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A CLINICAL STUDY OF ONE HUNDRED AND FIFTY CASES OF HYPERPHORIA.*

WENDELL REBER, M.D.,
PHILADELPHIA.

When this paper was first thought of, I went over all my records with a view of finding in what percentage of cases hyperphoria had been noted; also what lessons were to be derived from the study of these cases. After much examination I decided to limit myself to my last 700 cases, throughout which there has been almost absolute uniformity of study. The statistics resulting from any study of these 700 records will therefore be of much greater interest and more faithful to the facts than had I included my preceding 1700 records, many of which are more or less incomplete as to the muscular status.

In every case painstaking correction of all refractive errors was made, the tests for hyperphoria preceding and following the refraction. Out of these 700 records 525 were refraction cases, in every one of which the muscular status was carefully estimated with the Maddox rod—and sometimes with supplementary methods—before giving the prescription. It is my conviction that without full knowledge of the muscular status, it is absolutely impossible to come to an intelligent conclusion as to what lenses ought to be ordered in any given case of ametropia.

Hyperphoria is spurious or intrinsic. The former is almost invariably an outward expression of perverted accommodation. In such cases where the ciliary muscle has been cured of its bad habits by a mydriatic or the wearing of proper correcting lenses, the vertical deviation usually disappears. Particularly is this true when oblique astigmatism has been the fault. Moreover, lithemia, gout, neurasthenia and, I have sometimes thought, early tabes, will at times produce a transient hyperphoria which might easily lead the examiner astray if the general economy and the patient's life habits are not kept constantly in view.

Intrinsic hyperphoria, on the other hand, is uninfluenced by a mydriatic. Indeed in some cases I have seen a small but gradual daily increase under prolonged cycloplegia. This is no less true after wearing a proper correction, for, as far as my experience goes, intrinsic hyperphorias are little if any influenced by curing the ciliary muscle of its vicious habits. True, many such patients will be entirely relieved of all their symptoms by assuming the proper correcting glasses, in which case treatment directed to any coincident muscular imbalance is meddlesome ophthalmology. But, on the

other hand, I have known people to wear perfect corrections for some years, going from one to another ophthalmologist, still seeking relief, only to find it when a vertical prism had been combined with their curvature correction.

Whether a patient shall or shall not receive a prism correction should be entirely a matter of symptoms. Some people carry a vertical imbalance of 1 to 2 degrees all through a life of high tension and large usefulness without the slightest discomfort, while in others .5 to .75 degree is sufficient to make life an almost intolerable burden. Into this phase of the question enter such factors as heredity, temperament, life habits, worry, and all the rest of that intricate aggregation of facts that go to make up the personality of our patients.

But there seems a degree of hyperphoria below which it is trivial to go, and this I would fix at .5 degree—tentatively at least. Nothing less than this was recognized in the 700 records studied.

In the early part of my muscle work the diplopia test was unfailingly applied, either by means of a single rotary prism or the Stevens phorometer. But I soon found myself relying on the Maddox rod, and for the very good reason—shown by my colleague, Dr. Hansell—that any test that compared the behavior of a foveal image of one eye with a perifoveal image of the other eye could not but often lead the surgeon into false conclusions; this is because of the stimulation of non-corresponding points in the two retinas. The test that would so deform one image as to temporarily set aside the fusion impulse, and at the same time allow a certain portion of this deformed image to fall on the fovea of the eye seeing that image, certainly seems the best test so far at our command—hence the great value of the Maddox rod. On this test I have rested the diagnosis in these 150 cases.

There is a diplopia test that is of signal value in the detection of hyperphoria, namely, the use of a colored glass to detect, if possible, spontaneous diplopia, which if found is of great significance. But this is natural diplopia conforming to the proposition that a test must concern both foveæ centrales; hence this diplopia test is free from the objections made against those tests based on artificially produced diplopia.

Latent Hyperphoria.—An interesting feature in connection with this study is that out of the 150 times in which hyperphoria was found, it was partially or wholly latent in 49—or practically 33 per cent.—of the times it occurred. In glancing over the methods of various workers for the unmasking of latent hyperphoria I find that all of them are some manner of modification of the "blinder" test. It is now a fairly well accepted fact that in a certain number of cases, if a blinder is worn before one eye for fifteen to thirty minutes, a hyperphoria varying from .5 to 1 degree will often show itself for a few minutes after uncovering the eye.

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Some surgeons still resort to this method for the detection of supposed latent hyperphoria. Posey has modified this method by first working out the correction of the right eye and then with the correction still before that eye placing in front of it a blank disc. The left eye is now examined, and when the proper correction has been worked out, the Maddox rod is placed before the left eye. He now lifts the blank disc from in front of the right eye which has thus been under cover for from five to ten minutes. If latent hyperphoria exists, it is likely to show itself wholly or in part by this unveiling test.

My own maneuver differs but slightly from Posey's method. After the right eye has been corrected, I place in front of the correction the Maddox rod. When the left eye has been corrected, I immediately direct the patient's attention to the streak of light and after carrying the streak several times from the vertical to the horizontal position, have them tell me its relation to the point of light. I believe that by having the patients fix their attention on the streak in the vertical position and then suddenly swinging it into the horizontal position, the higher cerebral centers are taken by surprise and the detection of latent hyperphoria made relatively easy. The rod should be reversed a dozen times or more, each time questioning the patient as to the relative positions of the streak and light. The purpose is twofold: 1, by continually throwing the higher centers off their guard to unveil more of the hyperphoria; 2, frequent repetitions of the maneuver to educate the patient to assist in the determination by relaxing the impulse to binocular fixation as we relax the accommodation.

Any method that takes the higher centers off their guard will reveal the deviation. Binocular single vision is one of the brain's most complicated automatic functions. It concerns a cerebral—or governing—and a peripheral—or executing—apparatus in the most intimate association. The unconscious preservation of binocular single vision is so deeply rooted a thing in the human automatism that something, some trick, is needed that will temporarily set aside the dominating influence of the cortical centers over the eyeballs and their muscles. Only in this way can latent deviation tendencies in the peripheral portion—the eyeballs—of the binocular vision apparatus be revealed in many cases—and especially in young subjects.

Age seems to play no small part in this matter, for hyperphoria is much more commonly manifest after 40 years of age than before that time. It is difficult to put down the thought that along with the loss of ciliary elasticity there goes a similar loss of compensating elasticity in the extraocular musculature, that favors the easier detection of any previously existing lateral or vertical deviations. Certain it is that in presbyopes, hyperphoria is much more easily shown and vertical prisms are much more comfortably worn than by the generality of young people. In only 7 cases out of these 150 was hyperphoria found before the 20th year.

Between the 20th and 30th years hyperphoria occurred in 34 cases; 30th and 40th years, 50 cases; 40th and 50th years 29 cases; 50th and 60th years, 27 cases; after the 60th year 3 cases. These figures refer to true hyperphorias only, in whom binocular vision was invariably present. No instance of actual vertical squint was allowed to creep in.

As to the refraction conditions with which hyperphoria is associated, it was encountered 117 times out of

150 in hypermetropia and hypermetropic astigmatism; 17 times in myopia and its allied states; 8 times in mixed astigmatism; 6 times in antimetropia—commonly termed anisometropia—and twice in emmetropia. Anisometropia has not in my experience furnished the large percentage of hyperphorias noted by some other workers in this field. Perhaps this is because I found actual vertical squint not infrequent among this class—in other words the findings do not differ in kind but in degree.

In its relation to lateral muscular deviations, the hyperphoria in these 150 cases was associated with esophoria 86 times, with exophoria 55 times, and with lateral balance 9 times. This too is easily explainable on the basis of the predominance of hypermetropia as above shown.

In 73 cases the upward deviation occurred in the right eye and in 77 in the left, so that there is practically no difference in the frequency with which either eye deviates.

Hyperphoria.	No. of times found.	Corrected with prism.	No prism correc.
$\frac{1}{2}$ degree	40	12	28
$\frac{3}{4}$ degree	31	20	11
1 degree	43	35	8
$1\frac{1}{4}$ degrees.....	3	3	—
$1\frac{1}{2}$ degrees.....	10	8	2
2 degrees.....	10	8	2
3 degrees.....	4	2	2
4 degrees.....	5	5	—
5 degrees.....	4	4	—
	150	96	54

The results of prism correction were the following: Number of cases, 96; good, 78; improved, 6; no change, 6; unknown, 6. In the 54 cases in which no prism correction was ordered the results were fairly good. Prism corrections were not ordered in these 54 cases, 1, because, notwithstanding the existence of unmistakable hyperphoria, the patient had been previously wearing either no correction or a wrong one; 2, because convergence-training or repression dissipated the symptoms; or 3, because the patient took such high power plus or minus lenses as to make inadvisable the incorporation of any vertical prism. Such patients learn readily but unconsciously to correct their hyperphorias, generally by tilting one lens a little higher than the other. In prescribing these 96 vertical prisms, full corrections were ordered in 20 cases, three-quarter corrections in 60 cases, and half of the deviation was corrected in 16 cases. Full corrections were made only in glasses for near work, because hyperphoria is generally greater for the occupation distance than for infinity, and experience has shown that the prism that fully corrects the deviation for infinity can often be comfortably worn in all near work. This observation is almost invariably true for hyperphoria of .75 degree or more.

My figures show further that in 40 cases which I have been enabled to follow, the hyperphoria has increased from 33 to 50 per cent. after six-months wear of a vertical prism. I have no explanation to offer for this increase, or rather unmasking of a latent hyperphoria. I can but point to what seems to me a perfect parallel; i. e., the uncovering of a latent hypermetropia by age. When some member of this Section has discovered and made practical, the use of a myoplegic that will paralyze the extraocular muscles as a mydriatic does the intraocular muscles, we shall have made a giant stride toward solving the physiology of the extraocular muscles in particular and the act of binocular vision in

general. True, it would probably upset many of our pet notions of to-day and require us to build up new standards, but it would certainly give us some precious information as to the anatomic and functional positions of rest of the eyes.

The principal symptoms of vertical deviation tendencies are one-sided supraorbital, temporal and occipital neuralgias, photophobia, drowsiness, and tremendously exaggerated general fatigue after prolonged near work. I have been especially impressed with this last symptom in many cases. Too, hyperphoria stands in close relation, etiologically, to migraine; quite as close, I am sure, as oblique astigmatism.

In the effort to make hyperphorics comfortable without vertical prisms or tenotomies, too much attention can not be given to convergence deficiency or excess, the former to be met with convergence training after the method popularized by Gould, the latter with convergence-repression treatment by crowding on excessively strong convex lenses for ten-minute periods of work at the near point three or four times a day. These measures often suffice to wipe out all the symptoms. Moreover, as pointed out by Oliver,¹ sleep and exercise are splendid adjuncts in relieving the conditions of asthenopic hyperphorics. A few additional hours of sleep daily often rid a patient of much eyestrain that lenses, prisms, or tenotomies never fully reach. The benefit of exercise in the open is too patent for comment.

Tenotomy.—Out of the 150 cases studied, tenotomy was resorted to in only 5, all of which presented hyperphoria of 3 degrees or more. In no one of these 5 cases was there any marked difference in the refraction of the two eyes. Two were epileptics. One of them—a young man with left hyperphoria of 4.5 degrees—has been entirely free of epileptic manifestations for six months, whereas he had been having seizures daily. For three years he had worn a perfect correction of his low-grade ametropia without any effect on his minor epilepsy. In his case, I tenotomized the left superior and the right inferior rectus at the same sitting, securing 1 degree of overcorrection, which righted itself in four or five days. He has now absolute muscle balance.

The other, a 31-year-old married woman, has had but three epileptic seizures in four months, as against bi-weekly and sometimes tri-weekly attacks prior to operation. Her hyperphoria was 4 degrees.

The three remaining cases all presented distressing head symptoms not relieved by vertical prisms, but markedly benefited by free tenotomy of the superior rectus of the eye with the higher visual axis.

From the foregoing, the rational treatment of hyperphoria would seem to resolve itself into: *a*, wearing a proper correction four to six months; *b*, exclusion of causative diathesis or central nervous disease; *c*, convergence-training, if convergence be weak; *d*, convergence-repression, if convergence be excessive; *e*, if the hyperphoria is still annoying, prism correction of one-half to two-thirds of the vertical deviation for distance, and two-thirds to all of the deviation for near work; *f*, if the hyperphoria continues to produce urgent symptoms in spite of the preceding measures, tenotomy of the overacting elevators of one eye or the depressors of the other eye finally becomes justifiable.

Much more could be said in comment on the facts growing out of this study, but I shall have to content myself with recording the following convictions:

1. Hyperphoria occurs in about 1 out of 6 patients who seek counsel of the ophthalmologist.
2. It is present to the extent of .5 degree or more in 1 out of 3 refraction cases.
3. It becomes worthy of special attention in about 1 out of 5 refraction cases.
4. It is more likely to be latent before the 30th year—33 per cent. of all cases—and manifest after that time.
5. It occurs most frequently to the extent of about 1 degree.
6. The most frequent symptoms are supraorbital, temporal, and occipital neuralgias, photophobia, drowsiness, and abnormal physical tire after prolonged near work.
7. A full quota of sleep, daily open-air exercise, and a well-regulated life are all highly important factors in trying to make hyperphorics comfortable.
8. Hyperphoria is found a little oftener associated with esophoria—57 per cent.—than with exophoria—37 per cent. This is probably because compound hyperopic astigmatism with its associated convergence-excess outnumbers all other anomalies of refraction.
9. Convergence-training—if convergence be insufficient—and convergence-repression—if convergence be excessive—frequently relieve hyperphoric symptoms entirely.
10. *The vertical prism has a field of pronounced usefulness.*
11. Prism corrections will be of service in about 50 per cent. of all cases. One-half to two-thirds of the hyperphoria may be corrected for infinity and two-thirds to the full amount for work at the occupation distance.
12. When all these means have been exhausted, section of some one of the vertical muscles may be thought of. About 1 out of 20 hyperphorics whose deviation is 2 degrees or more will profit more by tenotomy than by any other treatment. Two or three degrees is likely to be the amount permanently obtained by vertical tenotomy.
13. The only fruit that can be borne of the attempt at hospital or dispensary treatment of hyperphoria—or for that matter any but the simplest muscular anomalies—is vanity and vexation of spirit.

1212 Spruce Street.

DISCUSSION.

DR. H. F. HANSELL, Philadelphia—Dr. Reber's paper is a conclusive one based on experience and is a most conservative expression of opinion founded upon a careful, thorough, clinical investigation. The subject is a very large one and open to discussion in various ways, but the part that seems to me to have the greatest weight is the diagnosis of hyperphoria. The paper is valuable, too, in that the author has carefully excluded hypertropia. Many writings on this subject are complicated and rendered less valuable because there has not been a close line drawn between hyperphoria and hypertropia. The conditions are different; in the one case we are exercising our brain, muscular force and all the nerve energy we can to cancel the defect, and in the other case we have yielded to this defect, and the main symptom we get is diplopia, readily corrected by simply covering one eye, whereas the mental symptoms of hyperphoria, which make it such an important part of our practice, can only be relieved by a careful study and a great deal of practical clinical experience.

DR. MARK D. STEVENSON, Akron, Ohio—Like most ophthalmologists I used to test for hyperphoria with the Maddox rod and prisms immediately after the patient had left the dark room, but now I not only make these tests before finding the refraction of the eye, but also with the proper lenses before the eyes and a Maddox rod before one: then by alternately covering and uncovering the eye which does not have the Maddox rod before it I can discover many cases of hyperphoria. Many cases of latent hyperphoria can be discovered by simply rotating the

1. Norris and Oliver: System of Diseases of the Eye.

Maddox rod from the vertical to the horizontal positions as suggested by Dr. Reber.

DR. G. C. SAVAGE, Nashville—Any muscle test that places the false images within the area of binocular fixation is full of error. The false image should be thrown entirely out of the area of binocular fixation and then we can at once get at the true condition. Monocular tests alone are trustworthy. Let me say that the hyperphoria which is corrected by convex lenses is a hyperphoria that is not due to any trouble in the superior or inferior recti, but is caused by error of attachment of the internal recti; the one muscle is simply attached too high, and the other too low. The esophoria you find in such cases is not an intrinsic esophoria, but a pseudo-esophoria. You correct the hypermetropia and at the same time cure the pseudo-esophoria and the hyperphoria disappears. Again the hyperphoria that is relieved by cylindrical correction is not due to any trouble in the superior or inferior recti. In such cases of astigmatism the meridians of greater curvatures are leaning in the same direction, but not parallel, and the leaning is toward the nose and greater in the hyperphoric eye. Correct this astigmatism and the hyperphoria vanishes like mist before the morning sun.

There was a very interesting case here yesterday in a man with less than 1 degree of right hyperphoria, whose eyes are subjected to 2 degrees of vertical prismatic effect. He gets more relief from that than from weaker prisms. This case was investigated first by an eminent ophthalmologist, who found no hyperphoria at all and who was later induced to operate on the internal rectus of the left eye for esophoria. (If he had divided the lower fibers of the internal rectus of the right eye I believe he would have corrected the trouble.) On examining this case yesterday I found he had a well-marked plus cyclophoria; and I found this to explain the relief he gets from those prisms. I tried first a 1-degree prism before each eye with no comfort; and then a 1½-degree prism, base down, before right eye and a ½-degree base up before the left; that combination gives him the most relief. He thus allows the eye to turn up and is getting relief, not because of any hyperphoria primarily, but because the prism thus placed enables the superior rectus of the right eye to torsion the eye in and help the weak superior oblique, while the prism before left eye causes it to slightly torsion out, but leaving the vertical meridians parallel. I will close with this remark, that I never operate on a case of hyperphoria without knowing where the cause resides and the conditions that complicate it, and never on a case of less than 2 degrees. If a man operates on the superior muscle for hyperphoria and divides more fibers on the outer side than the inner he will lead to more suffering than if he had done nothing. As a rule it is better to divide all the internal fibers of a superior muscle than even the central fibers, and if the outer fibers are divided the patient will be worse in the end than if he had never been touched.

DR. C. F. CLARK, Columbus—I can not help feeling that most of us are mystified by this muscle question. I have enjoyed Dr. Reber's paper very much and feel that it is deserving of credit, but that we need some foundation principles to base our study upon.

DR. JOHN T. CARPENTER, Philadelphia—I think Dr. Reber deserves credit for bringing forward, at this time, a paper dealing with the treatment of defects of the ocular muscles. I have been impressed by the manner in which any reference to this subject is met by many ophthalmologists. There is complete contempt for the question of the surgical treatment of muscular abnormalities. As an instance of extreme opposition I was lately told by a man well known in our special line, when speaking of a patient whom I had cured by tenotomy of the superior rectus, that I ought to have been prosecuted for doing the tenotomy. Dr. Reber has handled this subject in a calm, judicious manner, and we should all try to help clear up this "dark region" in ophthalmic science.

DR. WENDELL REBER, Philadelphia—I am gratified at this expression of opinion. Dr. Stevenson's modification of the cover test is one I have employed for a long time, and I am not sure that he and I have not talked about it. I think the Maddox rod test the most trustworthy yet offered. We know very

little about the physiology of the ocular muscles. When we reflect that there are six pairs of muscles, that no one is concerned alone in any movement of the eyes; and that according to Volkmann there are something near 200 possible combinations of these movements, we will perhaps realize how empirical our present knowledge is.

As to Dr. Thompson's question whether I cut all the muscle, I would say that I do not do partial tenotomies, but complete ones. I shall do partial ones only when clinical evidence of a convincing nature has been produced.

DR. SAVAGE—Do I understand that you do a complete tenotomy on the superior rectus for 2 degrees of hyperphoria?

DR. REBER—Yes.

DR. WILLIAM THOMSON—Have you had many occasions requiring a restricting stitch?

DR. REBER—I have had overcorrection in one case and I preferred to trust the re-establishment of normal conditions to the patient's brain, believing that the brain can take better care of such temporary apparent overcorrections than the surgeon can.

OCULAR COMPLICATIONS OF INJURIES TO THE HEAD.*

JOHN T. CARPENTER, M.D.

PHILADELPHIA.

The clinical fact that blindness may follow blows on the head is by no means new, but until Berlin, in 1879, cleared up the pathology of these cases, their etiology was clouded in vague conjecture. Beer attributed the amaurosis following head injuries to the reflex influence of the supraorbital nerve. The result of anatomic studies, however, by Berlin, Leber, and Von Hölder, proved that direct damage to the optic nerve or its sheath in the foramen opticum by fracture of the orbital wall frequently occurred. Von Hölder, in his cases studied post-mortem, found fracture of the orbital vault in 90 per cent., while the optic canal was implicated in about 60 per cent. There were hemorrhages into the nerve sheath in about 48 per cent. of the 88 cases. The line of fracture in some instances passed through both foramina. In Treves' "System of Surgery," illustrations show the line of fracture in two cases passing through both optic foramina, so that the blindness may be bilateral; indeed, it may even be on the opposite side to the injury by the contre coup.

Following Berlin's communication at the German Congress in 1879, there appeared reports of optic neuritis, of atrophy, and of hemianopsia resulting from blows or falls on the head. Most of the reported cases were of monocular blindness, caused by such blows or falls, the subsequent optic atrophy showing that the nerve had been severed or compressed, producing descending degeneration, with the ophthalmoscopic signs of simple atrophy, in about three weeks. Ocular complications have been recognized by surgical authors as of important corroborative value in fracture of the base of the skull; and conjunctival ecchymosis appearing some hours or days after the injury is one of the classic signs of these fractures. Emphysema of the orbit or lids proved the existence of a fracture of the orbital walls communicating with the air cells in the neighborhood of the orbit. In 1882, Von Bergmann called attention to the fact that rapidly increasing hemorrhage into the cranial cavity produced the same disturbance in intracranial pressure that was found in brain tumors; and thus a "choked disc" might accompany cerebral hemorrhage, as well as tumor of the brain. Optic neu-

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