Visual fields interpretation

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ABSTRACT — From the retina to the visual cortex, commonly encountered visual field defects are discussed. Retinal, optic nerve head, optic nerve, optic tract, lateral geniculate, radiations and cortex lesion examples are described with predicted outcomes and possible variations of field defects.

KEY WORDS — knees of Wilbrand, chiasmal syndrome, spontaneous venous pulse, papilledema, papillitis, Meyers loop, retinal filling.

Interpretation of visual fields results depends on information learned from a complete case history, through ocular examination and a basic knowledge of ophthalmic neurology. Ocular examinations performed without visual field screenings or testing are incomplete. Lesions involving the peripheral retina, asymptomatic retinal detachments, low tension glaucoma and other conditions will usually be missed or at least poorly documented without field testing. A routine complete ocular health examination should contain the following tests:

Evaluation of the optic nerve head
Pupillary study including afferent, consensual, direct and near responses
Extraocular muscle evaluation
Complete case history
Visual field screening (other than confrontation)

Each case history should include questions concerning blurouts or blackouts of vision, headache, nausea, paresis or paralysis, diplopia, reduced vision and "blind spots" in the visual fields.

This paper will begin at the retinal level, reviewing neuroanatomy and interpreting commonly seen visual fields while progressing from retina to the visual cortex.

Lesions affecting the retina and retinal/choroid system produce somewhat characteristic visual field defects, similar in configuration to the observed lesion. For example, a perimacular chorioretinitis scar one disc diameter superior to the macula produces a similar size scotoma in the inferior perimacular field. Naturally the size of the lesion must be sufficient to produce a defect. Histo spots are seldom plotted with commonly available clinical tools. In some cases of long standing, or healed lesions, the size of the defect is smaller that predicted. "Retinal filling" occurs to produce a smaller scotoma. Perhaps the actual damage to the retina itself is less than observed, because the retina is transparent, making it difficult to accurately determine the boundaries of the lesion. Juxtapapillary chorioretinitis is another example of you "plot what you see". The degeneration around the nerve head produces a blind spot enlarged in size similar to the peripapillary destruction.

Subretinal lesions commonly encountered include serous leakage. These serous choroidopathies (retinopathy is the older term) produce symptoms of distorted vision, visual loss, and in severe cases, scotomas. In the case of central serous choroidopathy, the vision will be distorted and reduced and a central sloping margin scotoma plotted. In such situations, Amsler grid will serve better to document as well as follow the patient's progress.

Retinal detachments usually produce large scotomas. Such detachments are not always symptomatic and can be missed if some sort of routine field study and/or dilation is not performed. An example of such a case is a 52-year

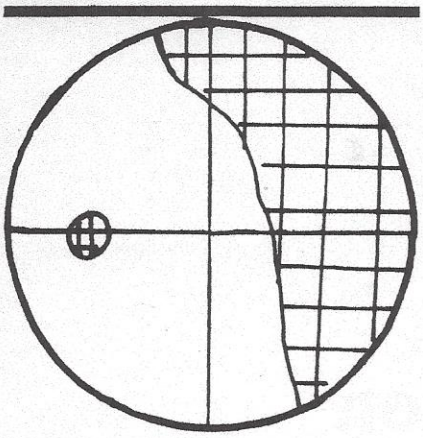


Fig. 1: Retinal Detachment

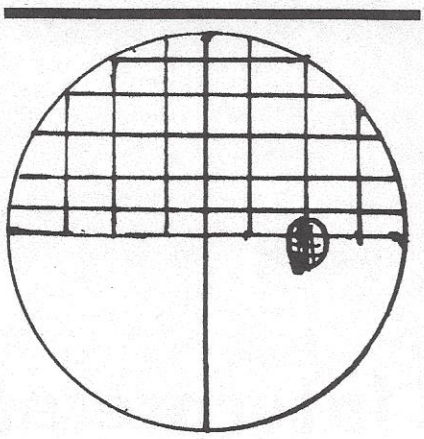


Fig. 2: Altitudinal Defect

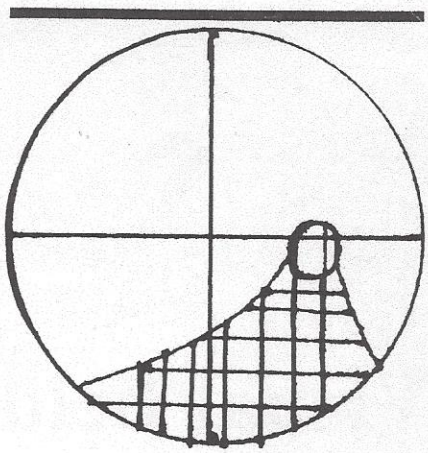


Fig. 3: Retinal Vascular Lesion

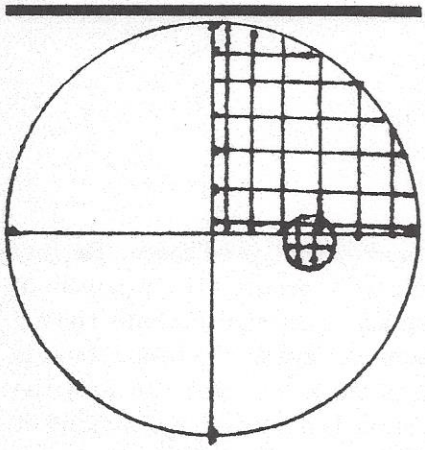


Fig. 4: Neurological Lesion

old female presenting for a routine examination. She was asymptomatic. Ophthalmoscopy was difficult since her pupils were less than 2 mm. Routine field screening with a multipatterned screener disclosed a large scotoma characteristic of a retinal detachment.

Such scotoma can cover any part of the visual field crossing either or both vertical and horizontal field separations.

Retinal arterial vascular tree defects

Retinal arterial vascular tree defects are predictable and consistent with their retinal location. Vessels emanate from the optic nerve head to the periphery in a superior nasal and temporal branch and an inferior nasal and temporal branch. The superior temporal branch remains superior, not crossing the horizontal raphe (separation of superior and inferior retinal halves) and the inferior temporal branch remains inferior. The nasal tree is not as organized or predictable since no anatomic division of superior and inferior exists. Devastating retinal arterial conditions, such as an inferior occlusion, produce defects affecting the superior field. The retina supplied by the occluded vessel dies and a resultant defect occurs. A total arterial occlusion would affect all arterial blood supplies with a resultant blind eye. Therefore, retinal vascular disease of the arterial variety produces defects which emanate from the optic nerve head[1] (Figure 3), depending on where the artery is occluded.

Branch arterial disease produces a more localized and smaller defect, but the scotoma if extrapolated would begin at the optic nerve. This is in diagnostic contrast to neurological lesions, which as you will discover, have the point of *fixation* as their imaginary apex (Figure 4).

Venous occlusions generally produce localized bleeding and if sufficient blood accumulates in the retinal tissue a corresponding scotoma may result. Devastating central vein occlusions usually produce severe vision loss.[2] In such cases, improvement in the visual field can occur. To monitor such a case, large targets are recommended. It is best to detect the presence of a scotoma with a small target (ie., 2 mm.) but to follow the progress with a larger target. The patient with a field defect undergoing change is more aware of a 20 mm. to 15 mm. than a 5 mm. to 2 mm.

Optic nerve head

Glaucoma is probably the most recognized disease of the optic nerve head. It too presents a defect which emanates or directs toward the disc. Atrophy of nerve tissue observed as cupping begins in the vertical meridian of the cup. Early notching is recognized at 12:00 and 6:00 with corresponding visual field defects from damaged fibers.

Note in Figure 5B, a more advanced glaucomatous disc, that the visual field "stops" at the nasal border of the horizontal raphe separating superior from inferior field halves. This nasal step may be the first indication of early glaucoma and perhaps these corresponding temporal retinal fibers are the most sensitive. Nevertheless, visual fields appear to be the best way of detecting early glaucoma. This is especially true in low tension glaucoma.

Drusen can affect the visual field when located at the level of the optic nerve.[3,4,5] An enlarged blind spot can result as well as a

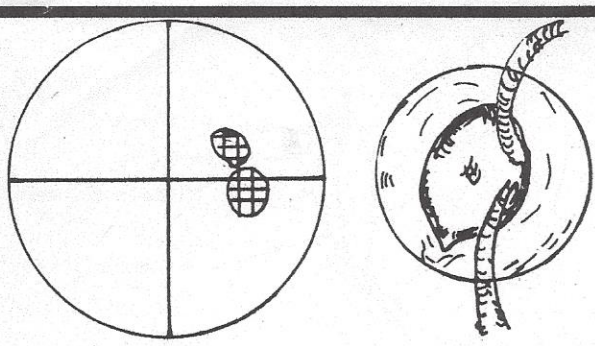


Fig. 5A: Early Glaucoma

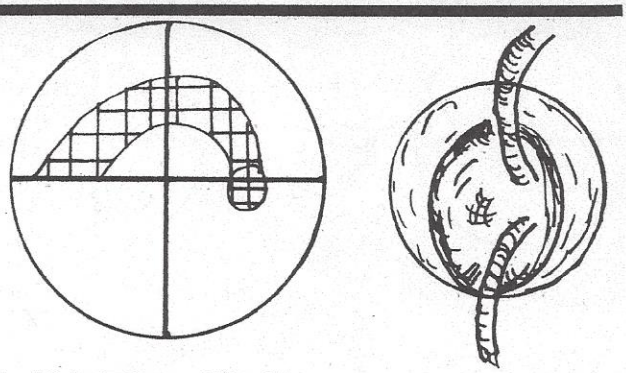


Fig. 5B: Late Stage of Fig. 5A

pseudoglaucomatous arcuate scotoma. Patz and Ornith report secondary serous leakage in the subretinal space as a result of drusenoid nerve head deposits presenting with unusual complaints and scotomas.

Peripapillary changes are found in several conditions:

1. Aging changes
2. Jensen's juxtapapillary chorioretinitis
3. Myopic and scleral crescents

All peripapillary defects result in an enlarged blind spot corresponding to the area of atrophic retina. This type of enlarged blind spot should not be confused with edematous etiology. Papilledema can occur from intracranial disease, hypertension grade IV, and orbital tumor. The two former conditions produce bilateral edema, the latter, unilateral.

In cases of papilledema, the spontaneous venous pulse (SVP) is absent. Normally, 80% of the population have an SVP, 20% of the normals do not.[6,7] So when evaluating an optic nerve head with swollen margins, note the presence or absence of a *previously* seen SVP. Conditions producing pseudopapilledema include drusen, hyperopia and medullated nerve fibers.

Optic nerve atrophy

Optic nerve atrophy produces reduced V.A., afferent pupil defects (unless both optic nerves involved equally), defective color vision for red-green, pale disc and a central scotoma. Optic neuritis affects the optic nerve in a similar way and should be considered in all cases of sudden reduction in central vision and pain with movement of the eye. Papillitis produces a moderately swollen nerve head, is usually unilateral and should be differentiated from papilledema. To compare the two conditions clinically, see Table 1.

TABLE 1

SIGN/SYMPTOM	PAPILLEDEMA (FIG. 6)	PAPILLITIS (FIG. 7)
vision	usually normal	reduced
pain	absent	on movement
SVP	absent	present
pupil	normal	afferent defect
disc swelling	2-5 diopters	up to 2 diopters
visual field	enlarged blind spot	central scotoma

In retrobulbar optic neuritis, all the same symptoms and signs as in optic neuritis occur except the optic nerve head appears normal (Figure 7).

When confronted with reduced vision in one eye and papilledema in the fellow eye, Foster-Kennedy syndrome should be considered. A tumor of the frontal lobe produces optic atrophy on the same side, and due to increased intracranial pressure, papilledema is produced on the opposite side. (Figure 7 & 8 combined). However, the examiner can be fooled. In older patients, a previously resolved ischemic optic neuritis can result in optic atrophy. When the second eye becomes involved, at a later date, the patient seeks care now noting reduced vision. Evaluation shows a mild swelling of the "fresh" eye and old optic atrophy of the fellow eye. Pseudo-Foster-Kennedy is the true diagnosis.[8]

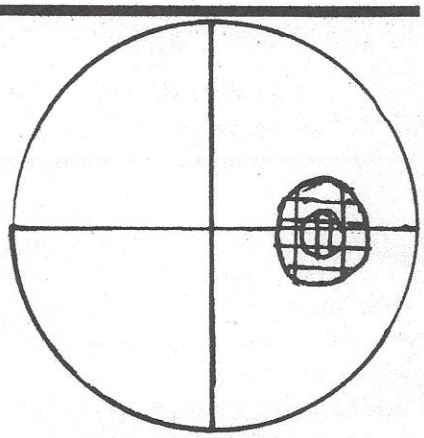


Fig. 6: Enlarged Blind Spot

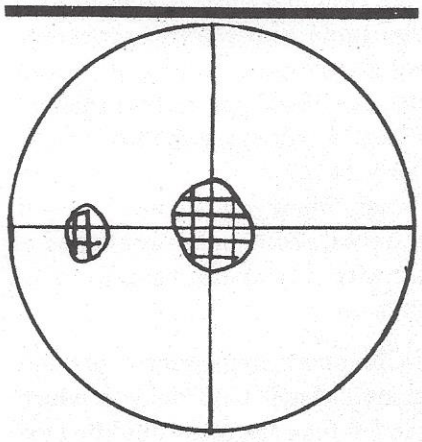


Fig. 7: Central Scotoma

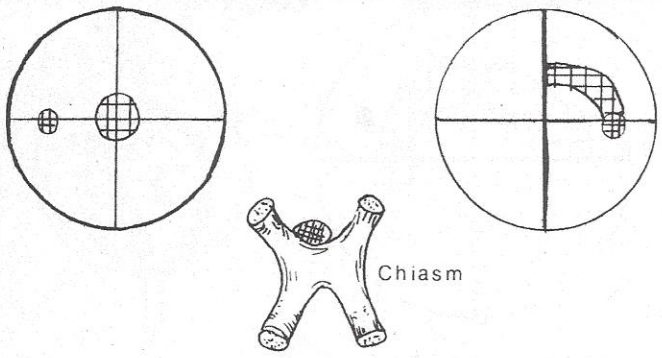


Fig. 8: Junction Lesion

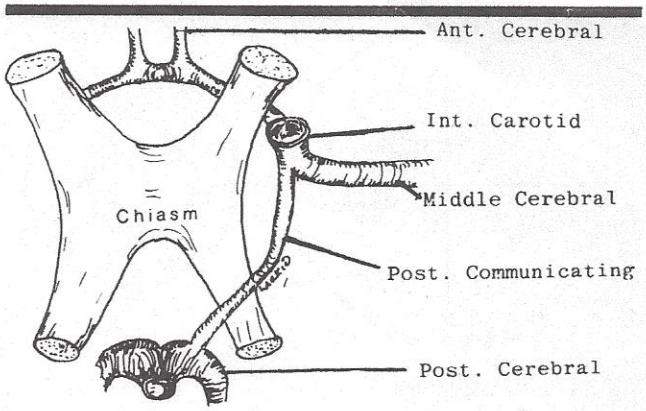


Fig. 9: Chiasm surrounded by the Circle of Willis — Viewed from below

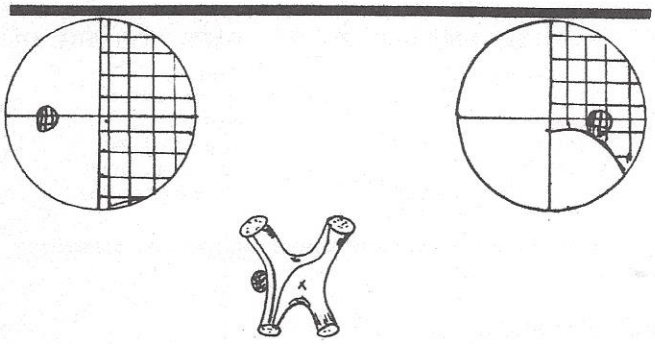


Fig. 10: Chiasmal Lesion

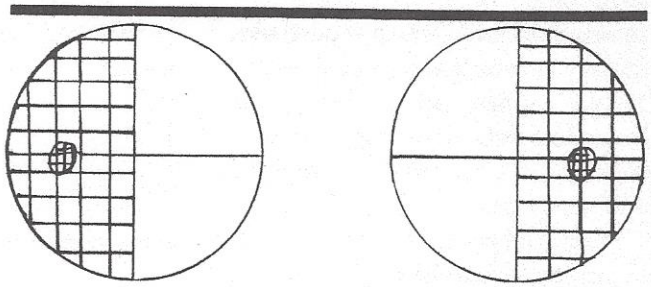


Fig. 11: Bitemporal Hemianopsia

Optic nerve

The optic nerve, for the purposes here, is considered to be that portion of the nerve beginning 1 mm behind the eye and ending at the chiasm. Lesions of this structure produce optic atrophy with the same signs and symptoms noted above. Commonly seen causes include optic nerve glioma. When a glioma expands posteriorly to affect the chiasm, a previously blind eye is now observed to have its companion eye presenting with a temporal hemianopsia.

Chiasm

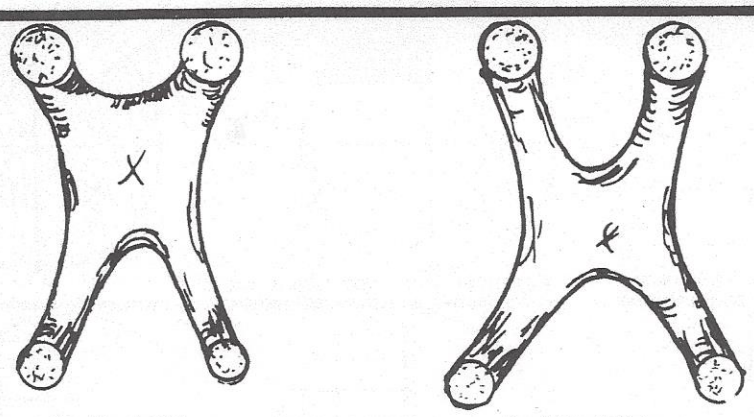
Chiasmal syndromes present unique visual field defects which can confuse the most qualified examiner. Peculiar variations in the visual fibers as they course through the chiasm are the culprits (Figure 8).

The left nasal fibers cross to the right side and the right nasal fibers cross to the left. The temporal fibers remain on the ipsilateral (same side). The chiasm is located in the bony saddle called the sella turcica, with the floor of the third ventricle making up the roof of the chiasm. (See Figure 10.)

Vascular supply is via the circle of Willis with blood flowing toward the circle in the internal carotids (Figure 9).

The most common factor producing field defects is a pituitary body tumor, located inferior to the chiasm. The typical chiasm field defect is the bitemporal hemianopsia (Figure 11). Rarely, simultaneous lesions on both sides of the chiasm produce binasal defects. A large craniopharyngioma can produce such a defect. In reality though, only 80% of the population have a chiasm located directly in the sella turcica with the pituitary directly below. From 4-10% have long optic nerves with the chiasm post-fixed[9] (Figure 12). With this variation, pituitary tumor can insult the chiasm in various locations. In the case of a post-fixed chiasm, a tumor from below will impinge on the anterior chiasm producing optic atrophy of one eye and an arcuate or upper temporal defect in the fellow eye (Figure 8). A hasty examiner could mistake the arcuate defect for glaucoma! Next, consider the macular fibers, they course through the chiasm in the inferior two-thirds, crossing at the posterior portion. A prefixed chiasm could result in a pituitary tumor demonstrating perimacular scotomas.

The "Knees of Wilbrand,"[10] are a variation in the clasically considered pathway through the chiasm. The nasal inferior fibers course thought the anterior chiasm and forward to form an anterior loop or knee in the contralateral optic nerve. The superior nasal fibers remain ipsilateral to the posterior optic tract (posterior to the chiasm) forming a posterior ipsilateral knee before crossing to the opposite side (Figure 13). Therefore, lesions located in or around



Pre-fixed Chiasm

Post-fixed Chiasm

Fig. 12

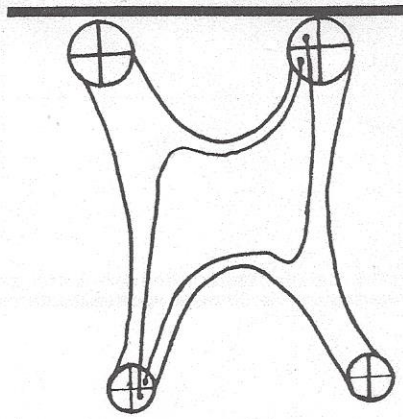


Fig.13: Knees of Wilbrand

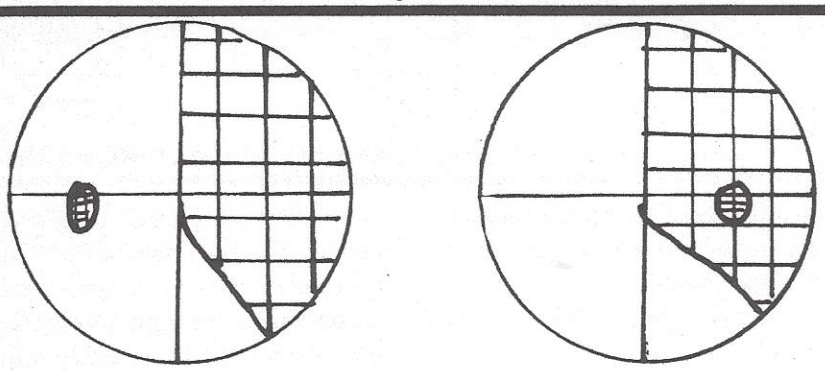


Fig.14: Optic Tract Lesion
Right Homonymous Incongruous Hemianopsia

the chiasm could produce atypical visual field defects. For example, an optic nerve glioma of the left side could produce a central scotoma in the left eye with secondary optic atrophy, and a superior temporal quadranopsia partial or complete, in the right eye.

Fibers emanating from superior retina remain superior all the way to the cortex, the inferior remain inferior. Temporal remain ipsilateral, nasal course contralateral. A natural neurological division results placing the fixation point, or the fovea, as the apex of neurological lesions compared to the optic nerve in vascular etiologies of the retina.

Superior lesions produce inferior defects, inferior lesions produce superior defects. But like every rule, there are exceptions. A visual field defect can be produced from direct insult on the pathway, or from displacement of the pathway to a firmer structure with the resultant damage opposite to the side of the lesion. The field result would be on the same side as the lesion! An example of this is found with increased intracranial pressure via the third ventricle displacing the chiasm inferior towards the sella turcica and pituitary. The resultant visual field defects mimic a pituitary tumor. The visual field defect is a bitemporal superior quadranopsia. The actual site of the cause is also superior! In such cases too, the examiner could miss a tumor in the posterior cranium causing increased CSF yielding displacement of the chiasm resulting in a pseudopituitary field defect.

Generally speaking, pituitary tumors produce optic atrophy. Other types of intracranial tumors sufficiently large or near a ventricle will cause papilledema. Therefore, a bitemporal hemianopsia with papilledema suggests a tumor in or near the ventricle system and generally not a pituitary lesion.

Optic tract

Optic tract lesions are difficult to localize based on visual field data alone. Resultant field defects can be similar to optic radiation lesions, that is, a homonymous hemianopsia. Generally, the optic tract lesions tend to be incongruous with a greater likelihood of macular splitting (Figure 14).

Since the optic tract is that portion of the pathway with the temporal ipsilateral fibers joining the nasal contralateral fibers, immediately posterior to the chiasm and anterior to the lateral geniculate, organization of the fibers is less than farther posteriorly.

A previously documented homonymous hemianopsia which now presents with severe vision reduction in one eye suggests a tract lesion expanding towards and involving the chiasm.

Lateral geniculate body

The lateral geniculate body (LGB) is a synapse of the visual fibers originating at the retinal level. From here, the fibers course into two divisions, a superior and inferior branch (Meyer's loop).[11,12] Meyer's loop is in the tip of the temporal lobe. Tumors in the temporal parietal area produce in addi-

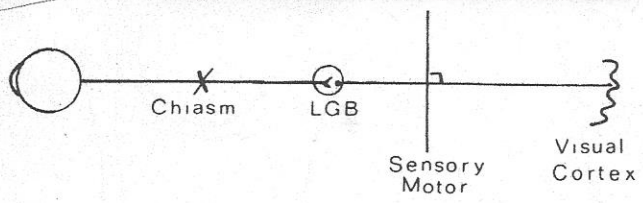


Fig. 15: Relationship of Sensory Motor and Visual Pathways

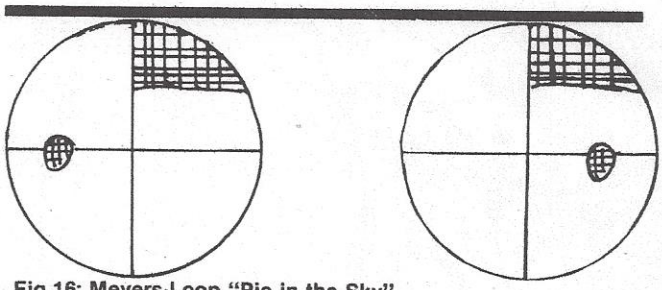


Fig.16: Meyers-Loop "Pie in the Sky"

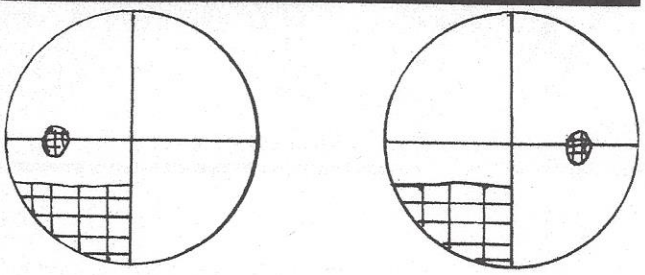


Fig 17: Superior Temporal Loop Defect

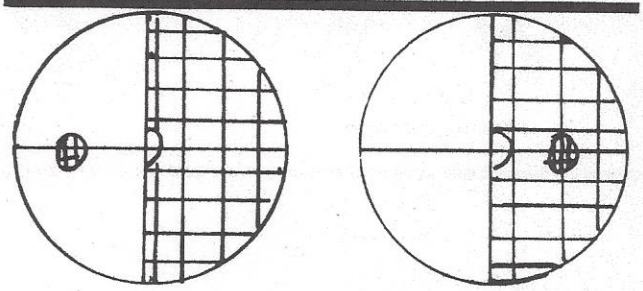


Fig.18: Right Homonymous Hemianopsia with Macular Sparing

tion to homonymous field defects, formed or familiar visual hallucinations.

The classic visual field defect from a lesion in Meyer's loop is the "pie in the sky" scotoma (Figure 16). Superior loop lesions produce inferior scotomas (Figure 17).

Slightly posterior to the LGB the visual fibers intercept the vertically positioned fibers of the sensory-motor pathway (Figure 15).

Lesions commonly seen in this area are cardiovascular accidents (CVA) or strokes. The resulting visual field defects of homonymous hemianopsias are accompanied by paresis or paralysis of the contralateral side of the body. The paralysis is on the same side of the scotoma, opposite the side of the lesion. Also a general rule is that patients look toward a hemianopsia and away from the lesion. A right sided hemianopsia may present difficulty for the patient during reading, therefore, the page may be turned 15 to 90 degrees away from the field defect towards normal field. Someone presenting with a reading habit as this, may be compensating for a scotoma.

As the fibers move posteriorly, they become more organized. As the visual cortex is approached, the more lateral fibers protect the more medial *macular* fibers which cover an extremely large area of the visual cortex along the calcarine fissure.

Therefore, the more posterior the lesion, the greater likelihood of a congruous homonymous hemianopsia with macular sparing (Figure 18).

Occipital lobe tumors

Occipital lobe tumors produce unformed hallucinations[13,14] similar to the type reported in migraine suffers. Since the visual cortex is well supplied by the vascular network, CVA's are common sources of lesions producing scotomas and symptoms. As a rule, vascular accidents produce absolute sharp bordered scotomas, and tumors produce sloping relative defects due to their slow progress.

Summary

Many of the common types of visual field defects have been described, along with some of the variations and unique differences. Interpretation begins with complete data, followed by additional specific testing culminating in the assessment through ophthalmic neurology knowledge. Like a chain, any weak area prevents a good diagnosis or evaluation. So it is essential that each optometrist not only produce high quality field data but also be thoroughly familiar with all of its interpretation.

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REFERENCES

1. Walsh TJ: *Neuro-Ophthalmology: Clinical Signs and Symptoms.* Philadelphia, Lea & Febiger, 1978, p 265.
2. *Ibid,* p 9.
3. *Ibid,* p 7.
4. Glaser JS: Topical diagnosis: Prechiasmal visual pathways, in Duane TD (ed): *Clinical Ophthalmology,* vol 2, ch 5. Hagerstown, Md., Harper & Row, 1978.
5. Webb NR, McCrary JA: Hyaline bodies of the optic disc and migrane, in Smith JL (ed): *Neuro-Ophthalmology Update.* New York, Masson, 1977, pp 155-162.
6. Glaser JS, pp 21-25.
7. Walsh TJ, p 3.
8. Glaser JS, p 27.
9. Harrington D: *The Visual Fields,* ed 4. St. Louis, CV Mosby, 1976, p 73.
10. Reed H, Drance SM: *Essentials of Perimetry: Static and Kinetic,* ed 2. London, Oxford University Press, 1972, p 8.
11. Glaser JS, pp 3-4.
12. Reed H, Drance SM, p 17.
13. Glaser JS, p 12.
14. Harrington D, p 354.