

ARCHIVOS DE LA SOCIEDAD ESPAÑOLA DE OFTALMOLOGÍA

www.elsevier.es/ofthalmologia



Editorial

Overall topographic frequency of glaucoma defects. In memory of Erich Seidel (1882–1948)

La frecuencia topográfica global de los defectos en el glaucoma. En recuerdo de Erich Seidel (1882-1948)

Manuel González de La Rosa,^{a,*} Julián García-Feijó^b

^aServicio de Oftalmología, Hospital Universitario de Canarias, Universidad de La Laguna, Canary Islands, Spain

^bServicio de Oftalmología, Hospital Clínico de San Carlos, Universidad Complutense, Madrid, Spain

It is well-known that glaucoma exhibits a preference for specific visual field areas. The lesion occurs in the optic nerve head, so that generally nervous fiber groups in the area are damaged at the same time, affecting more or less broad regions of the visual field.

In 1914 Seidel pointed out that one of the typical defects could be the lengthening of the sickle-shaped blind spot¹ (Seidel's original article is available at <http://www.springerlink.com/content/q3583550651442v9/fulltext.pdf>), while in relation to the first visual field lesions, Traquair and Dott² stated that generally this depression is expressed sometimes close to the blind spot and others in the form of a small initial nasal step («As a rule this depression is most manifest near the blind spot, constituting the barring of the blind spot; sometimes it is apparent at an early stage on the nasal side in the form of a small nasal step, in other cases both of these features may be present together»).

However, in his renowned classification, Aulhorn³ defined the first stages of glaucoma as isolated or arched scotoma separated from the blind spot and, in the early days of automatic perimetry, the paracentral and nasal scotomas were defined as the initial ones.⁴

At the time, some authors insisted on the possibility of initial defects close to the blind spot, but these would be less frequent than the nasal superior defects.⁵ Finally, other researchers pointed out that the initial defect could be

diffuse.⁶ The latter hypothesis did not attract many followers and for a long time the diffuse component of visual fields was suppressed as it was considered to be caused by cataracts.⁷ Quite recently we have verified that this opinion is not correct and that in fact the initial progress of glaucoma is characterized by a low focality.⁸

When the disease progresses, it does not always do so in the same way. Sometimes the defects are predominantly superior and others inferior, while in other cases it affects both hemifields. Likewise, on some occasions central fiber groups are affected and on others peripheral groups. Some years ago we attempted to classify all of these possibilities⁹ and this attempt matched quite well the descriptions of the main nervous fiber groups.¹⁰

Another classical idea to be emphasized is that generally the central vision survives together with the temporal field up to the terminal stages of the disease. However, the observation of each case does not provide an overall view of the capacity the disease has to damage or respect specific areas.

The mental representation we have of each glaucoma patient corresponds to that classical vision, derived from the case to case observation, and doubtlessly it is partially correct. However, there is another possible approach: not in all cases the same fiber groups are affected, which leads us to wonder in which regions the initial defect appears more

*Corresponding author.

E-mail: mgdelarosa@telefonica.net (M. González de La Rosa).

frequently and also which areas are globally more resistant to the damages caused by the disease.

The database of the San Carlos Clinic Hospital (Ramón Castroviejo Ophthalmological Research Institute, Complutense University) comprises a wealth of information to address research of this kind. In the course of 13 years, over 100,000 explorations have been gathered made with the same perimeter (Octopus 1-2-3. Haag Streit, Bern, Switzerland) and the same application (Tendency Oriented Perimetry, G2 mesh). We have utilized this database to obtain the information we were looking for.

In the first place, we discarded cases with isolated or associated neurological and macular pathologies. Finally, we selected the last exploration carried out for each patient, accepting the results for both eyes considering the size of the sample and interpolating data to transform them to format 32 (the most widely used format), totaling 18,397 visual fields.

Subsequently, we separated in a first group the cases exhibiting a mean defect (MD) below 3dB, accompanied by low irregularity (square root of the $sLV < 2.66$ dB loss variance)¹¹, mainly made up by ocular hypertensive patients (OHT) and glaucoma suspect cases (7,704 cases). The 4 remaining groups were formed with the MD value below 3dB (1,354 cases), between 3 and 6dB (3,603 cases), between 6 and 12dB (5,450 cases) or over 12dB (286 cases).

The mean threshold value in each point of the visual field per group is shown in figure 1.

Said results confirmed some of our axioms about the topography of glaucomatous defects and raised questions about others. Firstly, the initial defects seem to be related to the blind spot because the damage preferably occurs in the superior temporal region at nearby points.

Even in ocular hypertensive patients a small reduction of sensitivity in the area could be observed, which calls for additional research. For example, the mean defect of the 5 outlined points close to and over the blind spot was superior to the mean defect of the remaining points in 78% of ocular hypertensive patients and those suspect of glaucoma. This is an interesting information which even so cannot be afforded high specificity because it can occur randomly in 50% of normal subjects. However, we believe it could be a sign to be considered together with other risk factors.

The progression continues, forming an arch in the Bjerrum area, mainly in the upper hemifield but not centripetal to the papilla as we had assumed a few years ago,¹² but in a centrifugal manner so that the late effects concentrate at a distance, in the nasal region.

On the other hand, the data indicate that the resistance of the temporal field to glaucomatous damage is not as obvious as we had assumed. It cannot be doubted that this is a usual expression in specific cases, but overall glaucoma seems to affect the temporal field in a more acute manner than the inferior field.

On the other hand it is confirmed that the defects of the paracentral region do not evolve in parallel to peripheral defects. This area has a higher resistance to damage but only in an order of magnitude. Accordingly, the particularly relative paracentral scotomae are not only a fact but they

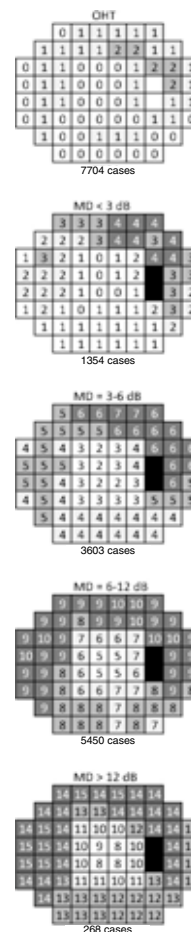


Figure 1 – Mean local defect in 5 groups of patients classified as per the magnitude of their mean defect (MD). The levels of gray are arbitrary in order to emphasize the depth of the defect within each group.

occur with great frequency. In relatively initial stages (MD between 3 and 6dB) the paracentral defect accounts for over 50% of the damage exhibited by the rest of points, and in the advanced stages of the disease this proportion exceeds 65%.

A few years ago we published an editorial¹³ reminding our readers about the need to honor the contributions of the masters and review not only recent literature but also that which provided the current groundwork of our knowledge. As the main conclusion of this analysis, it could be stated that even though Seidel's typical deep and elongated blind spot scotoma (fig. 2) is not the most frequent form of glaucomatous damage in its mean and advanced stages, we must admit that this author was right when pointing out the most frequent initial location of glaucomatous defects.

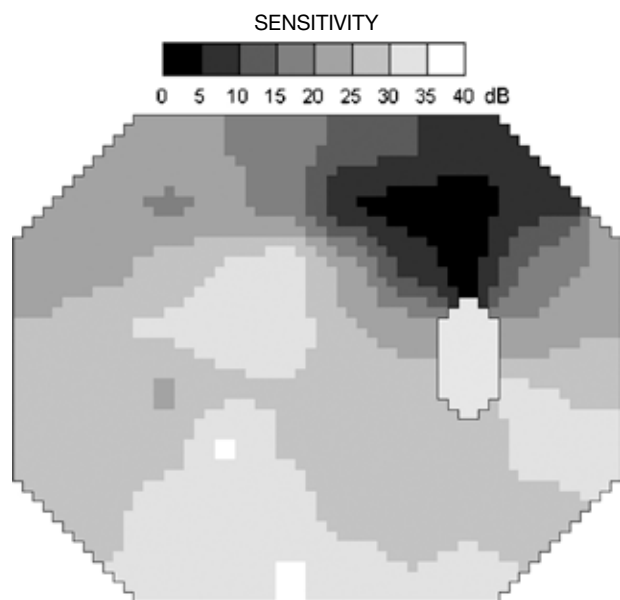


Figure 2 – Seidel's scotoma.

Funding

Manuel González de la Rosa has a commercial interest in the TOP application.

REFERENCES

1. Seidel E. Beiträge zur Frühdiagnose des Glaukoms. Arch f Ophth. 1914;88:102-57.
2. Traquair HM, Dott NM. An introduction to clinical perimetry. London: Henry Kimpton; 1942.
3. Aulhorn E. Glaukom-Gesichtsfeld. Ophthalmologica. 1969;158: 469-87.
4. Drance SM, Susanna R, Fairclough M. Frühe Gesichtsfeldausfälle bei Glaukomekrankung. Klin Monbl Augenheilkd. 1978;173: 519-23.
5. Gramer E, Gerlach R, Krieglstein GK, Leydhecker W. Zur Topographie früher glaukomatöser Gesichtsfeldausfälle bei der Computerperimetrie. Klin Monbl Augenheilkd. 1982;180:515-23.
6. Caprioli J, Sears M, Miller JM. Patterns of early visual field loss in open-angle glaucoma. Am J Ophthalmol. 1987;103:512-7.
7. Heijl A, Lindgren G, Lindgren A, Olsson J, Asman P, Myers S, et al. Extended empirical statistical package for evaluation of single and multiple field: Statpac 2. In: Mills RP, Heijl A, editors. Perimetry Update 1990/1. New York: Kugler & Ghedini; 1991. p. 303-315.
8. Gonzalez de la Rosa M, Gonzalez-Hernandez M, Sanchez-Mendez M, Medina-Mesa E, Rodríguez de la Vega R. Detection of morphological and functional progression in initial glaucoma. Br J Ophthalmol. 2010;94:414-8.
9. González de la Rosa M, González Hernández M, Aguilar Estévez J, Abreu Reyes A, Pareja Ríos A. Clasificación topográfica del campo visual glaucomatoso. Arch Soc Esp Oftalmol. 2002;77:87-94.
10. Garway-Heath DF, Poinoosawmy D, Fitzke FW, Hitchings RA. Mapping the visual field to the optic disc in normal tension glaucoma eyes. Ophthalmology. 2000;107:1809-15.
11. González de la Rosa M, González-Hernández M, Garcia Feijoo J, Morales J, Azuara-Blanco A. Diagnostic accuracy and reproducibility of tendency oriented perimetry in glaucoma. Europ J Ophthalmol. 2006;16:259-67.
12. González de la Rosa M, Arias Puente A, Morales J, García Sánchez J. Análisis del campo visual. In: Honrubia López FM, García Sánchez J, Pastor Gimeno JC, et al. Diagnóstico precoz del glaucoma. Madrid: Sociedad Española de Oftalmología; 1997; p 249-370.
13. González de la Rosa M. El maestro, el método y la memoria. Arch Soc Esp Oftalmol. 2003;78:183-4.