Threshold Static Automated Perimetry of the Full Visual Field in Idiopathic Intracranial Hypertension

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PURPOSE. To characterize visual loss across the full visual field in idiopathic intracranial hypertension (IIH) patients with mild central visual loss.

METHODS. We tested the full visual field (50° nasal, 80° temporal, 30° superior, 45° inferior) of 1 eye of 39 IIH patients by using static perimetry (size V) with the Open Perimetry Interface. Participants met the Dandy criteria for IIH and had at least Frisén grade 1 papilledema with better than −5 dB mean deviation (MD) centrally. Two observers (MW and AS) evaluated the visual field defects, adjudicated any differences, and reviewed optical coherence tomography data.

RESULTS. We found a greater MD loss peripherally than centrally (central 26 dB, peripheral 54 dB, P < 0.001). The median MD (and corresponding median absolute deviations) was −1.37 dB (1.61 dB) for the periphery and −0.77 dB (0.87 dB) for the central 26°, P < 0.001. There were about 30% more abnormal test locations identified in the periphery (P = 0.12), and the mean defect depth increased with eccentricity (P < 0.001). The most frequent defect found was a temporal wedge (25% of cases) in the periphery with another 23% that included this sector with inferior temporal loss. Although the presence of papilledema limited correlation, 55% of the temporal wedge defects had optical coherence tomography retinal nerve fiber layer deficits in the corresponding superonasal location. Other common visual field defects were inferonasal loss, superonasal loss, and superior and inferior arcuate defects. Seven patients (18%) had visual field defects in the periphery with normal central visual field testing.

CONCLUSION. In IIH patients, we found substantial visual loss both outside 30° of the visual field and inside 30° with the depth of the defect increasing linearly with eccentricity. Temporal wedge defects were the most common visual field defect in the periphery. Static threshold perimetry of the full visual field appears to be clinically useful in IIH patients.

Keywords: visual field, perimetry, idiopathic intracranial hypertension, pseudotumor cerebri, ocular coherence tomography

The visual loss detected in idiopathic intracranial hypertension (IIH) is highly dependent on the perimetric testing method used. In a retrospective study that used Goldmann perimetry with a nonspecific two isopter (14e and 12e) screening strategy, one-quarter of patients had visual loss.1 When a modified Armaly-Drance strategy was used that concentrated test location in areas of expected loss, 96% of IIH patients had visual loss.2 Also, it is well known that threshold static automated perimetry is more sensitive in detecting defects than manual kinetic perimetry. Its clinical use is limited by test duration and higher variability in threshold determination with stimulus size III.

The peripheral visual field outside 30° has largely been unexplored with threshold static automated perimetry in clinical practice, even though the untested area outside the central visual field represents over three times the area currently being clinically evaluated. The major reasons that testing outside 30° has been limited with threshold static automated perimetry is that with the size III stimulus, retest variability is often high and the test is longer, tedious, and difficult for the subject due to the small stimulus size. We have shown for the central visual field that the size V stimulus detects defects at least as well as using the size III stimulus.3,4 The size V stimulus is much easier for the subject to attend to especially in the visual field periphery. Also, current tests have a long testing time, as there are no Bayesian strategies with dynamic timing commercially available to rapidly test the far periphery.

In the few threshold static automated perimetry studies published for detection of glaucomatous visual field defects, it has been shown that visual field defects outside 30° are common and may occur when no defects are found within 30°.2 Similar findings have been shown using automated static suprathreshold perimetry.5

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In IIH, most treatment decisions are based on the extent and progression of visual field loss. To explore the far periphery in IIH patients, we used the Open Perimetry Interface (OPI) running on the Octopus 900 perimeter to develop testing strategies for the central and far peripheral visual field. We used the size V stimulus and used a Bayesian testing strategy with dynamic timing. Figure 1 shows the central (filled circles) and peripheral (open circles) test locations for the right eye using the custom OPI tests. After completing a normative database, we tested the central and far peripheral field of 1 eye of 39 patients with IIH and mild visual loss (mean deviation [MD] better than -5 dB on the central visual field examination). Our aim was to characterize visual loss across the visual field in these IIH patients.

**METHODS**

We tested 98 ocular healthy participants twice with normal vision to assemble a normative database with software developed using the OPI on the Octopus 900 Perimeter. Age-corrected pointwise normative limits and corresponding probability cutoffs were developed by linear regression formulas for each location derived by plotting sensitivity against age for each test location. Healthy participants were tested twice with a new perimetry test of the central and far peripheral visual field. The CRAN R visualFields package was used for the development of normative values.

Thirty-nine consecutive patients with IIH and mild visual loss were included in the study if their MD was better than -5 dB on the central OPI test after agreeing to be tested for the central and peripheral tests. Using the normative database from the healthy observers, we constructed pointwise probability plots and used them to determine patterns of visual field defects for those with IIH. All participants were tested twice with a new perimetry test of the central and far peripheral visual field. The CRAN R visualFields package was used for the development of normative values.

Further details of the testing methodology can be found in our report on these ocular healthy participants.

**Participants**

The visual testing protocol was approved by the University of Iowa institutional review board and followed the tenets of the Declaration of Helsinki. We offered patients treated at the University of Iowa Hospitals and Clinics Neuro-ophthalmology Service study admission if they met entry criteria. They all gave written informed consent to participate in the study. They were enrolled if they met the modified Dandy diagnostic criteria for IIH, had at least Frisèn grade 1 papilledema, and had an MD of better than -5 dB on the central OPI test. The patients did not have another disease affecting vision and were capable of reliably performing conventional automated perimetry. A total of 39 IIH patients were tested; 24 were tested twice for repeatability. When subjects were retested, the second test was used for analysis. The average age of the IIH patients was 31.6 with an SD of 8.3 years; the range was 20 to 63.

**Visual Testing**

Participants completed either Goldmann perimetry or standard automated perimetry and were then given two OPI tests: a test

**THE OPI TESTS**

The OPI, a standard for interfacing with visual field testing machines (perimeters), allowed creation of central and peripheral visual field tests by using the Octopus 900 perimeter. The Bayesian test procedure ZEST was used to estimate luminance thresholds at each location. The background luminance was set at 10 cd/m² (31.4 asb), as in conventional perimetry, and the maximum brightness was 1273 cd/m² (4000 asb). The coordinates for the test locations can be found in Figure 1; we used a polar grid with test point spacing that was concentrated in the central 10° and the spacing increased with eccentricity. We used repeatability data from version 1 of the software to eliminate test locations where repeatability was poor. Because values below about 15 to 20 dB are not repeatable, the thresholding algorithm was truncated at 15 dB. Blank presentations were used for false positive catch trials, and near normal thresholds were required for false negatives to be tested. Further details of the testing methodology can be found in our report on these ocular healthy participants.
of the central 26° with 64 test locations and a test of the far periphery out to 50° nasally, 80° temporally, 30° superiorly, and 45° inferiorly, also with 64 test locations. A size V stimulus of 1.72° in diameter was used for the OPI tests. We followed the standard automated perimetry testing recommendations and used a corrective lens for the central field when necessary; no lens was used for the peripheral test. Care was taken to prevent lens rim artifact. If both eyes qualified, the eye with the highest grade papilledema was chosen; if the two eyes were of the same papilledema grade, an eye was chosen at random. All visual field testing met the following reliability criteria: fixation losses less than 20%, a false positive rate less than 20%, and a false negative rate less than 20%. Fixation was monitored by the visual field technician. Our criteria for the presence of a visual field defect was three clustered test locations at the \( P = 0.05 \) level in a clinically suspicious area or two test locations in the area with one of the two at a \( P = 0.02 \) level or worse. We define a clinically suspicious area for IIH as an area where the pattern(s) of defects are known to occur in visual damage from IIH, that is nerve fiber bundle defects and enlarged blind spots.

### Statistical Analysis

We used the CRAN R visualFields package to determine the limits of normality and generate empiric probability plots from the ocular healthy participants.\(^{10}\) We then computed the total deviation probability maps. Using the total deviation probability plot outcomes, we then quantified the number of test locations with loss with a percentile lower than 1, between 1 and 2, and between 2 and 5, and then computed the weighted probability scores for the total deviation map as in Åsmann et al.\(^{15}\) (a value for each visual field test location is calculated by weighing the degree of abnormality as follows: \( P < 0.05 = 2, \ P < 0.02 = 5, \ P < 0.01 = 10 \)). Because the data were not normally distributed, repeated measures ANOVA on ranks was used to compare the number of normal and abnormal test locations among the three probability levels for the central compared to the peripheral test. We also compared the MDs of the central versus peripheral test by using a Wilcoxon rank sum test.

The decrease of sensitivity in decibels with eccentricity appeared fairly linear, so we fitted linear regression on distance from fixation determined by the Pythagorean equation. We used a two-tailed significance test of equal slopes\(^{16}\) to test for differences between IIH patients and healthy participants.

### RESULTS

We found a greater magnitude of visual loss of the MD peripherally than centrally. The median MD (and corresponding median absolute deviations) was \(-1.37 \text{ dB (1.61 dB)}\) for the periphery and \(-0.77 \text{ dB (0.87 dB)}\) for the central \(26°\) (\( P < 0.001\), Wilcoxon rank sum test). The most frequent visual field defect found was a temporal wedge defect (23% of cases) in the periphery, with an additional 23% that included this sector within inferior temporal loss. Other common defects were inferonasal loss, superonasal loss, and superior and inferior arcuate defects (Fig. 2). Seven patients (18%) had visual field defects in the periphery with normal central visual field testing. Two patients (5%) had a normal peripheral visual field and abnormal central visual field.

Although the presence of papilledema limited correlation, 55% of the temporal wedge defects had OCT RNFL deficits in the corresponding superonasal location; 55% of those with inferior temporal loss also had a corresponding OCT deficit. One patient had supranasal loss of the second superior OCT hump with no visual field defect. Other common visual field defects were inferonasal loss, superonasal loss, and superior and inferior arcuate defects. Seven patients (18%) had visual field defects in the periphery with normal central visual field testing.

The mean defect depth for each test location is shown in Figure 3. The colored polygons are a type of optimal quantization called centroidal Voronoi tessellations or cells, a partitioning of a surface into regions so that the center of each cell is its mean (center of mass). Every point in a given Voronoi polygon is closer to its generating point than to any other cell. Note the red values indicating the greatest defect depth is inferonasal, around the blind spot and in the temporal wedge regions. A similar pattern with the red symbols is found in Figure 4 that shows the relative number of abnormal test locations from the pointwise probability plot analysis.

The pointwise probability plot average weighted scores for abnormal test locations are shown in Table 1. Although there was 30% more loss in the periphery, the differences were not significant. To have 80% power to show this difference, we would need a sample size of 199 IIH patients.
FIGURE 3. Average depth of defect using total deviation values for the IIH dataset. Values greater than –2 dB are shown in white. Values smaller than –4.5 dB are shown in red. Values in between are shown in a yellow-orange-red scale. The colored polygons are a type of optimal quantization called centroidal Voronoi tessellations or cells, a partitioning of a surface into regions so that the center of each cell is its mean (center of mass). Every point in a given Voronoi polygon is closer to its generating point than to any other cell.

FIGURE 4. The proportion of eyes below percentile 5 in each location are shown. Values smaller than 20% are shown in light gray. Values greater than 35% are shown in red. Values in between are shown in a yellow-orange-red scale. Notice that the highest frequencies of visual field defects occur inferonasally, around the blind spot and in the temporal wedge area.
The participants’ average test times for the central 26° test (minutes:seconds ± SD) was 6:42 ± 1.02 minutes and for the peripheral test 8:16 ± 1.20. The longer peripheral test times were due to delay introduced by the stimulus projection motor.

The false positive and false negative catch trial rates were acceptable and are found in Table 2.

Figure 5 shows the effect of eccentricity on sensitivity. Sensitivities decreased by about 0.14 dB and 0.27 dB per degree of eccentricity for patients with IIH temporally and nasally, respectively; the values for the healthy participants were 0.10 dB per degree temporally and 0.17 dB per degree eccentricity nasally. The temporal and nasal differences between the IIH patients and healthy participants were highly significant ($P < 0.001$ for each). Sensitivities decreased two times faster with eccentricity nasally than temporally for patients with IIH. For healthy participants, the decrease was also more pronounced nasally than temporally.

Figures 6 through 8 show typical examples of temporal wedge defects confirmed with superior nasal RNFL loss with OCT. Note how limited the loss in the central visual field is with Figures 6 and 7. Figure 8 also demonstrates a discrete temporal wedge defect; also, a superior paracentral defect is found, likely related to the increased number of test locations in the central 10° using this visual field strategy. Although the presence of papilledema limited correlation, 47% of the temporal wedge defects had OCT RNFL deficits of the related superior nasal optic disc.

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DISCUSSION

We found that 23% of IIH patients had temporal wedge defects and an additional 23% had inferior temporal loss that included the temporal wedge sector. This is a higher frequency than other reports where it is seldom mentioned. It is likely that they are not commonly reported by others, as most perimetric strategies either fail to test the far inferotemporal visual field or they use kinetic perimetry, such as Goldmann perimetry, which is not as sensitive to detect shallow visual field defects due in part to statokinetic dissociation.

We attribute this improvement in sensitivity to our new testing strategy that tests the far peripheral visual field and concentrates test locations centrally. It was developed with the

![Figure 7](https://example.com/figure7.png)  
**Figure 7.** Examples temporal wedge defects in IIH patients. Yellow shows \( P < 0.05 \), orange shows \( P < 0.02 \), red shows \( P < 0.01 \), and black represents not seen at 15 dB. The OCTs show excellent clinical-pathologic correlation with superonasal disc damage (arrows); note the superonasal damage is relative in Figure 7. Figures 6 and 7 show a near normal and normal central visual field examination with a temporal wedge defect found in the far periphery.

Table 1. Number of Abnormal Test Locations for the Central 26° and the Periphery Tests

<table>
<thead>
<tr>
<th>Outcome Value</th>
<th>Central 26°</th>
<th>Peripheral</th>
<th>( P ) Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of eyes</td>
<td>39</td>
<td>39</td>
<td></td>
</tr>
<tr>
<td>Number of tested locations</td>
<td>2496</td>
<td>2496</td>
<td></td>
</tr>
<tr>
<td>Locations below percentile 1</td>
<td>275</td>
<td>358</td>
<td></td>
</tr>
<tr>
<td>Locations below percentile 2</td>
<td>72</td>
<td>87</td>
<td></td>
</tr>
<tr>
<td>Locations below percentile 5</td>
<td>148</td>
<td>184</td>
<td></td>
</tr>
<tr>
<td>Locations above percentile 5</td>
<td>2001</td>
<td>1867</td>
<td></td>
</tr>
<tr>
<td>Mean weighted score</td>
<td>1.36</td>
<td>1.76</td>
<td></td>
</tr>
<tr>
<td>SD of weighted scores</td>
<td>1.72</td>
<td>2.32</td>
<td></td>
</tr>
<tr>
<td>Median weighted scores</td>
<td>0.44</td>
<td>0.47</td>
<td>0.486</td>
</tr>
<tr>
<td>MAD of weighted scores</td>
<td>0.56</td>
<td>0.69</td>
<td></td>
</tr>
</tbody>
</table>

MAD, median absolute deviation.  
* Wilcoxon test.

Table 2. Results of Catch Trials With the Different Examinations and Groups

<table>
<thead>
<tr>
<th>Outcome Value</th>
<th>Central 26°, %</th>
<th>Peripheral, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean false positive rate</td>
<td>3.21</td>
<td>3.03</td>
</tr>
<tr>
<td>SD of false positive rate</td>
<td>5.15</td>
<td>5.16</td>
</tr>
<tr>
<td>Median false positive rate</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>MAD of false positive rate</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Mean false negative rate</td>
<td>0.95</td>
<td>6.67</td>
</tr>
<tr>
<td>SD of false negative rate</td>
<td>2.85</td>
<td>6.67</td>
</tr>
<tr>
<td>Median false negative rate</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>MAD of false negative rate</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Percentage of subjects with FPR &gt;0</td>
<td>30.77</td>
<td>28.21</td>
</tr>
<tr>
<td>Percentage of subjects with FNR &gt;0</td>
<td>10.26</td>
<td>38.46</td>
</tr>
</tbody>
</table>

FPR, false positive rate; FNR, false negative rate.
OPI, an open source perimetry development system. Incorporating a Bayesian test strategy with dynamic timing similar to the Swedish interactive threshold algorithm (SITA) enabled us to develop a test with a reasonable test time. Using the size V stimulus, we enabled an extended useful dynamic testing range, better repeatability, and better patient acceptance.

Visual field defects in IIH are very similar in frequency to those in glaucoma except for the commonly occurring enlarged blind spot of papilledema. The lack of OCT damage relating to the enlarged blind spot is likely due to the fact that most of this loss is related to hyperopic retina and is, therefore, refractive in origin. Thus, there is little or no optic nerve damage related to the enlarged blind spot. Areas of RNFL defects most frequently damaged by glaucoma, as shown by OCT, cluster in three regions: (1) the temporal portion of the superior optic disc (superior arcade), (2) the temporal portion of the inferior optic disc (inferior arcade), and (3) the superonasal optic disc. Nerve fiber layer thinning related to the superonasal optic disc causes visual loss in the temporal wedge region and falls outside the central visual field. This area of loss is present in the examples shown in Figures 6 through 8, and the superonasal optic disc RNFL thinning likely contributes to the vision loss present in the inferotemporal portion of the IIH patients’ peripheral visual fields. Fifty-five percent of our IIH patients with temporal wedge defects had related superonasal optic disc damage. We suspect that the reason more defects did not have an OCT correlate was the presence of optic disc edema that was sometimes marked.

The most common types of defects previously reported in IIH patients have been a loss of inferonasal portions of the visual field, constriction, and arcuate scotomas, paracentral scotomas are less common, and temporal sector and altitudinal defects were notably uncommon. Comparing the manual (Goldmann) and automated perimetry strategies, we found that manual perimetry is superior at defining step defects along the nasal horizontal meridian. Generalized loss on the nasal side of the visual field is more common with automated perimetry. The detection of shallow scotomas, especially in the Bjerrum area, is superior with automated threshold static perimetry due to statokinetic dissociation. Our study shows, in addition, that temporal wedge defects are commonly found in IIH patients with mild visual loss when testing the full visual field.

Temporal wedge defects have been reported uncommonly in a variety of other optic neuropathies, including glaucoma, nasal optic disc hypoplasia, and optic neuritis. They can also occur in the absence of a demonstrable cause.

Visual field loss, like the loss associated with glaucoma, may be mild and go unnoticed in IIH; its importance is that it serves as an important marker to guide therapy. In standard practice,
most clinicians use a 24-2 strategy that tests the central 21° with two nasal locations at 27°. Therefore, it is noteworthy that we found a significantly greater MD in the periphery along with the detection of 30% more abnormal test locations outside the central 26° of our test grid. Also, 7 of the 39 IIH patients (18%) had normal central visual field testing with a visual field defect present in the far periphery. Our results demonstrate that testing this largely unexplored peripheral area allows better detection of visual field defects.

We have reported that there was a linear decrease in sensitivity of healthy participants with increasing eccentricity with threshold automated periphery of the full visual field. In this study, we found that IIH patients with mild visual loss also have a linear decrease in sensitivity with eccentricity, and when compared to normals, this falloff in sensitivity is accelerated (Fig. 5). The nasal visual field had a greater rate of decline than the temporal visual field. These findings are similar to prior reports of visual loss across the visual field that show central visual field loss is accelerated with eccentricity.26,27

Limitations of our study included difficulty in correlating OCT defects due to the concomitant optic disc edema. Also, we limited our cohort to those with MDs of the central field of better than −5 dB, so our findings are not generalizable to IIH patients with more severe damage.

In summary, we found significantly more visual loss outside 30° of the visual field than inside 30°, with the depth of the defect increasing with eccentricity. Surprisingly, temporal wedge defects were the most common visual field defect present in the far periphery. We found testing of the full visual field can be done in a practical manner and may have a place in the evaluation of IIH with mild visual loss. The OPI is a useful platform to develop and test new perimetry methods.

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