A New Clinical Method for Visual Function Evaluation Including Estimation and Measurement of Therapeutic Effectiveness: Lessons From Infantile Nystagmus Research

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ABSTRACT

Purpose: To translate infantile nystagmus system (INS) research into easily understood, clinically relevant terminology and suggest modifications to research and clinical testing, data and clinical interpretation, and therapeutic choices and evaluation.

Methods: A clinical method is presented using only three best-corrected visual acuity measurements of patients with INS, whereby (1) a measure of the quality of visual acuity across the visual field is possible; (2) pre-therapy estimates of post-therapy improvements in peak acuity and the high-acuity range of gaze angles are possible; and (3) more realistic visual function outcome measures of therapy are available to the practitioner.

Results: The application of the high-acuity field quality spreadsheet to the analyses of patients with INS (before and after therapy) results in a quantitative measure of visual function based on three visual acuity measurements.

Conclusions: The clinician can now duplicate adequate functional visual acuity descriptions in patients with INS along with their pre-therapy estimates and outcome measures. Previously, these have only been available to researchers or the rare clinicians who have access to both eye movement data and the expanded nystagmus acuity function analysis of INS waveforms.

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INTRODUCTION

The past 50 years witnessed a "golden age" of eye-movement research, especially in nystagmus of infancy. It was brought about by the confluence of accurate eye-movement recordings (EMR), an efficient paradigm to collect eye-movement data, and a biomedical engineering/modeling approach. As interest in the ocular motor system grew, neurophysiologists, experimental psychologists, optometrists, and ophthalmologists (pediatric ophthalmologists and neuro-ophthalmologists) made significant contributions to the field. In the area of infantile nystagmus syndrome (INS, also known as congenital nystagmus) research, a waveform-measurement function, the eXpanded Nystagmus Acuity Function (NAFX), was developed that allowed identification of the motor portion of visual acuity to be extracted from EMR. That coupled with measured best corrected visual acuity (BCVA) allowed the sensory portion to be deduced. Unfortunately, most clini-

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cal facilities did/do not have access to EMR (even off-site). Thus, they cannot accurately quantify the actual deficits to stationary or dynamic visual function; single BCVA measurements at one gaze angle (usually the INS "null") fail to identify the most important INS factor limiting static visual function.

This tutorial is aimed at the practicing pediatric ophthalmologist, neuro-ophthalmologist, and general ophthalmologist. In it, I will try to translate some of the key research findings and conclusions into practical terms, discuss their clinical utility, and introduce a new clinical method that accurately measures the static visual deficits in INS, predicts the improvements to expect from therapy, and provides accurate outcome measures of therapy. The hundreds of citations that are the foundations for information provided in this tutorial, as well as the details of the methodology and analyses presented, may be found elsewhere in an extensively indexed single source.[1]

THE AGE OF OPHTHALMOLOGICAL MYTHOLOGY

Because I had INS from birth, my first steps into nystagmus research were observations made throughout early childhood. They provided insights that guided my subsequent research. Early in my "formal" research into nystagmus in the early 1960s as part of my PhD research fulfillments, I was astonished to read definitions and descriptions in medical texts that I could only ascribe to misinterpretations of clinical observations or fiction invented to "explain" the many confusing observations made in patients with nystagmus. Together, they formed a body of imprecise, unclear, contradictory, or simply erroneous statements that violated the laws of basic physics. Yet, this collection of illogical and patently false statements was the foundation of both the diagnosis and treatment of nystagmus appearing in infants and children; amazingly, some are still believed (and taught) by ophthalmologists today.

BCVA can be good (even "normal" = 20/20) despite the oscillation, which would be impossible at the high eye velocities present if the eyes actually crossed the target sinusoidally where physics determines velocity is maximal. EMR proved that the oscillation is actually biased in such a way that the eyes come to rest on the target before accelerating away from, and back to, it. Patients with INS of all descriptions have the same nystagmus waveforms and variations regardless of whether or not they have a visual sensory deficit. Also, the direct cause of these waveforms has been demonstrated (via a behavioral computer model) to be a failure of calibration of the smooth-pursuit damping mechanism, which is underdamped in normal individuals and undamped in those with this type of nystagmus. That normal, precarious underdamping explains the common occurrence of INS in many individuals; at most, visual sensory deficits may facilitate the improper calibration of smooth-pursuit damping.

There are many treatments for INS that improve target foveation characteristics over a broader range of gaze angles and, by doing so, improve BCVA over that broad range of gaze angles. They include surgical, vergence prisms, soft contact lenses, and both systemic and topical drugs. Surgically shifting the INS null to correct a head turn can improve either the INS or BCVA. The laws of physics and the neurophysiology of vision determine that BCVA is possible only when the target is imaged in the foveal area and is relatively stationary for some minimum amount of time. During the rest of the nystagmus cycle, the target image is too far from the fovea and moving at too high a velocity for good acuity. Thus, BCVA depends on the quality of the foveation periods alone, independent of the rest of the nystagmus waveform (ie, "well-developed" foveation periods have accurate position, low velocity, and sufficient time). Therefore, improving the quality of the foveation periods will increase potential and measured BCVA, as has been repeatedly demonstrated. Gross measures such as amplitude, frequency, or slow-phase velocity are cosmetic; they neither determine nor are inaccurate outcome measures of BCVA.

DATA-DRIVEN DEFINITIONS, CHARACTERISTICS, AND CAUSALITY

It is important to properly define the specific types of nystagmus being referred to and clarify the above misconceptions about INS. All nystagmus seen in infancy is not the same and the term "congenital nystagmus" does not distinguish between different types; erroneously lumps them into a single entity; and improperly implies that all are strictly "con genital" or "with birth." Using EMR, these nystagmus types were definitively identified and defined as INS, fusion maldevelopment syndrome (FMNS), nystagmus blockage syndrome (NBS), and spasmus nutans (SNS). The major type of nystagmus seen in INS is pursuit-system nystagmus, which is responsible for most of the common pathog-

nomonic waveforms. Some patients exhibit a dual-waveform nystagmus in which a high-frequency, low-amplitude sinusoidal nystagmus (nucleus of the optic tract [NOT] nystagmus) is superimposed on the common high-amplitude, low-frequency pendular and jerk INS waveforms. Thus, if a dual waveform is present, it is the result of simultaneous oscillations in both the pursuit system and the NOT circuitry. Additionally, some patients with INS may exhibit several transient waveforms with linear slow phases that are due to tonic imbalance in the visual vestibular system. In different individuals, even within the same family, INS can be present at birth, become manifest in infancy, or not be manifested at birth but due to a condition present at birth. In each case, the waveforms and characteristics remain the same with the same underlying mechanism and cause.

CHARACTERISTICS AND DIFFERENTIAL DIAGNOSES

Identifying INS characteristics that may be clinically exploited to increase BCVA is dependent on accurately diagnosing INS and differentiating it from other types of nystagmus seen in infancy. The combinations of waveforms and their characteristic variations with gaze angle, fixating eye, attempted pursuit, vestibulo-ocular reflex, or optokinetic reflex, or even time (Asymmetric, (a)Periodic, Alternating Nystagmus [APAN]) have been documented using EMR, the "gold standard." EMR allows easy, objective, and repeatable differential diagnosis between INS (with or without NOT nystagmus), FMNS, NBS, SNS, or combinations of INS and FMNS. There are some patients whose INS characteristics require EMR for definitive diagnosis; the same applies to those with combinations of INS and FMNS. However, many patients with INS can be properly diagnosed and their therapeutic possibilities determined using careful clinical observations and following a specific paradigm. The exact steps in this complex endeavor are spelled out in OMLAB Report #061214.[2]

CLINICAL EVALUATION AND THERAPEUTIC OPTIONS

Clinical evaluation should be conducted independently from that of the patient's associated visual sensory deficits (eg, after refracting and measuring each eye's BCVA monocularly, INS evaluations must be made binocularly because occlusion may significantly alter the nature of the INS). Also, BCVA provides a measurement of the total (sensory plus motor) effects of these respective deficits on acuity and without a separate measure of either of the contributing deficits, evaluation of the other is impossible. If a measure of the motor effects of INS on BCVA is available, the sensory effects can be deduced. Furthermore, knowing the motor effect on acuity, we can predict, a priori, the expected benefits of ocular motor therapy. Fortunately, using EMR, the NAFX provided these capabilities for the first time; the relevant methodology has been extensively presented elsewhere.[1] Although the peak value of the NAFX (NAFXpk) is affected by associated, sensory visual deficits, gaze-angle changes in NAFX are independent of sensory deficits. Finally, a single BCVA measurement (whether in primary position or at the INS null angle) provides only an inadequate, partial description of how INS impacts real-world acuity and visual function. Unfortunately, that is the extent of the acuity assessments commonly performed by practitioners today. Not only does INS usually result in a lowered peak BCVA (BCVApk) but it also (and more importantly) results in "tunnel acuity," whereby merely attempting to look at (image on the fovea) a target slightly lateral to the INS null (where BCVApk occurs) results in a drastic drop in BCVA, sometimes reducing it to levels of "legal blindness." That severely limits attempts to locate a familiar face in a crowd, checking your side-view mirror, or playing sports.

Suppose you measured the BCVA of two patients, one with no ocular motor deficits and the other with INS, and both had "normal" (ie, 20/20 = 1.00) acuity in primary position. Would you say that both had normal visual acuity? Normal visual function? Would the patient with INS be eliminated as a candidate for therapy (standard surgical or nonsurgical)? If any of your answers were "Yes," you might try the tunnel acuity simulation[3] and then revisit them, especially the last one. Note: the correct answers are "No, No, and No."

A single measure of distance BCVA suffices in normal individuals only because wherever the target is located throughout the normally usable visual field (eg, ±30°), acuity remains constant and equal to the BCVA measured at 0° (presuming that no end-point nystagmus occurs). A good measure of the quality of high acuity across that field (HAFQ) is the area under the curve within the ±30° limits or the product of measured BCVA and the 60° extent of the field; for normal individuals, HAFQ = 1.00 × 60 = 60 (**Figure 1**). The area under the curve is a common

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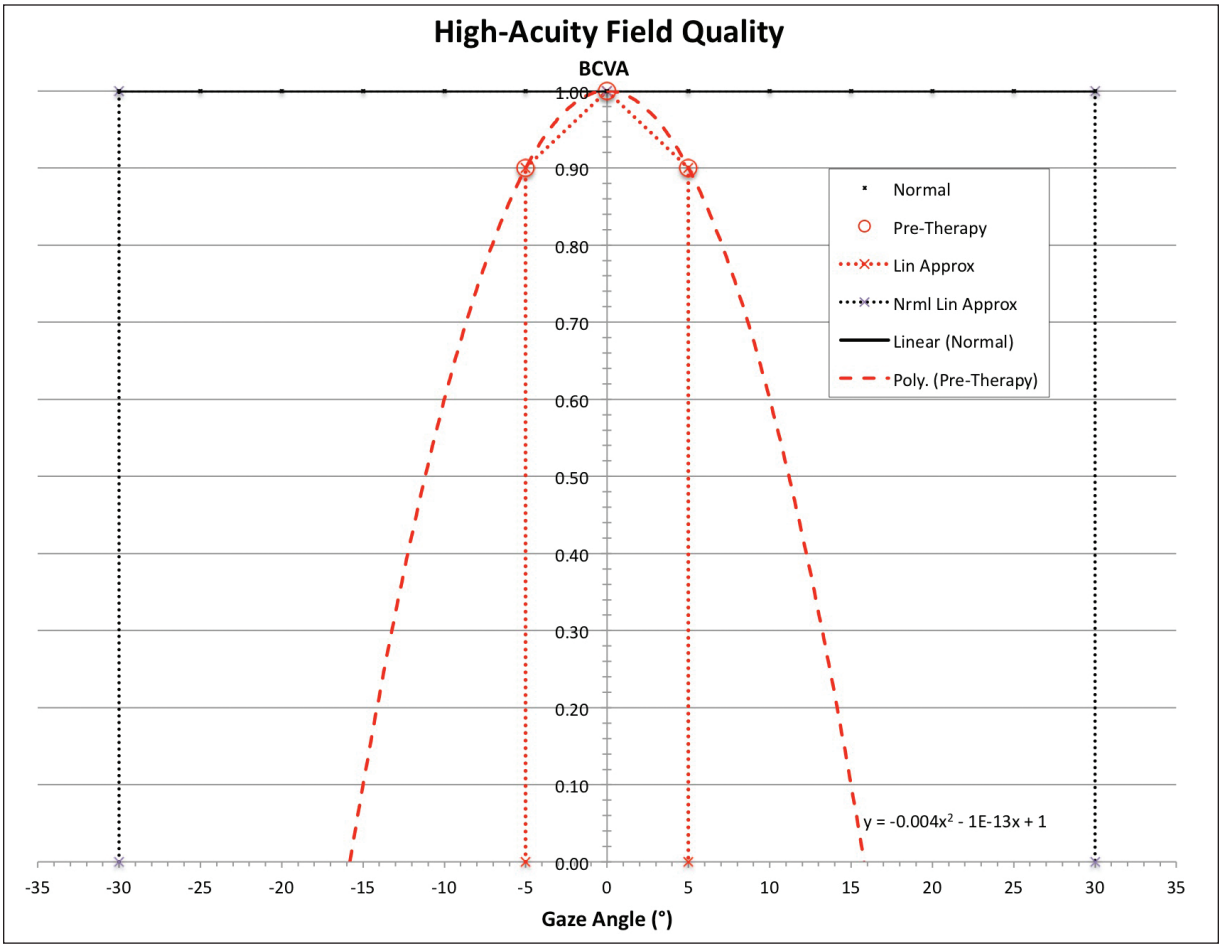

Figure 1. Comparative examples of visual field acuities for normal individuals and patients with infantile nystagmus syndrome (INS) (the curve for the hypothetical INS example is based on data from hundreds of patients). Both are assumed to have a best corrected visual acuity (BCVA) of 20/20 = 1.00 in primary position; the normal individual exhibits that same BCVA across the ±30° range of gaze angles considered. For the INS example, the three measures of BCVA show a typical reduction at lateral gaze angles. The spreadsheet automatically fits a 2° polynomial (dashed) to the three measured acuities. The area under that curve enclosed by the gaze angles at which BCVA is ≥ 0.90 of the peak BCVA is the high-acuity field quality and is approximated by summing the areas of the rectangle and triangle formed by the linear fit shown (dashed); the ±30° limits of the normal acuity plot are shown dotted.

method to compare two functions and is an intuitively simple concept to grasp. Similarly, if a patient without nystagmus has a BCVA of 0.80, HAFQ = 0.80 × 60 = 48. Based on the techniques developed using EMR-derived NAFX values of only three measures of BCVA, we can now obtain a spreadsheet "trendline" of the BCVA function. In INS, acuity decreases at gaze angles lateral to the null, where BCVApk is measured. Suppose primary-position BCVApk was 1.00 (ie, "normal") in a given patient but at 5° to either side, BCVA decreased to 0.90. For that patient, HAFQ is the integral of the acuity function (**Figure 1**) between the ±5° limits using the same definitions for high acuity as we apply to the NAFX in obtaining the longest foveation domain (LFD) (ie, gaze-angle range in which NAFX ≥ 0.90(NAFXpk)). Fortunately, this area can be approximated as HAFQ = 0.90(10) + 0.05(10) = 9.5, using the areas of the rectangle and triangle within the linear approximations shown in **Figure 1**. The simple reduced equation for this area is, HAFQ = 0.95(BCVApk)(LFD). Clearly the useful range of high acuity of even this patient with high-peak-acuity INS is far less than that of a normal individual with the same BCVA, measured at a single point (9.50 << 60; 9.5 = 15.8% of 60).

This pre-therapy HAFQ calculation is a more representative measure of high-acuity quality across this gaze-angle range. Moreover, it can be compared to normal individuals and to both the predicted and measured post-therapy calculations of HAFQ. This is a far more accurate description of functionally useful acuity in patients with INS than BCVApk. It docu-

ments the complete nature of their static visual acuity deficits and should clarify them for both the physician and the patient. Therefore, distance acuity examinations of patients with INS should consist of at least three measurements. In addition, near acuity must be determined to identify those patients whose BCVA improves with convergence. Not only will these measures adequately document the total static visual function deficits in each patient with INS, but they also will suggest the best therapeutic approaches and allow prediction of best possible outcomes for that patient.

A simple methodology has now been developed to approximate the above using only three distance acuity measurements easily made in the office: measuring binocular BCVApk at the INS null and at 15° to the right and left of that null. This can be done using a head-angle measurement device or, more easily, by fixing the patient's head to the chair (eg, using a chair-mounted chin cup and head rest) and rotating the chair to each desired position. The choice of ±15° from the null is arbitrary and useful for relatively centered null angles. For more lateral null angles, asymmetric ranges (eg, -20° and +10° from a null at +15°) work equally well (ie, BCVA measurements at -5°, +15°, and +25°).

Each of these distance measurement pairs (angle and BCVA) can then be entered into a simple spreadsheet (HAFQ Spreadsheet available at http://www.omlab.org/software/software.html). The spreadsheet is set up to accept up to 13 pairs of BCVA measures as angles separated by 5°; although seven were suggested in OMLAB Report #061214,[2] a minimum of three measures ensures a good curve fit. The fourth measurement, near BCVA (BCVAn), should also be entered into the spreadsheet. By using the instructions in the spreadsheet, the following pre-therapy data will be automatically provided: BCVApk, BCVAhi (= 0.90 BCVApk), LFD where BCVA remains within 0.10 of BCVApk, and HAFQ. The spreadsheet also includes the charts that allow estimates of post-therapy BCVA and LFD values based on linear approximations using these three easily obtained clinical data points. Although not as accurate as estimates derived from EMR and NAFX values, these provide good upper limits for improvements.

If BCVAn is > BCVApk, it is most probable that therapies exploiting convergence damping of INS will provide higher BCVA than other therapies. In non-binocular patients with INS (ie, those with strabismus), BCVA should be measured using the preferred, better eye.

THERAPEUTIC INTERVENTION AND OUTCOME EVALUATION

INS research has documented the therapeutically beneficial effects of: (1) null-shifting (Anderson-Kestenbaum recess and resect procedures on four rectus muscles in the plane of the nystagmus); (2) Anderson recessions plus tenotomy and reattachment of the remaining two rectus muscles; or (3) tenotomy and reattachment of all four rectus muscles in the plane of the nystagmus. For those binocular patients whose INS damps with convergence, the bimedial rectus recessions (BMR) plus bilateral rectus tenotomy and reattachment procedure has been shown to provide improvements in NAFX, LFD, and BCVA measures that are superior to four-muscle, null-shifting, or tenotomy and reattachment procedures. The use of soft contact lenses, systemic or topical drug application, or base-out prisms has also been demonstrated to improve these measures when applied to the appropriate patient population.

These surgical therapies are all four-muscle procedures that affect the small-signal gains of each involved, push-pull extraocular muscle (EOM) pair. No studies exist that directly compare the results of these procedures to two-muscle procedures (eg, Anderson recessions of only the two conjugate muscles). Due to the large INS damping effects of convergence (increasing NAFX and BCVA), it is probable that the bimedial rectus recession procedure to take advantage of convergence damping of INS may suffice without the addition of the tenotomy and reattachment of the lateral rectus muscles. The non-surgical therapies (drugs, contact lenses, or base-out prisms) also affect all four muscles in the plane of the nystagmus.

Because INS is a small-signal oscillation that does not affect saccades, therapies should be confined to reduction of small-signal gains without causing saccadic or gaze-holding deficits. This eliminates from rational consideration overzealous therapies such as maximal recessions or, even worse, EOM-destroying/crippling, irreversible therapies such as EOM extirpation; the latter not only failed to broaden the LFD (BCVAhi) but also reversed the broadening produced by prior tenotomy and reattachment.[4] Also, both are known to cause symptomatic deficits not produced by standard INS therapies. The symptomatic deficits of extirpation can have serious detrimental effects on education, exclude some occupations, impair or eliminate some sports, and dangerously impair safe driving. This

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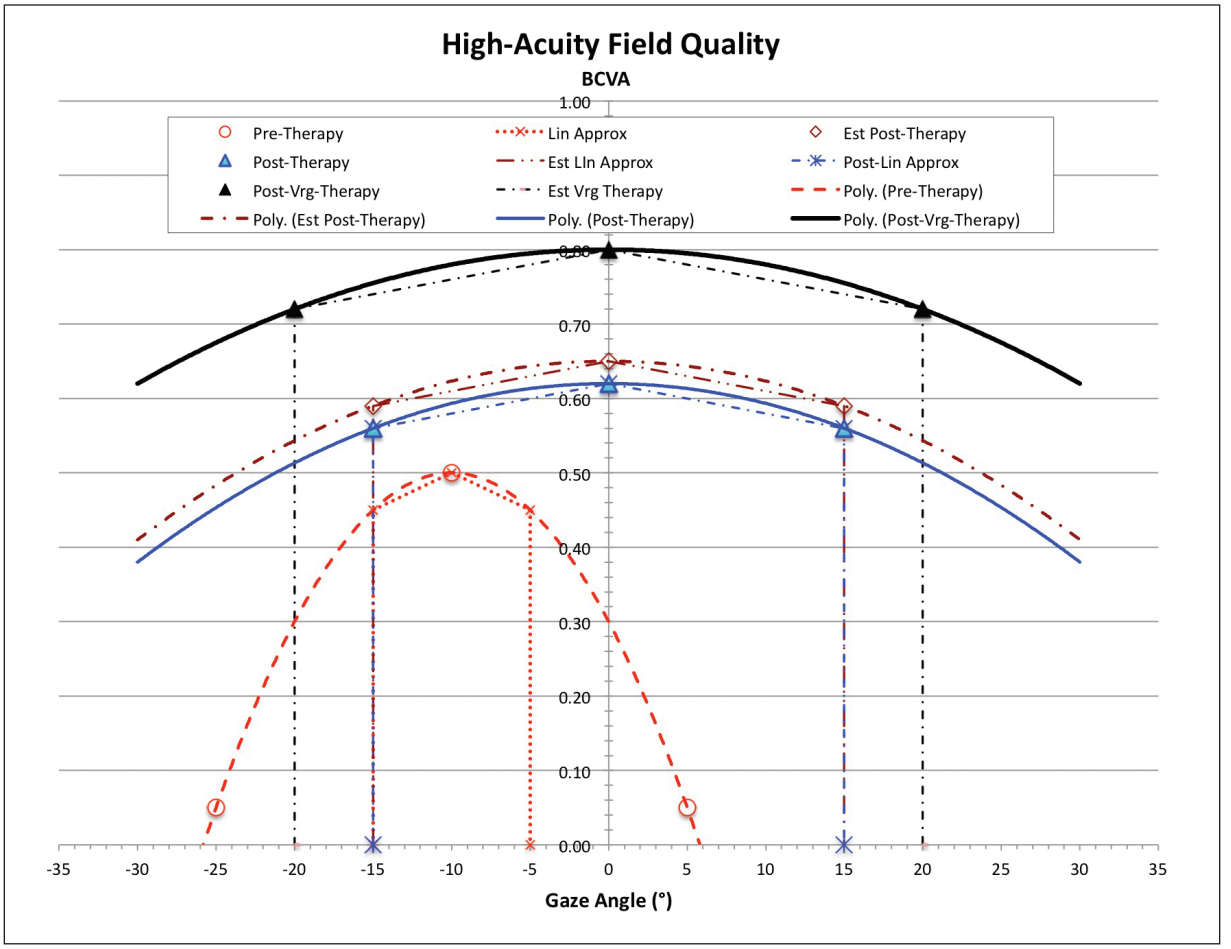

Figure 2. An example of a typical pre-therapy, visual field acuity plot (red, dashed) formed from three acuity measurements taken at the null angle and ±15° laterally (the curve for the hypothetical infantile nystagmus syndrome [INS] example is based on data from hundreds of patients). Also shown (red, dotted) is the linear approximation used to calculate the area under the high-acuity portion of the curve (the high-acuity field quality). Using the peak acuity (BCVApk), curves used for eXpanded Nystagmus Acuity Function (NAFX), and longest foveation domain (LFD) prediction, the predicted post-therapy plot is shown (brick, dash-dot) with its high-acuity, linear approximation (brick, dash-double dotted). It is presumed that the therapy accurately centered the null region. The post-therapy best corrected visual acuity (BCVA) measurements are shown along with the resulting visual field acuity plot (blue, solid) and its high-acuity, linear approximation (blue, dash-dotted). Finally, post-vergence therapy BCVA measurements are shown along with the resulting visual field acuity plot (black, solid, thick) and its high-acuity, linear approximation (black, dash-dotted).

high-risk/low-reward nature of extirpation should preclude its use in adult patients with INS and exclude it in infants or children, who are incapable of giving informed consent.

Figure 2 is an example of a more representative patient with INS containing plots of the measured pre-therapy, estimated post-therapy, and measured post-therapy BCVA functions over a ±30° gaze-angle range. Each of the measured functions were generated from three distance BCVA measurements, whereas the estimated function was derived using the pre-therapy BCVApk and LFD values in conjunction with the NAFX- and LFD-prediction curves in the literature (these are reproduced in the supplemental spreadsheet along with the curve used to determine millimeters of resection and recession needed to shift the INS null via the homeostatic, even recessions and resections of the Flynn four-muscle Anderson-Kestenbaum procedure). As shown in **Figure 2**, the three pre-therapy BCVA measurements produced a curve with a peak of 0.50 at -10° and LFD-determining 0.90 BCVApk points at -5° and -15° (LFD = 10°). The 0.50 BCVApk value suggested an estimated post-therapy peak of 0.65 (a 30% increase) and the 10° pre-therapy LFD suggested an estimated post-therapy LFD of 30° (a 200% increase). The pre-therapy HAFQ of 4.75 increased 290% to 18.53. These BCVA values were plotted and used to generate the estimated post-therapy function shown (brick, dash-dotted).

Also shown in **Figure 2** are representative measured post-therapy BCVA values, the resulting function (blue, solid), and linear approximation (blue, dash-dotted). Because we expect the estimations from BCVA measurements to be higher than those from NAFX values, post-therapy values are plotted slightly lower than the estimations (see Discussion).

For those patients with INS who are binocular (have neither strabismus nor amblyopia), if their nystagmus damps with convergence, exploitation of that damping will have greater therapeutic effects than either null-shifting or tenotomy and reattachment procedures. This is illustrated by the highest curve in **Figure 2**, which can be achieved by either a BMR surgical procedure or 7.00 diopters (D) base-out prisms in the left eye (with -1.00 D sphere added to the refraction of patients before presbyopia). The pre-therapy LFD of 10 improved 300% to 40 and the HAFQ of 4.75 increased 540% to 30.40.

Although greater damping can be achieved surgically or optically with additional convergence, the 14.00 D value for distance vision provides significant damping and acuity improvement while still allowing further convergence for near viewing.

In the research laboratory, measures derived from the EMR data (NAFX and LFD) have proven to be accurate indicators of corresponding static improvements in BCVA over a large range of gaze angles or with convergence. In the office, using only clinically available BCVA data at three distance gaze angles and at near, the same post-therapy BCVA measures provide a direct comparison to pre-therapy and estimated post-therapy values. Another research-produced outcome measure of INS therapy is "target acquisition time" and is related to how long it takes for the patient with INS to repeatedly foveate a new target. This is a good dynamic measure of visual function during driving or sports. Unfortunately, no simple office procedure to measure dynamic acuity is currently available or in common use.

DISCUSSION

Evaluation and treatment of patients with INS require: (1) diagnosis of the exact type of nystagmus present and (2) having an adequate measure of the effects of the nystagmus on visual function. The first is by far the most difficult. EMR provides both and ensures accurate, repeatable diagnoses, measures of the motor effects of nystagmus on BCVA (ie, the NAFX and LFD), pre-therapy estimations of the therapeutic improvements in foveation quality, and post-therapy outcome measures of visual function.

Clinicians have often expressed surprise at the "better vision" reports from patients after null-shifting surgery for INS, especially when their only outcome measure, BCVApk at the null, may change only minimally or not at all. Such patient satisfaction is often attributed to a "placebo effect." However, as shown above, HAFQ can significantly improve even if BCVApk does not. I conclude that it is predominantly the ability to see "more" (high acuity at a greater range of gaze angles) rather than "better" (a higher BCVApk) that is responsible for this measurable improvement in real-world visual function that patients are actually experiencing. For this reason, BCVApk alone is an inadequate and misleading outcome measure of the effectiveness of INS therapies. To more accurately assess pre-therapy and post-therapy INS visual function, the standard clinical work-up of all patients with INS should include measuring BCVA at three points to assess the respective HAFQ values.

The NAFX is a mathematical function that assesses the quality of nystagmus foveation periods and whose output is a linear equivalent to potential best-corrected, decimal visual acuity in patients with INS who have no significant associated visual sensory deficits. Thus, it is a measure of the motor effects of visual acuity. The curve formed by NAFX values taken at gaze angles lateral to the INS null where NAFXpk is measured is generated solely by the effects of waveform changes on visual acuity (ie, it, and the LFD calculation, are independent of any associated visual deficits). In the clinical method outlined in this tutorial, the BCVApk value reflects both the motor and sensory components of visual acuity but the BCVA-determined LFD remains purely a motor measurement and is the foundation for the methodology described.

How does the accuracy of this new clinical method in estimating post-therapy improvements in BCVApk and LFD compare to those using NAFX values? If the patient has only INS with no associated sensory deficits, BCVApk = NAFXpk and this clinical method has the same accuracy as the EMR method using NAFX values. However, many patients with INS do have associated visual deficits of varying degree. The smaller the sensory component of the BCVA is, the more accurate the method will be because BCVApk ≅ NAFXpk. For those

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with a larger sensory deficit, NAFXpk > BCVApk and using BCVApk on the curve generated to estimate NAFXpk improvement will produce a higher estimate than if NAFXpk was used. In the limit, where foveation-period quality is perfect (NAFXpk = 1.00), BCVA is due solely to the visual sensory deficit and no motor-therapy–based improvement is possible. Few, if any, patients with INS fall at that end of the spectrum and BCVApk-based estimations will be at the high end of actual improvements. That is, the actual improvement in BCVApk will be somewhat less than clinically estimated. However, because LFD is independent of associated sensory deficits, this clinically based method is as accurate as the EMR-NAFX–based method. Thus, using this clinical method, the estimated broadening of high-acuity, gaze-angle range that is so important to the patient's functional visual acuity will be accurate for all patients with INS.

There is no substitute for EMR and the NAFX for repeatable, accurate nystagmus diagnoses or INS evaluation, therapeutic determination, prediction, or evaluation. However, given the dearth of EMR facilities available to the practitioner, the measures and simple steps outlined herein provide a clinical path for better diagnosis, treatment, and visual function improvements for patients with INS. The ability to clinically estimate therapeutic improvements for most patients with INS should enable the practitioner (who is limited to clinical measures) to choose and provide therapies with confidence similar to those who have access to EMR. A full description of the distance visual function deficits in INS requires both BCVApk and LFD. At a minimum, office procedures should be expanded to include these two additional measures of distance BCVA to describe the total deficits and to predict possible, and assess actual, therapeutic outcomes.

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