

ETIOLOGY OF COMITANT CONVERGENT STRABISMUS*

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THE standards of the body parts are set by predominance, utility, and concept of beauty. The eyes conform to these when they look forward; they work with ease when the visual axes are parallel when at rest and cross on the object fixed, both far and near; and being in conformity to this they satisfy the esthetic sense.

Strabismus is a deviation of the eyes from the normal position to an observable degree. When an eye turns inward it is known as the convergent variety; and when the other changes its position to the same extent, on fixing with the squinting eye, it is called comitant.

To acquire a knowledge of this anomaly requires a study of the entire visual system. This essay is for the purpose of advancing some views at variance with the present theories, and, to make the presentation clearer, it may help to trace briefly those that have been brought forward in the past and down to the present time.

For the sake of brevity, references and long citations have been omitted; these are well covered in recent books on the subject.

Varieties.—According to its behavior comitant convergent strabismus is found as three kinds: occasional, alternating, and monocular. The occasional may remain as such, but it is usually premonitory to an alternating or monocular. The alternating and monocular become permanent as a rule. In a few instances they may disappear later, known as a spontaneous cure. The squinting found in babies is called occasional or periodic but as it may have no relation to the true form it might be termed infantile strabismus.

History.—The past history, of necessity gained from the parents, may give little information. It may be learned that the child had the usual diseases and accidents occurring at this period of life. The presence of strabismus in parents and relatives may be established.

Course.—If seen at the onset the patient may be as young as two years but the majority are between three and four, and rarely over five. If first seen when older the history of its inception coincides with these ages.

The mother's recital is apt to be reliable. The information given is that one eye started to turn in, usually for a short time at first, and later it became permanent. They often volunteer that the first turning was when the child was tired; in the latter part of the day; or it followed an illness, accident, or fright.

Examination.—At the beginning many are too young for accurate testing of the acuity of vision. When it is possible to make it most

* Read before The American Homoeopathic Ophthalmological Otolological and Laryngological Society, at the 86th Annual Convention of the A. I. H., Atlantic City, N. J., June 16, 1930.

cases show a reduction from normal and usually more in the squinting eye.

There is also, as a rule, an error of refraction; usually of considerable hyperopia and astigmatia. A few are almost emmetropic and a slighter number myopic.

Type of Child.—A child of sound constitution and good inheritance may develop strabismus, but it is more apt to be present in one showing some physical or mental defect. If precocious they may be high strung and nervous. On the other hand they may range from a slight mental dullness to an extreme of retarded development. The physical defects present may be minor or an extreme abnormality, as Mongolian idiocy, as an example. I have noted also the presence of stuttering, asthma, harelip, and cleft palate.

Theories of Strabismus.—A review of the different theories is presented with an effort to confine it to such points as may throw some light on the subject.

Heredity.—Heredity has been considered a factor from ancient times, as noted in the works of Hippocrates. Today it is seen to occur in families but this fact gives no solution as it still must be explained in the progenitors and kindred.

Muscular Theory.—The most outward and tangible things were ascribed by the earliest observers as the cause of departures from the normal. For many centuries an over-strong muscle was deemed the correct and plausible explanation. Later muscular conceptions as weakness, shortness, wrong insertion, and shape of the orbit were added. The muscular cause has been discarded as the principal element but one condition, an abnormal insertion, may have some influence.

The writer advances as a conjecture the probable prevalence of abnormal insertions from a study of the heterophorias. As the two eyes are elevated, and lowered together, they should receive the same innervation from the same center, for each movement. Always working together they should have the same strength. This and the ability to overcome only a weak prism, base up or down, would indicate that hyperphoria is probably due to an abnormal insertion.

In a study of 1000 consecutive cases, where it was possible to make a muscle test, the results as to hyperphoria were as follows:

Some form of heterophoria was present in 728 persons, and of these 446 showed hyperphoria, a percentage of 61.2 of this fault. Those having two degrees and over were 12.7 per cent.

If this deduction is logical then, by analogy, it may be that the external and internal recti muscles have about the same amount of misplacement. This would be divided, of course, among four muscles.

Accommodation Theory.—Before the middle of the eighteenth century is found the first departure from the muscle theory, and a few writers advance the idea that refractive errors may be the cause.

About one hundred years later Donders stated positively that hyper-

metropia and astigmatism were the etiological factors of convergent strabismus. He also attributed the poor vision in the squinting eye to non-use, termed amblyopia ex anopsia.

Donders' theory was universally accepted for some years, and has fixed the idea of excessive accommodation in the medical mind.

With the progress of ophthalmology and the accumulation of statistics it became evident that this could not be the sole cause. There are too many exceptions; we constantly meet patients who by these rules should squint, and do not. Also some squint with only slight hyperopia and astigmatism and occasionally the person is myopic.

Fusion Theory.—A deeper-seated cause was later advanced, placing it in the nervous system. This, from his able presentation, has become known as Worth's fusion theory. This in turn had, and still has, many adherents but some have raised a dissenting voice.

The fusion theory is based on a hypothetical fusion center. At some time in the future this special center may be located and undoubted evidence be produced that squint is due to a fault in this area, but until then I think the cause must be placed in some part of the known visual system.

Pathology as a Factor.—The absence of any visible lesions in the fundus in strabismus has led to the belief that the eye was free from pathology, and the reduced vision was an amblyopia ex anopsia.

Ralph I. Lloyd, from findings with his stereocampimeter, has pointed the presence of central scotomata in the fields. He has found the perimetric outline to be regularly contracted for white and red. The field of the fixing eye is somewhat contracted, while that of the squinting eye will usually run twenty degrees smaller than normal. The better eye will show a moderately enlarged blind spot and the deviating one may have a huge loss; or a long wavy defect up and out, or down and out, from the blind spot. This macular defect is in harmony with the loss of vision.

J. N. Evans has examined twenty consecutive selected cases and found a scotoma at the fixing point in all.

Heine, using the Schloesser method of binocular perimetry, found 94 per cent had central scotoma. Uthoff reported 54 per cent.

This is strong evidence of the presence of a pathology before the onset of the squint, and must be considered as one of the causative factors. It may also change the conception of amblyopia ex anopsia.

Functional Nervous Causes.—The presence of functional nervous disorders in these children is emphasized by Oscar Wilkinson in his recent book, "Strabismus." He quotes many authors who have noted such manifestations.

Divergence Insufficiency as a Cause.—Divergence insufficiency, or weakness, has been mentioned as a cause in some cases by a few authors, but has been ignored by the large majority. It is inserted here to complete the list of ascribed causes but will be considered at greater length later.

Some Preliminary Considerations.—Before passing to the arguments as to the writer's idea of the cause of strabismus there are several things to be considered.

Nomenclature of Ocular Movements.—For the sake of uniformity and clearness the suggestion is made for the following designation of the cardinal ocular movements:

Rotation: for the movement of one eye; outward, inward, upward, and downward.

Vergence: for the conjugate and disjunctive movements; dextrovergence, sinistrovergence, sursumvergence, deorsumvergence, convergence, and divergence.

Duction: the ability to overcome prisms; adduction, abduction, supraduction, and infraduction.

Orthophoria.—The ideal state of the eyes is that of orthophoria, and is present when the visual axes are parallel when the eyes are at rest. There are two principal planes of the visual axes, horizontal and vertical. The visual axes are parallel in the horizontal plane; one is not higher than the other. The vertical plane is midway between the eyes and the visual axes when parallel are as far apart as distance between the two foveae; or, to measure the angles, the line connecting the center of rotation of each eye.

During the waking hours, and especially indoors, the eyes are almost constantly fixing on near points and the axes are converged toward the vertical plane.

Convergence.—Convergence is the function which is in constant play to fix on objects, from infinity to the nearest point of its ability. At the near point it works in harmony with accommodation. The presence of convergence and its working are so self evident that there is nothing to be considered; except one point. Does it function alone or in conjunction with the divergence function?

Divergence.—Divergence would seem to be a much less important function, as it is not necessary for the eyes to assume this position. But it must play an important rôle as a check function, as is evident from the following:

In paralysis of the sixth nerve the eye rotates inward, with the production of diplopia.

The presence of a divergence function is shown by the ability to overcome a prism held base in before the eye. This abducting power is usually put at 4 to 8 prism diopters. The results of a series of tests I made showed an average of 7 diopters. Occasionally it may be as high as 14 diopters.

Cause of Comitant Convergent Strabismus.—I advance the opinion that the fundamental cause of comitant convergent strabismus is a weakness of the divergence function. Contributing causes are abnormal insertion of a muscle, hyperopia and consequent excessive accommodation, and amblyopia. There may be one or more of these present in a case. Amblyopia may be due to pathology and I think

astigmatism of a high degree has the same effect in the weakening of the foveal guide.

The opinion that divergence weakness plays such an important rôle is based on the following argument:

In orthophoria there is a balance between the convergence and divergence functions. There is often a perfect balance in the presence of refractive errors, and amblyopia. Abnormal insertions may be present, but impossible to prove.

Divergent strabismus is conceded to be a weakness of convergence, which argues for a similar cause in the convergent form.

There are four types of eyes in the convergence anomaly:

I. Emmetropic and normal vision.

II. Ametropic.

III. Amblyopic.

IV. Ametropic and amblyopic.

In the first type, with no refractive error and with good vision—but possibly an abnormal insertion—the divergence function is too weak to maintain a balance.

In the others there is the added burdens of overacting accommodation and loss of the foveal guide.

The first type is more apt to be of the occasional or alternating variety, and the fourth the constant monocular.

The clinical history develops some significant facts which point to divergence weakness. The eye begins to turn in toward the latter part of the day. It is noticed to occur when the child is tired. The squint follows an illness, accident, or shock. Is it not more logical to consider that the innervation of divergence is weakened by these conditions rather than that the convergence is strengthened?

The type of child also denotes a depression of other functions, as evidenced by the various nervous manifestations. Also the physical defects and retarded developments which point to weakness rather than strength.

I think that the methods of treatment, which have been successful in many cases, are an additional argument in favor of divergence weakness. The occlusion of one eye has rested a tired innervation; a cycloplegic has had the same effect; the correction of the hyperopia has removed the added load; and the corrected hyperopia and astigmatism have aided the foveal guide. The stereoscope and amblyoscope may have increased the divergence power by exercise.

It would seem, therefore, that there is a need for even a more diligent use of these means. It also leads to a suggestion of the trial of the use of prisms, base out, in some cases. When an operation is considered, it may be a good reason for advancing the external rectus muscle.

In conclusion, the writer would say that the final word in the ocular muscle problems has not been written, but he desires a consideration of the opinions presented and welcomes any criticism.