CONGENITAL NYSTAGMUS: BASIC ASPECTS

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ABSTRACT

Studies of the underlying mechanisms responsible for oscillations of the ocular motor system have provided insights into the normal functioning of this system and resulted in therapeutic benefits to patients afflicted with one such instability. Congenital nystagmus (CN) is the manifestation of a high-gain instability in the slow eye movement (SEM) subsystem which is initiated by fixation attempt. The CN oscillations cause the eyes to move away from and back to the target with any of several waveforms. It is possible to identify a distinct portion in each of these waveforms which corresponds to target foveation. Waveform analysis enables us to distinguish true CN from another congenital form of nystagmus which is mechanistically and phenomenologically different. Accurate detection of null angles in patients with CN can be used to therapeutically intervene to reduce their nystagmus and thereby increase their visual acuity.

KEYWORDS

Congenital nystagmus, genesis, foveation, waveforms, SEM instability, nulls, and acuity.

INTRODUCTION

The last decade has seen the application of two powerful tools to the study of congenital nystagmus (CN): a) accurate ocular motility recording techniques, and b) control system analysis. Preceding this unique two-pronged attack on a mystifying clinical problem was, with a few isolated exceptions, a collection of anecdotal observations, unsubstantiated myths, and benign clinical neglect. The clinician was not in an enviable position because he lacked the following: the ability to differentiate the various CN waveforms; the equipment to accurately locate null angles or to differentially diagnose CN from another common, but largely unrecognized, congenital form of nystagmus; and knowledge of the relationship between the CN waveform and target foveation. Thus, attempts at therapy were few and success stochastic. The level of understanding at that time is best illustrated by the following:

"Never write on nystagmus; it will lead you nowhere." Wilbrant (1921).

"A lot of intensely intelligent and highly dedicated workers have given their lives to this subject of nystagmus and very little has come out of it." Distinguished American neurologist (1965).

and, with tongue in cheek,

"So much has been written about nystagmus that there are only two unresolved questions about nystagmus: 1) the origin of the slow phase, and 2) the origin of the fast phase." Unknown source.

In this chapter, I will confine myself to two of the forty-one distinct varieties of nystagmus which have recently been identified (Dell'Osso, 1980). My discussion of the basic aspects of CN will be divided into several sections: genesis, foveation, waveforms, mechanism, nulls, and acuity. In addition, latent/manifest latent nystagmus (LN/MLN) will be discussed. MLN is a congenital form of nystagmus, but it is not a form of CN; although both are present at birth, they are two distinctly different types of nystagmus. Without the aid of accurate ocular motility recordings MLN is often difficult to distinguish clinically from CN and therefore, is often misdiagnosed as CN.

GENESIS

As with all forms of true nystagmus, a slow eye movement (SEM) is responsible for the genesis and continuation of the oscillation (Dell'Osso, 1980). The cause of the SEM is one of the parameters which differentiate the various types of nystagmus. It may be a rotating visual field, rotation of the subject, a vestibular imbalance, or a brainstem lesion. In 1973 fixation attempt was identified as the stimulus responsible for generating the initial slow eye movements of CN (Dell'Osso, 1973 a,b). Prior to that time, the literature contained contradictory anecdotal observations which attempted to link the genesis of CN with such things as lid position and/or retinal illumination. Since most patients do not attempt to fixate behind closed lids but could be expected to do so with eyes open in a darkened room (especially if not totally dark) it was not unusual to observe that CN "disappeared behind closed lids but remained with lids open in darkness." By carefully controlling fixation attempt, we showed that neither of these conditions were related to the genesis of CN. As increased fixation attempt results in a nystagmus with increased intensity, so does the presence of various psychophysiological inputs. A chain consisting of increased fixation attempt causing increased nystagmus causing decreased acuity causing further increased fixation attempt results in a vicious positive feedback loop which makes it difficult for the patient with CN to see clearly details which are near the level of his maximal acuity (Dell'Osso, 1973b). The clinical implications of this are that one must minimize fixation effort in whatever therapeutic method is employed so that visual acuity can be maximized.

In the past, it was assumed that one type of CN was caused by an afferent defect in the visual system: this was called, "sensory-defect CN." The inference that a sensory defect could cause CN was made because of the association between CN and one or more afferent defects which were present in many, but not all, patients. This confusion of genetic association with true causality was unfortunate and resulted in further muddying of the waters when attempts were made to relate etiology to assumed waveform; this will be discussed later. It should be clearly stated, however, that an afferent defect and the presumed failure to develop fixation reflexes cannot possibly explain a motor oscillation which is present at birth. The reasoning behind the assertions that non-

vectorial defects such as aniridia, blurring due to cataracts, ocular albinism etc., should invariably result in the highly-vectorial horizontal CN, is obscure at best. Since many patients with CN have absolutely no sensory defect, it is obvious that such a defect is not a necessary condition for CN. Also, since many patients who have the above sensory defects do not have CN, these defects are not sufficient conditions for CN. Since they are neither necessary nor sufficient, they are not causal. Further discussion of the genesis of CN appears in the section on mechanisms.

FOVEATION

Early descriptions of CN, based on clinical observations, stated that the oscillation was centered about the target or intended gaze angle. Thus, the image of the target on the retina of a person with CN supposedly slid rapidly back and forth across the fovea, never coming to rest in this most important area. Such a description completely ignored a commonly known piece of clinical evidence; patients with CN could have visual acuities in the 20/20 to 20/40 range. Since it is necessary for a target to be imaged in the foveal area for approximately 100 msec to achieve an acuity of 20/20, this clinical description of the CN oscillation was clearly in error. Using both retinal cinematography (with a laser beam as a target) and accurate eye movement recordings, the exact relationship between foveal motion and target position was discovered (Dell'Osso, 1973a). It was shown that in CN the eyes oscillate away from and back to the target such that one peak of the oscillation (be it either pendular or one of the many jerk waveforms) corresponds to target position. Only in this way can the eyes come to rest when the image of the target is directly on the fovea and good visual acuity be possible.

The preferred side of this fixation bias is idiosyncratic as is the frequency with which this bias reverses (i.e. the eyes oscillate to the other side of the target). The bias is conjugate, as are the fixation bias shifts, so that the image of the target always falls on corresponding parts of both retinae. Furthermore, one can see with accurate eye movement recordings that, in the interest of target foveation, most CN waveforms are flattened at those peaks which correspond to target foveation. Thus, the ocular motor system maximizes the amount of time during which the eyes are motionless and the target is foveated (Dell'Osso, Flynn, and Daroff, 1974; Dell'Osso and Daroff, 1975). Note that maintaining target foveation in the face of an instability capable of driving the eyes off target with SEM velocities of several hundred degrees per second requires extremely powerful fixation reflexes not poor, underveloped reflexes as was postulated by the proponents of the sensory-defect idea. In many CN waveforms, target foveation is obtained by foveating saccades which are part of the waveform (Daroff, Troost and Dell'Osso, 1978). Immediately following such saccades, one finds the flattening of the waveform described above (See Fig. 1).

The discovery of the existence of a fixation bias and of shifts in the side of the fixation bias in patients with CN, has uncovered a method to assess foveal function in these patients (Dell'Osso and Daroff, 1975). If the fovea is functioning normally, then the flattened peaks of a CN waveform which correspond to target foveation (with the fixation bias to one side) are at a corresponding gaze angle when the flattened peaks are on the opposite side of the oscillation (after the fixation bias has shifted to the other side). If, in addition to CN, the patient also has foveal dysplasia, there would be a difference of several degrees between where the target is foveated when the oscillation is biased to one side and the position in space where foveation occurs when the bias shifts to the other side. Thus, by observing the shifts in fixation bias, one can assess foveal function in a patient with CN.

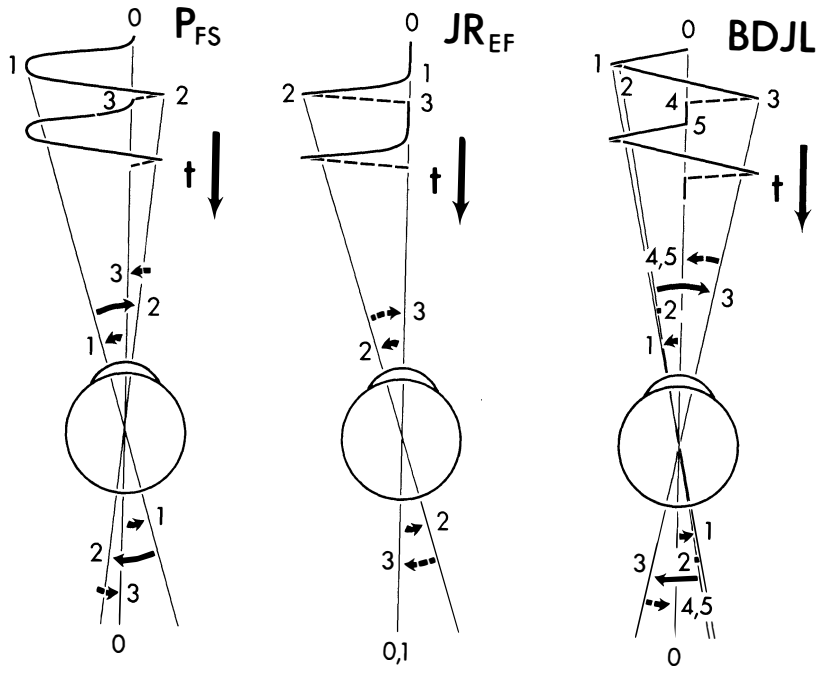

Fig. 1. Illustration of the foveation "strategy" employed during pendular nystagmus with foveating saccades (P_{FS}), jerk right with extended foveation (JR_{EF}), and bidirectional jerk left (BDJL) nystagmus. Target is foveated for extended periods of time resulting in good acuity. Saccades 2-3 (P_{FS}) and 1-2 (BDJL) are braking saccades. Saccades 2-3 (JR_{EF}) and 3-4 (BDJL) are foveating saccades. t, time scale.

WAVEFORMS

Accurate eye movement recordings have been used to identify the twelve distinct waveforms exhibited by patients with CN (Dell'Osso and Daroff, 1975; Dell'Osso, 1976). They fall into three categories: there are 3 pendular waveforms, 8 jerk waveforms, and 1 dual-jerk waveform. Furthermore, the 8 jerk waveforms consist of two groups; 4 are unidirectional and 4 are bidirectional. The classic pure jerk waveform (sawtooth) is extremely rare in CN and more commonly consists of an increasing-velocity SEM off target (slow phase) followed by a foveating saccade (fast phase) which returns the eyes to the target. There are many subtleties about these waveforms which can only be revealed through accurate ocular motility recordings and which are impossible to see clinically. One of the most important of these subtle movements is the small braking saccade. Braking saccades serve to stop (or brake) the classic runaway SEM characteristic of CN. The identification of braking saccades in the study of CN waveforms pointed out a stimulus for saccades which was unknown prior to that point. That is, saccades could serve to stop a runaway SEM in addition to their normal role of repositioning the eyes to bring a target to the fovea (Dell'Osso and Daroff, 1976). In CN waveforms, braking saccades are sometimes also foveating saccades (Fig. 1).

Since the four bidirectional jerk waveforms and pseudo-cycloid (a unidirectional jerk waveform) are clinically indistinguishable from the three pendular waveforms, it became clear that, prior to accurate ocular motility recordings, there was a strong tendency to over-diagnose pendular CN and under-diagnose jerk CN (Dell'Osso and Daroff, 1975). This point becomes extremely important when one considers the attempts which have been made in the past to infer etiology from waveform. The erroneous couplets of sensory-pendular and motor-jerk, which unfortunately still permeate the thinking of many ophthalmologists, became extremely suspect when we realized that these misconceptions were the result of clinical observations alone. They are not supported by accurate eye movement recordings which differentiate the truly pendular waveforms from bidirectional or pseudo-cycloid forms. In fact, no such correspondence exists (i.e. one cannot infer etiology from waveform). We have shown, by the study of a single family with a common genetic heritage, that the manifestation of their CN could take many forms, both pendular and jerk (Dell'Osso, Flynn and Daroff, 1974). Subsequent studies of over two hundred CN patients have confirmed our initial findings.

MANIFEST LATENT NYSTAGMUS

While studying the waveforms of CN, it became clear that there existed another congenital form of nystagmus which was not CN. Many patients referred to our laboratory with the diagnosis of CN had, in fact, a latent nystagmus which was present with both eyes open. Although both eyes were open, the patient was only using one eye to view the world (i.e. he was suppressing the other). Since image suppression is equivalent to occlusion, nystagmus present under these conditions was designated, "manifest latent nystagmus" (MLN) by Kestenbaum (1961). We studied thirty-one patients with MLN (Dell'Osso, Schmidt and Daroff, 1979); some, whose MLN was clearly visible, were misdiagnosed clinically as having CN and others, who appeared to have no nystagmus with both eyes open, were diagnosed clinically as having latent nystagmus. In fact, all had MLN and we have not yet recorded a patient with true LN (i.e. no nystagmus recorded with both eyes open). Since the slow phase of the MLN oscillation is a decreasing-velocity exponential, it is easily differentiated from CN with the aid of accurate eye movement recordings but it is almost impossible to do so by clinical observation alone. MLN is, therefore, nystagmus present with both eyes open, but with only

one eye viewing, which has a decreasing-velocity exponential slow phase and a fast phase in the direction of the viewing eye.

The high probability of a misdiagnosis in these cases is important clinically when one is considering therapy (especially surgical therapy). Another way in which accurate eye movement recordings can differentiate between CN and MLN is in the behavior of the oscillation at the various gaze angles. CN usually has a null at some gaze angle and the oscillation increases in intensity to either side of that gaze angle. MLN does not have a null except that, in accordance with Alexander's Law, it may diminish as gaze is directed in the direction of the slow phase. Knowing the distinctions between the slow phase waveforms and the behavior of the nystagmus with different gaze angles, it becomes relatively easy to use accurate ocular motility recordings to diagnose differentially CN, MLN, or various forms of acquired nystagmus (Dell'Osso, Schmidt and Daroff, 1979). Since CN and MLN are distinct types of nystagmus, it should be noted that a patient may have both. We have recorded many patients with both CN and MLN to varying degrees.

Important facts which relate to the mechanism of MLN are as follows: a) the direction of the conjugate slow phase exponential drift is always nasal to the viewing eye and is independent at gaze angle, strabismus angle or tropia type (i.e. ET or XT); b) in some patients MLN can also be recorded in the dark with no retinal input. Thus, any hypotheses aimed at explaining MLN must apply equally to ET's and XT's, must apply with the fixing eye in full adduction as well as abduction and should not rely on a mechanism requiring retinal input. Consistent with these constraints we have put forth the hypothesis that MLN is caused by a confusion between the perceived egocentric direction associated with binocular vision ($\theta_P = \frac{\theta_R + \theta_L}{2}$) and that associated with monocular vision ($\theta_P = \theta_R$ or θ_L). Such a confusion will always result in a conjugate nasal exponential drift toward the improperly perceived version angle (a simple step-response of the ocular motor plant). This nasal drift is independent of gaze angle or tropia and is independent of retinal input. Theories which rely on pursuit asymmetries and/or lateral gaze-holding inabilities fail to explain the directional constancy of MLN under all combinations of the above variables. The MLN of an XT (25% of patients we have seen with MLN are XT's) is exactly the same as that of an ET (i.e. direction, waveform and variation with gaze angle). All evidence points to a single mechanism causing MLN in both ET's and XT's that is distinctly different from the mechanism causing CN.

MECHANISMS

The initial evidence supporting the hypothesis that a high-gain instability of the SEM subsystem caused CN, came from observations made in a variable feedback experiment (Dell'Osso and colleagues, 1972). It was found that a patient with CN was able to pursue more rapidly moving targets than normal subjects could. This prompted the use of variable artificial feedback to change the overall loop gain. A normal subject will brake into a repetitive series of back and forth saccades under these conditions; the patient with CN developed a high-gain pendular oscillation. This response indicated that his SEM subsystem was responding in the abnormal experimental situation. Additional evidence, given the fact that the SEM subsystem and fixation are closely linked (if not unitary), consists of the following: fixation attempt is responsible for generating the nystagmus oscillation; the CN patient is locked in a positive feedback loop in which increasing his effort to see results in an increased nystagmus oscillation; and the waveforms of CN are either pendular (sinusoidal) or consist of increasing-velocity SEM runaways. Taken together, these data suggest to the control system engineer, two distinct types of feedback system instability.

One can describe a feedback control system by its system transfer function, which is the ratio of two polynomials in the variable (s) which describe input and output behavior (gain) over all frequencies. The polynomials can be factored into roots where the roots of the numerator polynomial are called, "zeroes", and the roots of the denominator polynomial, "poles." As the gain of a feedback control system is changed, so are the positions of the poles when plotted on the s-plane (i.e. real $s=\sigma$ on the X-axis and the imaginery $s=j\omega$ on the Y-axis) (Daroff and Dell'Osso, 1979). The mathematical background to understand clearly the positions and migration of these system poles and their relationship to oscillations in a closed-loop system is too extensive to be discussed here. Suffice it to say that when two of these poles are on the imaginery axis ($j\omega$-axis poles) the system oscillates in a sinusoidal manner and, when a pole exists on the positive-real axis (σ-axis poles) an increasing-velocity SEM runaway results. Since these poles migrate with various values of gain, it is not surprising that a system (or a patient) may manifest either one or both types of instability. We have observed many patients with CN who have both pendular and jerk waveforms.

The exact manner in which high gain affects SEM stability is unknown. If the instability merely results from high gain and a negative feedback loop, then the transfer function must be non-minimum phase (i.e. a pole or a zero must exist on the positive σ-axis). It is also possible that the rising gain alters the basic nature of the loop by changing a normal inhibitory junction into an excitatory one and, in so doing, creates a local positive feedback loop which would allow for positive-real pole positions without having been a non-minimum phase system. In a patient, increasing the gain (or fixation attempt) appears to drive the poles up the $j\omega$-axis, since the frequency of the oscillation may increase along with its amplitude. In a similar manner, increasing effort (gain) seems to drive a pole further into the right-half plane (on the σ-axis). The behavior of CN patients with a dual-jerk nystagmus waveform supports the contention that the pendular and jerk waveforms are independent oscillations caused by different types of instability. Usually dual-jerk is a high-frequency, low-amplitude pendular oscillation superimposed on a low-frequency, high-amplitude jerk nystagmus. One or the other component can be independently reduced or eliminated or, one component may spontaneously diminish or increase.

Initially, it was thought that the high gain of the SEM subsystem resulted from a primary defect in the saccadic subsystem (Dell'Osso and colleagues, 1972). This hypothesis arose from observations of saccades which were smaller than those required to refixate new targets. However, subsequent studies, made after identification of the relationship between waveform and target foveation, showed conclusively that the new targets were always accurately foveated by a combination of the ongoing oscillatory waveform and a saccade. Thus, the calculation of required saccadic amplitude was accomplished by the saccadic subsystem using information from the oscillating SEM subsystem, truly an impressive feat requiring precise amplitude and phase information. Whatever the reason for the high gain in the SEM subsystem, it can safely be said that it is not due to a deficient saccadic subsystem. Pendular CN may merely result from increasing the loop gain of an otherwise normal SEM subsystem with its inherent transport lags. Similarly, jerk CN may be the result of too much gain occurring in the normally present positive feedback loop around the neural integrator. Zee, Leigh and Mathieu-Millaire (1980) proposed the existence of such a loop when discussing cerebellar-induced gain changes which caused increasing-velocity runaway slow phases in nystagmus acquired by a patient with cerebellar dysfunction. Since no lesions have ever been associated with CN, it is reasonable to hypothesize that CN results from high gain in an otherwise normal neurological control loop.

The clinical phenomenon of inversion of the optokinetic reflex and the relationship between the head oscillation of patients with CN and their CN

waveforms are topics which deserve mention in this section on mechanism. Over the past ten years, I have observed that the null angle of a CN patient (normally stationary unless he has periodic alternating nystagmus) was a function of eye velocity as well as position. That is, during SEM tracking the null would shift in a direction opposite to the pursuit direction (unpublished observations of many CN patients). The amplitude of the shift need not be equal in both directions and could be related to the pursuit velocity. Such a shift can be seen in Fig. 6 of Dell'Osso and colleagues (1972) but it was not discussed and, until recently, has remained only a curiosity tucked away in my bin of interesting findings which should be studied further. Since I and several other colleagues in our laboratory failed to connect this observation with any clinical findings or significant mechanistic hypotheses, it remained a low-priority item. However, Halmagyi, Gresty and Leech (1980) have recently made the critical connection. They discovered this virtually unknown phenomenon and used it to show that the reversed optokinetic nystagmus found in patients with CN was due to the patient's own gaze-modulated nystagmus shifted to the primary position by the optokinetic stimulation. These workers demonstrated the modulation of a CN patient's nystagmus by attempted smooth pursuit. Thus, the null angle in patients with CN is a function of both intended eye position and intended eye velocity. It should be noted here that presentation of an OKN stimulus to a CN patient cannot be equated with an adequate OKN stimulus to his retina. The latter requires stabilizing the image motions due to the CN and then imposing the OKN retinal image drift (a very difficult task). Thus, inferences about the functioning of the basic OKN system of a CN patient made by studying responses to simple OKN stimulus presentations must be highly suspect. The resulting eye movements may be modified CN, CN plus OKN or modified CN plus OKN but they will not be OKN alone. As a final thought on this subject, it would not be surprising to discover (when we are able to properly study it) that the basic OKN reflex of a CN patient has been modified since the retina has constantly been exposed to horizontally moving images which must be perceptually stabilized. Such a finding, which has not yet been satisfactorily shown, would in no way imply cause and effect (i.e. an OKN asymmetry causing CN). CN is present at birth (before foveation reflexes develop) and in the dark (where the possible existence of an OKN asymmetry is irrelevant).

It has long been claimed that the head motion, seen in some patients with CN, was compensatory. For this to be true, the patient's vestibulo-ocular gain would have to be reduced to a value of zero (Daroff, Troost and Dell'Osso, 1978). It has now been shown that the head oscillation is not a compensatory movement (Gresty, Halmagyi and Leech, 1978). Rather, it has a common pathological origin with the CN and does not affect the periods of target foveation found in the CN waveform because of the normal vestibulo-ocular reflex acting to cancel the motion induced by the head oscillation.

Since CN is caused by a high-gain instability in the SEM subsystem, one might expect persons with CN to be able to pursue moving targets at least as well as normals. Indeed, CN patients have no trouble (personal observation) with such sports as handball, racquetball, baseball, etc., all of which make high demands on the pursuit subsystem. However, uncritical observation, by either man or an unsophisticated computer algorithm, of ocular motility records on attempted pursuit by CN patients might lead one to doubt that they can pursue at all. The apparent paradox is easily resolved if one understands the mechanism of CN as it relates to actual target foveation. It is only during the flattened portions of most CN waveforms that the eyes are motionless and on target. During the remaining portions they are either being irresistably drawn off target with ever-increasing velocity or they are being directed back towards the target by a saccade. Thus, the only times one might accurately assess the integrity of smooth pursuit in the CN subject is during these otherwise motionless foveation intervals. If one imagines a simple jerk waveform (constant in amplitude and

frequency) added to a perfect ramp tracking eye movement, what results bears no resemblance to the good smooth pursuit of a normal but would reflect the good smooth pursuit of a patient with such an ongoing nystagmus. But we know that with gaze angle changes, as well as attempted pursuit, CN waveforms change in type, amplitude, frequency and direction. Although the resultant eye movement records look nothing like good normal pursuit, they may actually represent good pursuit, given an ever-present CN. Evaluation of smooth pursuit (or the response to an OKN stimulus) must be done with full recognition of the inherrent changes in CN which accompany the responses of a CN patient to either stimulus. It cannot be done by simple computer algorhithms which identify saccades (excluding small braking saccades, which they miss) and construct simple linear links between the end of one saccade and the beginning of the next. Such artificial data are unrelated to the true eye movements of a CN patient and conclusions drawn from analysis of such data are so suspect as to be meaningless.

NULLS

The use of ocular motility recordings has enabled us to accurately define the null angle and/or convergence angle which results in a nulling of a patient's CN (Dell'Osso and colleagues, 1972). This can be directly translated into the size of prism required or, in cases where the null angle is too large, the exact amount of surgery to be performed. Accurate eye movement recordings have been used to identify the shift in null position with occlusion of each eye, commonly called the latent component in CN, and are also useful in documenting the null shift with time in periodic alternating nystagmus (Daroff and Dell'Osso, 1974; Dell'Osso, Flynn and Daroff, 1974). Additionally, ocular motility recordings have documented the stationarity (during fixation) of the null angles in patients with CN both pre-operatively and post-operatively (Dell'Osso and Flynn, 1979). Anecdotal comments concerning a mysterious revival of a new null angle, after surgical intervention had supposedly shifted the original null to the primary position, have been laid to rest. When properly done, surgery permanently shifts the null angle to the primary position; this has been documented by ocular motility recordings made immediately post-operatively and null stationarity has been documented by periodic recordings. If the initial surgery was inadequate, the new null angles will also appear immediately post-operatively; the adoption of a new head position may take several days. These will remain stationary until a second operation is performed. Information about other effects of CN surgery has also been provided by accurate ocular motility recordings. It has been found that, in addition to shifting the null, surgery broadened the null and decreased the intensity of the nystagmus at off-null gaze angles (Dell'Osso and Flynn, 1979). Follow-up on some cases that we have studied pre- and post-operatively has shown the null to remain stationary at the new position for many years (Flynn and Dell'Osso, 1979).

ACUITY

One of the important results of our studies of CN using accurate ocular motility recordings has been the realization that acuity of CN patients could be improved; in many cases they had previously been the objects of benign neglect. The therapeutic benefits resulting from such studies of CN have been significant. They range from very simple things, such as seating a child in the center of a classroom to maximize angle at which he views the board (thus minimizing the horizontal shrinkage caused by his horizontal oscillation) to the more complex realization that post-operative visual acuity can be greater than pre-operative acuity at the null (Dell'Osso and Flynn, 1979). Surgical rotation and prism rotation are not mere substitutions for head turning. Both considerably reduce the "effort-to-see" or "fixation attempt" and permit optimal acuity at-a-glance;

this is the aim of good CN therapy and is also why post-operative, primary position acuity can be better than pre-operative acuity at the null angle. Studies of waveforms (Dell'Osso, Flynn and Daroff, 1974; Dell'Osso and Daroff, 1975) revealed the importance of foveation time and the distortion of the oscillatory waveform which was imposed by the ocular motor system in an effort to increase foveation time. An analysis of foveation time and CN has also been presented by Abadi and Sandikcioglu (1974).

In addition to the use of prisms and the utilization of CN surgery, auditory biofeedback has been proposed as a method to diminish CN intensity and thereby permit greater acuity (Abadi, Carden and Simpson, 1979). This latter method takes advantage of the effects of psychophysiological inputs on the intensity of CN (Dell'Osso, 1973b). While useful in the laboratory, biofeedback has not yet been shown to be beneficial over long periods of time. Although the use of biofeedback is promising, it must be demonstrated that CN patients can voluntarily diminish their oscillation without the aid of biofeedback in real life situations. It should be noted that, if this can be demonstrated, the ability to calm oneself and diminish an existing CN is not dependent on the biofeedback which facilitated the learning process. Thus, other methods (yoga, tanscendental meditation, etc.) could also be used. The success of such "mind-set" altering therapies would probably be highly idiosyncratic.

FUNCTIONAL BASIS OF OCULAR MOTILITY DISORDERS

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