

PARACENTRAL SCOTOMA

Authored by
mohammad looti

October 31, 2025

RECOMMENDED CITATION

mohammad looti (2025). *PARACENTRAL SCOTOMA*. PSYCHOLOGICAL SCALES.
Retrieved from <https://scales.arabpsychology.com/?p=63487>

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Primary Disciplinary Field(s): Ophthalmology; Neuroscience; Clinical Psychology

1. Core Definition and Visual Anatomy

The **paracentral scotoma** is defined as a specific type of visual field deficit characterized by a **small area of blindness** located within the central visual field. This blind spot is unique because it encompasses the visual area immediately surrounding, but specifically **not consisting of, the fovea** itself. The fovea is the anatomical center of the retina responsible for the highest acuity vision, necessary for precise tasks such as reading and detailed object identification. Consequently, the paracentral region, though not the absolute center, is vital for the initiation and maintenance of sharp central vision, making deficits in this area profoundly disabling.

The location of the paracentral scotoma typically falls within the 10 to 20 degrees eccentricity from the central fixation point. Anatomically, this region corresponds to the nerve fiber bundles that arc around the fovea as they proceed toward the optic disc. Damage to these specific fibers, often stemming from conditions affecting the intraocular pressure, circulation, or inflammation of the optic nerve head, results in the characteristic curved or wedge-shaped scotoma that defines this condition. The depth of the scotoma--whether it is absolute (no light perception) or relative (diminished vision)--provides crucial diagnostic information regarding the severity and chronicity of the underlying pathology.

Understanding the retinotopic map is essential for diagnosing the **paracentral scotoma**. The central 30 degrees of the visual field receives massive representation in the primary visual cortex, known as cortical magnification. Because the paracentral area is part of this magnified representation, localized lesions can cause disproportionately significant visual impairment. The presence of a scotoma always indicates a pathological interruption of the visual pathway, confirming the observation that the **paracentral scotoma is not a normal occurrence** but a sign of disease affecting the delicate neural transmission process.

2. Neurophysiological Basis of Scotomas

A scotoma, fundamentally, is an island of absent or severely impaired vision surrounded by a field of relatively normal vision. The neurophysiological basis relies on the disruption of the neural circuitry responsible for visual perception, which extends from the photoreceptors in the retina to the calcarine sulcus of the occipital cortex. The specificity of the **paracentral scotoma** means that the damage is highly localized, affecting only the neurons or nerve fibers responsible for processing that particular region of the visual field.

The mechanism of vision loss can involve direct neuronal death, as seen in ischemia (lack of blood

flow) or trauma, or temporary functional impairment due to inflammation, compression, or metabolic disturbance. In the retina, the highly organized distribution of nerve fibers makes the paracentral region susceptible to conditions that preferentially affect the inner retinal layers or the specific vascular supply to the optic nerve head. For instance, in optic neuropathies, swelling or demyelination of the optic nerve fibers can selectively impair the bundles serving the paracentral field, leading to a temporary or permanent blind spot.

The clinical distinction between the two major classifications of **paracentral scotoma**--monocular and homonymous--hinges entirely on the location of the lesion relative to the optic chiasm. Lesions occurring before the chiasm (pre-chiasmal) affect the visual input from only one eye, resulting in a monocular deficit. Lesions occurring after the chiasm (post-chiasmal), where visual input from both eyes is merged and segregated based on the side of the visual field, result in a homonymous deficit, affecting the corresponding left or right visual field in both eyes simultaneously.

3. Classification of Paracentral Scotoma: Monocular Type

The **monocular type** of paracentral scotoma is characterized by a visual field defect that is restricted entirely to one eye. As confirmed by clinical findings, this deficit **stems from disabling of the peripheral visual system**, which refers to the visual structures anterior to the point where the nasal fibers cross--namely, the retina and the optic nerve. Because the damage is unilateral and pre-chiasmal, the visual field of the unaffected eye remains completely normal, allowing the patient to compensate easily by using the good eye for central tasks.

The most frequent causes of the monocular paracentral scotoma include serious ocular diseases such as glaucoma and optic neuritis. In **glaucoma**, elevated intraocular pressure damages the optic nerve head, leading to progressive atrophy of the nerve fibers. The resulting scotomas often begin in the paracentral region as arcuate defects (Bjerrum scotomas), reflecting the pattern of fiber loss. In **optic neuritis**, often associated with demyelinating diseases like multiple sclerosis, the inflammation causes temporary or permanent dysfunction of the optic nerve, frequently resulting in a central or paracentral blind spot accompanied by pain and reduction in color vision.

Other etiologies include vascular occlusions, such as retinal vein or artery occlusions that affect the macula and surrounding area, or toxic/nutritional optic neuropathies where the damage to the papillomacular bundle (a dense bundle of fibers responsible for central vision) can extend into the paracentral area. Diagnosis of the monocular type requires meticulous examination, including advanced techniques like optical coherence tomography (OCT) to assess nerve fiber layer thickness and perimetry to accurately map the boundaries and density of the blind spot, distinguishing it from other types of retinal or optic nerve pathology.

4. Classification of Paracentral Scotoma: Homonymous Type

The **homonymous type** of paracentral scotoma presents as a visual field loss that affects the same side of the visual field in both eyes, meaning the patient loses vision in either the left half or the right half of the paracentral field. This phenomenon occurs because the damage is localized to the central visual processing system, specifically **due to damage to the central visual system behind the lateral geniculate nucleus (LGN)**. The LGN is the thalamic relay station, and all visual information posterior to it—including the optic radiations and the primary visual cortex (V1) in the occipital lobe—is organized homonymously.

A typical cause of the homonymous paracentral scotoma is a localized cerebrovascular accident (stroke) or a small, non-compressive lesion in the occipital cortex. Because the most central part of the visual field (the foveal representation) often has a wider blood supply and a larger cortical representation, it may be spared (known as macular sparing), leaving a visual field defect that borders the fixation point but does not engulf it, perfectly matching the definition of a paracentral scotoma. Such lesions are often subtle and require high-resolution neuroimaging to identify, particularly when the patient presents only with minor visual complaints.

The functional consequences of the homonymous paracentral scotoma are severe, especially concerning detailed visual tasks. As noted in the clinical literature, patients with the homonymous type **often have trouble reading**. Reading involves tracking letters sequentially, and the visual system relies on the paracentral field to pre-process the upcoming words, guiding the rapid, ballistic eye movements known as saccades. When the paracentral field is blind, the ability to rapidly acquire the target word and plan the next eye movement is impaired, leading to frequent misalignments, loss of place on the page, and reduced comprehension speed, necessitating specialized visual rehabilitation strategies.

5. Clinical Presentation and Functional Impact

The clinical experience of a patient with a **paracentral scotoma** can vary widely depending on its size, density, and speed of onset. An acute scotoma, such as one caused by an ischemic event, presents dramatically as a sudden loss of part of the central viewing area. Conversely, a chronic scotoma, such as those associated with slowly progressing glaucoma, may go unnoticed for years until the patient performs a task that demands high central visual integrity, like reading or focusing on a distant sign, making regular screening a critical component of preventative medicine.

Patients often describe the defect not as a black hole, but rather as a region of visual blur, distortion, or an area where objects simply vanish when they attempt to look directly at them or immediately adjacent to them. This phenomenon is related to cortical completion, where the brain attempts to fill in the missing visual information based on surrounding visual context, which can lead to visual illusions or profound difficulty distinguishing details within the scotoma area. If the

scotoma is unilateral (monocular type), the patient may not notice it at all when both eyes are open, relying on the healthy eye to compensate for the central loss.

The functional impact of this defect is pervasive due to the vital role the paracentral field plays in spatial orientation and visual motor integration. Beyond the documented difficulty with reading, patients experience challenges with tasks requiring accurate hand-eye coordination, driving (particularly peripheral warning signs or instruments near the center of gaze), and recognizing fine details in complex scenes. Effective management requires not only addressing the underlying pathology but also providing comprehensive occupational therapy to help the patient adapt to the permanently altered visual landscape.

6. Differential Diagnosis and Etiology

The diagnostic workup for a patient presenting with a **paracentral scotoma** is focused on differentiating the location and nature of the pathology. Key conditions that must be ruled out or confirmed include diseases of the optic nerve (optic neuropathies), diseases of the retina (maculopathies), and lesions in the posterior visual pathways (cortical or tract lesions). Testing typically involves detailed visual field mapping (perimetry), fundus examination to view the retina and optic nerve head, and neuroimaging (MRI or CT) if a neurological cause is suspected.

From an ocular standpoint, the most common differential diagnoses are **glaucoma**, which causes characteristic arcuate defects, and age-related macular degeneration (ARMD), which often begins as drusen accumulation in the paracentral macula leading to subtle scotomas. Other retinal conditions, such as central serous retinopathy or various inherited dystrophies, can also target the paracentral area. The monocular presentation strongly suggests an ocular or optic nerve etiology, directing the initial investigation toward the eye itself.

When the visual field defect is homonymous, the focus shifts immediately to the central nervous system. The primary etiologies here are vascular events (strokes), which often produce sudden, dense defects, and tumors or demyelinating diseases that cause more gradual, sometimes fluctuating, visual loss. The specific location behind the **lateral geniculate nucleus** must be precisely identified, as management and prognosis differ significantly between lesions affecting the deep optic radiations and those affecting the surface of the visual cortex.

7. Prognosis and Management

The long-term prognosis for a patient diagnosed with a **paracentral scotoma** is dictated by whether the underlying cause is progressive, acute and reversible, or acute and permanent. If the scotoma is secondary to a treatable condition, such as inflammation in optic neuritis or elevated pressure in glaucoma, aggressive medical or surgical intervention aimed at the primary disease can often stabilize or even partially reverse the visual field loss, particularly if treatment is initiated

promptly.

However, many scotomas, especially those resulting from vascular strokes or advanced neurodegeneration, represent irreversible loss of neural tissue. In these cases, the focus of management shifts entirely to rehabilitation and adaptation. This includes low vision therapy, where the patient is trained in specific compensatory strategies to navigate the blind spot. For the homonymous type, training often involves large, systematic visual scanning movements toward the side of the field loss to detect potential hazards or locate text cues.

Technological aids are also critical elements of management. Devices ranging from specialized reading telescopes to digital magnification tools and sophisticated visual displacement prisms can help redirect images from the blind paracentral region onto the preserved foveal or surrounding visual area. Ultimately, while the structural damage leading to the scotoma may be permanent, rehabilitation aims to restore functional independence and mitigate the significant impairment caused by the loss of vision in this critical region adjacent to the fixation point.

Further Reading

[Scotoma - Wikipedia](#)

[Lateral Geniculate Nucleus \(LGN\) - Wikipedia](#)

[The Human Visual System - Overview](#)