Retinal Microperimetry as a Means to Assess Visual Field Expansion in Visual Restoration Therapy

Mohamad Chmayssani, Brandon Minzer, Nina Saxena, Roy Arogyasami, Ronald Lazar, Vivienne C Greenstein, Randolph Marshall
Columbia University Medical Center, New York, NY

Methods Cont'd

We defined relative-defect as cells where detection occurred 25% or 50% out of 4 trials at pre-treatment, whereas cells detected zero times were defined as absolute-defect. We excluded locations seen 3 out of 4 times to evaluate for improvement only in more severely affected regions.

We quantified performance within each cell and compared change over time for both relative and absolute zones among the six patients using a paired t-test. Furthermore, we translated the performance, stimulus detection rate, into a visual map using the following color code:

1) Black voxels represent all blind (absolute defect) fields (0) that did not improve.
2) Grey voxels represent blind field (0) that improved either to 1 or 2
3) Lime voxels represent relative defect zones (1, 2) that worsened or did not improve.
4) Lavendor voxels represent relative defect zone (1, 2) that worsened or did not improve.
5) White voxels represent normal defect (3, 4) at baseline.

Results

For the group, there was improvement in stimulus detection at both absolute and relative defect zones (P<0.038). Improvement was seen in each patient, in contiguous cells along the borderzone and blind regions. Table 1 represents the stimulus detection rate at baseline and follow-up including both absolute and relative defect cells.

Figure 2 (a, b) illustrates one run of the Microperimetry at each of the time points for patient 2. Consistent fixation is maintained throughout both trials. Figure 1 (a, b) illustrates one run of microperimetry tests at each of the time points (pre and post VRT respectively) for patient 1. Location of fixation during stimulus presentation is a key displayed on the color digital photograph acquired by the MP-1 color camera (center of blue dots), indicating consistent fixation within 1 degree of the central fixation spot. The MP-1 allows for improvement in the visual field using the color code described above.

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Table 1

<table>
<thead>
<tr>
<th>Patient</th>
<th>T1</th>
<th>T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient1</td>
<td>0.29</td>
<td>0.57</td>
</tr>
<tr>
<td>Patient2</td>
<td>0.33</td>
<td>0.50</td>
</tr>
<tr>
<td>Patient3</td>
<td>0.14</td>
<td>0.18</td>
</tr>
<tr>
<td>Patient4</td>
<td>0.30</td>
<td>0.45</td>
</tr>
<tr>
<td>Patient5</td>
<td>0.22</td>
<td>0.72</td>
</tr>
<tr>
<td>Patient6</td>
<td>0.23</td>
<td>0.60</td>
</tr>
</tbody>
</table>

Conclusion:

● Using microperimetry we showed that visual field maps improved in 5 patients undergoing VRT. Our data suggest that with the use of microperimetry, visual field expansion can be demonstrated to be independent of eye movements.

● Although the mechanism of visual field expansion following visual field training with VRT is not understood by this study, our findings of visual field expansion are consistent with animal models showing changes in cellular receptive fields after injury 2. Other animal studies have shown that training of the visual system may result in plasticity at the cellular level 3. In humans, we previously showed that home training of hemianopic stroke patients on the VRT program resulted in increases in the BOLD signal in regions related to visual processing, and these changes were specific to stimulus in the trained (contralateral) field compared with the untrained field. The biological basis for the remapping of new RFs to neurons within a previously silent cortical region is thought to be long-range horizontal connections in superficial layers of primary visual cortex 4.

References:


Method:

 Patients: Six patients (25-79) with retro-chiasmatic brain injury producing homonymous visual field defects underwent VRT 1-6 months following stroke.

Visual Restoration Therapy: Therapy was done at home twice daily for 20-30 minutes, 6 days a week. VRT targets specific regions of the visual field while the patient maintains central fixation. With the chin supported on a frame 15 inches from an LCD screen, the patient fixates on a central stimulus (diameter=3.5°) and presses a single mouse button when either the central stimulus changes color or an eccentric stimulus appears in the peripheral field. The color change (yellow to green) was designed to be large enough to require saccades for discrimination. The microperimetry tests during therapy consisted of a white square 2 degrees in width which appeared sequentially along a horizontal path from a location in the seeing field 6 degrees from the border of the blind field, into the blind field 8 degrees, and then back into the seeing field. The interstimulus interval varied between 1000 and 1800 msec to minimize anticipation of the next stimulus. Eighty percent of the eccentric stimulus appeared in the visual borderzone, 20% appeared at random locations in the seeing and blind fields to reduce the predictability of target location.

Microperimetry (MP-1): The Nidek MP-1 (Nidek Technologies, Padova, Italy) uses a reference frame for stimulus presentation based on a photograph of the retina vessels. The borderzone is defined as the central 12° of the retina. The location of the stimulus is therefore possible. Following pupil dilation (1% tropicamide and 2.5% phenylephrine hydrochloride) and adaptation to dim room illumination for 30 minutes, the patient maintained fixation on a centrally placed red cross (2° in diameter) while responding to suprathreshold white squares (stimulus size Goldman I, duration 200 msecs, 0 dB) presented on a dim white (background: 1.27’stimulus) background. The non-seeing eye was occluded throughout the procedure. Stimuli locations spacing an area 2.5” in diameter were tested. Stimulus locations were spaced 2 degrees apart from each other and were centered around the fovea. The results of the microperimetry tests and the location of fixation during stimulus presentation were displayed on color digital photographs acquired by the MP-1 color camera.

Fig 1

Fig 2a Fig 2b Fig 2c

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Retinal Microperimetry

HRP map, eyes still

Fig 3

Fig 4

Fig 5

Fig 6

Fig 1a Fig 1b