

PARACENTRAL SCOTOMA

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The Nature and Definition of Paracentral Scotoma

A paracentral scotoma is defined precisely as a small, circumscribed area of functional blindness or diminished sensitivity situated within the central visual field. Crucially, this defect is located immediately adjacent to the point of fixation, which corresponds to the **fovea centralis**. While the scotoma is positioned in the highly sensitive paracentral region and therefore greatly impacts central vision, it is technically distinct from a classic macular scotoma, though their clinical effects often overlap. The distinguishing feature is its location: it encompasses the area surrounding the fovea but does not necessarily obliterate the foveal fixation point itself, although the proximity ensures severe functional impairment. This specific location disrupts the intricate processing necessary for high-resolution tasks such as facial recognition and reading, which rely heavily on the integrity of the immediate central visual span.

The term **scotoma** itself originates from the Greek word for darkness, signifying an island of visual loss within a generally preserved field of vision. Paracentral scotomas are significant because the paracentral area, while slightly less acute than the fovea, still possesses an extremely high density of photoreceptors and retinal ganglion cells, making it critical for detailed visual tasks. The disruption of this small area results in a disproportionately large functional deficit. Understanding the precise boundaries and density of the visual loss--whether it is an absolute scotoma (complete absence of vision) or a relative scotoma (diminished sensitivity)--is vital for accurate diagnosis and determining the underlying pathophysiology. Defects in this region often indicate highly localized damage, either in the peripheral sensory apparatus or the central processing pathways.

The clinical presentation of a paracentral scotoma is often subtle initially, but its central location quickly alerts patients to its presence, particularly during activities requiring precise visual targeting. Unlike peripheral field loss, which may go unnoticed until advanced stages, the paracentral defect interferes immediately with the ability to maintain continuous visual fixation and gather detailed visual information. From an anatomical perspective, the central 10 degrees of the visual field are represented by a vast portion of the primary visual cortex (V1) due to the concept of **cortical magnification**. Consequently, even a small lesion affecting the central pathways that project to this highly represented cortical area can manifest as a clinically significant paracentral scotoma, underscoring the delicate balance required for maintaining central visual function.

Neuroanatomical Foundation of Central Visual Field Integrity

Maintaining the integrity of the paracentral visual field relies upon an unbroken chain of neural transmission, beginning at the specialized layers of the retina and extending deep into the occipital lobe. The paracentral retina, immediately surrounding the fovea, is uniquely dedicated to fine detail and color discrimination. Signals generated here are carried by specialized bundles of nerve fibers, collectively known as the papillomacular bundle, which travel directly to the **optic nerve head**. Any

compromise to these specific fibers, whether through compression, inflammation, or vascular insufficiency, results in a corresponding paracentral defect in the visual field. This initial peripheral segment is crucial for the monocular type of scotoma.

The visual pathway continues posteriorly through the optic chiasm, where fibers from the nasal retina cross, and then proceeds through the optic tracts to the **Lateral Geniculate Nucleus (LGN)** of the thalamus. The LGN acts as a critical relay station, organizing and projecting visual information via the optic radiations to the primary visual cortex (V1) located in the occipital lobe. It is significant to note that the central visual field, including the paracentral area, has a massively disproportionate representation within the V1 cortex, specifically concentrated at the posterior tip of the occipital lobe. This highly localized cortical representation means that a relatively small, specific lesion in the posterior visual pathways can selectively produce a paracentral scotoma while sparing other visual fields.

When damage occurs to the visual system posterior to the LGN--affecting the optic radiations or the visual cortex itself--the resulting visual defect is typically **homonymous**, meaning it affects the same side of the visual field in both eyes. The fibers responsible for carrying paracentral information are anatomically vulnerable, especially within the optic radiations (Meyer's loop or the superior fibers) as they fan out toward the cortex. Furthermore, the vascular supply to the occipital pole, often derived from the terminal branches of the posterior cerebral artery (PCA), is susceptible to ischemic events. Occlusion of a small PCA branch specifically targeting the macular representation in the cortex can lead directly to a homonymous paracentral scotoma, often resulting in a highly debilitating visual impairment.

Classification and Differential Etiology

Paracentral scotomas are primarily classified based on the anatomical location of the lesion, which dictates whether the defect is monocular (affecting one eye) or homonymous (affecting corresponding parts of the visual field in both eyes). This distinction is fundamental for differential diagnosis. The **monocular type** arises from pathology anterior to the optic chiasm, involving the retina or the optic nerve itself. Common etiologies include ocular diseases such as chronic open-angle glaucoma, where nerve fiber layer damage begins subtly in the paracentral regions, or various forms of optic neuropathy, including those related to inflammation (optic neuritis) or ischemia (anterior ischemic optic neuropathy).

Conversely, the **homonymous type** of paracentral scotoma is invariably linked to damage within the central visual system, specifically posterior to the crossing point at the optic chiasm. This involves the optic tract, the LGN, the optic radiations, or the visual cortex. The primary causes here are vascular events, such as strokes (ischemic or hemorrhagic) affecting the posterior circulation, or compressive lesions like tumors, abscesses, or arteriovenous malformations located in the

temporal or occipital lobes. Since the central visual field is represented bilaterally after the chiasm, damage to the left optic tract or visual cortex results in loss of the right paracentral field in both eyes, and vice versa. Identifying the homonymous nature of the defect is a strong indication that the pathology is neurological, not ophthalmological.

The differential diagnosis must carefully consider the clinical context. For example, a patient presenting with an acutely developing, painful monocular paracentral scotoma might suggest demyelinating disease (multiple sclerosis), leading to optic neuritis. In contrast, a patient with sudden, painless onset of a homonymous paracentral scotoma, especially if associated with other neurological signs like hemiparesis or aphasia, strongly suggests a cerebrovascular accident affecting the occipital lobe. The shape and density of the scotoma are also highly informative; a paracentral scotoma associated with glaucoma, for instance, often assumes an arcuate or arc-shaped pattern reflecting the trajectory of the damaged retinal nerve fibers, whereas a homonymous scotoma from a cortical lesion may respect the vertical midline precisely.

Monocular Paracentral Scotoma: Peripheral System Dysfunction

The occurrence of a monocular paracentral scotoma points directly toward a disabling process affecting the **peripheral visual system**--that is, the eye itself, the retina, or the optic nerve prior to its junction with the chiasm. One of the most common and clinically relevant causes is primary open-angle glaucoma, a progressive optic neuropathy characterized by the death of retinal ganglion cells, typically due to elevated intraocular pressure. Glaucomatous damage often manifests initially as subtle paracentral defects, such as Seidel's scotoma or Bjerrum's scotoma, which are highly localized arcuate defects originating from the blind spot and arching around the point of fixation, impinging upon the paracentral field.

Beyond glaucoma, various forms of **optic neuropathy** can selectively impair the central nerve fibers, leading to a monocular paracentral or centrocecal scotoma. Ischemic optic neuropathies, particularly anterior ischemic optic neuropathy (AION), result from insufficient blood flow to the optic nerve head, causing localized infarction of the nerve tissue. While AION often leads to altitudinal defects (loss in the upper or lower half), involvement of the papillomacular bundle can specifically create a scotoma adjacent to the fovea. Similarly, inflammatory conditions like optic neuritis, characteristic of demyelinating diseases, cause inflammation and destruction of the myelin sheath surrounding the nerve fibers, leading to acute visual loss that frequently involves the central or paracentral field.

Other peripheral causes include retinal vascular diseases, such as retinal artery or vein occlusions, which, depending on the vessel involved, can cause localized retinal ischemia or edema affecting the paracentular region. Conditions like central serous retinopathy, where fluid accumulates beneath the retina, can also distort the paracentral photoreceptor layer, leading to a relative

scotoma accompanied by micropsia (objects appearing smaller) or metamorphopsia (distortion of vision). The monocular nature of the defect is the key identifier, necessitating a thorough ophthalmological examination, including fundoscopy to assess the optic nerve head and retina, to pinpoint the precise peripheral etiology.

Homonymous Paracentral Scotoma: Damage to the Central Visual Pathways

The presence of a homonymous paracentral scotoma signals damage to the **central visual system** located behind the lateral geniculate nucleus (LGN), primarily involving the optic radiations or the visual cortex (V1) in the occipital lobe. This pattern of visual field loss, where corresponding portions of the visual field are lost in both eyes, results from the highly organized retinotopic map maintained throughout the posterior pathways. If a lesion affects the right visual cortex, for example, the patient loses vision in the corresponding left paracentral field of both eyes, severely impacting the ability to perceive stimuli entering that specific spatial location.

The most common cause of homonymous scotomas is **cerebrovascular accident (stroke)**, particularly those affecting the posterior circulation supplied by the posterior cerebral artery (PCA). The terminal branches of the PCA supply the occipital pole, which contains the dedicated cortical representation for the macula and, crucially, the surrounding paracentral region. A highly selective, small infarction in this area can lead to an isolated homonymous paracentral scotoma, often sparing the very center of the foveal representation due to dual blood supply (known as macular sparing), though involvement of the paracentral area remains debilitating.

Furthermore, lesions within the **optic radiations**--the extensive pathway connecting the LGN to the visual cortex--can also produce this specific defect. While larger radiation lesions typically result in complete quadrantanopias or hemianopias, partial damage, especially near the temporal lobe (Meyer's loop) or the parietal lobe, might manifest as an incomplete or sectorial homonymous defect that impinges upon the central field. The precise localization of the lesion using advanced neuroimaging techniques, such as MRI, is paramount for distinguishing between a cortical lesion, which tends to respect the vertical meridian precisely, and a radiation lesion, which might present with less defined borders.

Clinical Presentation and Diagnostic Procedures

Patients presenting with a paracentral scotoma often report significant subjective visual disturbances that are disproportionate to the small size of the defect. Symptoms typically include difficulty maintaining smooth visual fixation, the sensation of a shimmering or blank area near the center of vision, and a pronounced struggle with tasks requiring precise central alignment. For patients with homonymous defects, this blind spot moves with the eyes, meaning the patient cannot look around the defect, as it follows their gaze. This phenomenon is particularly disabling

because it interferes with the primary area used for gathering visual information.

The cornerstone of diagnosis is **visual field testing**, specifically high-resolution perimetry. While basic screening tests like the Amsler grid can alert the clinician to central or paracentral defects--patients will often report missing or distorted lines near the center--formal automated static perimetry is required to map the exact size, shape, and depth of the scotoma. Standard perimetry (e.g., Humphrey Visual Field Analyzer 24-2 or 30-2 protocols) may be sufficient, but specialized central 10-2 testing is often necessary to provide the granular detail required for accurately mapping a paracentral defect, ensuring proper monitoring of progression, especially in conditions like glaucoma.

Once a paracentral scotoma is confirmed by perimetry, subsequent diagnostic steps diverge based on whether the defect is monocular or homonymous. For monocular defects, ophthalmological evaluation including **Optical Coherence Tomography (OCT)** is critical for assessing the thickness of the retinal nerve fiber layer (RNFL) and the ganglion cell complex (GCC), allowing for early detection of glaucomatous or neurodegenerative thinning. If the scotoma is homonymous, immediate **neuroimaging (MRI or CT scan)** is mandatory to localize the lesion in the central nervous system, confirming whether the damage resides in the optic tracts, LGN, optic radiations, or the occipital cortex, guiding neurological management and prognosis.

Functional Impact: Reading Disability

One of the most profound and frequently reported functional consequences of a paracentral scotoma, particularly the **homonymous type**, is severe difficulty with reading, often termed acquired dyslexia or alexia depending on the associated pathology. Reading is a highly complex visual task that relies not only on clear central fixation but also on the successful processing of information in the immediate parafoveal and paracentral areas. Normal reading involves rapid, sequential eye movements (saccades) followed by brief fixation periods. During fixation, the brain processes the currently fixated word and simultaneously gathers preparatory information from the words immediately following (the visual span) within the paracentral field.

A paracentral scotoma critically impairs this preparatory stage. If the scotoma falls into the area immediately to the right of fixation (for English readers, where reading proceeds left-to-right), the patient cannot efficiently gather information about the next word or group of letters. This prevents the normal guidance of the subsequent saccade, forcing the patient to rely on inefficient, short, and often regressive eye movements. The disruption of the **parafoveal preview** mechanism leads to significantly slowed reading speed, increased errors, and frequent loss of place on the page, transforming a seamless process into a laborious and frustrating task.

In cases where the scotoma is monocular, the reading difficulty may be less severe, as the non-affected eye can compensate, although binocular summation is impaired. However, the

homonymous paracentral scotoma is uniquely disabling because the defect is present in the corresponding field of both eyes, meaning there is no way to move the head or eyes to circumvent the blind spot without moving the critical fixation area itself into the defective zone. Therefore, rehabilitation efforts specifically target strategies to shift the preferred retinal locus for fixation (eccentric viewing) and utilize visual scanning training to maximize the use of the remaining functional visual field, allowing the patient to re-establish a functional reading rhythm.

Management, Prognosis, and Rehabilitation Strategies

The management of a paracentral scotoma is fundamentally dependent upon addressing the underlying etiology. For monocular scotomas caused by glaucoma, management centers on rigorous intraocular pressure control through medications, laser treatment, or surgery to halt the progression of nerve fiber damage and prevent the enlargement of the scotoma. In cases of inflammatory optic neuropathy, high-dose corticosteroids may be administered to reduce inflammation and promote recovery of nerve function. If the cause is vascular (e.g., AION), controlling systemic risk factors such as hypertension, diabetes, and hyperlipidemia is paramount to prevent recurrence in the affected or fellow eye.

For homonymous paracentral scotomas resulting from central nervous system damage, particularly stroke, acute management focuses on minimizing neurological injury (e.g., thrombolysis or thrombectomy). Long-term prognosis for these central defects is variable; while some recovery of visual function may occur, particularly in the first six months following the injury, residual scotomas are often permanent. The stability of the visual field loss dictates the type of rehabilitation strategies employed, as stable defects allow for the successful implementation of compensatory training.

Visual rehabilitation strategies are crucial for improving the quality of life when the scotoma is permanent. These strategies often include the training of **eccentric viewing**, where patients learn to deliberately position their gaze slightly off-center to place the target object (like a word) onto a preserved, functional area of the retina adjacent to the scotoma. Furthermore, specialized optical aids, such as base-in prisms, can sometimes be used to shift the image slightly away from the blind spot. For reading, training programs focus on anchor placement and visual search strategies, helping the patient overcome the loss of parafoveal preview and regain reading independence, thereby mitigating the severe functional impact of the paracentral visual loss.