

VISUAL FIELD DEFECT

Authored by
mohammad looti

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VISUAL FIELD DEFECT (VFD)

Primary Disciplinary Field(s): Neuro-Ophthalmology, Neurology, Ophthalmology, Neuropsychology

1. Core Definition

A **Visual Field Defect** (VFD) is defined as a measurable reduction or loss in the typical extension of the visual field, leading to areas of partial or complete blindness within the boundaries of peripheral vision. This impairment signifies a disruption in the processing of visual information at some point along the complex visual pathway, which stretches from the sensory receptors of the retina to the occipital lobe of the brain.

The visual field itself represents the entire spatial area that an individual can see when the eyes are fixed in a specific forward direction. A defect, therefore, manifests as a 'hole' or area of diminished sensitivity within this field, often referred to as a **scotoma**. VFDs are crucial clinical indicators because they reflect specific localized damage to the neural structures responsible for sight. Unlike simple refractive errors, which diminish visual acuity uniformly, VFDs are topographical, meaning the location and shape of the defect directly correlate with the anatomical site of the underlying lesion.

The severity of VFDs ranges widely, encompassing minor, isolated patches of reduced sensitivity (relative scotomas) to massive, complete loss of vision across half of the visual space (absolute hemianopia). Understanding the precise nature of the visual field loss--whether it respects the vertical or horizontal midline, affects one eye or both, and whether it is congruent--is paramount for the diagnosis, as it allows clinicians to pinpoint the lesion with high accuracy. The presence of VFDs often necessitates immediate investigation because they are commonly associated with serious neurological conditions, including stroke, tumors, and multiple sclerosis, requiring rapid medical intervention.

2. Neuroanatomical Basis of Defects

The highly organized structure of the visual pathway dictates the manifestation of VFDs. The journey of visual information begins at the retina, where light is converted into neural impulses. These impulses travel via the optic nerve to the optic chiasm, a critical juncture where fibers from the nasal (inner) half of each retina cross over (decussate) to the opposite side of the brain. Fibers from the temporal (outer) half remain uncrossed. This anatomical arrangement ensures that the left hemisphere processes the entire right visual field, and the right hemisphere processes the entire left visual field.

Damage occurring **prior to the optic chiasm**, specifically to the optic nerve of one eye, results in

unilateral defects that only affect that eye, potentially leading to monocular blindness or central scotomas. Damage **at the optic chiasm** typically affects the crossing fibers, which carry information from the temporal visual fields of both eyes, resulting in the classic bitemporal hemianopia, where peripheral vision is lost on both sides. Lesions here are often caused by pituitary tumors or aneurysms pressing on the chiasm.

Lesions occurring **posterior to the optic chiasm**--in the optic tracts, the lateral geniculate nucleus, the optic radiations, or the visual cortex (Area V1)--result in **homonymous field defects**. Homonymous means the defect affects the same side of the visual field in both eyes (e.g., loss of the entire left field in both the left and right eyes). The further posterior the lesion is located along the pathway, the more congruent (identical in shape and size) the resulting homonymous defect tends to be, reflecting the tight bundling and reorganization of visual fibers as they approach the occipital lobe.

3. Classification by Lesion Location

VFDs are clinically classified based on the anatomical location of the lesion, which produces distinct and recognizable patterns of visual loss. This classification system is fundamental to neuro-ophthalmological diagnosis.

Pre-chiasmal Defects: These involve the retina or the optic nerve (Cranial Nerve II). Damage here results in defects confined to the ipsilateral eye. Common examples include central scotomas (often seen in optic neuritis or macular disease) and altitudinal defects (typically caused by occlusions of the central retinal artery or vein). Since the optic nerve fibers have not yet crossed, the unaffected eye retains its full visual field.

Chiasmal Defects: These occur at the junction where nasal retinal fibers cross. The most pathognomonic finding is **bitemporal hemianopia**, where vision is lost in the temporal halves of both visual fields. This pattern arises because the lesion (usually an expanding mass like a pituitary adenoma) compresses the central, crossing fibers. Less commonly, compression from the sides can cause binasal defects.

Post-chiasmal Defects: These defects arise from lesions in the optic tract, lateral geniculate nucleus, optic radiations, or the primary visual cortex. They always result in **homonymous defects**, meaning the same half of the visual field is affected in both eyes. Examples include homonymous hemianopia (loss of one entire side), and homonymous quadrantanopia (loss of one quarter of the field). The specific pattern within this category--such as superior vs. inferior quadrantanopia--helps localize damage to specific parts of the optic radiations (Meyer's loop for superior defects, or parietal radiation for inferior defects).

The concept of **macular sparing** is a crucial detail in post-chiasmal defects. This phenomenon, where the central 5-10 degrees of vision (corresponding to macular processing) is preserved even

with a full homonymous hemianopia, strongly suggests a lesion affecting the visual cortex (occipital lobe). It is hypothesized that macular sparing occurs because the macular region is often supplied by dual blood supply (from both the middle cerebral artery and the posterior cerebral artery), or because the representation of the macula is extensive and often unilaterally localized in the posterior tip of the occipital cortex, allowing partial function to remain intact even after vascular insult.

4. Etiology and Underlying Pathophysiology

The causes of VFDs are diverse, ranging from acute vascular events to chronic degenerative diseases, all culminating in damage to the visual neuronal tissue. The most common cause of acute VFDs, particularly homonymous hemianopias, is **cerebral vascular accident** (stroke), which involves either ischemic blockage or hemorrhagic rupture of blood vessels supplying the posterior visual pathway. Ischemic strokes affecting the posterior cerebral artery (PCA) are frequent culprits, as the PCA supplies the primary visual cortex.

Secondly, **mass lesions**, such as tumors, abscesses, or aneurysms, are significant causes, particularly for chiasmal and pre-chiasmal defects. Tumors like pituitary adenomas or craniopharyngiomas grow and compress the optic chiasm, leading to the characteristic bitemporal field loss. Expanding tumors along the optic tract or within the temporal or parietal lobes can compress the optic radiations, leading to homonymous defects that are often less congruent initially but progress over time.

Other causes include **trauma**, which can directly sever optic nerve fibers or cause cerebral contusions that impact the visual cortex, and **inflammatory/demyelinating diseases** such as Multiple Sclerosis (MS). Optic neuritis, a common manifestation of MS, causes inflammation and demyelination of the optic nerve, typically leading to a temporary, painful, unilateral VFD, usually a central scotoma. Finally, certain toxins, nutritional deficiencies, and severe glaucoma can also cause progressive visual field loss, though these typically present as chronic, slowly evolving defects.

5. Diagnostic Methods: Perimetry

The definitive diagnosis and mapping of a VFD rely on **perimetry**, the systematic measurement of the visual field. Perimetry determines the boundaries of peripheral vision and identifies areas of depressed sensitivity (scotomas). Diagnostic methods range from simple bedside checks to highly sophisticated automated testing.

Confrontation Field Testing: This is a quick, initial screening method performed at the bedside. The examiner compares the patient's peripheral vision to their own, testing the four quadrants of the visual field by introducing a moving target (e.g., the examiner's fingers). While crude, it is

effective in detecting large, dense defects like hemianopias.

Automated Static Perimetry: The most common clinical test is the use of automated devices (e.g., Humphrey Field Analyzer). These devices present stationary lights of varying intensity across a standardized bowl. The patient presses a button when a light is detected. The computer generates a topographical map of visual sensitivity, providing quantitative data that is essential for monitoring progression and confirming the specific anatomical location of the lesion.

Goldmann Kinetic Perimetry: This older but still valuable method uses a moving light spot of known size and intensity. The boundaries of the field are mapped manually as the patient reports when the target is first seen entering their visual field. This technique is often preferred for mapping steeply sloped field defects or for patients who have difficulty cooperating with automated tests.

Accurate perimetry is indispensable because the resulting map of the defect guides subsequent neuroimaging studies (MRI or CT) by providing a precise anatomical hypothesis regarding the location and extent of the underlying pathology. Furthermore, consistent monitoring of the VFD over time helps track the efficacy of treatment for conditions like glaucoma or mass lesions.

6. Significance and Management Strategies

The significance of a VFD extends beyond simple visual impairment; it acts as a silent indicator of underlying neurological distress. Early detection can lead to life-saving interventions, such as the surgical removal of a growing brain tumor or the rapid management of an acute stroke to limit neuronal damage.

Management of VFDs is two-pronged: treatment of the underlying cause and rehabilitation of the visual deficit. Treating the cause may involve medication (e.g., steroids for optic neuritis), surgery (e.g., tumor resection), or vascular interventions (e.g., stroke management). If the damage is permanent, the focus shifts to rehabilitation, primarily directed at maximizing the patient's residual vision and enhancing navigation safety.

Rehabilitation strategies for patients with persistent homonymous hemianopia often include **Visual Restoration Therapy (VRT)**, which attempts to stimulate the border zone between seeing and non-seeing areas, and **compensatory training**. Compensatory training teaches patients strategic eye and head movements (scanning techniques) to efficiently search the blind field and increase awareness of objects in the neglected space. Additionally, optical aids, such as Peli Prisms, can be incorporated into eyeglasses. These specialized prisms shift images from the blind field into the seeing field, providing a wider field of view and crucial assistance with mobility and obstacle avoidance.

7. Further Reading

[Visual field defect - Wikipedia](#)

[Optic Chiasm - Wikipedia](#)

[Optic Radiation - Wikipedia](#)

[What is a Visual Field Defect? - American Academy of Ophthalmology](#)

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