PRISM AND BINOCULAR STRESS*

Raymond R. Roy†
Portland, Oregon

The pendulous nature of conflicting opinion regarding the use of prism as a valid therapy for certain binocular dysfunction, reflects the limitation of our understanding of the complex forces involved in the act of maintaining binocular vision. This alternation of thought has extended from the concept of a liberal use of prism to complete abstention.

The use of prism as a therapeutic agent has been treated very lightly in ophthalmic literature and as a result has been very limited in routine clinical practice. There are even those who have considered that the use of prism was an inferior substitute for visual training or orthoptics. Hence, in the minds of many there has been an "either-or" concept, rather than the recognition that with proper differential diagnosis, there is no substitute for prism therapy when the need is present.

THE BINOCULAR COMPLEX

The primary position of an upright man is that natural position in which the head is erect upon an erect body, and the gaze is directed exactly forward to the horizon. In this position the median plane of the head and body is perpendicular to the horizon and parallel to lines of gravitational force. A transverse plane extending through the centers of rotation of the two eyes is parallel with the horizon and perpendicular to the force of gravitation. In this position the center of gravity will be found midway between the two feet and the stress of body weight will be carried by the skeletal structure.

Man as an upright biped, constantly beset by gravitational forces, must utilize many sensory stimuli to maintain proper equlbrium for efficient motility. One of the major stimuli, upon which he must rely, is that which is received through the binocular complex.

Associated with binocularity is the labyrinthine and tonic neck reflexes which together play a major role in the machinery of postural set. So interdependent are these functions that they have been referred to as the great triad, or head, neck, and eye reflex. Duke-Elder[1] in emphasizing this states:

---

*Read before the annual meeting of the American Academy of Optometry, Section on Binocular Vision and Perception, Chicago, Illinois, December 12, 1961. For publication in the September, 1962, issue of the AMERICAN JOURNAL OF OPTOMETRY AND ARCHIVES OF AMERICAN ACADEMY OF OPTOMETRY.

†Optometrist. Fellow, American Academy of Optometry.

> In the normal condition all these reflexes are summated, the one supplementing the other with the result that there is an extremely well-developed correlation of ocular, labyrinthine, and neck reflexes, by means of which, both in movement and at rest, and in the various physiologically possible positions of the head with respect to the body in space, the correct visual attitude and the suitable correlation of the two eyes are ensured.

Any muscle or group of muscles used to effect movement of the body must be constantly modified depending upon changes in the posture of the entire body or of its separate parts. This is especially true of the eyes, as any desired movement must be integrated with the position of the head in space. For example, the muscles used in sursumvergence, when the head is in the primary position, vary considerably from those used for the same act when the head is tilted.

To these involuntary compensatory movements relating to changes in position of the head in respect to gravity, may be added other impulses affecting binocular movement. These could be briefly mentioned as: (1) stato-kinetic reflexes caused by movement of the head through space, (2) sensory impulses (i.e., auditory or pain), (3) visual impulses, (4) proproceptive sensation, and others.

Thus we see that even in a theoretically perfect human organism under ideal environmental circumstances, the oculomotor muscles are kept in a constantly changing state of tone during the entire waking hours. As a result, the coordination centers for binocular control are in a continuous state of excitation even under the most ideal circumstances.

However, the perfect human organism does not exist and we are dealing with imperfections of structure and function anywhere from a mild to a radical degree. Therefore, if anything prevents a perfect alignment of the eyes, the fixation reflex must correct the maladjustment if single binocular vision is to be maintained. These imperfections which influence binocular control are most certainly not limited to the structure of the eye or its adnexa, even though in this area alone there are innumerable combinations of structural imperfections. Outside the localized area of the eye, one of the most marked developmental structural anomalies to affect binocular control is that of a unilateral short leg. With minor deviations of but an ⅛ inch or ¼ inch in leg length, the body center of gravity is shifted enough that compensatory action must be taken. A common compensation is to develop a slight torticollis of the head and neck, which in turn alters the position of a transverse plane connecting the two eyes. It is now no longer parallel with the horizon, so from peripheral visual stimuli, from labyrinthine control, and from tonic neck reflexes come the compensatory changes in the tonus of the oculomotor muscles. This compensatory action is found in disjunctive vertical movement as well as torsional movement.

Thus to the already complicated reflexes used in maintaining binocular fixation, must be added these compensatory reflexes which alter and influence oculomotor muscle tone in every postural position which the eyes must take. Collectively, the direct, indirect, and compensatory reflexes affecting binocular control might be referred to as *The Binocular Complex.*

RECIPROCAL INNERVATION

The question of how antagonistic muscles cooperate in the performance of active movement was first answered by the observations of Galen.[2] His early pronouncement, as translated by Daremberg in 1856, is as follows:

> The contracted muscle is shortened while the relaxed muscle is extended simultaneously with the limb. For this reason, the two muscles are in action throughout both movements of the member. However, we are not concerned with both, for the activity consists in the tension of the limb which moves itself, and not in the passive action.

This, in essence states that when one muscle of an antagonistic group acts to produce movement, its antagonist remains inactive. This concept held until 1662 when Descartes[3] suggested that when the external rectus muscle of the eye contracted, the medial rectus simultaneously relaxed.

In 1776 Winslow[4] presented another point of view in which he felt that the antagonist also contracted to some degree in order to offer a moderate resistance. This was perhaps the first recorded concept of co-contraction of opposing muscle groups.

In 1872 Duchenne[5] was even more emphatic than Winslow and claimed a simultaneous contraction of agonist and antagonist muscles during the production of motion.

Beaunis[6] in 1885 and 1889 and Demeny[7] in 1890 presented results of the first laboratory tests to demonstrate that in the main, during the movement of voluntary muscles, there is simultaneous contraction of antagonistic muscles.

Then in 1891 and 1904 Beevor[8] claimed that in strong movements against resistance, the antagonists are always relaxed. This was the beginning of the English school; which was finally crystallized by the writings of Sherrington[9] and the concept known as Sherrington's law of reciprocal innervation.

In arriving at this conclusion Sherrington sectioned the third and fourth cranial nerves of two experimental animals and then observed the resultant eyes movements. Such meager observations under very unnatural conditions led to the establishment of a theory which has been labeled a law.

In 1925, Tilney and Pike[10] using electromyography to compute the action of agonist and antagonist found that by not destroying the functional integrity of the antagonistic group, they seemed to get results which were directly opposite from Sherrington's. Their summary held that: "Under normal conditions, we have not observed the contraction-relaxation phenomenon which is generally known as Sherrington's reciprocal innervation." Their results indicate that co-contraction is the normal status in smooth ocular movement.

In 1952 Levine and Kabat[11] further substantiated this view and contended that co-contraction was the rule rather than the exception in all voluntary movements of the limbs.

NEUROMUSCULAR HYPERTENSION

Chronic or persistent states of residual co-contraction in large groups of sketetal muscle result in neuromuscular or nervous hypertension. It is variously described as an anxiety tension reaction, neurasthenia, "nervousness" or nervous tension. This is an increasing problem, especially in America, and is the basis for the tranquilizer form of pharmacotherapy which is so prevalent today. Investigators in this field, however, are learning that extensive and protracted use of tranquilizers is not the answer to this problem of residual tension. In fact further research is bringing to light that prolonged use of tranquilizers may even have an adverse reaction in certain instances.

Through the pioneer work of Edmund Jacobson,[12] much has been learned of this protracted tension state and means have been utilized to relax progressively these pathologically tense individuals. In psychiatric offices throughout the land, this progressive relaxation therapy is finding its place as a major weapon against anxiety tension states.

Here again, as in any branch of the healing arts, good differential diagnosis is needed to determine if this residual co-contraction state is autogenetic or if it could be the result of improper function. Or again, could it even be the result of structural asymmetry. The influence of psychiatry on the health-care professions today has created an era which may someday be referred to as the "psychosomatic age." It is often far easier to label a chronic syndrome as "psychosomatic" rather than to plunge into exhaustive analyses. If proper investigations were made, many so-called psychosomatic syndromes would ultimately prove to be somatopsychic in nature and with proper therapy could be eliminated.

For a good example of this, let us again turn to the aforementioned problem of the unilateral short leg. When such a seeming small discrepency exists, there must of necessity be a compensatory tension state of skeletal musculature to hold the body center of gravity in its proper place. This chronic tension state was created to maintain an erect organism in proper gravitational alignment. As the defect is structural, the tension state will exist until the asymmetry is corrected. If allowed to exist for any length of time, symptoms of a protracted tension state will soon appear.

Perhaps the currently most celebrated example of this problem is found in the person of the president of the United States, who some years ago complained of chronic backache, cervical tension and the related symptoms. The physician who diagnosed this condition and prescribed ¼″ lift in one shoe made such an impression that she is now the attending physician for the first family of the White House.

In a recent popular magazine there appeared a medical information type of article which told of the story of a man who had repeated chest pain which had been diagnosed as a heart ailment. When another physician discovered a unilateral short leg and prescribed a shoe correction, the so-called "heart condition" left. This was nothing more than a compensatory tension state due to structural asymmetry.

Wiles,[13] in commenting upon the postural changes which will effect stress in erect skeletal musculature, shows that while in a standing primary position, moving the hands forward just one inch displaces the center of gravity sufficiently to electromyographically record a compensatory contraction of the erector spinae.

Now it is true that most muscles involved in stress conditions are capable of withstanding a considerable stress for short periods or a milder amount for more extended periods, but few muscle groups are able to withstand protracted stress indefinitely and remain asymptomatic.

BINOCULAR STRESS

All paired motor groups of the human organism must hold some degree of coordination, but nowhere is there the extreme finesse found in binocular vision. Here there must be a most complete harmony between bilateral muscle groups if fixation and fusion are to be maintained. The binocular complex yokes the eyes into virtually a single organ, or functional cyclops. Worth refers to it as a "physiological binoculus."

The reference points which determine all coordinated oculomotor activity are the two maculas. These two areas, approximately one millimeter in diameter, must be held in perfect superimposition by the fused occipital image, in whatever postural position the eyes find themselves. If there were a deviation of but one millimeter there would be subjective diplopia. Now in addition to these points of reference there is a plane of reference formed by corresponding peripheral retinal points. These peripheral retinal points are deeply integrated into hemispherical juxtaposition by partial chiasmal decussation. Thus there must of necessity be a torsional globular compensation if there is to be a complete bond between peripheral reception, cerebral fusion and postural orientation.

This fine degree of binocular coordination must exist in spite of anomalies which may produce abnormal binocular action. The potential of anomalies which may impair stress-free coordination is much greater than is generally agreed. Referring to ocular muscle anomalies, Fink[14] states they are probably more frequent than is usually supposed. He feels that the frequency with which anomalies are encountered in the dissecting room should lead one to believe that more should be observed clinically. Bielschowsky[15] concluded that more than eighty per cent of human beings would have heterotropia were it not for the fusion process preventing it.

Fink, in reviewing the developmental anomalies of the oblique muscles, refers to many authors who have presented cases which represent such variables as: (1) absence of one or more muscles, (2) anomalous development (of tendon, muscle, insertion, etc.), (3) congenital palsies, and (4) anomalous development of the adjacent fascia. With this realization of the almost legion number of variables which may exist in the oculomotor mechanism of the two eyes and the further realization that each variable will produce equally variable compensatory response, it becomes clear that any human organism not exhibiting some degree of compensatory stress would be very rare indeed. Scobee[16] refers to orthophoria as the exception rather than the rule.

Nevertheless, many phoria findings are recorded as "orthophoria" on many prescription forms. Why this apparent contradiction to a very probable fact? The answer lies in our modus operandi of visual analysis. It is extremely rare that any two tests of heterophoria ever give identical readings in the same individual and often variable answers are received with even the same test on repeated trials. Yet in the face of this evidence, the mathematicians have formularized and graphed our refractive routine until we have come to believe in a numerary diagnosis. We have become so enamored with the accommodative-convergence relationship that we seem incapable or unwilling to recognize the existence of a stress in the fixation mechanism which may not be dependent upon accommodation. There seems to exist the feeling that any disharmony between accommodation and convergence must be overcome by the use of accommodative inhibition.

LATENT BINOCULAR STRESS

Scobee,[16] Fink,[14] and others designate heterotropias as manifest binocular deviations, and heterophorias as latent deviations. This designation leaves no room for the phoric position found after prolonged disruption to fusion.

A better understanding between these two conditions would be that a tropia and a phoria are not the same. A tropia is not merely an increase of a phoria or the result of a high phoria. In a tropia, there is rarely an awareness of diplopia. If there is, then the fusion impulse will make a supreme effort to eliminate this annoyance and the visual axes will again be forced into parallelism. In the presence of a full-functioning fusion center, a tropia is almost non-existent under normal conditions. Traumatic sequelae are of course exceptions.

With the realization that a co-contraction of the antagonistic oculomotor system is the means by which smooth motility is achieved, and with the realization that this normal co-contraction is altered by imperfect body symmetry and that residual tension is increased, it is understandable that most routine phoria measurements are not measurements of the position of rest of the two eyes, as is so commonly supposed. A phoria measurement taken during a routine refraction is merely the position manifest by the neuromuscular system under a temporary disruption to fusion. When fusion is made inactive for a prolonged period, this phoric response is generally considerably altered and can be referred to as the latent phoria. It is this latent heterophoria which can induce pain syndromes often described as psychogenic.

For this reason, the vision specialist should feel constrained to take an adequate case history on every patient to determine the possibility of a syndrome suggestive of a binocular stress.

PRISM AND STRESS

Prism in a lens prescription can either stimulate or inhibit stress in the binocular complex. Unwanted prism induced during binocular seeing demands that a stress be set up in the oculomotor system to prevent diplopia. This can so easily be demonstrated by holding two or three diopters of vertical prism before one eye. With many subjects, there would be diplopia which would last from a few seconds to a few minutes. The alleviation of this diplopia can only result from a disjunctive vertical movement of the two eyes and result in stress and eventual symptoms.

Conversely, a vertical prism of the proper amount held base-down before a hyperphoric eye will inhibit an existing vertical disjunctive stimulation and relieve binocular stress. It might be said that the inhibitory effect of a relieving prism does to the extraocular muscles what a tranquilizer may do to general skeletal musculature. It can be further stated that, in certain cases, a prism is the only known method in which a binocular stress can be eliminated while still maintaining single binocular vision. The use of drugs can not induce this inhibitory effect, as diplopia would be the end result. Thus then, the awareness of diplopia would again induce a tension state and the cycle again repeated.

An interesting observation is, however, that the application of prism to induce an inhibitory action does not always give an immediate response. Also if the compensatory stress has been in existence for a long time, perhaps only a small amount of inhibition may be obtained at one time. After the neuromuscular system has been able to relax to this first amount, then a second phase may be entered if the symptoms have not entirely abated.

Any sketetal musculature subjected to a prolonged tension state cannot immediately find relief when the tensor is released. The longer the muscle has been subjected to this tension state, the longer it will take to resume normal function. For this reason, it may take weeks or even months to obtain relief from a binocular stress which has been present for many years.

In the case of binocular stress, there is not only the tension state of the muscle fibre itself, but there is the frustration set up at cortical levels. One portion of the brain is seeking relief from the chronic abnormal co-contractile state of the oculomotor system. Another part is demanding single binocular vision and here develops an impasse. Scobee explains that muscles do what nerves tell them to do and that nerves only transmit messages from motor nuclei and that these nuclei are controlled by a supranuclear mechanism. Of this, he says: "It is this *supranuclear mechanism,* then, which when it receives messages from all sources, including the voluntary centers of the brain, arranges the proper pattern of stimuli to the nuclei to execute a well-chosen response; in other words, a reflex, but a special type of reflex involving the will, that is, a psycho-optical reflex."

In referring to the results of this frustration and stress, Scobee[16] says:

> Several writers have commented upon the association of neuroses with heterophoria, particularly with exophoria (Mellick, 1950; Livingston, 1946; Beccle and Kitching, 1945). The implication apparently is that the neurosis precedes the heterophoria, but our experiences have been exactly the reverse, namely, that the heterophoria seems largely responsible for the neurosis. This observation is based upon the often marked personality change for the better in these patients once their heterophoria is corrected either by surgical or non-surgical means.

The fact that one part of the human organism cannot be subjected to a prolonged stress without soon affecting other parts drew this observation from Jones:[17]

> Since the central nervous system is a continuous structure, tension in one area must be transmitted in varying degree to all others. Conversely, for the same reason, relaxation of tension may cause corresponding release at distant points.

Thus we see that the use of prism, when properly used to inhibit binocular stress, is not only furnishing relief of a protracted tension in the oculorotary muscles themselves, but is inhibiting a stress at cortical levels, and then by the complex interconnection through the central nervous system, is relieving stress at even distant points as well. Truly, no other form of vision therapy can show such marked and dramatic results as prism, when properly used.

318 S. W. Alder Street
Portland 4, Oregon

REFERENCES

1. Duke-Elder, W. Stewart, Text-book of Ophthalmology, Vol. 4. St. Louis, Mo., C. V. Mosby Co., 1949.
2. Galen, Del' utilite des parties, *in* Oeuvres de Galien, edited by C. Daremberg, Vol. 2, 1856.
3. Descartes, R., De Homine. Leyden, Moyard & Leffen, 1662.
4. Winslow, J. B., Exposition Anatomique de la Structure du Corps Humain, Vol. 2. Paris, 1776, p. 43.
5. Duchenne, G. B. A., Orthopedic physiologique et pathologique on deductions pratiques des recherches electro-physiologiques et pathologiques sur les mouvements de la main et du pied, Bulletin de Therapie, Vol. 2, 1857.
6. Beaunis, H., Recherches physiologiques sur la contraction simultanée des muscle antagonistes, Archives de Physiologie Normale et Pathologique, Series 5, 1: 55, 1889.
7. Demeny, Du role mechanique des muscles antagonistes, Archives de Physiologie Normale et Pathologique, Series 5, 2: 747, 1890.
8. Beevor, C. E., On some points in the action of muscles, Brain, 14: 51, 1891.
9. Sherrington, C. S., Integrative action of the nervous system, J. Physiol., 17: 28, 1894-1895.
10. Tilney, Fredrick, and Frank H. Pike, Muscular coordination experimentally studied in its relation to the cerebellum, Arch. Neur. and Psychiat., 13: 289-334, 1925.
11. Levine, Milton G., and Herman Kabat, Cocontraction and reciprocal innervation in voluntary movement in man, Science, 116: 115-118, Aug., 1952.
12. Jacobson, Edmund, Progressive Relaxation. Chicago, Ill., University of Chicago Press, 1956.
13. Wiles, Philip, Essentials of Orthopaedics. Boston, Mass., Little, Brown & Co., 1955.
14. Fink, Walter H., Surgery of the Oblique Muscles of the Eye. St. Louis, Mo., C. V. Mosby Co., 1951.
15. Bielschowsky, A., Lectures on Motor Anomalies. Hanover, N. H., Dartmouth College, 1943.
16. Scobee, Richard G., The Oculorotary Muscles, 2d ed. St. Louis, Mo., C. V. Mosby Co., 1952.
17. Jones, Laurence, The Postural Complex. Springfield, Ill., Charles C. Thomas, 1955.

BIBLIOGRAPHY

Adler, Francis Heed, Physiology of the Eye; Clinical Application, 2d ed. St. Louis, Mo., C. V. Mosby Co., 1953.

Anderson, J. Ringland, Ocular Vertical Deviations and the Treatment of Nystagmus, 2d ed. Philadelphia, Pa., J. B. Lippincott Co., 1959.

Breinin, Goodwin M., Electromyographic evidence for ocular muscle proprioception in man, A. M. A. Arch. Ophth., 57 (2): 176-180, 1957.

Brooks, V. B., and V. J. Wilson, Localization of stretch reflexes by recurrent inhibition, Science, 127: 472-473, 1958.

Friedman, Arnold P., and H. Houston Merritt, Headache; Diagnosis and Treatment. Philadelphia, Pa., F. A. Davis Co., 1959.

Haugen, G. B., H. Dixon, and H. A. Dickel, A Therapy for Anxiety Tension Reactions. New York, Macmillan Co., 1958.
Maddox, Ernest E., Tests and Studies of the Ocular Muscles, 2d ed. Philadelphia, Keystone Publishing Co., 1907.
Miller, Ronald G., Intraocular pressure changes associated with effects upon the intra and extraocular muscles, Am. J. Optom. and Arch. Am. Acad. Optom., 39 (1): 17-22, 1962.
Peter, Luther C., The Extra-ocular Muscles, 2d ed. Philadelphia, Lea & Febiger, 1936.
Roy, Raymond R., Ocular migraine and latent heterophoria, Archivos de la Sociedad Americana de Oftalmologia y Optometria, 2: 229-235, 1959.

IN MEMORIAM

DR. GORDON L. WALLS

Dr. Gordon L. Walls, 57, professor of physiological optics at the School of Optometry, University of California, and one of the Nation's outstanding authorities on vision, died of a heart attack in Berkeley on August 22, 1962.

A native of Malden, Massachusetts, Dr. Walls took his undergraduate work at Tufts College, received his master's degree from Harvard and his Sc.D. degree from the University of Michigan.

He came to the University of California as an associate professor in 1947 after having taught at the University of Michigan, the University of Iowa, and Wayne University. He also had been on the research staff of the Bausch and Lomb Company in New York.

Ten years ago Dr. Walls was advanced to a full professorship at the University of California. He also lectured at the University of California Medical School in San Francisco.

He was internationally known for his research on color vision and was author of the book, *The Vertebrate Eye*. His latest studies were on the hereditary aspects of vision. He was a frequent contributor to optometric, optical, psychological and ophthalmological journals, acted as associate editor of SURVEYS OF OPHTHALMOLOGY and was a fellow of the American Academy of Optometry and the American Association for the Advancement of Science.

Dr. Walls is survived by a daughter, Istar, 12, and a son, Charles, now in Chicago.

The Alumni Association of the School of Optometry, University of California, Berkeley, is opening a Library fund in Dr. Walls' memory.