabismus where suppression may augment, or be, the overall contour decrement ingredient.

Shutting one eye in bright lights may occur in any situation where there is a washing out of both macular and peripheral contours of one eye relative to its fellow. This type of "photophobia" may be

mistakingly diagnosed as residual inflammatory activity, long after the inflammatory process subsides in an injured eye, when the injury has resulted in a diffused retinal contour.

Why is it that shutting one eye in bright light is such a dominant feature of intermittent exotropia? We point out here that this is not a feature of constant alternating exotropia which characteristically has equal vision; an interesting exception that fits the hypothesis.

We have previously speculated that shutting one eye in bright lights is associated with a nasal-temporal difference in conjunction with the unilateral diffusion process.

Binocular interactions are of paramount importance in the sensory motor defects of strabismus. Only one example will be cited, which is pertinent in elucidating the further decrement of perception from the poorer eye under the binocular competition of a normal fellow eye. A patient with unilateral deep amblyopia may perceive an after image from that eye when this eye is tested alone (with the fellow good eye occluded). However, under the binocular circumstance of an after-image from each eye, that after-image perceived by the good eye completely washes away the percept of the after-image which "would have been seen" by the amblyotic eye. The "good" eye inhibits the "bad" eye.

We would speculate that when one eye's vision is diffused, the emerging superiority of the nasal retina (temporal field) which occurs as a monocular phenomenon, is perhaps further enhanced under the competition of binocular viewing conditions. Thus, when the normal eye is open, the fellow eye with diffused imagery has its temporal-retina (nasal field) further washed away, leaving a still further superiority of the nasal-retina (temporal field) which is now further enhanced. The consequences of this in strabismus we believe to be far reaching. Diffusion of the overall image in one eye where the nasal field of the diffused eye is relatively degraded from its own unilateral process, is still further degraded by the binocular interaction process. This type of competitive difference between the sensory inputs of the two eyes forms "the adequate retinal stimulus" for the optomotor changes which result in slow divergence (increasing exodeviation). We would like to suggest that this vergence is a creditable and genuine addition to the list of vergences elicited by retinal inputs. This is an inter-retinal diffusion-divergence. This retinal (optomotor) reflex, like all others, is active as long as the adequate stimulus persists. In this situation it alters the basic deviation in the direction of increasing exodeviation.

Secondary anatomic changes in the abducting group of muscles usually take place when the degree of exodeviation is marked. We have previously alluded to the overaction-contractures of both agonist and antagonist obliques (inferior oblique and superior oblique) and the lateral rectus in the same eye consequent to an exotropia.

Thus, there are both neural and anatomic factors which are symbiotically active in gradually increasing the basic exodeviation.

BACKGROUND FOR HYPOTHESIS

Adler[4] states, "It is well known in general physiology that light exerts tonus on the musculature of the body." Cogan[3] more specifically discusses the tonic effect of light on ocular muscles and believes this to be a special case of its effect on the general body musculature present in man as well as animals. He states that it can be shown "that illumination of one eye, or of homonymous halves of the retinas, results in an increase in extensor tone of the contralateral limbs and a decrease in tone of the homolateral limbs."

Elsewhere, Cogan[20] states "It is also known that the intensity of light impinging on the retina appears to have a special tonizing effect in such states as alternating hypertropia and latent nystagmus; little more than this is known."

Kestenbaum[9] believes that latent nystagmus is related to the character of the macular contours, and the character of fixation and perhaps attention. Further, he considers that altered fixation is the main determinant of the tonic changes in the fellow eye in such conditions as latent nystagmus and occlusion hypertropia.

This writer has observed a patient where light per se appeared to have a direct tonizing effect upon the oculorotary muscles. This patient was an adult male with right hypertropia and deep amblyopia consequent to a traumatic cataract at age two. There was overall gross diffusion of the light entering the eye by virtue of a dense secondary cataract membrane. The 20 prism diopters of hypertropia could be made to decrease to a degree proportional to the intensity of light made to enter the amblyoptic hypertropic eye. Increasing the light flux into the involved eye only caused it to rotate downward as far as the horizontal meridian, and to resume its hypertropic position when the light was removed. The fixing eye remained undisturbed throughout. This patient exhibited a direct light-tonus effect.

The possibility of an indirect effect of light upon oculorotary muscle tonus has received scant mention in the literature. The only specific reference found relative to the influence of light upon the basic devia-

tion is that of Brecher[66] who asserted that heterophoria (no mention of which type of phoria) was increased when light, especially diffused light, was increased in either the fixing eye or nonfixing eye. Gillott[67] states that when the illumination of one retinal image is decreased relative to the other there are significant distortions in subjective visual space, as may be seen in the Pulfrich phenomenon, the Trincker phenomenon and the Weale asymmetry effects in the Pulfrich phenomenon.

Thus, we see that light which enters the human eye has some known effects upon the visual space percept, and upon the oculorotary muscle tonus in certain clinical situations.

Let us briefly examine some phylogenetic points of interest. There are several steps in the phylogenetic development of phototropism. In lower animals a cutaneous light sense develops in the right and left halves of the body. The animal detects whether the two halves are equally or unequally illuminated; if unequal, it then turns in the appropriate direction.[68]

This primitive animal tropotaxis of orientation is a step up from the phototaxic reaction of purposive response to a difference of intensity. The latter in turn is a step above the phototropic response mediated by growth regulating hormones in lower plants and animals.

It should be noted that phototropisms and phototaxes "are invariably of biological utility, and it would appear that the essential and primary function of vision was the control of movement in order to attain an optimum environment as efficiently as possible . . . the state at which these motorial responses to light evolve beyond purely reflex acts below the level of consciousness and become endowed with awareness is impossible to conjecture."[68] "The difference, however, between the primitive response to light in plants and animals is merely a difference of method; the reaction is fundamentally the same, the transformation of a photochemical change into a motorial response."[68]

Much has been written about the interrelationship between the choroidal pigment of the eye, the light incident upon it, and the effect of light upon the pituitary-hypothalamic pigmentary regulating mechanism. Indeed, every clinician is aware of pigment changes in the skin consequent to certain eye diseases. It will suffice for our purpose here to relate this discussion to the phylogenetic history of the effect of light upon the body tonus, and from the primitive pigment "eye" spot on the skin, to the advanced pigment cup, the human eye.

We wish here to propose a very tenuous bridge by calling attention to some similarities between vision in the human with interretinal diffusion competition, and our visual ancestry. The nasal-retina superi-

ority (temporal field), which we have noted as an essential ingredient of our hypothesis, may reflect a normally latent atavistic type of vision which may become exaggerated under the condition of diffusion in one eye. The loss of the primitively essential peripheral retinal orientation, and also of macular orientation in such an eye (mislocalization, "faulty nasal projection") appears to be intriguingly related to the outward turning of this eye relative to its fellow eye (interretinal diffusion-divergence).

We wish to point out that washing out of the nasal field of the diffused eye causes loss of binocularity of the overlapping part of the visual fields, since only the temporal field remains available as a functional localizing perceptual area. It is "as if"* the diffused eye abducts, and looks to the temporal field where light is correctly perceived.

There is an interesting statement by Posner[69] in this regard:

"Since binocular vision arose at a fairly late state of phylogenetic development, it may be assumed that even in man there are present potential tonus regulating centers which control the postural muscle tonus of one eye without reference to the other. Superimposed upon these are the centers controlling binocular innervation, both tonic and kinetic varieties. In the normal exercise of binocular vision the monocular influences are held in abeyance, being completely inhibited by the higher centers."

Riesen,[70] in animal experiments, has pointed out the fundamental difference between the visual circumstance induced by diffusion of one image, and that induced by total occlusion. The motor learning ability of cats with one eye's image diffused was considerably poorer than of cats with one eye completely occluded. Further, transfer tests to the fellow eye showed immediate correct responses when one eye was occluded, but not when one eye was diffused.

One might speculate as to the possible relationship of these findings to the documented motor learning difficulty in children with unilateral congenital cataracts; that is, with a diffused image in one eye relative to the other eye.

Riesen (personal communication) remarked that in some further experiments the majority of the animals with one eye diffused became exotropic!

Diffusion of the imagery of one eye relative to the other is a unique sensory circumstance which has far reaching developmental consequences in the visual system. And, we suggest, it has far reaching impli-

*Vaihinger's "Philosophy of the 'As IF' as quoted by Crutis and Greenslet from Havelock Ellis' 'Dance of Life' ".

cations in strabismus by virtue of the retinally elicited (optomotor) divergence which persists as long as this adequate stimulus persists, even in visually mature adults. There is a real difference between diffusion of light, and complete occlusion or blindness. One recalls Milton's words, "No light, but rather darkness visible."

PRACTICAL CLINICAL APPLICATIONS

Let us now attempt to apply some of these concepts to the clinical course of events in intermittent exotropia. The intermittent exotrope characteristically shuts one eye in bright light probably to avoid the many perceptual visual field changes which take place in bright light diffusion. The bright light triggers or augments the latent temporal hemiretinal suppression which indeed characterizes intermittent exotropia. The superiority of the nasal-retina, which is thus precipitated in the usually deviating eye, is further augmented by the binocular interretinal interaction in the dazzled retinas. All of these factors reduce the normal overlap of the visual fields, distorting visual space, unlocking both macular and peripheral fusion, and activating interretinal diffusion-divergence.

This neural divergence influence may be further augmented by secondary anatomic changes in the abducting group of muscles, as previously described. The divergence may be not only initiated, but maintained by the interretinal diffusion-competition. We believe that this type of divergence represents the basis for the gradually increasing degree of exodeviation so frequently observed in this entity.

In the manifestly exotropic state the patient is relieved of conflict between the two eyes, since in this status he suppresses the temporal-retina of the deviating eye and loses the overlap of his visual fields.

The maximum disengagement of the two eyes occurs in patients with alternating constant exotropia and equal vision. We have previously called attention to the fact that such a patient does not shut one eye in bright lights, and this is an important exception. Patients with alternating exotropia and equal vision have almost dissociated eyes, with non-overlapping visual fields. In our experience the after-image test in such patients frequently reveals true alternation rather than simultaneity of percept. We believe this to be the reason why the determination of retinal correspondence in such patients is so frustrating, and fraught with confusion, as is reflected in the variable and confused reports in the literature. Indeed, retinal correspondence cannot be determined if there is alternation, rather than simultaneity of vision, since correspondence loses its very meaning in this circumstance.

The secondary overaction-contracture of the abducting group of muscles is nowhere better seen than in alternating constant exotropia (where the exodeviation is characteristically of large degree). When either eye fixates in the primary position it does so with greater effort of the medial rectus and its allies, pulling against the overacting-contracted abducting group of muscles. Hering's Law dictates an increased activity of the yoke abducting muscles of the fellow eye, which in turn further increases the exotropia. Alternation of fixation increases this binocular overaction-contracture of the abducting group in each eye, further augmented by the fact that a significant degree of adduction is seldom necessary, or indeed attained. Hence, patients with equal vision and alternating constant exotropia usually exhibit a very large angle of deviation, and enjoy comfortable vision, albeit almost a completely dissociated monocular status. There is no reason to shut one eye in bright lights!

The author's attempt to relate the hypothesis and its physiologic basis to the explanation of all of the details and nuances of exo-deviations is admittedly not entirely satisfying in all facets. The real relevance of any hypothesis is its predictive value in real life situations. Therefore, let us attempt to do this.

Clinicians are well aware of the admonition that an esodeviation in an infant is likely to decrease if the following factors are present: (1) high hyperopia (even though fully corrected), (2) amblyopia, and (3) visual immaturity. Indeed, not only may the esotropia diminish, but exotropia of an increasing amount may result, without benefit of surgery. This is an actual change in the basic deviation in an exo direction. Secondary consecutive nonsurgical exotropia usually does not occur unless some combination of these three factors is present. The writer suggests that the reason for this is the overall blur (diffused perceived image) in the amblyopic eye as a result of the defined macular amblyopia, and what we will term peripheral amblyopia; (i.e., the degraded peripheral retinal contours), as result of the optical aberrations present in corrected high hyperopia. Some amelioration of this condition may be obtained with contact lenses because of the increased clarity of peripheral imagery. The immediate measurable benefit is an increase in fusional amplitudes because of improved peripheral retinal imagery.

In the small group of patients with these three ingredients who do become secondarily exotropic, there may be transient diplopia as the esodeviation gradually becomes an exodeviation. We suggest that this activity and increasing divergence may be predicted because of

the adequate stimulus of macular and peripheral blur in one eye: interretinal diffusion-divergence. The diplopia may be transient as the esodeviation turns to exodeviation, and may be almost unnoticed if it occurs in the very young, since readaptation to the new hemiretinal suppression is not difficult. In adults the diplopia may be more noticeable.

What if both eyes are equally diffused, so that the retinal images (both maculas and peripheries) in both eyes are considerably degraded, but there is not complete blindness? With equal blurredness, as with equal normal clarity, there is no competition or difference between the two eyes. Thus, there is not the adequate stimulus situation which our hypothesis describes, and hence no activated divergence. A clinical example is to be found in uncorrected bilateral hyperopia of very high degree (8–10 diopters) in young otherwise normal (unoperated) individuals. Such individuals do not ordinarily exhibit a strabismus.

Additionally, there is a statistically significant increase in incidence of exophoria in patients with anisomyopia (uncorrected). Isomyopia is not so correlated. Uncorrected anisomyopia is, of course, a situation wherein the overall imagery in one eye is blurred relative to its fellow eye, especially at distance fixation. We have already suggested that other psychophysical parameters of the visual scene at very remote distances may be germane (as in remote-divergence), such as the increased amount of illumination ordinarily present in the very far distance fixation coupled with the frequent paucity of structured visual surround. All of these "distance factors" may augment an uncorrected refractive error image difference between the two eyes. It would be our feeling that the "differential cerebral impressiveness" in such a situation may be the genesis of the increased exotonus in these patients.

It is of practical clinical importance to recognize that unilateral "photophobia" is an expected consequence of an interretinal diffusion difference. And further, that shutting of one eye in bright lights, although very characteristic of intermittent exotropia, may occur in other individuals wherein there is a significant difference in dominance between the two eyes, or wherever there is overall diffusion of the imagery in one eye.

We are presently conducting a practical clinical test of this hypothesis, by purposely inducing an overall diffusion of the imagery in one eye relative to the other eye, in patients with small or moderate degrees of esodeviation (avoiding patients with contractured medial

rectus) in an attempt to shift the basic deviation in an exo direction by simulating the adequate stimulus situation for interretinal diffusion-divergence.

CONCLUSIONS

(1) Nonretinally derived basic ocular tonus is inately organized as a version system, which retinal fixation can control.

(2) Retinally derived convergence is associated with visual co-ordination at near.

(3) A fusion vergence is a relatively simple coupling which may employ the above two mechanisms.

(4) Nasal versus temporal retinal sensory differences account for some differences in horizontal motor vergences.

(5) Interretinally derived divergence is associated with visual competition at distance.

(6) The basic deviation, or the clinical starting point for ocular deviations, is determined by the non-retinally derived ocular tonus, when stabilized by monocular distance fixation of the dominant eye alone, completely devoid of other retinal stimuli (fusion, accommodation, etc.)

(7) Alteration in the general level of non-retinal ocular tonus may elicit mechanically disjunctive horizontal eye movements.

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