Abstract for the Imaging and Perimetry Society.

Central and Cecocentral Scotomas and Neuro-Ophthalmic Revisionist Approach

Steven A. Newman, University of Virginia Health System

Purpose

Although it is possible that visual field defects (homonymous hemianopsia) were recognized in ancient Greece, the advent of perimetry as localization, stems to the early portion of the 19th century. During much of the 19th century, it was hoped that specific visual field defects would be diagnostic of etiology. Although cecocentral scotomas have been traditionally felt secondary to neuro-op disease, even Lawton Smith in 1979 in a retrospective study out of Bascom Palmer recognized this was probably fallacious, Toxic metabolic and hereditary causes do produce central and cecocentral scotomas.

Methods

Retrospective study of 193 patients recorded with central or cecocentral scotomas seen through the Neuro-ophthalmology Service between 2008 and 2013. Twenty-nine patients were excluded for transient occurrence or paracentral defects without central fixation loss. Patients were studied with 24-2 program on and most of the patients also had a 10-2 program done.

Results

At least 80 out of 150 patients' fields that had adequate material for evaluation, did have evidence of retinal pathology and no evidence of optic nerve pathology. Unfortunately, at the time this study was begun, we did not have OCT. Thirteen of these patients were felt to have retinal vascular disease, including macular branch artery occlusion and 67 clear evidence of maculopathy. In the later group of these patients, OCT became available and absence of optic nerve pathology was confirmed by normal nerve fiber layer thickness on OCT testing. Optic nerve pathology certainly was possible (24 patients had evidence of optic neuritis and an additional 24 patients had evidence of compressive optic neuropathy). When 10-2 programs were available, one could determine that the central defect was denser. A third optic neuropathy, that of anterior ischemic optic neuropathy could produce central defects and did so in 17 patients.

Interestingly, in 15% of patients as we reported at ARVO had macular edema. Two patients had evidence of Leber's optic neuropathy with confirmation by mitochondrial DNA testing. Two were nutritional, one had dominant optic atrophy, and one had toxic optic neuropathy.It was not at all uncommon that patients had more than one process going on.

Even in a neuro-ophthalmology office, the most frequent cause of central and cecocentral scotomas was retinal pathology was retinal pathology. Just because the macula looked normal, did not necessarily mean that it functioned normally. More than one etiology wa possible, several patients with obvious evidence of optic neuropathology also had evidence of retinal pathology. It should be noted that this study was done over a period of five years and, in many cases diagnosis was presumptive. Some of the findings here are not surprising as in a 1981 study by Samples and Young s a group of patients diagnosied as tobacco alcohol amblyopia with cecocentral defects were re-evaluated Only four had poor nutrition and only 15 drank Of the 27 patients with central defects, 18 were classified as poor nutrition; 19 drank some; and, only 15 smoked. This change in thinking showed up in Morton Smith's publication in 1979 of 65 cases published in Focus in 1980, of 14 bilateral cases were attributed to genetic causes (usually laborers). Unilateral cases were often due to inflammatory causes, including optic neuritis.

Conclusions

1) The pattern of visual field defect is not specific for any etiology.
2) Arcuate visual field defects, if not due to vascular pathology, does indicate the strong possibility of optic nerve pathology. The advent of OCT as a quantitative way of looking at the anatomy of the anterior visual pathways, makes it much easier to define the component due to optic nerve pathology (NFL and ganglion cell thinning on OCT).
3) In a number, clearly gross appearance of the macula was inadequate to exclude macular disease as many of these only showed up on multifocal ERG. OCT and more recent of these was particularly useful as it allows specific comments about retinal pathology and optic neuropathology.
4)"Just because the macular looks normal does not mean it works normal."
5) Clearly, the papillomacular bundle is particularly sensitive to nutritional stress and hereditary pathology.
6) It is likely that cecocentral scotomas per se will not have any specific diagnostic significance.
7) "When the retina service sends you a patient because it's not the macula, it's still most likely the macula."

Properties of measurements from the S-MAIA microperimeter in people with intermediate age-related macular degeneration in the MACUSTAR study

Purpose: To assess test-retest variability and discrimination performance of the S-MAIA in a large cohort of eyes with intermediate age-related macular degeneration (iAMD), as part of the multicentre MACUSTAR study conducted in seven European countries.

Methods: Cross-sectional data from 258 participants with iAMD (n168; age 71±8 years; 63% female), early AMD (n34; age 72±6years; 79% female) and visually healthy peers (n56; age 68±6 years; 59% female) were acquired from a baseline and short-term follow-up (FU) visit (~14 days). AMD status was defined on multi-modal imagining by a dedicated reading centre using the Beckman classification. S-MAIA (CenterVue, Italy) data (mesopic and scotopic average thresholds and pointwise data [PWD]) were collected at 18 centres and screened for procedural errors and unreliable data. Bland-Altman 95% limits of agreement (LoA) were calculated for the average threshold data and PWD stratified by eccentricity. Area under the receiver operating curve (AuROC) assessed how well the different measures discriminated between iAMD, early AMD and healthy peers.

Results: Reliable examination rates were equivalent for mesopic and scotopic microperimetry despite scotopic testing taking longer. Test-retest assessment indicated that both measures (average thresholds and PWD) had equivalent levels of variability, with no systematic differences between baseline and FU (Fig1). Mesopic and scotopic microperimetry could discriminate people with iAMD from controls (AuROC [95% confidence intervals] 68[60,77] and 69 [60,77]% respectively) to a fair level. Yet, both methods could not discriminate between people with iAMD and early AMD (AuROC 50[38,62] and 53[40,65]% respectively).

Conclusion: Mesopic and scotopic microperimetry have similar levels of test-retest variability and only fair discriminatory power for separating iAMD and visually healthy peers. There were no obvious perimetric learning effects likely mitigated by the standard operating procedure (SOP) including a short practice test. 71% of high-quality data were collected in this multicentre setting. This information on properties of measurements from mesopic and scotopic microperimetry will be useful for those planning clinical trials using these modalities. The longitudinal component of MACUSTAR will assess how well these measures perform as markers of progression in iAMD.

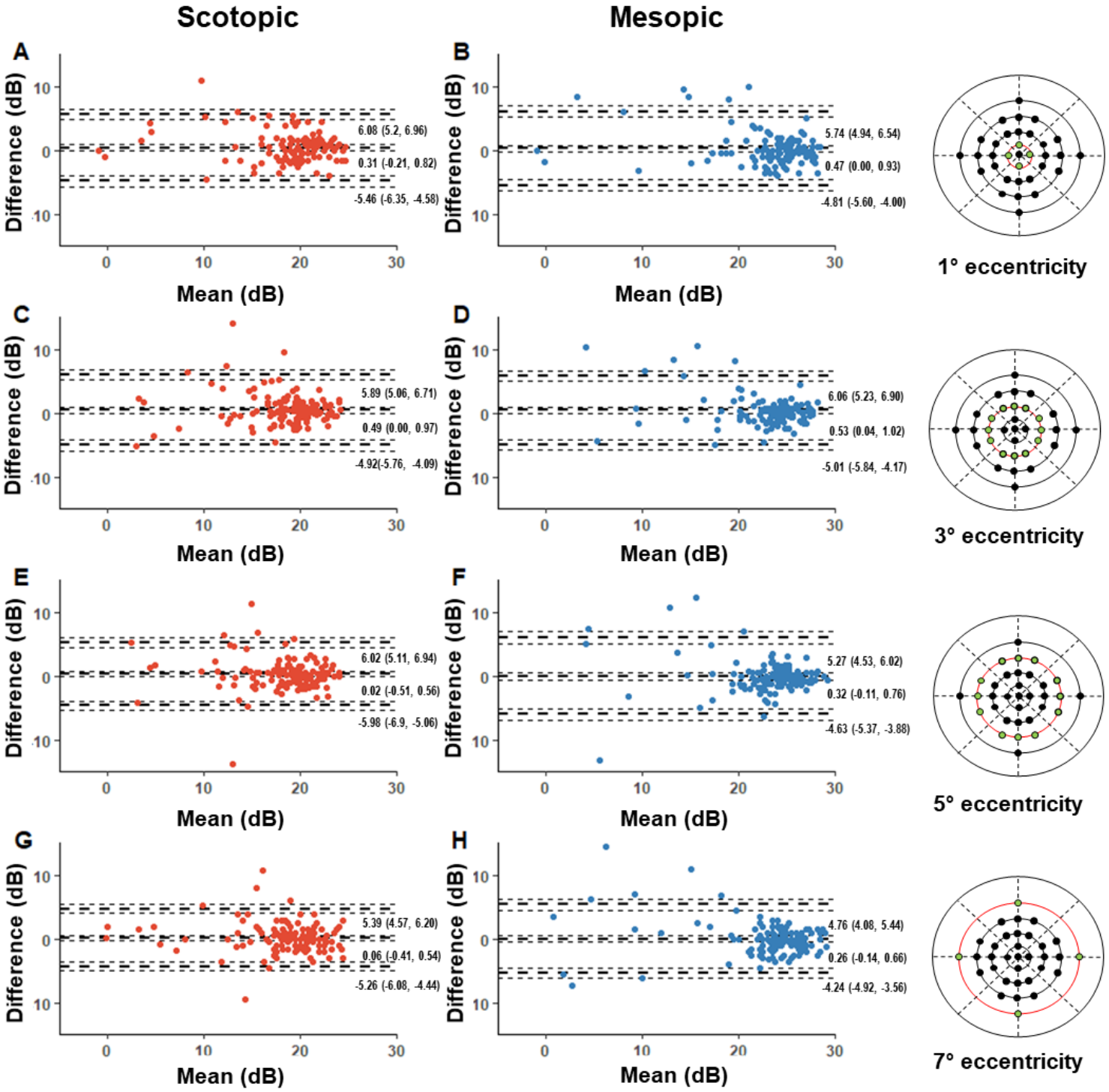

Figure 1. Bland Altman plots to show the between test agreement for mesopic and scotopic point-wise threshold sensitivity, grouped by eccentricity for people with iAMD. Bias, lower and upper limits of agreement are given (95% confidence intervals).

Authors:
Bethany E. Higgins, City, University of London
Giovanni Montesano, City, University of London; Moorfields Eye Hospital NHS Foundation Trust, London, UK
Hannah Dunbar, University College of London Institute of Ophthalmology, London, UK
Alison M. Binns, City, University of London
Deanna R. Taylor, City, University of London
Charlotte Behning, University Hospital Bonn, Institute for Medical Biometry, Informatics and Epidemiology, Bonn, Germany
Amina Abdirahman, University College of London Institute of Ophthalmology, London, UK
Jan H. Terheyden, University Hospital Bonn, Department of Ophthalmology, Bonn, Germany
Nadia Zakaria, Translational Medicine, Novartis Institute for Biomedical Research, Cambridge
Stephen Poor, Ophthalmology Research, Novartis Institute for Biomedical Research, Cambridge
Robert P. Finger, University Hospital Bonn, Department of Ophthalmology, Bonn, Germany
Sergio Leal, Bayer AG, Berlin, Germany
Frank G. Holz, University Hospital Bonn, Department of Ophthalmology, Bonn, Germany
Matthias C. Schmid, University Hospital Bonn, Institute for Medical Biometry, Informatics and Epidemiology, Bonn, Germany
Ulrich F.O. Luhman, Roche Pharmaceutical Research and Early Development, Translational Medicine Ophthalmology, Roche Innovation Center Basel, Switzerland
Gary S. Rubin, University College of London Institute of Ophthalmology, London, UK
David P. Crabb, City, University of London
On behalf of the MACUSTAR consortium

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Nadia Zakaria, (E) Novartis Institute for Biomedical Research
Stephen Poor, (E) Novartis Institute for Biomedical Research
Robert P. Finger, (C,F) Bayer, (C,F) Novartis, (C) Roche/Genentech, (C) Alimera, (C) Böhringer-Ingelheim, (C) Santhera, (C) Ellex, (C, F) Novartis, (F) Zeiss, (F) Heidelberg Engineering, (F) CenterVue, (F) Biogen;
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