

Ocular Migraine and Prolonged Occlusion

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Von Storch once paraphrased Oliver Wendell Holmes with these words, "If I wished to show a student the difficulties of medical practice, I should give him a headache to treat."

If this statement was true in the days of Holmes, it is perhaps an under-statement of truth in the light of today's medical advancement. Moench refers to the headache patient as a medical orphan. He is perhaps more than this: he is the medical football; he is the man without a friend. It seems that any symptom or complaint that cannot be seen, X-rayed, felt, measured, graphed or analyzed eventually is relegated to the great medical wastebasket—neurasthenia. In this giant rotunda is found the last remains of many a medical diagnosis, either for lack of further interest or knowledge on the part of practitioners or for lack of funds and discouragement on the part of the patients. With specific regard to head pains (headache or cephalalgia), the mild form goes into the "nerve tension" basket and the severe form goes into the "migraine" basket. In these categories, they can be so easily forgotten—so easily explained; but for one thing—the patient must still suffer the many times excruciating symptoms.

Migraine Cephalalgia

As the title of this article will undoubtedly elicit much skepticism on the part of those who are very exacting in their concepts of migraine cephalalgia, let us first consider the meaning of the term "migraine."

The word "migraine" itself first began as the Latin word *hemicranium*, meaning unilateral or possible "splitting" headache. This was later corrupted to the low Latin *hemigrænea*, then to *emigrænea*, *migrænae*, *migræna*, the French *migraine* and the English *migraine*, *megrism*, *migrim* and *megrim*.

In the strict sense of the word, the term "migraine" is accepted to describe only a classic symptomatology. This syndrome is a violent, unilateral head pain, generally preceded by visual disturbance called teichopsia, or scintillating scotoma and hemianopsia on the affected side. The pain is accompanied by nausea and vomiting and followed by exhaustion and sleep. In most cases, the patient is in good health and no systemic malfunction is evident. There is general evidence of another member of the family suffering in like manner. In many cases there are prodromal symptoms that vary

greatly. Hours or days before there may be a lack of a sense of well-being, malaise, irritability, restlessness or emotional depression. It is perhaps during this period that many have seriously contemplated suicide, rather than continue through life suffering such periodic anguish. (This fact has been confided to the author on numerous occasions in cases of very frequent migraine symptomatology or in cases of extreme constant cephalalgia.) The duration of the attack varies from 15 minutes to 10 days and will average from 12 to 24 hours.

In summation of hemicrania simplex, it may be said that although numerous experiments have alleviated some cases of suspected migraine and much has been learned of the symptomatology, nonetheless, no responsible organism or pathology has as yet been demonstrated. Thus, a true, unattenuated migraine patient can look for little lasting relief from medication.

In addition to the classic type of migraine, there are sub types with special features and variations which are accepted in medical circles and must be considered.

Abdominal Migraine: This term is employed to describe a condition occurring largely in children of migrainous parents in which abdominal pain is associated with migraine or a substitution for the headache. This type of periodic abdominal pain may persist after the migraine has vanished, while at times cerebral and abdominal syndromes alternate. Kinnier Wilson stated the belief that if abdominal symptoms occur periodically without a history of any cerebral migraine, the diagnosis of abdominal migraine is extremely difficult and may well be wrong.

Ophthalmoplegic Migraine: This term is applied to a syndrome characterized by attacks of severe headache and paralysis of the ocular muscles, with ptosis as a very frequent symptom. The course of the attack is about as follows:

The patient develops a severe headache, which is localized to one side, starting as a rule at the temple and spreading to the occipital and frontal regions. The headache is not neuralgic in character, but is of the constant, dull, harrowing type as occurs in migraine and brain tumor. It is accompanied by nausea and vomiting, but not by scintillating scotoma, nor any of the visual aura common to migraine. After suffering with the headache any-

where from one day to a week, a paralysis of the oculomotor sets in on the side of the pain. The headache and nausea usually precede the paralysis and abate as the latter develops, but there are cases in which this order is reversed. The pain is localized on the side of the paralysis and if it extends to the other side, is more intense on the paralyzed side.

In most cases, the muscles supplied by the oculomotor are affected. The paralysis is usually complete, but cases of isolated paralysis of a single ocular muscle supplied by the third nerve are reported.

The term "ophthalmoplegic migraine" was first suggested in 1890 by Charcot. However, the first recorded case of this type was reported by Gubler in 1860. In 1884, Moebius, in his paper, referred to "recurrent oculomotor paralysis." The newer term of Charcot's, however, was so descriptive of the clinical picture, it found ready acceptance and since then, practically all cases of this type have been reported under this term.

Precardial Migraine: This type of migraine is characterized by intense pain around the heart without any evidence of disease which might produce the pain. This condition was reported by Fitz-Hugh as occurring in 20 per cent of 880 patients with migraine. It may occur as a "substitute" for headache and can be confused with angina pectoris.

Faciolegic Migraine: This is an uncommon type associated with Bell's palsy. It commonly involves the chorda tympani, and may produce a metallic taste. When there is involvement of the tensor tympani innervation, there may be a roaring or clicking sensation in the ears. However, some recent research would indicate the distinct possibility that a subluxation of the temporo-mandibular joint may be the causative factor of the aural symptoms.

Ophthalmic Migraine: This term is used to describe the ocular manifestations of migraine, such as recurrent scintillating scotomas, teichopsia and hemianopsia without headache. Such forms either may occur or be preceded or followed by hemicrania simplex.

It is the intent of this article to add to this classification the new designation: *ocular migraine*. To understand this term, there must of necessity be a slight departure from the classic syndrome of simple or common migraine. It is to be noted that even the above-accepted sub-classifications depart materially from the symptomatology of hemicrania simplex. It will be further noted that there is a greater disparity than will be found in ocular migraine.

As is so often true with a descriptive term, common usage and acceptance over a period of years many times changes the original meaning. English has had what is known as *semantic change*, affecting the meaning of words. Many words, while usually retaining

earlier meanings, have developed new ones, together with figurative uses, specialized uses and differences of various other kinds. The whole history of the language has been one of change. This is by no means a disturbing factor because the new concept may be more descriptive and more inclusive than originally was conceded. As has been brought out heretofore, the original meaning of migraine was unilateral cephalalgia, or hemicrania, of severe intensity, with accompanying visual aura. Popular usage and concepts, however, have come to include as migraine any severe, incapacitating headache of previous undetermined origin, whether unilateral or bilateral. This is especially true if nausea, vomiting, vertigo and photophobia are present.

To take the original Latin word *hemicranium* and to think of its alternate translation, that of a "splitting" headache, is to better understand the modern concept of migraine. It is common to hear of a headache of severe intensity described as a "splitting" headache, with no regard to unilateral or bilateral definition. In taking hundreds of case histories of these medical "orphans," it is common to hear of numerous past medical diagnoses of migraine, with no due regard to location, duration or time of onset of the particular headache. Many patients report little or no case history having been taken specifically concerning the headache. Many report a very languid attitude on the part of internists and other specialists in reference to the complaint of headache and invariably come away with the usual prescription for empirin or codeine to alleviate the pain.

Another popular misconception in medical, ophthalmological and optometric circles is the supposition that a severe chronic headache, with many times a symptom syndrome closely allied to common migraine, is not and cannot be of ocular origin. Such headaches not only can be of ocular origin, but also if a thorough medical examination does not reveal the source, they undoubtedly are of an ocular origin or partial ocular origin, which can only be discovered by prolonged monocular occlusion. When reference is made to a thorough medical examination, there is not implied a thorough, one-visit examination, but a most careful analysis to include histamine, allergies, foods, circulatory, brain tumors, sinusitis, etc. These investigations may cover many months and even years of conscientious work.

Rea in his book "Neuro-Ophthalmology" states that constant headache is of grave prognostic significance, but he continues by stating that any headache which is paroxysmal and wakens the patient at night is not the type that is produced by disturbances of the ocular apparatus.

While this statement is correct in that these headaches are of grave import and should be carefully screened for possible cerebral involvement, nonetheless it is entirely false to

state that such headaches cannot be produced by ocular disturbances. In my files are numerous cases of this type that have been completely or materially helped by ocular therapeutics, prescribed following prolonged occlusion.

New Concept of Vision

It is strongly felt that one of the major reasons for this widespread belief that ocular disturbances do not produce migraine cephalalgia is the mistaken concept that vision is limited to the organ of sight—the eye. Gesell, in his monumental work entitled "Vision—Its Development in Infant and Child" brings out that vision is a dominant act of a being which controls the very development of the organism. It is an integrated activity bringing into play all other senses. Spatial orientation is a complex factor involving far more than just the act of seeing. Vision is the over-all concept that includes enormous areas of the cerebrum and the autonomic nervous system and it is identified directly and reflexly with the skeletal musculature from head to foot. Vision is so pervasively bound up with the past and present performances of the organism that it must be interpreted in terms of a total, unitary, integrated action system.

If one thinks of the act of seeing in this much greater scope, he will realize there is far more to visual anomalies than hyperopia, myopia and astigmatism. Vision is more than a camera-like focusing mechanism for light. It is a complex sensory-motor response to a light stimulus mediated by the eyes. It explains the great variance in response to sensory impulse. One organism may be very critical of a disturbance to a receptor or sensory mechanism. It may set up a chain of motor responses quite complex to make adjustments to these sensory stimuli; while another organism may make a much simpler adjustment. It may be much less disturbed by the same stimuli.

These inherent variables undoubtedly account for the wide variation of the pain syndrome in different individuals. Case histories will reveal that some patients can hardly describe a headache. It is practically an unknown quantity to them. Regardless of sickness, dissipation, visual anomalies or any other variation from normalcy, they just do not suffer from cephalalgia. Conversely, there are people who suffer with almost constant headache, varying anywhere from a light or dull pain to an extreme incapacitating type. These organisms have a very complex adjustment to any sensory stimuli, and this factor must be taken into consideration when dealing with the headache patient. The more severe the pain, the more exacting must be the technique of the physician, ophthalmologist or optometrist in dealing with the problem. He must realize that a minute variation to that individual may be more important than a much greater disparity to another.

Specifically relating this thought to vision, Gesell brings out the idea that lays a foundation for this concept of ocular migraine. He states that fixation becomes the most basic or primary visual function. Fixation is the directing or orienting of the organism so that a stimulus or an image falls in optimal relation to the visual receptor. All other visual functions are subsidiary to fixation or they are refinements of fixation. Fixation is the single unifying action system to which every act of vision must be referred. He further states that "an optimal adult visual mechanism might be defined as one in which the basic skeletal, visceral and cortical functions have attained full stature and operate in balance and harmony."

Thus, we see that if binocularity is in discord, if it is out of balance in relation to optimum performance, then corrections must be made through the cerebral cortex. Gesell states it thus: "The cerebral cortex thus serves a triple purpose in the organization of visual behavior. It corrects imperfections, when possible, in the primitive mechanisms; it reinforces these mechanisms through energy discharges; it introduces subtleties, substitutions and suppressions or accentuations. It organizes visual acts in terms of the optimal needs of the action system. Indeed, in last analysis, the cortex becomes the seat of action for the action system. It funnels and organizes the electrodynamic forces which culminate in adaptive behavior."

Now, the principal reason for efficient binocularity is fusion. It is a basic inherent desire of man to maintain fusion and single binocular vision. If there is an innate or acquired binocular disparity, the cortex will exert every force available to correct the imperfection and still maintain fusion. If this is impossible, then, as a last resort, monocular suppression or amblyopia are the only means available to prevent diplopia.

As fixation is primary and as fusion is mandatory for efficient binocular fixation, any binocular anomaly that creates a handicap to fusion then becomes an irritant which demands constant cortical supervision. It is this frustration set up in cortical levels that contributes to the migraine of ocular origin.

Vision as a Focal Point

Following this supposition that vision is incorporated into the total structure of the organism, we can further demonstrate the reason why ocular dysfunction can bring about cephalalgia to the degree of a migraine intensity. Nowhere else in human behavior can be found the intimate coordination between voluntary, sympathetic and cortical reactions. It is unique only to vision and brings to a great focal point the linkage between visceral, skeletal and cortical action. These three functional fields operate independently and conjointly, but not necessarily uniformly.

In vision, these three fields may be represented by the triplex of fixation-focus-fusion. Vision then becomes a sense that is in almost constant use and that must utilize all three of these independent neural systems. To get a clear understanding of this integration, we might liken vision to the long-distant flight of a modern six-engine bomber. There are primarily three men who fly this giant monster: the pilot, the navigator and the flight engineer. Each operates independently in his own specific specialty, yet each must operate with precision in coordination with one another. The moment the wheels leave the ground and normal climb is maintained, the engineer must change propeller pitch, change fuel-air ratio, watch manifold pressure and numerous other mechanical details that make for coordinated engine operation. The pilot must retract the wheels, watch air-speed, altitude, course and direction, maintain radio contact with ground stations and attend to many other flight details. The navigator must watch course and direction, must maintain constant radio vigilance for the "beam" signal, must plot speed, direction, wind drift and watch for check points as the flight progresses. Thus, we see that the pilot flies the plane, the engineer operates the engines and the navigator plots and maintains the course. The entire job is too big for one man, so if one should become inefficient and allow error to creep into his actions, then the success of the flight must depend upon the adaptive capabilities of the other two.

Thus, we see in vision that if there is any lack of harmony between fixation, focus or fusion, there must of necessity be inefficiency or excessive stress on reciprocal structures. If the organism can accept inefficiency in one realm or the other, there will be little or no discomfort. If, however, that organism still tries to maintain high efficiency in the face of malfunction, then disturbing symptoms will appear to warn of the conflict.

Although the human organism, through its numerous paired organs, seems to be constructed on the basis of bilateral symmetry, it shows a consistent trend toward functional unilaterality. This is found in dominant tendencies of eyes, hands, feet and torsal sidedness. In the development of ocular and manual behavior, this means recurrent alternation and reciprocal interweaving of right and left components and of monocularly and binocularly. Two pairs of opposing trends are in mutual rivalry—bilateral versus unilaterality, and right versus left.

Sherrington's law of reciprocal innervation describes a physiological mechanism. He demonstrated that the inhibition of one set of muscles, while the opposing muscles are in excitation, is a condition for effective movement. However, if two opposing muscles are in excitation at the same time, or even if excitation of minor degree should exist in one,

there is set up a frustration or an impasse resulting in a nervous tension. These neural prostrations invariably result in irritating symptoms, with the degree depending upon the intensity and duration of the impasse and the sensitivity threshold of the organism. They account for many headaches, rightfully classified as neurasthenic or migraine, but wrongfully allowed to continue due to lack of proper investigation as to the source of the tension. It is in this area that a careful investigation of latent binocular function, by means of a proper prolonged monocular occlusion, brings to light a faulty reciprocal innervation involving bilaterality and unilaterality. This faulty innervation results in a visuo-neurasthenia, which in turn sets off a cephalalgia, with the intensity depending upon the sensitivity threshold of the individual, the amount of binocular deviation and the length of time the condition has been present.

It is here that consideration must be given to this word *neurasthenia*. It is used herein in its original interpretation, that of a "nerve debility." Dorland's Medical Dictionary identifies the word as a "nervous prostration." The definition continues, "It is usually due to prolonged and excessive expenditure of energy and is marked by tendency to fatigue, lack of energy, etc."

However, in consulting with internists, neurologists and psychiatrists as to the commonly accepted meaning of the word in medical circles, we find it has grown to imply a psychic origin. It is generally associated with emotional or mental tensions, which more accurately would be described by the word *neurosis*. It is unfortunate that the word *neurasthenia* should imply the psyche or mind because its technical meaning is unique in describing nerve fatigue.

There are many words that imply the possibility of nervous exhaustion or refer to nervous energy disassociated from mental origins. For example, the word *neurolysis* means, "exhaustion of nervous energy." The word *neurorrhoea* (nerve-flow) means, "nervous energy." *Neuricity* describes "the specific energy peculiar to the nervous system."

From the study of these words describing an innate function we call nervous energy and also this complex function we call *vision*, which is the over-all concept that includes skeletal, visceral and cortical functions, we can conceive of a nerve fatigue, or neurasthenia, due to faulty binocularly and not to an emotional or mental problem.

For this reason, we shall refer to a visuo-neurasthenia as a basis for ocular migraine and its related symptoms. Perhaps the following discussion of the psycho-ocular reflex will further explain the possibility of visuo-neurasthenia.

Psycho-Ocular Reflex

Lancaster and Kenelly estimated the force of the pull required to rotate the eyeball to

be from 1.0 to 1.75 Gm., calculated on a basis of a diameter of 24 mm. and a weight of 8.0 Gm., and neglecting friction. Even making an extra allowance for the extra weight and drag of the optic and ciliary nerves, the arteries, veins, conjunctiva and fascia, a safe estimate would be 5 Gm. to move the eye at its observed velocity. Thus, speaking purely of muscular energy, we find the extra-ocular muscles powerful enough to pull 100 times the amount needed to move the eyeball.

This gives us ample evidence to prove that heterophorias are by no means due to a muscle weakness, as is so commonly stated. We find from this fact a reason why a person should be able to move the eyes back and forth across a page for hours without fatigue. Lancaster estimates that less than five per cent of the oculorotary muscle fibers are made to contract at any one time. Scobee estimates less than one per cent, although he grants an extra allowance for tonus. Therefore, in movements created by the oculorotary muscles before the first set of fibers become fatigued, the task is shifted to a different set, and so on. By the time it becomes necessary for the first set to act again, they may have long since recuperated completely. This is in complete accord with the work of Mosso and others on finger muscles. In this work, they found with a recording ergograph that the finger could raise a light weight up and down indefinitely with no falling off in amplitude. Only when the weight exceeded a critical magnitude did the curve show a falling off.

From this data, it would be inconceivable to think of heterophorias in terms of muscle weakness, as we can easily see that muscles merely do what nerves tell them to do. Going further in this reasoning, we realize that nerves merely transmit messages from motor nuclei. Then, these nuclei are coordinated and controlled by a supra-nuclear mechanism that receives messages from all sources and arranges the proper pattern of stimuli to the nuclei to execute a well-chosen response. In other words, we find a complete reflex, but in this case, a very special type of reflex involving the will—that is, a *psycho-ocular reflex*.

Ocular Migraine Syndrome

The symptomatology of the cephalalgia of ocular origin takes on any and all so-called "classic syndromes" known to medicine. I have endeavored to classify the pattern of the pain as to location, onset, duration, etc., so that a simple diagnosis of ocular migraine might be established and there seems to follow no correlation.

This fact has been verified by George A. Woodruff, M.D., to whom I am eternally indebted for his collaboration on this study of ocular migraine. After working and counseling together over chronic cephalalgic patients for a period of three years and listening to hundreds of histories, we are unable to know in

advance if the cephalalgia is of ocular origin, except in some cases where an exhaustive history will reveal increased discomfort following specialized visual tasks. Our experience has shown only one sure way of knowing if a particular therapy will alleviate a headache and that is from the patient's report after the particular therapy has been applied.

This is true because in headache therapy we are dealing with a symptom and not a specific condition. In this type of clinical research, we are tabulating symptoms and changes of symptoms, with only secondary regard to any specific tests or findings. It is based purely on the patient's feelings; hence, a close working relationship must be established between doctor and patient and both must sense an appreciation of what is being done. In laboratory experimentation, the practitioner can rule out the patient as an individual and establish a therapy based on the patient as a human organism with certain responses to laboratory tests not involving the individual's feelings. This is impossible in dealing with headaches, as we are confronted only with symptoms.

To aid in this symptomatology, we find that a chart accurately tabulating these symptoms and kept over a given period of time is not only valuable, but in some cases absolutely necessary. This "Headache Diary," as it is called, is a mimeographed sheet ruled off to include daily entries of the following information: date, time headache began, location, time headache left, time retired the night before, nausea, vomiting, vertigo, intensity of pain, medication and remarks. This information is all contained on one line and can easily be sketched-in each evening.

The following are some typical histories taken from my files. They illustrate ocular migraine as being a true medical entity for which there is no cure except through a most accurate visual analysis and a prolonged monocular occlusion test to establish the nature and amount of latent binocular dysfunction which has been causing the visuo-neurasthenia.

Mrs. H. S. B. Dec., 1950. Housewife, age 39.

Had been suffering with severe cephalalgia for three years prior to this case study. Had had very extensive and exhausting medical tests. Was even confined to hospital on several occasions while tests were given and the pain was kept under control by opiates. The extreme pain was generally accompanied by nausea and vomiting. On many occasions, there was a very annoying vertigo. Numerous times the pain was so intense that her physician was called to the home to administer sedatives intravenously. There was intense photophobia.

The migraine attacks would come in waves, extending over a period of perhaps three or four months at a time, then would recede for a short while, then reoccur. The two months prior to this case study, she was spending from three to four days out of each week in bed, with no relief other than opiates. In be-

tween these intense attacks, she had almost a constant dull background cephalalgia that was relieved with large dosages of aspirin.

These headaches would awaken her between three and five o'clock each morning, beginning in the occipital area and working up to the vertex and then seemed to be "all over." They were always bi-lateral, but the pain was so intense, localization seemed impossible. It was also noted that under these migraine attacks, the eyes themselves were sore.

For one year she had worn the following Rx constantly: O.D. -25 -50 × 180 O.S. -75 × 180. This was her first lens prescription and although she reported slight improvement in acuity, there was no recognizable relief of the cephalalgia.

First analysis showed subjective findings of: O.D. -50 × 5 O.S. -75 × 5, with distance lateral phoria of 4Δ exophoria and distance vertical phoria negative. The vertical ductions were O.D. 2:1/2:1 O.S. 1:0/2:1. Prolonged monocular occlusion was instituted and final results revealed 5½Δ exophoria at distance with 1Δ right hyperphoria. Vertical ductions were: O.D. 1:0/2:1 O.S. 2:1/1:0. On the basis of these findings, the following lenses were prescribed in No. 2 shade tint: O.D. -50 × 5 1½Δ down and 1Δ in. O.S. -75 × 5 1Δ in.

These were given with instructions that they would be temporary lenses while keeping a headache diary to observe the pain syndrome. Ten days later, the patient reported absolutely no headache, with not even a tendency to one. These were neither the constant dull type, nor the severe morning headaches which had been so frequent. There was no soreness around the eyes and she reported she was feeling wonderful. A month later the headache diary was abandoned as no entries were made subsequent to her lens prescription. Periodic reports still showed no recurrence of the migraine syndrome and the last analysis was made March 17, 1952. There was no change necessary in this correction and she still reported freedom from headaches as long as the lenses were very accurately placed before the eyes. Any tilting or sliding of the frame had a tendency to bring back light headaches immediately, much as in the cases involving aniseikonia.

Mrs. M. G. January 15, 1951, Housewife, age 42.

This woman has always enjoyed sewing and is an artist in any phase of sewing. She makes her own clothes and those of her daughters in a most beautiful and professional manner. Her history of cephalalgia dates back from 12 to 14 years when she was doing very fine and fancy sewing and doing it almost constantly. She had spells of vomiting, lasting 30 days at a time and no etiology was discovered. Her husband being in the Navy, she was treated in Naval hospitals, spending many weeks going through extensive tests.

During these past 12 to 14 years, she has been to numerous physicians and in no case could systemic malfunction be found to cause the headaches, nausea and vertigo. These pains were the same in winter as in summer, yet she always had a more severe setback each spring. During the spring of the year, she experienced loss of weight and nervous exhaustion. She had had numerous eye examinations during this period, with only a routine visual problem discovered at any time.

Ten years ago, she gave birth to a baby girl by Caesarean section. Following this she had constant cephalalgia for 10 and one-half months with never a letup. She was never free from pain and the physicians never found a reason. In 1946, she had a complete hysterectomy in the hope that this might alleviate the cephalalgia, but it did not.

Her symptoms, as recorded in her history at this time, included bilateral occipital, vertex and frontal cephalalgia. She was very photophobic. Also post-cervical tension of fluctuating degree. The cephalalgia was mostly of the A.M. type, causing her to awaken anywhere from 3 a.m. to the usual time, with varied degrees from a mild to a severe pain. About three weeks prior to this appointment, she had a most severe attack, at which time the right side of the face was numb. There was frequent vomiting and she, of course, was confined to bed. She reports she had difficulty in thinking clearly following these migraine attacks and she had to grasp for words when talking—descriptive of verbal aphasia. She had frequent spells of vertigo that would come in cycles. At times being so bad she would fall if not supported.

Her current prescription, which was about 18 months old and a result of refraction under cycloplegics, was: O.D. +300 -50 × 100 ½Δ down; O.S. +325 -100 × 90. (Note: This vertical prism was evidently there as a result of the optician's work, not as a part of the prescription.)

The first visual analysis showed subjective findings of: O.D. +300 -50 × 90 O.S. +275 -50 × 90. Lateral distance phoria was 2Δ exophoria and vertical phoria was negative. Ophthalmoscopy was negative.

Monocular occlusion was instituted the last of January for six days, causing the following changes: first and foremost was that her symptoms almost entirely disappeared under occlusion. There was only a slight ache of the occluded eye and very little nausea. There was no vertigo and in general she felt much better. Second, the exophoria at far jumped to 6Δ exophoria and the vertical phoria changed to 1Δ left hyperphoria. Prism fit-overs were used to get an effective 3Δ in and 1Δ down O.S., and the headache diary kept each day until into April. This diary is on file with a complete record of all appointments.

This prism alleviated all headaches while doing distance seeing. Even went to movies

and could sit through a whole feature with no discomfort. However, because of her beginning presbyopia and increased exophoria at near, she could still not read and sew without producing some headache. Therefore, on March 13, visual training was instituted and added plus in fit-overs for near work. One week later she reported noticeable improvement even in near work and her general symptoms. By the first of April, she reported practically no headache of any kind; also no vomiting, which had been a daily procedure prior to monocular occlusion. Her last training session was April 13 and on April 18, the following corrections were prescribed: distance: O.D. +275 -75 × 90 1½Δ in O.S. +250 -75 × 90 1½Δ in and 1Δ down with tint No. 2; near: O.D. +375 -75 × 90 2Δ in O.S. +350 -75 × 90 2Δ in and 1Δ down.

By then, she reported that during this spring she had actually gained weight and had so much more vitality and reserve, while before, it was always a severe depletion after a winter of extensive sewing. She reports only an occasional mild cephalalgia, but nothing of any consequence. No more nausea nor vomiting and no more vertigo of any degree. She is seen regularly for frame adjustments and at every visit is questioned, but always the same result—no recurrence of the chronic symptoms of the previous 14 years.

Mrs. E. S. May 9, 1951, Housewife, age 47.

Referred by the last physician who saw her. This woman for four and one-half years had been afflicted with a constant unilateral cephalalgia that was present night and day. She never experienced relief no matter what hour of the night or day. Sleep came only from utter exhaustion and no amount of medication gave any relief. In the four and one-half years previous to this visit, she had been to 25 of the finest medical doctors in this area. These included internists, psychiatrists, neurosurgeons, ophthalmologists, etc. All were highly recommended; all tried every therapy they knew, yet not once did any therapy lessen the intensity of the pain to even a slight degree. Needless to say, she had spent hundreds of dollars in her effort to obtain relief.

The pain was left unilateral involving frontal, temporal, occipital and aural areas and extending down into the maxillary bone and even into the teeth. Also she had a skin sensitivity that prevented her going to the hairdresser. Along with the pain was a most intense photophobia. Her last refraction had been a year previous, at which time no lenses were prescribed. Her history shows her unable to do any reading or sewing—she could take no interest in either.

Visual analysis showed O.U. plano on subjective. Distance lateral phoria was 1Δ exophoria. Vertical phoria was negative with vertical ductions O.D. 1:0/3:2 O.S. 2½:1/1½:½. Horizontal ductions were within normal limits. Cross-cylinder test at 16 inches revealed pres-

byopia of +125 O.U. with 10Δ exophoria. Maddox rod test showed no cyclophoria. Amplitude of triangulation revealed diplopia at 5 inches. Rotations and versions were a series of jerks. Cover test revealed no tropia. Motion fields and blind spots were within normal limits, but form and all colors were restricted. Telebinocular DB series showed into exophoria column, with fusion and stereopsis normal.

The first six days of monocular occlusion revealed 10Δ exophoria at distance and 2Δ right hyperphoria with only a very slight lessening of the headache. However, one very important note was that the pain was more intense in the left eye, but lessened elsewhere. Of significance was the fact that this was the first therapy or test in four and one-half years that had altered the syndrome even slightly. Also, it was significant that the last two or three nights under occlusion she slept better than previously. If she awakened, she could get to sleep again more quickly.

Not being satisfied with the results, we occluded the O.D. again for another six days and kept a dark green lens over the O.S. At the conclusion of this period, her headache diary revealed that the last four days of this 12-day period, the headache was completely gone, except for a light residual ocular pain. Her sleeping was almost normal and needless to say, her general attitude and spirits were greatly increased. At this time, a temporary Rx of 2¼Δ down on O.D. and 4½Δ in O.S. in tint No. 4 was prescribed.

There followed, from the last of May to the first of July, intermittent headaches, which slowly increased in frequency. On July 3, a re-examination showed vertical phoria was now 4Δ right hyperphoria, so new lenses were made with 4Δ down O.D. and 6Δ in, equally distributed in both lenses. These again gave relief for a short time. By October 16, the intensity of the headache was back again, almost to the severity of before beginning occlusion. However, we had a period of about two months of almost complete relief. This encouraged us to keep on. Thereafter followed several refractions and an attempt at visual training, which failed because the pain was too intense.

In the last part of 1951, while at lunch with a dentist friend, we were discussing migraine headache and he mentioned a new problem to dentistry concerning a subluxation of the temporomandibular joint and its relation to headache and nerve tension. It is a new subject, called centric relation, and is unknown and unheeded by many dentists and very little is written on the subject. Yet, he knew of cases of headache that had been relieved by proper therapy. As a result, Mrs. E. S. was referred to him, still wearing the 4Δ D.O.D. and 6Δ in, in No. 4 shade color. His tests revealed that there was subluxation of the temporomandibular on the left side and temporary plastic bite raisers were inserted for a trial period. Almost immediately all of the remain-

ing headache disappeared. He then did the permanent work on the teeth. Then new lenses were made to cut the color down to a No. 2 shade. Immediately headaches returned and there was no relief until the darker lenses were again prescribed, thus showing the import of photophobia control.

Her final prescription was: distance: O.D. +50 -50 × 35 with 1¼Δ down and 3Δ in; O.S. +50 -25 × 135 with a 1¼Δ up and 3Δ in with No. 4 tint. For near she wears: O.D. +150 -50 × 35 with 2Δ down and 5Δ in; O.S. +150 -25 × 135 with 2Δ up and 5Δ in, in white. She is still perfectly comfortable, with no trace of the original symptoms. Again, however, her lenses must be adjusted like aniseikonic lenses when it comes to accuracy.

This most interesting case well illustrates the fact that migraine headaches may frequently be a result of multiple etiological factors rather than a single factor. In this instance, the discovery of the latent phoria was the key to the problem, yet the centric relation and photophobia control were equally essential for total relief.

It may well be noted here that if a headache case study on the part of the optometrist reveals a latent phoria and then prism therapy fails to relieve the complete cephalalgia, *that prism therapy is necessary as a part of a total problem and should remain as a part of the visual therapy.* These cases of multiple etiological factors can be very disheartening, unless there is a close cooperation from practitioners in allied professions.

The above-described history also reveals a departure from our established standardized technique of monocular occlusion and is one of the rare exceptions that proves the rule. Because an exact technique is so important, the next phase of this article deals with prolonged monocular occlusion and the standardized procedure used.

Prolonged Monocular Occlusion

In 1920, Marlow published a paper entitled, "Prolonged Monocular Occlusion as a Test for Muscle Balance." This indeed was a monumental work, but evoked a storm of protest from his colleagues who immediately claimed the unreliability of the test. With very little support and a vast amount of protest, the validity of the prolonged monocular occlusion test was questioned by ophthalmologists and optometrists alike. A typical sentence that carries the finality of the last rites is quoted from Krinsky as recently as 1948; "Beisbarth and Abraham and others exposed the unsoundness of the occlusion test and it required a wealth of literature to finally show that prolonged occlusion is of no value in diagnosis." The next sentence refers to Swan's experience of esotropia following occlusion that resulted in three lawsuits. He then refers to Marlow's 1938 writings as being a final admission that his occlusion was in reality producing artefacts

that had no relation to the normal physiology of the eye.

Scobee, in his 1952 edition of "The Oculomotor Muscles," devotes one paragraph to Marlow's prolonged occlusion and quotes a few of Marlow's critics, such as Cridland, who said, ". . . it has evoked a voluminous outpouring of unscientific hyperbole and almost hysterical vituperation."

Maddox, objecting to it, said, ". . . a derelict machine is not so informative as a functioning one, although we can learn something from it."

Abraham (1931) studied six cases in which he occluded each eye in turn and every occluded eye developed hyperphoria. He said, bluntly, ". . . it is a subjective test for demonstrating Bell's phenomenon and it is not a test for latent heterophoria."

Scobee's comment, in conclusion of the above quotations, is: "It is clear that the position of rest revealed by prolonged occlusion is close to the physiologic position of rest, but can never attain it because the fixation reflex (monocular) comes into play. The hyperphoria thus revealed in nearly every case very strongly suggests a persistence of the protected position of the eye in sleep, the persistence of any abnormal position being well established. It is a method which demands the greatest caution in its interpretation."

In the face of such overwhelming authority as to the impractical value of monocular occlusion as a diagnostic test, it would only be the stouthearted or the curious who would attempt any practical therapy based on occlusion. This is indeed a tragic mistake—a costly mistake that has set back the progress of headache therapy by at least three decades because a properly performed prolonged monocular occlusion test, rightly interpreted, is the key to relief for multitudes of chronic headache patients.

Cephalalgic symptomatology is a complicated and highly specialized field that demands the closest cooperation of practitioners of allied professions. It is a field which the average practitioner in any profession rather avoids because of the tremendous complexities and yet, when success ensues, is a field which is tremendously gratifying to both the patient and practitioner. Headache therapy demands perhaps more patience on the part of both doctor and patient than any other diagnostic procedure. A systematic routine may extend over several years of periodic testing and trying pharmacotherapy, physiotherapy, psychotherapy and visuo-neurotherapy. It is with this last phase that the optometrist becomes identified because of his high specialization in the visuo-neural field. Without the optometrist and this investigation, headache therapy is very incomplete.

Visuo-Neurasthenia

Visuo-neurasthenia is the cause of ocular migraine and other related neural symptoms

and can be discovered in many cases only by prolonged monocular occlusion. Many patients who on the first visual analysis reveal absolutely no alteration from accepted norms of postural binocularity will reveal, after prolonged occlusion, enormous amounts of deviation.

Going back to Sherrington's law of reciprocal innervation, we find that the extra-ocular muscles serve as agonist and antagonist in monocular movement, thus constituting a unilaterality even in a binocular act. This unilateral dominance is integrated into the binocular act by an extremely complicated cortical supervision.

As has been brought out before, a neurasthenia is produced when there is a fault in reciprocal innervation resulting in an impasse. Should the antagonist innervation for any reason be stimulated so as to oppose the stimulation of the agonist, then movement and efficiency are impaired. Thus, to achieve the original goal, the agonistic innervation must be increased to compensate for the lack of inhibition in the antagonistic sphere.

To illustrate simply this unilateral impairment of a binocular act, let us take the example of a small degree of hyperphoria. There is no tropia, there is no diplopia and yet under disassociation there is evidenced this faulty bilateral coordination. On the hyper eye, under disassociation, the superior rectus receives greater stimulation than does the inferior rectus—yet under associated binocular stimulation, there is no evidenced diplopia, hypertropia, suppression or amblyopia. There is only one reason for this single binocular vision and that is that the organismic demand forces cortical supervision into service and extra stimulation is sent to the inferior rectus as a compensatory mechanism. However, there has been no inhibition to the superior rectus and, consequently, neurasthenia will develop in time.

It is here that the secret of latent phorias can be understood and explained.

Should an individual have an increased unilateral stimulation to one or more of the extra-ocular muscles and organismic demand creates the counter cortical stimulation to maintain fusion and this situation exists for many years, then the constant repetition of this stimulation creates a fixed pattern that even under disassociation merely reveals this habitual cortical pattern to maintain normal response. This is why so many patients suffering from migraine cephalgia, extreme vertigo, nausea, vomiting, motion sickness, inability to read over prolonged periods, extreme post-cervical tensions extending to spine and shoulders and general symptoms of malaise and neurasthenia, must continue to suffer these symptoms because even the most careful visual analysis will not reveal these latent phorias. Consequently, it has been felt that there is no correlation between such symptoms and the visual act.

Standardized Technique for Prolonged Occlusion

Perhaps one reason why there has been so much criticism of monocular occlusion is because no one has attempted to standardize a practical, useful procedure that could be a part of general office routine. Marlow advocated a period of two weeks. Other authors have advocated anywhere from a few hours to two weeks. Some have occluded only one eye. Others have done both alternately.

Therefore, in standardizing a technique, there are two factors of import: (1) it must get results that are practical and useful; and (2) it must cause only a reasonable amount of discomfort and time-loss to the patient. If the time involved is so extensive that the patient feels it is impractical, then the test loses its meaning because it cannot be used. If it is not long enough to produce the desired results, it will be discarded as unscientific, impractical and archaic. Therefore, I have used various methods, from one day to three weeks, and from occlusion of only one eye, to alternate occlusion of both. There has been a constant endeavor to create simplicity, along with efficiency.

The result has been a six-day occlusion period equally divided between the two eyes. After the first analysis, the patient is given an occluder pad with the following instructions: the date to apply the occluder; the date of the second analysis on the fourth day; and the date of the third analysis on the seventh day. This gives a period of three full days of complete occlusion on each eye. In some cases, an additional two days are required back on the first eye, but this will be explained later.

Materials Used

Before attempting any prolonged occlusion, there are certain materials that the optometrist should have on hand ready to facilitate the procedure.

First, would be the material for the actual occlusion. To date, I am dissatisfied with all currently marketed occluder pads; however, the most satisfactory seems to be the Elastoplast Eye Occluder, manufactured by the Duke Laboratories. These are much on the order of a band-aid type of adhesive dressing, with the patient removing the gauze backing and exposing the adhesive surface. The material is flesh colored and shaped to fit the orbit, with a gauze center to cover the eyelid. It firmly adheres to the skin all around the orbit and is easily removed—much easier and with less residue than adhesive tape. An adhesive dressing is necessary because complete occlusion is necessary.

Secondly, there must be a solution to remove any adhesive residue on the face of the patient when the occluder is removed. Some use isopropyl alcohol or ether, yet the strong odors make them objectionable. I use a solution called "Solitine," a liquid cream solvent produced by the Luralite Laboratory of Portland, Oregon,

and purchased through the local dental supply. It has a pleasant odor and is very effective in removing adhesive from the skin.

Third, there must be a good supply of plano prisms in 42 and 44 round, with base and apex etched with small marks, and also have the power etched on so they are easily filed. These should range in powers from $\frac{1}{2}\Delta$ to 6Δ . It is best to have these in $\frac{1}{2}\Delta$ steps to about 4Δ and then 1Δ steps to 6Δ . There should be a good supply of $\frac{1}{2}\Delta$, $\frac{3}{4}\Delta$, 1Δ , $1\frac{1}{4}\Delta$, $1\frac{1}{2}\Delta$, $1\frac{3}{4}\Delta$ and 2Δ as these are most commonly used. These trial plano-prisms are then used in clip-over frames or in regular flesh plastic frames and any combination of base-down and base-in can be obtained. They can be readily changed from one to the other and in just a few minutes, your first amount of prism is ready for trial. In many cases, the patient may receive several changes of prism correction before the headache is completely eliminated. These changes may be as little as $\frac{1}{4}\Delta$ change if it is for a vertical phoria.

Order of Occlusion

It was first felt that the order of occlusion was not important; therefore, some time elapsed before a semblance of order came out of the experimentation. Originally, we allowed the patient to occlude first whichever eye he wished. In this way, there can be many puzzling experiences. For example, one might occlude the right eye three days and find 3Δ right hyperphoria. Then, after the subsequent three days on the left eye, find a negative reaction. At the conclusion of such a test, it would be impossible to apply base-down over the right eye or diplopia would ensue—yet, had it been applied at the conclusion of the first three days, it would have been worn successfully.

To rectify such a phenomenon as described above, we now find that to satisfactorily consummate this case, it would be necessary for two more days occlusion again on the right eye. Had we occluded the left eye first in this instance, the six days would have been adequate, as the last three days occlusion would have been on the hyperphoric eye. Therefore, our proper order of occlusion, if it can be adequately determined beforehand, is most important in cutting down the time of monocular vision to the patient. It is also important to the practitioner because it cuts down the number of appointments with any one patient.

We now determine the order of occlusion by any evidence of hyperphoria we can glean in the original analysis. If the original vertical phoria findings are negative and the ductions read: O.D. 2:1/2:1 O.S. 1:0/3:2, we then have some evidence of a tendency to a left hyperphoria. Knowing that the last eye to be occluded should be the eye showing the greater hyperphoria, we would in this case occlude the right eye first. This meager evidence of a left hyperphoria is by no means factual, however,

and many cases, as illustrated, eventually will show a right hyperphoria and can even show an extensive right hyperphoria. Nonetheless, using this technique has cut down a great amount of needless trial-and-error.

In the event the vertical phoria is $\frac{1}{4}\Delta$ left hyperphoria and the ductions would read O.D. 2:1/2:1 and O.S. 2:1/2:1, we would still occlude the right eye first, unless there was a distinct right torticollis. In that event, we would be biased by the torticollis and occlude the left eye first. While on this subject of torticollis, this is a most vital factor to note on your case history. To date, we have never found a patient with chronic torticollis who did not reveal a hyperphoria following occlusion. This subject will be dealt with in a subsequent section.

In the event the vertical phorias are negative, the ductions are negative, there is no vertical deviation on the Jaques bichrome scopes, there is no evident torticollis and there is no diplopia manifest on a pencil-head-tilt test, then we routinely occlude the right eye first.

Even with this care, we periodically have a patient who must go two days longer than the basic six, merely because the hyper-eye was the first occluded and proper therapy cannot be instituted until the occluder has been removed from the eye revealing the greater hyperphoria.

Hyperphoria Following Occlusion

Perhaps one reason monocular occlusion has not been more universally accepted as a diagnostic routine is the fact of the heterogeneous nature of the findings following occlusion. Some, perhaps, have tried occlusion and because the result was so unpredictable, have become disheartened or felt that the answer was unreliable. If the human organism were mechanical or if all of its reactions were mathematical in nature, then this reaction would be permissible.

Each of us is cognizant of the human element. We are each a separate entity within ourselves. We are each a vast array of complex integration. It is this element that has been largely forgotten in optometry and ophthalmology. We have become so accustomed to refracting a patient, getting an "exact" prescription and expecting that patient to be happy with it, that the feasibility of trial-and-error does not enter our mind. We could take a great lesson from the allergist, who must patiently try one therapy after the other until results are obtained. If you wish to enter into this specialized field of headache therapy, you must develop untold patience and not become disheartened with one or two tries.

Upon reviewing well over 800 cases of prolonged monocular occlusion, the following types of hyperphoria are revealed:

(1) A hyperphoria will be manifest only when one eye is occluded, with a negative response following occlusion of the other eye.

(2) Each eye deviates upward and comes to rest above the horizontal plane. This is a bilateral vertical deviation, commonly known as a double hyperphoria or anaphoria. Sometimes, the degree of deviation is the same for both and other times, one will be greater than the other.

(3) The same kind of hyperphoria is found when either eye is occluded, but is usually greater on the side of the hyperphoria. For example, after right occlusion may be found 1Δ left hyperphoria, while left occlusion may reveal 2Δ , 3Δ or more left hyperphoria.

(4) In the fourth type, the relative position of rest of the two eyes is the same in kind and degree, whichever eye is covered. It is here that one can elicit a negative response after occluding each eye. Or, one can find a pre-occlusion 1Δ left hyperphoria and 1Δ left hyperphoria following occlusion of both right and left eyes.

In commenting on the above types, it can be said that type No. 1 is by far the easiest for which to prescribe.

Type 2 is by far the most difficult, especially if the amount of hyperphoria should be exactly the same for either eye. It is this type that would create the greatest pessimism and discouragement, yet there have been numerous occasions in which complete relief has been afforded to a patient responding in this manner.

Type 3 would be the second easiest type to handle, yet many times will demand several tries on prism power before success ensues.

Type 4, if response is negative after either eye has been occluded, will no doubt indicate the cephalalgia is not of ocular origin. This would be true, however, only if the symptoms continued throughout the period of occlusion. If the symptoms abated or ceased during the occlusion period, then cyclophoria or aniseikonia would be suspicioned.

Lateral Phorias Following Occlusion

Up to this point, all stress has been laid on the vertical phorias. This, perhaps, is true because it is the more important of the two, not because we have ignored this phase. We agree with Marlow wholeheartedly in that both vertical and lateral phorias must be taken into consideration, with the lateral often of as much and sometimes more importance than the vertical. Most often, the final prescription is a combination of both vertical and horizontal prism. Many cases show complete relief of the headache with just base-in prism and a very limited few with base-out. This is because the great majority of patients will show increased exophoria following occlusion, yet a few have gone into esophoria, with one patient showing a very high esophoria following occlusion.

It perhaps should be stated here that there is on record in Oregon two lawsuits of malpractice, created by an esotropia that was manifest following prolonged occlusion. In

these cases, the occlusion was for amblyopic children who exhibited an esophoria before occlusion. There is, therefore, some risk involved in occluding a high esophore.

When to Occlude?

The answer to the question, "When to occlude?" is found exclusively in the case history. Too much emphasis cannot be laid on this, the most important finding in the optometric routine. If your prescription sheets do not have ample space to take and record an extensive history of that patient, then do not attempt headache therapy. As has been said heretofore, a headache is only a symptom; therefore, every fact about that symptom must be recorded accurately before attempting any therapy. I have written history for 45 minutes on the first visit of some patient with very severe and frequent migraine headaches. Then, on each visit, a short history is taken of these symptoms during the period of occlusion or the period of wearing the trial prisms. Only when the patient reports that the original symptoms are gone can one consider the case a success.

The subject of taking a proper history of the chronic cephalalgic patient would entail an article devoted exclusively to that matter and will not be attempted herein.

How Much Prism to Prescribe?

It is the answer to this question, not only as it relates to prolonged monocular occlusion, but also in any manifest binocular disparity, that has been long lacking in optometric and ophthalmological literature. The reason it has been lacking is the same reason as I explained earlier: the vision specialist is so accustomed to the comparative simplicity of obtaining an "accurate" prescription for myopia, astigmatism and hyperopia that he is looking for a mathematical equation to assist in prescribing prism. When he tries repeatedly to find a formula and none is adequate, then he feels that because it is not mathematical, prism is of no value. Hence, thousands are referred to psychiatrists for neurasthenias and chronic and migraine cephalalgias, merely because the ophthalmic specialists are afraid to try the second prescription on a patient. We have so long trained ourselves that we are either prescribing "glasses," or "selling glasses," that to think of a lens prescription as a medication to be tried in various dosages has not entered our minds.

Why does one hypothyroid patient respond to one-half grain of thyroid medication, while another may need one grain and another two grains? Why must the internist try repeatedly with prescriptions of varying dosages to accomplish a cure? The answer is simple—we are dealing with the human organism, not a mechanical machine where all parts either fit or do not fit.

When you prescribe prism, you are affecting the psycho-ocular reflex; you are releasing tre-

mendous areas of the cerebrum from crippling neurasthenias; you are affecting personalities; you are preventing suicides, which often result from acute depressions. Therefore, is it any wonder there is no mathematical solution to such a complex structure?

However, complexity does not absolve us from our responsibility. For this reason, I have advocated the use of prisms in clip-over frames or in temporary plastic frames to be changed as many times as necessary to obtain relief. There are several cases in our files that extended over or near a year before final success was obtained. It depends upon many factors, but the sensitivity threshold of the patient predetermines it to a great degree. There have been instances of $\frac{1}{4}$ of a diopter of prism being the difference between partial and complete relief. There are numerous cases where adjusting the angle of the glass differently in front of the eye has been the final answer or perhaps bringing the lenses closer to the corneal vertex. For this reason, even the style of the patient's frame will determine one's success, as the vertex-lens distance takes on even greater import when prism is a part of the lens prescription. For this reason, some experience in adjusting aniseikonic lenses would be valuable for the practitioner wishing to enter this field of headache therapy.

Perhaps only one rule can be stated regarding prism application: prescribe the least amount possible to relieve the symptoms, whether that be $\frac{1}{2}$ degree or 14 degrees.

Ocular Torticollis

No article on migraine headache would be complete without a word about torticollis. Let us consider for a moment the tension involved in torticollis.

The head has a weight of approximately 15 to 20 pounds. This is properly carried on the bony column of the spine when the median plane of the head is perpendicular to the horizontal plane of the shoulders. When there is any deviation of this, immediately the force of gravity pulls on the head in the direction of tilt. To compensate for this, the opposing neck and shoulder muscles are placed in a state of tension directly proportional to the pull of gravity. This means a constant flow of nerve impulse to these muscle fibers to increase their tonicity and there is never a chronic waste of nerve impulse without neurasthenia ensuing.

Now, if this torticollis is the result of an organism endeavoring to overcome a latent vertical phoria, then three problems exist which cause neurasthenia: (1) the tension of the extra-ocular muscles in each orbit; (2) the neurasthenia resulting from the cerebral control of fusion; and (3) the tension from the torticollis. These three added together can result in very severe cervical tension and cephalalgia.

In taking a case history, the patient should be observed very carefully for torticollis, with-

out mentioning the possibility to him. Then, if any doubt remains as to its existence, the patient should be instructed to look up old snapshots taken in informal poses, preferably front views only. By this means, you have a check on how long the torticollis and the accompanying latent phoria have existed.

If there is a distinct torticollis, it must be treated along with the latent phoria for best results. The more chronic has been the torticollis, the more difficult to correct; but after the vertical prism has been prescribed, the patient is instructed to draw a large cross on a mirror at home. This can be done with grease pencil or soft chalk and the horizontal line should be at shoulder level. Periodically during the day, he should stand in front of the mirror with the shoulders parallel to the horizontal mark and the median plane of the head parallel to the vertical line and learn the "feel" of this new position. With practice and a new consciousness of the problem, plus a realization of its merit, a patient will earnestly endeavor to correct this situation.

This torticollis will produce a scoliosis, which is a lateral curvature of the spinal column. When this scoliosis is due to an ocular deviation, it is referred to as ocular or ophthalmic scoliosis. However, there may be the possibility that a faulty posture in childhood, due to improper desks in school and improper lighting, may have first produced the scoliosis and, thus, caused the vertical phoria. A static scoliosis is that which is caused by a difference in the length of the legs. This is remedied by a heel lift being inserted on the shorter leg.

At the present time, Dr. George Woodruff has advanced the theory that the scoliosis produced the vertical phoria, while I have felt the reverse to be true; however, it could be either way. Undoubtedly, there is no definite pattern in all cases, yet this subject bears investigation.

Summary

A complete survey of all credited types of migraine has been presented, showing that the term migraine is more inclusive than merely the classic pain syndrome of migraine simplex. To this group of accepted migraines, I have added the new designation "*ocular migraine*." Most writers in referring to chronic severe cephalalgia have ruled out the ocular mechanism and have stated that headaches of ocular origin are merely of the mild to average intensity. There has been presented herein substantial evidence that the most severe head pain syndrome known to medicine has been completely eradicated by proper vision therapy. Also, in a cephalalgia of multiple etiology, it is shown that success has not been complete with medical therapy because the latent visual problem has not been found. When both are treated together, complete success ensues.

As the key that unlocks the door to these latent visual problems is a prolonged monocular occlusion test, the previous evidence of its impractical value is presented. This is

greatly overshadowed, however, by the overwhelming evidence of practical value, provided the technique is standardized. The balance of the article goes into a step-by-step analysis of a standardized technique for prolonged monocular occlusion.

It is felt that the role of the optometrist in the field of headache therapy and related neurasthenia problems is just beginning. The proper technique and interpretation of prolonged occlusion opens a whole new field of research for the specialist in vision. There are countless thousands who suffer with chronic cephalalgia merely because their problem is hidden to all diagnosis and, hence, incurable. Surely, the day is not far distant when great inroads into this "lost battalion" of sufferers will be made by men of vision.

Bibliography

Bannon, Robert E., "Diagnostic and Therapeutic Use of Monocular Occlusion," *American Journal of Optometry*, 20:345, 1943; "Heterophoria and Aniseikonia," *American Journal of Optometry*, 16:96, 1939; "Headaches and Aniseikonia," *American Journal of Optometry*, 17:448, 1940; "Vertigo and Aniseikonia," *The Columbia Optometrist*, Dec. 1939.

Bielschowsky, J., "Disturbances of the Vertical Muscles of the Eye," *Archives of Ophthalmology*, 20:175-200, 1938.

Blumenthal, Lester S., "Headache: Diagnosis and Treatment," *American Journal of Optometry*, 27:611, Dec., 1950.

Charnwood, Lord, "An Essay on Binocular Vision," The Hatton Press, London, 1950.

Dally, R. K., "Ophthalmoplegic Migraine," *Texas State Medical Journal*, 36:802-806, April, 1941.

Day, William Henry, "Headaches, Their Nature, Causes and Treatments," Lindsay & Blakiston.

Donahue, H. C., "Migraine and Its Ocular Manifestations," *Archives of Ophthalmology*, 43:96-141, 1950.

Duke-Elder, W. Stewart, "Text-Book of Ophthalmology," Vol. IV, C. V. Mosby Co., 1949.

Dunnington, J. H., "Hyperphoria—Its Etiological Diagnosis and Treatment," *American Journal of Ophthalmology*, 1931, 1140-1144.

Dutton, Walton F., "Headaches and Head Pains," F. A. Davis Co., 1939.

Foster, Clarence B., "Headache as a Symptom of Visual Disability," *American Journal of Ophthalmology*, 33:773-776, May, 1950.

Friedman, Arnold P., "Modern Headache Therapy," C. V. Mosby, 1951.

Gesell, Arnold, "Vision, Its Development in Infant and Child," Paul B. Hoeber, 1949.

Gordon, B. L., "Importance of Cephalalgia in Ocular Diagnosis," *Archives of Ophthalmology*, 11:769-796, May, 1934.

Granger, Ernest R., "Centric Relation," *Journal of Prosthetic Dentistry*, Vol. II, No. 2, 1952, 160-171; "Biometrics" Lecture, (not in print); Personal conversation.

Gubler, cited by Wilson, S. A. K., "Migraine" (Hemicrania: Megrim; Sick or Billious Headache), in *Neurology*, edited by A. Ninian Bruce, vol. 2, 1940, p. 1580.

Hewitt, Robert S., "Torsional Eye Movements," *American Journal of Ophthalmology*, 34:290-292, 1951.

Jaques, Louis, Sr., "Corrective and Preventive Optometry," Globe Co., 1950; Personal correspondence and conversation.

Kilby, Ralph A., "Ocular Vertigo," *American Journal of Ophthalmology*, 34:290-292, 1951.

Krimsky, Emanuel, "The Management of Binocular Imbalance," Lea and Febiger, 1948.

Marlow, F. W., "A Tentative Interpretation of the Findings of the Prolonged Occlusion Test on an Evolutionary Basis," *Archives of Ophthalmology*, 19:194, 1938.

Marlow, F. W., "Recent Observations on the Prolonged Occlusion Test," *American Journal of Ophthalmology*, 16:519, 1933; "Prolonged Monocular Occlusion as a Test for Muscle Balance," *Trans. American Oph. Sec.* 18:275, 1920; "The Technique of the Prolonged Occlusion Test," *American Journal of Ophthalmology*.

Mead, Sterling V., "Diseases and Disorders of the Mandibular Joint and Muscles of Mastication," from *Diseases of the Mouth*, C. V. Mosby Co., 1940.

Mobius, P. J., "Ueber periodische wiederkehrende oculomotoriuslahmung," *Berl. Klin. Wchnschr.* 21:604-608, 1884.

Moench, Louis G., "Headache," Chicago Year Book Pub., 1947.

Musser, John H., "Internal Medicine," Lea and Febiger, 1945.

Posner, Adolph, "The Prescribing of Prisms for Hyperphoria," *American Journal of Ophthalmology*, 34:197-199, 1951.

Rea, R. Lindsay, "Neuro-Ophthalmology" (Headache and Amaurosis, chap. 17), C. V. Mosby Co., 1938.

Schmidt, Rudolph, "Pain," 911.

Scobee, Richard G., "The Oculorotary Muscles," C. V. Mosby Co., 1952.

Simmerman, Harold, "Headaches," presented before American Academy of Optometry, Dec., 1950.

Sluder, Greenfield, "Nasal Neurology, Headaches and Eye Disorders," C. V. Mosby Co., 1927.

Snell, A. C., "The Relation of Headache to Functional Monocularity," *Archives of Ophthalmology*, 51:5, 1952.

Sugar, H. Saul, "The Extrinsic Eye Muscles," *American Acad. of Oph. and Otol.*, 1947.

Walsh, Rita, "The Measurement and Correction of Hyperphorias in Refractive Cases," *American Journal of Optometry*, 23:373, Sept., 1946.

White, J. M., "Hyperphoria, Diagnosis and Treatment," *Archives of Ophthalmology*, 7:739-747, 1932.

Wolff, Harold G., "Headache and Other Head Pain," Oxford University Press, 1948.

Zimmerman, M. W., "Ocular and Other Reflexes," *New York and Philadelphia M. J.*, 78:973, 1903. 318 S.W. Alder street