Etiologies of functional horizontal strabismic deviations

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Abstract
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Degree Type
Thesis

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ETIOLOGIES OF FUNCTIONAL HORIZONTAL STRABISMIC DEVIATIONS

by

ALISON D. FUJISAKI

A thesis submitted to the faculty of the
College of Optometry
Pacific University
Forest Grove, Oregon
for the degree of
Doctor of Optometry
May, 1994

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SIGNATURES

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The etiologies of strabismus are numerous and widely varied ranging from accommodative to reflexologic to neurologic. Although many of the proposed theories are plausible explanations, some are more powerful than others. There is not one proposal in particular, that can account for all types of strabismus. In fact, there may be several, depending upon the condition and individual. Future studies and research are indicated which will better enable us to understand and treat each condition appropriately.
Alison D. Fujisaki was born and raised in Chicago, Illinois. She received her Bachelor's Degree in Biology from DePaul University in Chicago in May, 1987. She will receive the Doctor of Optometry Degree from Pacific University, College of Optometry in May, 1994. Her academic and professional affiliations include the American Optometric Student Association, American Academy of Optometry, and Beta Sigma Kappa International Honor Society. Future plans for Alison include an associateship in a general practice with emphasis on vision training and contact lenses. In her leisure, Alison enjoys cooking, fine art, music, and reading.
ACKNOWLEDGEMENTS

First, I'd like to thank Marina for suggesting the title of this paper and getting me started on the research.

I'd also like to thank Stan for his support and encouragement towards the end of this project.

Finally, thanks to Dr. Samson for his suggestions, patience, and input.
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INTRODUCTION
The purpose of this paper is an attempt to review the major theories proposed which explain the causes of horizontal functional deviations. Functional being defined here as those cases not attributed to any specific physical defect, pathology, or syndrome. Hence, anatomical and mechanical theories, in addition to strabismus secondary to systemic and ocular syndromes will not be covered in the following text.

For years, numerous theories have been proposed in an attempt to explain the etiologies of strabismus. "Until the late 18th century, understanding of all strabismus remained rudimentary. Variously considered the cause of an angry God, the effect of malposition in the cradle, or the work of imaginary vices." (13) Later, theories were proposed that seemed more reasonable. In 1903, Worth attributed such ocular deviations to a "defect in the fusion faculty." (7) Subsequent studies, however, have demonstrated the presence of fusion following surgical ocular alignment (41, 43, 45) This was prompted on the basis of Chavasse's theory which involved the development of fusion through use of the mechanism itself. Prism compensation is another method through which fusion can be attained.

Genetics, while not the most consistent certainly plays a role if only a minor one. Evidence has suggested a multifactorial mode of inheritance (20) based upon monozygotic twin studies (22, 23) Identical twins demonstrating an incidence four times greater than dizygotic siblings. (22) Information regarding the heritability of eso- or exodeviations may serve as an integral tool in determining surgical outcomes (6).
Still another theory introduced by Zeeman and continued by Keiner involves a maldevelopment of the optomotor reflexes necessary to sustain ocular alignment. (4) Research conducted by Mitsui, et al, supports this proposal, however further studies need to be executed to lend credence.

**ACCOMMODATIVE ESOTROPIA**

The most common type of ocular deviation in children and infants is esotropia. (16) Several reasons can account for this nasalward tendency and one of the first theories proposed was by Donders in 1864. He discovered the link that exists between accommodation and convergence. Thus, in looking from a distant to a near object, one must clear the image through accommodation, simultaneously converging the eyes nasalward. Hyperopes, in particular, to maintain clarity, must accommodate for the object's distance as well as their refractive error. This results in excessive convergence and, in certain cases, esotropia may ensue. This is referred to as accommodative esotropia. Characteristically, it manifests at approximately 2-3 years of age, sometimes later with the deviation being greater at near than at far. It develops gradually through an uncorrected hypermetropia coupled with inadequate divergence reserves.

Donders' proposal is quite a plausible explanation for the incidence of esotropia, particularly in children. The fact that most infants are born with an average of 2-3 diopters of hyperopia lends further credence to his theory. But, it's not without wrinkles. Critics have questioned the mismatch between refractive error and magnitude of deviation. For example, emmetropes and myopes presenting with esotropia. What about those high hyperopes who don't squint at all? The reason for the discrepancy lies with each patient's AC/A ratio.
Those individuals exhibiting a high versus low AC/A will demonstrate an extreme change in convergence upon small amounts of accommodative stimuli, thus accounting for the discrepancy between refractive error and deviation.

A study was conducted by von Noorden, et al, (30) to investigate orthotropic uncorrected hyperopes (Group A) versus corrected hyperopes who deviated without their spectacles (Group B). The nine subjects in Group A ranged in age from 5-16 years, all with a negative history of any types of strabismus. Their refractive errors extended from +4.25 - +15.00 diopters OD and +4.00 - +15.00 OS, all attaining visual acuities of 20/20 or better in the distance. The second group consisted of 30 individuals with refractive accommodative esotropia corrected with spectacles. Age ranged from 6-13 years and refractive error from +3.50 - +9.25 OD and +3.75 - +9.50 OS. All patients attained VAs of 20/40 or better.

Each patient was evaluated through a complete visual examination and cycloplegic and an AC/A was determined. The results were such that those subjects in Group A were found to have an AC/A below normal values of 2.76-4.50. von Noorden concluded that "an intrinsically low AC/A ratio may protect certain patients with uncorrected hypermetropia from developing esotropia or esophoria." In contrast, Group B was shown to have ratios within expected values.

Although individuals who exhibit high amounts of hyperopia must accommodate an excessive amount to obtain a clear image, if their convergence response to accommodation is very small, they will not manifest an esotropia. If, on the other hand, their ratios are normal or high, they will respond by increasing convergence resulting in an esotropia.

Nonrefractive accommodative esotropia results when an individual's accommodative convergence response exceeds normal values of 4-6 prism diopters/diopter and is independent of refractive error. "The hypermetropia factor
alone must be large to incite esotropia, whereas a small amount will precipitate the esotropia if it occurs with an abnormal AC/A." (36) As a rule of thumb, those patients with a high AC/A have an average of +2.75 D of hyperopia and those with a normal ratio, an average of +4.75 D. (26) This evidence provides explanation for the reason why not all hyperopes become esotropic and also why certain emmetropes as well as myopes develop esotropia.

**INFANTILE ESOTROPIA**

Essential infantile esotropia is another category that should be addressed. Originally referred to as congenital esotropia, it is now accepted as infantile simply because most cases develop at approximately six months of age or less, not at birth. It is characterized by a constant large angle deviation of 30 prism diopters or more, sometimes associated with an asymmetric optokinetic nystagmus, defective abduction, DVD, or dysfunction of the obliques with an otherwise normal functioning central nervous system. (24) Current literature suggests a prevalence rate of .1%, it being the most common type of esotropia. (40)

The etiology of essential infantile esotropia to date is unknown, but many have provided us with their insight and speculations. Most noteworthy is Claud Worth, who firmly believed that strabismus is due to an inborn defect in the ability to fuse, this ability being either altogether absent or developmentally delayed. Worth refers to this skill as "fusion faculty". "When the fusion faculty has begun to develop, the instinctive tendency to blend the images formed in the two eyes--the desire for binocular vision, as it is called--will keep the eyes "straight". When the fusion faculty is fairly well developed, neither hypermetropia, nor anisometropia, nor heterophoria can cause squint."(7) Without fusion, "the eyes
are in a state of unstable equilibrium ready to squint either inwards or outwards on slight provocation." (7)

A study was conducted by Pratt-Johnson (5) in which a total of 148 patients all with "congenital" esotropia were aligned surgically within 10 prism diopters of orthophoria before age 2. Of the 148 subjects that participated, only 118 remained aligned for at least one year. None obtained central fusion, however 53 gained peripheral fusion. This evidence supports Worth's hypothesis and/or the idea that surgery alone may be the proper cure. "It is possible that congenital esotropia is a syndrome caused by an innate defect of fusion which, if complete, prevents the development of fusion and, if incomplete, may allow the development of peripheral fusion only." (5)

**ESOTROPIA ASSOCIATED WITH MOTION PROCESSING**

Tychsen and Lisberger (16) suggest that a maldevelopment of visual motion processing which apparently develops alongside binocularity may be responsible for esotropia. They believe that if such a defect exists, the nasally-biased motion processing commonly found in infants will predominate. (5,16) They evaluated 7 subjects ranging in age from 22-38 years of age who underwent surgery by age 3 for ocular alignment caused by infantile strabismus. The purpose of the study was to monitor and compare each patient's eye movements against normal findings.

Eye movements were recorded through a magnetic search coil placed on the surface of the eye. Pursuits were tested monocularly and were plotted with eye acceleration (degrees/second) against target position. In normal participants, equal eye acceleration was found between nasally and temporally directed movement. Additionally, acceleration increased when the target was displaced laterally toward versus away from the fixation point. "Thus, leftward eye
acceleration was largest when the target started at 3 degrees right, and rightward eye acceleration was largest when the target started at 3 degrees left." (16) In contrast, when strabismus subjects were evaluated, very different results were obtained. For example, a bias toward nasally-directed movement was found. "Eye acceleration was larger for rightward pursuit when viewing through the left eye and for leftward pursuit when viewing through the right eye." (16) Temporal movements were much weaker. Also, upon attempted fixation of a steady, straight-ahead target, a nasally-directed (slow phase) nystagmus was noted with an equal conjunctive response in the occluded eye. The nystagmus seemed to depend on the velocity of the target, it being larger for slowly-moving as opposed to higher velocity targets. Strabismus subjects also perceptually judged nasally-directed targets to move faster than those directed temporally.

From the data gathered, all of the strabismic subjects studied demonstrated abnormal motion processing ability with a nasal bias. These researchers suggest that the "pathways mediating motion processing are immature at birth and that they develop normally only if the animal experiences binocular correspondence throughout early life." (16) In conclusion, they believe that the normal nasallyward bias is naturally opposed by a temporal force in an effort to achieve ocular alignment. If this drive is somehow prevented, through, for example, maldevelopment of temporally directed motion processing, the tendency of the eyes to position inward will take precedence. "The normal nasally directed bias of the infant motor system favors the development of esotropia and could explain the fact that esotropia is at least 30X more common than exotropia in cases of strabismus with onset in infancy." (16) Tychsen, et al, however, are not conclusive as to the cause and effect of strabismus and such poorly developed motion processing abilities. Further study and research is necessary.
In situations where esotropia manifests very early in life, surgical alignment has been recommended before age two in an effort to obtain maximum visual benefit. "If esotropia is caused by an inborn defect of the fusion faculty, no treatment, no matter how early in life will restore normalcy. If, on the other hand, no such defect exists, a cure could be accomplished by aligning the eyes as early as possible." (40) In a study done by Ing (45) to determine the optimum age to operate, 106 infants participated, all of whom were diagnosed with infantile esotropia and then aligned within 10 prism diopters of orthotropia at various stages in their lives. Subsequently, motor and sensory tests were performed approximately 7 months after surgery. Included among the testing battery were the cover test, Bagolini striated lenses, Worth 4 lights, and the Titmus vectograph stereotest.

Eighty nine of 106 subjects demonstrated both fusion and/or stereopsis. This evidence refutes the idea of a congenital absence of the fusion faculty. Similarly, Morris, et al. (46) followed 24 subjects ranging in age from 8-36 years who also were diagnosed with infantile esotropia. Of the 24, 12 were not aligned surgically at any time previously while the remainder were. However, alignment within 8 prism diopters was never achieved. Preoperatively, all subjects were unable to demonstrate fusion with the red filter glass test, Worth 4-dot, or Titmus stereotest. Postoperatively, all participants were aligned within 8 prism diopters of orthotropia and demonstrated peripheral fusion using the Worth 4-dot test. In addition, 50% achieved steropsis of 200 arcseconds or better.

Again, this evidence refutes Worth's hypothesis and seems to favor that of Chavasse who believed fusion developed through use and conditioned reflexes. He believed that any interference with the development of such binocular reflexes will result in ocular deviation, and, if not rectified within the plastic stages of
development will not be correctable with any form of therapy. "Indeed, it was Chavasse's thinking that triggered the era of early surgery that began with Costenbader" in 1934. (10)

REFLEXOLOGIC THEORIES

Another reflexologic theory proposed by Zeeman and carried out by his student, Keiner, held that the gradual development of optomotor reflexes in infancy are responsible for ocular alignment. "This directing and coupling process, initiated by the optomotor reflexes, leads eventually to the full motor and sensory cooperation of the eyes; from the state of dissociation in the newborn infant, the eyes change to a state of association." (2) They believed that any disturbance or abnormality of such reflexes was responsible for any manifest deviations.

The development of the optomotor reflexes begins immediately postnatally when light acts as the initial stimulus. They are gradually integrated with existing sensorimotor reflexes, vestibular and proprioceptive, and ultimately lead to full coordination of both eyes. Zeeman spoke of a binocular reflex superimposed onto the vestibular reflex; a monocular reflex associated with the proprioceptive pathway and a convergence reflex associated with the monocular reflex. He believed that if all of these reflexes developed in a concerted, methodical fashion, binocular vision would follow. If, on the other hand, a normal foundation is not established, some form of strabismus would result.

It was concluded by Keiner that "all children are born with a potentiality to squint and an almost total dissociation of the two eyes." (4) He also believed that the concept of congenital strabismus is nonexistent since in the absence of light, strabismus simply cannot occur. In a study based on 984 cases of squint in children under age 2, the frequency of occurrence was highest during the first 6 months of life, gradually dropping off by 1 year, and more rapidly by age 2. Between
the ages of 6 months and 1.5 years, a directing and coupling process is actively functioning as a basis for orthophorization. (4)

This process of orthophorization initially begins in the periphery and gradually progresses to the central zone of fusion. Keiner theorized that the manifest deviations seen in infants at about 6 months of age represented a temporary imbalance in the developing reflexes. If, by internal or external influences, an equilibrium between the two eyes is prevented, constant or alternating strabismus will result and normal binocularity will be absent. Esotropia, then, can be accounted for by a dominance of monocular adductive reflexes over abductive and conjunctive movements, and vice versa for exotropia.

Although Keiner and Zeeman did not provide us with solid evidence for their reflexologic theory, subsequent investigators have made an effort to support their proposal. In particular, Mitsui, et al, researched 214 cases of constant and intermittent exodeviations. Specifically, the "magician's forceps phenomenon" discovered by Mitsui was studied. By definition, the magician's phenomenon is said to occur when a "slight adductive force is applied by forceps to the straight or master eye in exotropia, the deviated or slave eye assumes the straight position." (53)

The procedure and criteria of results were such that a slight adductive force was applied to the straight eye by forceps after 2% procaine was applied to the subconjunctiva of the master eye. If the slave eye did not respond after 20 attempts of forced adduction, the forceps test was regarded as negative. If the slave eye assumed the straight position under the forceps test, the master eye was released from the forceps. If the slave eye then promptly returned to the exodeviation position, the forceps test was regarded as positive." (53)

An EOG was used to record eye movements and an oscillator to apply repeated forced ductions of 1-9 Hz to the straight eye of each participant. The
results were such that 85% of the 214 cases examined exhibited the magician's forceps phenomenon. Even if the deviating eye had no sight, the phenomenon occurred, however, if attempted in the dark the same results rarely were elicited, suggesting the need for visual input. "In all probability the phenomenon is a result of interaction between proprioceptive and visual input, where the proprioceptive impulse chiefly constitutes the signal and the visual input restricts the threshold of the reflex pathway." (53)

From the results of this study, it is still questionable whether the magician's phenomenon is due to a reflex excitation. Both intermittent and constant exodeviations decrease under dark conditions. Also, through anesthesia of the master eye, deviation of the slave eye disappears. "All of these findings can be explained if it is assumed that in cases of exodeviation an abnormal proprioceptive impulse persists that originates from the master eye to cause abnormal contraction of the slave eye lateral rectus, and that the forceps phenomenon is the result of cancellation of this abnormal standing impulse of proprioception." (53)

Although Mitusui, et al, emphasized exodeviations, some investigation was also devoted to esodeviations. Many of the findings were similar to the exotropia with some exception. In studies to observe the reaction to spot-lighting in which a small circle of light is projected onto a screen and the subject is asked to track its movement, many differences were noted. In exodeviations, the reaction to spot-lighting is slow and seen in the opposite eye, whereas in esodeviations, just the reverse is noted, i.e., the reaction is quick and in the same eye that is stimulated. "These findings may show that esodeviations are the result of a kind of light-motor reflex from the retina to the medical rectus muscle of the same eye through the brain as suggested by Keiner, and that exodeviations are the result of a proprioceptive reflex from the master eye to the lateral rectus muscle of the slave eye, where the light stimulus on the master eye acts like a bias for a switching
transistor, placed in this reflex pathway. . . " (28) To support this theory, it was observed that in the dark, exos had unilateral deviation while esos exhibited symmetrical deviation. The question, however, remains why esotropia manifests in the dark if, in fact, it is a light-motor reflex. Further investigation must be conducted to lend credence.

**GENETICS**

Still another etiology of strabismus lies in the area of genetics. From the early days of Gregor Mendel, the father of modern day genetics, it was discovered that similar traits, such as hair and eye color commonly run in families. Hence, blonde-haired individuals often give way to blonde-haired offspring, so why not the same principle for strabismics?

It is currently believed that a multifactorial mode (20) of transmission is responsible for this particular trait, and, depending upon the degree of penetrance, manifestation of an ocular deviation will result. In a study by Richter (22), the incidence of strabismus in monozygotic twins is nearly 4x greater than in dizygotics. The factor of four between concordance of monozygotic and dizygotic twins is consistent proof of a multifactorial mode of inheritance." (22)

Considering that inheritance does play a role in the development of strabismus, what is its significance? The importance seems to lie in the treatment of the deviation and may act as a predictive tool in surgical management. "The behavior of the squint in one member of the family may serve as a fair guide to the response of another member to similar conditions, if the types of strabismus are the same in the two members." (6) Hence, the magnitude of the deviation and response of one individual may determine the amount of correction required in another member of the same family.
EXOTROPIA

Exotropia like esotropia has many proposed etiologies. This type of deviation, perhaps, is more difficult to explain and understand. It seems that most theories point to neurological factors. For example, one proposal suggests that the tendency to seek one's natural position of rest is one of divergence "as evidenced by the position of the eyes in death, sleep, and during anesthesia." (1) Other theories point to convergence and divergence mechanisms, some advocating a diminution of the former while others support an active process of the latter.

Divergent types of deviations frequently are divided into intermittent and constant classifications. Divergence excess and convergence insufficiency are headed with the former while congenital, and anatomical are placed under the latter.

DIVERGENCE EXCESS EXOTROPIA

Several theories have been postulated to explain the genesis of divergence excess exotropia. Generally, it is characterized by a deviation that is greater in the distance than at near with normal stereopsis at 40cm, normal visual acuities since alternate stimulation of both eyes is possible, a general absence of amblyopia, and a chief complaint that is of cosmetic concern.

One of the more interesting theories is purely based upon functional reward. "The divergence excess type of intermittent strabismus seems to be a compromise between using binocular
stereoscopic vision when it is an advantage and deviating one of the eyes with panoramic viewing when stereopsis is not needed." (3)

Since stereopsis is only utilized within a certain viewing distance, patients who have divergence excess exotropia will maintain binocularity at near, fully able to judge depth of three-dimensional objects as a normal individual can. However, when viewing two-dimensional objects such as a book or newsprint, the need for stereopsis is no longer a requirement and as such the eyes will deviate. "Since stereopsis is more important at nearpoint and is probably associated with detail information processing and attention mechanisms, auditory stimuli, touch, accommodation, changes in visual stimuli, or movement may serve to trigger the restoration of binocularity. On the other hand, when no definitive identification process is needed, the eyes deviate and passively scan for information from a larger binocular field of view." (3)

Hence, in the distance, divergence excess exotropes will have greater angles of deviation in comparison to near distances where fine detail and concentration is necessary. Additionally, in the distance when the eye deviates, the individual theoretically will enjoy a wider field of view setting him at a greater advantage. "Excluding cosmetics, the divergence excess probably represents the best of both worlds—an expanded binocular field and good stereoscopic judgment." (3)

Another theory proposed to explain this type of exotropia involves the accommodative mechanism. The average AC/A value is approximately 15/1. (32) Such high values allow the deviation at near to be reduced significantly in comparison to the distance angle.
However, a large variation of ratios have been found among many different researchers ranging from normal to high values. For example, von Noorden found a range of 3.3 to 9.0 (57), Brown, a ratio of about 13.0 or greater (58), and Moore, et al, found one of 4.7 (59).

It’s difficult to obtain an accurate AC/A value simply because each individual will accommodate to various degrees based upon the stimulus given, the effect of proximal convergence, the method used, and the adjustment needed for individuals with binocular vision abnormalities. In an attempt to determine AC/A ratios under different conditions, Cooper, et al, (11) studied four divergence excess intermittent exotropia patients.

The subjects ranged in age from 20-26 years all exhibiting a deviation at near of 10 prism diopters less than at distance. For all participants, visual acuities were 20/20 OU and stereopsis of 40 arcseconds was attained.

The four methods utilized were as follows: 1) Distance/Near Cover Test where each subject was neutralized using prism bars at 6m using a 20/25 Snellen letter and at 40cm using J1 print size. 2) Gradient Test at Near (40cm) was utilized where the subject viewed J1 print and lenses of +2.00, +1.00, plano, -1.00, and -2.00 were interposed before both eyes, their horizontal deviations being neutralized with prism. 3) Gradient Test in the Synoptophore where the subject viewed a superimposed target of 1 degree while lenses of plano, -1.00, -2.00, and -3.00 were used. Each time the angle was neutralized based on subjective and objective responses. 4) Phorometric Method involved use of a phoroptor to measure phorias
at 6m and 40cm while the subject viewed a 20/25 target and J1
print size respectively.

Following the above testing, a standard optometer was used to
measure dynamic accommodative changes of the left fixating eye.
All testing was done under dark conditions, and accommodative
response, vergence responses, as well as stimulus were recorded
and used to calculate AC/A ratios. In addition, after 45 minutes of
monocular occlusion, subjects were retested so as to determine any
changes postocclusion.

In conclusion, the findings indicated relatively normal AC/A
ratios among the subjects examined using the near gradient and
objective tests. In particular, the evidence supports the fact that
proximal as well as fusional vergence play important roles in the
near fusion response as opposed to the accommodative component. In
the clinical setting, most divergence excess intermittent exotropes
present with very high AC/A ratios, however, many factors tend to
compromise the accuracy of these values. Specifically, fusional and
proximal components as well as sensorial aspects.

"Over 75% of all patients with divergence excess exhibit a
dramatic increase in the near deviation following a short period of
monocular occlusion." (60) This results in a decreased AC/A ratios
due to an increased near deviation coinciding with the values
obtained by Cooper, et al. If accommodation plays a key role, how
does occlusion alter the stimulus to accommodation? It is believed
that this postocclusion change is a direct manifestation of a
fusional convergence after-effect resulting from sustained fusional
convergence required to bring the eyes from a divergence position to one of binocular alignment on the object of interest." (11)

Further experimentation with near gradient ratios led to the conclusion that proximal convergence also plays a significant role. It was found that "near gradient stimulus AC/A ratios were typically much reduced relative to the distance/near values." (11). The accommodative component and thus the AC/A ratio cannot account for the deviation.

ACTIVE DIVERGENCE

Another theory proposed by Jampolsky concerned active divergence, hence, exotropia. Basically, the idea being one of increased innervation to the lateral rectus muscles. Evidence to support this proposal stemmed from research with electromyography. Tamler, et al, quantitatively evaluated the activity using electromyographic equipment, and further defined active divergence as "divergence beyond the fusion-free position, as in response to base-in prisms while maintaining fusion with both lateral rectus muscles showing a simultaneous increase in electrical activity" and also through "recovery of fusion by an esophoric or intermittent esotrope from the fusion-free position" with the same increased activity as demonstrated above. "In these instances the divergence function is working either to maintain fusion against odds or to restore fusion." (61) These researchers have thus provided evidence to support the theory of active divergence.
Research conducted by Breinin also revealed increased innervation to the lateral rectus muscles of patients exhibiting intermittent exotropia. "A remarkable finding in all instances was the fact that only the deviating eye showed increased innervation of the lateral rectus." (60) This suggested an inconsistency with Hering's Law of Innervation, however, this phenomenon can be explained by the nature of convergence upon a specific fixation point.

Hering explained that "convergence on a fixation point which lies on the fixation axis of one eye in the primary position (in contrast to midline convergence) can be analyzed into two movements. There is convergence of both eyes to the midline and then a version to the side of the fixation object. This is synthesized into a single movement such that the eye on the side of the fixation object does not move because its convergence is neutralized by the version. The other eye must execute twice as great a movement to maintain its fixation, and this movement is compounded of convergence augmented by version." (60)

Electromyographically, the above has been demonstrated and validated by repeated trials. In primary position, both medical and lateral recti are active, however, with a shift in gaze to the right or left, an increase or decrease in innervation to the lateral rectus or medial rectus is observed, respectively. With the fixation point placed at the fixation axis of the right eye, for instance, the objective is to "restore the exact balance and level of innervation which existed prior to the convergence, and which it habitually exhibits in the primary position." Hence, the left eye will exhibit an
increase in activity of the medical rectus with a simultaneous decrease in the lateral rectus activity.

The above evidence can, thus, be applied to tropias. For example, in exodeviations, one eye deviates temporally while the other eye maintains proper fixation. The explanation is that the yoked muscles receive equal innervation, but the fixating eye executes an opposite version, hence, maintaining fixation while the other eye receives double the amount of innervation.

The opposite of the above theory is an abnormality in the convergence mechanism. This theory was advocated by Adler who firmly believed that "all squints are due fundamentally to an abnormal convergence innervation," (9) excluding those due to paralysis. Although, the location of the center of convergence is unknown, he considered a few possible areas, for example, the ocular muscles, vestibular apparatus, cortical as well as subcortical centers. The most probable location is in the cortex. The mechanism through which such a center is activated or withdrawn is not known and, thus, further experimentation and research must be conducted.

**CONCLUSION**

As evidenced by the above text, the etiologies of strabismus are numerous and widely varied. From the available literature, however, it seems that accommodative esotropia, as opposed to exotropia, can be more readily accounted for through accommodation and refractive state. This particular theory, proposed originally by Donders, is the most plausible explanation and "is solidly based on
physiologic and clinical facts. It has led to the important practical conclusion that in every patient with heterotropia the refractive error has to be determined and fully corrected before other therapeutic steps may be taken." (2)

Because Donders' theory cannot explain each and every case of strabismus, other proposals have been formulated. They include nonrefractive accommodative esotropia in which the deviation can be attributed to the patient's AC/A ratio. Other theories point to defects in the fusion faculty or simply a congenital absence thereof.

Recent studies speculate that a maldevelopment of visual motion processing may be responsible for esodeviations since abnormal eye movements and a tendency toward nasally directed movement was found in strabismic vs. "normal" patients. This may account for the fact that esotropia is 30x more common than exodeviations in infants.

It was hypothesized by Keiner that all individuals are born with the potential to squint and he based his theory on the development of optomotor reflexes. He believed that a process integrating sensorimotor, vestibular, and proprioceptive reflexes gave way to binocular vision. If, however, by some disturbance or abnormality of the above process, strabismus would manifest.

Proposals that account for exodeviations also are numerous and varied. Most of the theories are based upon neurological factors or convergence and divergence mechanisms. One of the more interesting hypotheses assumes intermittent exotropia to be purely functional whereby the patient utilizes stereopsis when needed, demonstrating orthophoria. However, when binocular vision is of no
benefit, for example, in the distance or when viewing two-dimensional images, the eyes will deviate allowing for panoramic viewing, hence, a wider field of view.

Other theories seem to parallel those of esotropia. For example, faulty accommodative mechanisms have also been used to explain exodeviations. AC/A ratios do not seem to play a significant role. Active divergence is yet another explanation that has been proposed. Electromyographic studies seem to provide evidence for this theory, however, further research should be conducted to lend further credence. In contrast, an abnormality in the convergence mechanism may account for both eso- and exodeviations as proposed by Adler but again evidence is lacking.

In conclusion, the above theories are all plausible explanations for the etiologies of strabismus, some more powerful than others. There is not one proposal in particular, however, that can account for all types of strabismus. In fact, there may be several, depending upon the condition and individual. Future studies and research will, therefore, contribute to our knowledge and help us to understand the origins of strabismus, thus, better enabling us to treat each condition appropriately.
REFERENCES


