Asymmetric Papilledema in Idiopathic Intracranial Hypertension: Prospective Interocular Comparison of Sensory Visual Function

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Purpose. Visual loss is the main morbidity of idiopathic intracranial hypertension (IIH). The relationship between papilledema grade and visual loss is unclear. The goal of this study was to determine whether there is a relationship between papilledema grade and visual loss.

Methods. Fundus photographs of 478 patients with IIH were reviewed, and their degree of papilledema was graded using Frisen’s scheme. We identified 46 patients (10%) with IIH and highly asymmetric papilledema, as defined by an interocular difference of two or more grades. Nine of these patients with active asymmetry agreed to return for a series of visual tests. They underwent three visual field tests—Humphrey visual field analyzer 24-2, motion perimetry, and ring perimetry. The perimetry outcome measures were mean deviation, foveal threshold, and means for eccentric zones (3°, 9°, 15°, and 21°). The patients participated also in visual acuity, Farnsworth-Munsell 100-hue, Pelli-Robson contrast sensitivity, and foveal flicker fusion testing. Their relative afferent pupillary defect was graded using neutral density filters.

Results. The intereye comparisons showed vision to be worse in the eye with the high-grade papilledema for all outcome measures. The magnitude of the loss with the perimetry tests increased with eccentricity. The measures of central visual function, although in the normal range, were relatively depressed in the eye with high-grade papilledema.

Conclusions. Visual loss in patients with asymmetric papilledema caused by IIH was most pronounced in the eye with the higher grade of papilledema. Foveal visual functions, although they remained in the normal range, were also decreased in patients with high-grade papilledema. In patients with high-grade papilledema, visual loss appeared to affect the entire visual field, and the peripheral field showed the most deficit. Our findings showed that high-grade papilledema was associated with visual dysfunction in patients with IIH. (Invest Ophthalmol Vis Sci. 1998;39:134-142)

Idiopathic intracranial hypertension (IIH) is a disorder of elevated intracranial pressure of unknown cause. Visual loss caused by papilledema is the most important complication of IIH. Patients at risk for permanent visual loss include those with severe or high-grade papilledema.1-3 Those with moderate or less severe papilledema, however, also demonstrate visual loss using sensitive psychophysical testing techniques.4,5 Rush reported no correlation between papilledema grade and degree of visual loss.6 Others have reported an association between higher grades of papilledema and more severe visual dysfunction.1-3

Our main goal in this study was to investigate the question: Is the amount of visual loss related to the degree of papilledema? Our secondary goal was to look for evidence of selective loss of either magnocellular or parvocellular functions. To accomplish this we analyzed sensory visual function data from patients with highly asymmetric papilledema. Our premise was: If there was no difference in visual function between the two eyes with highly asymmetric papilledema, then there was no relationship between visual loss and papilledema grade. However, if visual functions were systematically worse in the eye with high-grade papilledema, then the amount of visual loss was related to the degree of papilledema. We retrospectively identified patients with highly asymmetric papilledema and analyzed their visual functions. We then prospectively tested nine patients with highly asymmetric papilledema with a battery of sensory visual function tests.

Subjects and Methods

We reviewed the optic disc photography files and charts of 478 patients with the diagnosis of IIH evaluated at the University of Iowa Neuro-opthalmology Clinic from 1972 to 1995. Patients were excluded if they failed to meet the five modified Dandy criteria for idiopathic intracranial hypertension8; there was presence of signs and symptoms of increased intracranial pressure; the patient was alert and oriented; the neurologic examination was normal except for papilledema and its associated visual loss and sixth nerve palsies; neurodiagnostic studies were normal except for increased cerebrospinal fluid pressure;
and there was no secondary cause of intracranial hypertension found.

The IIH patients all had fundus photography, and each patient’s papilledema was graded according to Frisen’s scheme. Grade 0 defines an optic disc without swelling; Grade 5 represents severe papilledema. Nine patients from these 38 that still had asymmetry of two or more grades present agreed to take part in the study. All participants were reimbursed for their time and travel. The mean patient age was 31.8 ± 9.2 years.

The protocol was approved by the University of Iowa Investigational Review Board and fulfilled the tenets of the Declaration of Helsinki. Normal subjects for the perimetry tests were randomly chosen from our database of normal control subjects and matched to the patients by age (two control subjects to each patient). All normal subjects had a normal neuro-ophthalmologic examination and no history of eye disease except refractive error and normal automated perimetry results (Program 24-2, Humphrey Field Analyzer; Humphrey Instruments, San Leandro, CA).

The subjects were tested for the following: visual acuity using the Early Treatment of Diabetic Retinopathy Study chart, foveal flicker fusion, Pelli–Robson contrast sensitivity, relative afferent pupillary defect quantitation using neutral density filters, Farnsworth-Munsell color, and three types of perimetry. We used conventional automated perimetry with the Humphrey visual field analyzer 24-2, high-pass resolution perimetry (Frisen’s ring test), and motion perimetry.

Conventional Automated Perimetry

Using the Humphrey visual field analyzer, and following the manufacturer’s recommendations, we performed conventional automated perimetry. The patients’ appropriate near correction was used. Subjects were tested with a program using a 6°-spaced grid of the central 21°. Rest periods were given when requested.

High-Pass Resolution Perimetry

High-pass resolution perimetry was administered according to the recommendations of the manufacturer (Ophthimus system; HighTech Vision, Göteborg, Sweden) except that, inadvertently, we calibrated the background to 100 cd/m² brighter than the standard level. Therefore, we will only report differences between the eyes with this test. The patients’ appropriate near correction was used and care was taken to prevent lens rim artifact. Patients’ fixation was monitored by the visual field technician and with intermittent blind spot testing (Heijl-Krakau method).

Motion Perimetry

In a darkened room, motion perimetry was conducted using an IBM-compatible 486 computer with software we have developed previously. The patients’ appropriate near correction was again used. Care was taken to prevent lens rim artifact by asking each subject if he or she could see each corner of the video display while looking at the fixation target. The details of this testing method can be found in other publications.

In summary, the test background is composed of 5000 random dot cinematograms within which 50% of the dots move in one of four directions relative to fixation (up, down, left, or right) and 50% move in random directions. The stimulus presentation time was 128 msec. The targets were of 20 different sizes. The angle subtended by the targets ranged from 0.25° to 21°.

The size of the stimulus window varied from trial to trial, and a 2 to 1 staircase procedure was used to bracket the threshold. The test, therefore, continued until the smallest circle size seen (size threshold) at each test point was bracketed by the staircase procedure. We tested 44 locations. These matched the 24-2 Humphrey perimetry test points, except for the absence of the top and bottom rows (y = 21° and −21°) and the two points along the nasal horizontal (x = −27°). The test time for the 44-test loci was approximately 15 minutes.

The perimetry measures were analyzed by calculating the mean score for the ring test and motion perimetry and tabulating the mean deviation for conventional automated perimetry and by compacting the data for the three tests into concentric zones. We excluded the region of the blind spot and the adjacent-test locations for this analysis so that visual loss from elevation of peripapillary retina would be minimized.

The patient’s best-corrected visual acuity was recorded using the Early Treatment of Diabetic Retinopathy Study chart and method. The total number of letters read correctly for each eye was summed. Foveal flicker fusion was tested three times, and the mean for each eye was recorded. The details of this method can be found in a publication by Brenton and coworkers. The patient’s relative afferent pupillary defect was quantitated using the method reported previously by Thompson.

The Pelli–Robson contrast chart is a contrast letter chart with three letters for each contrast. A score was recorded at the lowest contrast line at which the patient could read at least two letters correctly. The line number for each eye was used as the subjects’ outcome measure.

Color vision testing was performed using one rack of the Farnsworth-Munsell 100-hue test monocularly and Type C lighting (as provided by Munsell) which provided a luminance targets are circular random dot cinematograms within which 50% of the dots move in one of four directions relative to fixation (up, down, left, or right) and 50% move in random directions. The stimulus presentation time was 128 msec. The targets were of 20 different sizes. The angle subtended by the targets ranged from 0.25° to 21°.

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of 500 lux evenly distributed across the testing area. The chips were scrambled, and the patient was instructed to arrange the colored caps in order according to their hue. Bar codes on the undersurface of the ordered chips were scanned into a personal computer for scoring and plotting with custom software. We used only one rack (21 chips, numbers 22-42), because if the patient and one other did not perform the ring follow-up. This patient and one other did not perform the ring perimetry test.

Because of the small number of patients, a predefined number of questions for statistical analysis were designated as study outcomes to compare the eyes of the patients and the normal subjects. These were: mean and concentric zone perimeter scores for the three perimeter tests, Early Treatment of Diabetic Retinopathy Study acuity, flicker fusion scores, relative afferent pupillary difference with neutral density filters, and defect Farnsworth color vision total error scores. Other statistical tests were performed for description and hypothesis generation.

Differences between scores of the nine patients from the various tests between the high-grade and low-grade papilledema groups were tested for statistical significance. A two-tailed paired t-test was used for normally distributed quantitative data. The Wilcoxon test was used for corresponding nonparametric data. An ANOVA test was used for differences between the high-grade and low-grade papilledema results and the age-matched normal results. A Kruskal–Wallis test (ANOVA on ranks) was done for data that were not normally distributed or had unequal variances. Differences among groups of the test results were interpreted as significant if the probability of their occurrence was less than 0.05.

### RESULTS

#### Retrospective Data

Of the 478 charts reviewed, we identified 46 patients (10%) with IIH and highly asymmetric papilledema, as defined by an interocular difference of two or more Frisen grades. Two patients had amblyopia, one had traumatic optic atrophy, and one had Fuch’s dystrophy. These four patients were eliminated from the analysis. Four others did not have elevated cerebrospinal fluid (CSF) pressures recorded and were excluded. The remaining 38 patients met the entry criteria for the study. Twenty-seven (71%) were women; 11 (29%) were men. The mean age was 35.7 ± 9.8 years. Their mean CSF opening pressure was 358.9 ± 102.5 mm H2O. At the initial visit the median papilledema grade in the eyes with the highest grade was 3 (range, 2–5) and was 1 (range, 0–2) in the low-grade eyes. At the final visit, the high-grade eye values fell to 2 (range, 0–4); the low-grade eyes improved to a median of 0 (range, 0–1).

The visual acuity in the eyes with high-grade papilledema at the initial visit, in Snellen equivalents, had a median of 1.00 with a range of 0.5 to 1.33; in the low-grade eyes it was 1.00

<table>
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<th>Visual Field Zone</th>
<th>Mean</th>
<th>SD</th>
<th>Minimum</th>
<th>Maximum</th>
<th>P value</th>
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<tr>
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$P$ values are from comparisons of high-grade papilledema eyes with low-grade papilledema eyes. (SD = one standard deviation).
FIGURE 2. Box plots showing measures of central visual function in nine prospectively tested patients comparing eyes with high-grade papilledema with eyes with low-grade papilledema. Box shows 75th, 50th, and 25th percentiles. Error bars show 90th and 10th percentiles. In each example, the high-grade eye showed less sensitive visual function than the low-grade eye. (A) Early Treatment of Diabetic Retinopathy Study acuity scores. (B) Foveal critical flicker fusion. (C) The second box of the Farnsworth-Munsell 100-hue test.

with a range of 0.8 to 1.54. At the final visit these values improved to 1.00 (range, 0.8–1.67) and 1.25 (range, 0.67–1.67), respectively. Flicker fusion values in the eyes with high-grade papilledema at the initial visit was 30.29 ± 2.69 Hz, and in the other eyes it was 31.17 ± 2.36 Hz; these values changed with treatment to 30.08 ± 2.03 and 30.83 ± 2.06, respectively. The mean relative afferent pupillary defect was 0.26 (median 0.1, range 0–1.2) at the initial examination and was 0.23 at the final examination (median 0.2, range 0–0.9).

Twelve patients had conventional automated perimetry (Humphrey visual field analyzer, programs 30-2 or 24-2) at an early and final visit. No subjects had cecocentral or central scotomas. Figure 1 and Table 1 show the results of the initial and final perimetry results analyzed by eccentricity. Data for the age-matched normal subjects was not shown, because it was similar to the data of the low-grade eyes at the final visit. Surprisingly, the initial visit results show a generalized depression of the visual field in the eyes with high-grade papilledema, most marked peripherally but including the foveal threshold. At the final visit, sensitivity improved but the results for the high-grade eyes still showed more visual loss.

Prospective Data

The mean age of the nine patients was 35.7 ± 9.8 years. Their mean CSF opening pressure was 347.2 ± 90.9 mm H2O. The mean relative afferent pupillary defect was 0.3 log units (median 0.3, range 0–0.6); all but one patient (with no relative afferent pupillary defect) had a relative afferent pupillary defect on the side of the eye with high-grade papilledema. The visual testing results in this group are similar to the retrospectively collected results (Tables 2–5, Figs. 2, 3). We found visual function to be worse in the high-grade papilledema eyes for all visual function tests, although statistical significance was not reached for every one. This finding occurred whether the tests were biased for the magnocellular or parvocellular system.

The data shown in Figure 3 support the findings of a generalized depression of the visual field and extend the findings of the retrospective data by showing this depression to be present to motion testing and peripheral acuity testing (high-pass resolution perimetry). The differences in the foveal threshold are not as marked with the prospective as with the retrospective data. The most likely explanation for this is: The retrospectively collected data were obtained during the acute phase of the patient's presentation, whereas the prospectively collected data were obtained during the chronic, treated phase of the patient's disease in which the magnitude of the papilledema severity and, consequently, the visual loss were less.

To evaluate visual loss as a function of visual field eccentricity, we used the slope of a regression of visual field sensitivity on eccentricity. We looked for differences among the results of eyes with high- and low-grade papilledema and nor-
FIGURE 3. Visual field sensitivity by concentric zone for (A) conventional automated perimetry, (B) motion perimetry, and (C) high-pass resolution (ring) perimetry. Note how visual sensitivity was lowest for high-grade eyes, intermediate for low-grade eyes, and highest for age-matched normal eyes and how sensitivity decreased with eccentricity.

mal eyes. For all three types of perimetry, the slopes were significantly different (Table 4). In all cases, the steepest slope was found with the high-grade eyes, and the flattest slope was found with the normal eyes. The low-grade eyes were intermediate, indicating an increasing magnitude of visual loss with eccentricity. Post-hoc comparisons showed significant differences in the slopes of the high-grade eyes compared with the normal eyes in all three perimetry types; with motion perimetry, the comparison between the high- and low-grade eyes was also significant.

There was a weak relationship between mean visual field sensitivity (excluding the test locations around the blind spot) and papilledema grade for the three perimetry tests. Although the $r^2$ values ranged from 0.4 to 0.18, in all cases there was a trend of increasing visual loss with increasing papilledema grade.

A typical example of one of our patients is shown in Figure 4. This woman, aged 36 years, had transient visual obscurations of vision in her left, high-grade papilledema eye (Fig. 4; left). Her conventional automated perimetry results are shown in Figure 4; right. Note the generalized depression of the visual field, which is most evident peripherally in the patient’s left, high-grade papilledema eye.

DISCUSSION

Our main finding is that there was a generalized depression of the visual field in eyes with high-grade papilledema. The visual loss increased in magnitude with increasing visual field eccentricity. However, this finding is only appreciated when compared with low-grade papilledema eyes. The results of the foveal visual function tests in high-grade papilledema eyes are, for the most part, still in the normal range.

Our data showed that the amount of visual loss correlated with the severity of disc edema—eyes with more disc edema have more visual loss. However, there was considerable inter-individual variation. That is, some patients with marked optic disc edema appeared to have mild or no visual loss unless the loss was compared with a fellow eye with less disc edema. This relationship of degree of papilledema with visual loss implied that visual loss in III was caused by papilledema and not by a retrolaminar mechanism.

Based on these results, we propose the following scenario for visual loss in III (Fig. 5). High CSF pressure at the optic nerve head causes high tissue pressure there.19,20 This leads to blockage of slow axoplasmic transport (axoplasmic flow stasis)21,22 and resultant optic disc edema. There appears to be related compression of the intraneuronal microvascular supply with resultant nerve fiber ischemia.21 This may result in diffuse damage to optic nerve fibers. An explanation for the character of the visual loss observed might be as follows. Because there appears to be more redundancy of visual processing elements subserving the central visual field,23 testing may not show as much visual loss as that found in the periphery. The loss we
have observed is so mild in the central visual field that it usually falls within the normal range. It is only by comparison with the fellow eye, with less disc edema, that some degree of central visual function can be confirmed. This analysis is based on summing data from nine patients. In this analysis, localized defects such as arcuate scotomas and nasal step defects contribute to the eccentric zone in which they occur, but their spatial relationships within that zone are lost. Our patients did have these localized defects. They presumably occurred when intraneuronal ischemia affected nerve fibers in groups (bundles) instead of diffusely.

Highly asymmetric papilledema in IIH is not rare. We observed it in approximately 10% of patients with IIH. Unilateral or highly asymmetric papilledema in IIH has been reported by others.24 32 These case reports provide few data regarding interocular comparisons of sensory visual function in patients with asymmetric papilledema.

Lepore29 reviewed his case records over a 10-year period, and, using subjective criteria for asymmetry, found 26 patients with unilateral papilledema were significantly older (37.5 versus 25.7 years). Six patients with asymmetric papilledema had enlarged blind spots, and two had temporal constriction of the visual field. Our mean age was similar at 35.7 years. Of the 18 patients with asymmetric papilledema identified from the perimetric examinations, five have more visual loss in the eye with high-grade papilledema (excluding blind spot enlargement).26 30 32 33 However, our perimetry was manually performed; we did not use automation.

Why does asymmetric papilledema occur in patients with raised intracranial pressure? The occurrence of papilledema is thought to be dependent on the relationship of three factors: CSF pressure, intraocular pressure, and systemic blood pressure.34 Either elevated CSF pressure, low intraocular pressure, or low perfusion pressure can cause axoplasmic flow stasis, optic disc edema, and resultant intraneuronal ischemia.
TABLE 5. Relationship between Papilledema Grade and Visual Field Sensitivity

<table>
<thead>
<tr>
<th>Comparison</th>
<th>$R^2$</th>
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<tr>
<td>Conventional Automated Perimetry</td>
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<td>High Grade eye to mean sensitivity</td>
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<tr>
<td>Low Grade eye to mean sensitivity</td>
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<td>Motion Perimetry</td>
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</table>

First, it was unlikely that there was any difference in optic disc perfusion pressure in the patients in the present study. Our group of patients was young; therefore, a postulate, that enough premature vascular disease was present to cause sufficient carotid or ophthalmic artery stenosis, that would in turn account for the asymmetry, was not plausible. Also, Kirkham did not find any difference in ophthalmodynamometry values between the eyes of his patients with asymmetric papilledema to implicate asymmetric perfusion pressures. This finding implies that there is no difference in ophthalmic artery pressure between the two eyes. It does not negate the suggestion that optic nerve damage can be the result of CSF pressure-related optic nerve head edema with resultant intraneuronal ischemia, because factors that are distal to the ophthalmic artery are likely important in the latter.

Second, it is possible that intraocular pressures between the eyes could be asymmetric. Kirkham and co-workers studied five patients with chronic unilateral papilledema and found intraocular pressures were normal and symmetric in all. This leaves higher CSF pressure at the optic disc as the remaining possibility. The effects of optic nerve sheath fenestration in monkeys with experimental papilledema and humans prove that relief of the elevated pressure within the optic nerve sheath is associated with defervescence of papilledema. Therefore, the effect of asymmetric CSF pressure at the optic nerve heads is a viable mechanism. However, in patients with asymmetric or unilateral papilledema, optic nerve sheath width

**FIGURE 4.** Typical example of conventional automated perimetry testing results from a patient with IIH with highly asymmetric papilledema showing gray scales and difference plots. High-grade papilledema was shown in the left eye, and low-grade papilledema was shown in the right eye (left). Note the generalized depression of the visual field, most evident peripherally in the high-grade papilledema left eye (right).

**FIGURE 5.** Model of the visual islands (plots of sensitivity versus eccentricity) for visual loss in IIH comparing high-grade papilledema (dashed line), low-grade papilledema (dotted line), and normal visual island (solid line). Note the depression of the visual island, most evident peripherally with increasing papilledema grade.
is increased and symmetric. This appears to suggest that CSF pressures must be equal in the two optic nerve sheaths or the sheath width would be wider in the sheath under higher pressure. However, Hayreh showed that the nerve sheath is composed of fibrous tissue and, after it unfolds, it cannot expand any further. Therefore, there may be a high enough pressure within the sheath to distend it but not high enough to cause optic disc edema. The pressure could be elevated in both nerve sheaths but could be higher on one side. If this is the case then what is the mechanism of the asymmetric pressures?

The width of the optic nerve is greatest just behind the globe and narrowest within the optic canal. Thick fibrous bands within the canalicular nerve sheath interrupt the subarachnoid space. Hayreh found that the number of these bands varied from animal to animal and was occasionally scanty. He observed in monkeys and humans that when dye is injected into the optic nerve sheath, it usually passed easily into the cranial cavity. The force needed appeared to depend on the quality of the fibrous bands within the subarachnoid space. Although the dye usually flowed freely, it concentrated in the subarachnoid space adjacent to the optic canals. We suggest that there may be differences among subjects in the trabecular meshwork of the optic nerve sheath subarachnoid space contiguous with the optic canal. When there is elevated CSF pressure, the pressure in the nerve sheath rises enough to distend the sheath, but there is not enough pressure at the optic nerve head to cause high-grade papilledema with resultant high tissue pressure and intraneuronal ischemia in the eyes with low-grade papilledema.

We and others have suggested that, as in glaucoma, there may be selective large fiber loss in patients with papilledema. In these reports, sensitive tests of parvocellular function were not used for comparison. Our data show a loss of many different visual functions when high-grade papilledema eyes are compared with low-grade eyes. This is true whether the tests are biased toward the magnocellular system (motion parame-}