

QUADRANOPIA

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1. Core Definition

Quadrantopia is a specific type of visual field defect characterized by the irreversible loss of vision in one quadrant, corresponding to one-fourth (25%) of the total visual field. This deficit is typically homonymous, meaning the loss occurs in the same relative quadrant of the visual field for both eyes. The visual field, defined by the area seen when the eye is directed straight ahead, is conventionally divided into four quadrants: superior nasal, superior temporal, inferior nasal, and inferior temporal. Because the visual pathway decussates (crosses) at the optic chiasm and then again within the brain, damage occurring posterior to the chiasm--specifically within the optic radiations or the primary visual cortex (V1)--results in homonymous field loss, affecting the contralateral side of the visual world. A pure quadrantopia indicates a highly localized and restricted lesion that affects only the superior or inferior bundle of the optic radiations, contrasting sharply with hemianopia, which involves the loss of an entire half (50%) of the visual field due to more extensive pathway damage. The precise location of the lesion determines whether the superior or inferior quadrant is affected, following the principle of retinotopy, where specific regions of the retina and visual field map systematically onto corresponding parts of the visual pathway and cerebral cortex.

The definition mandates that the borders of the remaining visual field respect the vertical midline (the macula), which distinguishes homonymous defects caused by post-chiasmal lesions from pre-chiasmal defects like those involving the retina or optic nerve. For example, a patient suffering from a lesion in the right hemisphere's visual pathway will experience a loss of the left visual field quadrant. This defect is a clinical manifestation of the highly organized neuroanatomy of the visual system, where fibers carrying information from the superior retina (representing the inferior visual field) separate spatially from those carrying information from the inferior retina (representing the superior visual field) as they pass through the white matter of the brain toward the occipital lobe. Damage to these separated bundles of axons, collectively known as the optic radiations, causes the distinct pattern of quadrant loss. The clinical presentation of quadrantopia can vary widely, sometimes being immediately obvious to the patient, but often remaining subtle, particularly if the lost quadrant is peripheral or if the patient naturally compensates by turning their head or employing visual scanning techniques.

Understanding quadrantopia is crucial in neurological diagnostics because the pattern of visual loss serves as a highly reliable neuroanatomical signpost, enabling neurologists to localize the exact site of the underlying pathological process. The defect is classified based on both its location (superior or inferior) and its laterality (left or right homonymous). For instance, an **Inferior**

Homonymous Quadrantopia (I-HQ) signifies damage to the superior portions of the visual pathway, typically in the parietal lobe, while a **Superior Homonymous Quadrantopia** (S-HQ) points toward damage in the inferior portions of the pathway, often involving the temporal lobe. The identification of quadrantopia is achieved through formal perimetry testing, which plots the boundaries of the patient's remaining vision and definitively maps the scotoma (area of vision loss).

2. Anatomical Basis and Retinotopic Mapping

The neuroanatomical underpinnings of quadrantopia rely entirely on the retinotopic organization maintained throughout the visual pathway from the lateral geniculate nucleus (LGN) to the primary visual cortex (V1, or Brodmann area 17). The visual input originating from the superior visual field is processed by the inferior halves of the retinas and subsequently travels through the inferior optic radiations (Meyer's loop), which sweep anteriorly and inferiorly into the temporal lobe before turning posteriorly to reach the lower bank of the calcarine sulcus. Conversely, input from the inferior visual field is processed by the superior halves of the retinas and travels through the superior optic radiations, which take a more direct route through the parietal lobe, terminating on the upper bank of the calcarine sulcus. A localized lesion affecting one of these distinct bundles, but not the other, results in the specific quadrant deficit. This strict separation of fibers--superior versus inferior--is the defining feature that allows for a quadrant rather than a half-field loss.

Damage to the temporal lobe structures, specifically the white matter containing **Meyer's loop**, is the classic cause of Superior Homonymous Quadrantopia (S-HQ). Because Meyer's loop contains the fibers representing the superior visual field, a lesion here, often caused by a temporal lobe tumor or vascular insult, destroys the representation of the upper contralateral visual quarter. This anatomical vulnerability in the temporal lobe is exploited by mass lesions which, due to the large, sweeping arc of these fibers, can selectively compress or destroy the inferior pathway without affecting the superior pathway running through the parietal lobe. This precise localization offers clinicians a powerful diagnostic tool, linking S-HQ directly to temporal lobe pathology.

In contrast, lesions within the parietal lobe, which house the more dorsally located superior optic radiations, lead to **Inferior Homonymous Quadrantopia** (I-HQ). These superior fibers carry information corresponding to the inferior visual field. Damage in the parietal region, frequently associated with posterior middle cerebral artery (MCA) or anterior posterior cerebral artery (PCA) territory strokes, selectively ablates the representation of the lower contralateral visual quarter. Furthermore, the clinical significance extends beyond just vision, as damage to the parietal lobe often co-occurs with other neurological deficits, such as difficulties with spatial awareness, arithmetic (acalculia), or language comprehension (Wernicke's aphasia, if the dominant hemisphere is involved), thereby providing additional confirming evidence for the lesion location.

3. Key Types and Associated Lesions

Quadrantopia is categorized primarily by the location of the lost quadrant, which directly dictates the location of the neurological lesion. The distinction between superior and inferior loss, and the consistent homonymous nature, form the core of the classification system used clinically and academically. The most critical point of differentiation is recognizing that visual field loss is always contralateral to the cerebral lesion.

The key classifications of quadrantopia include:

Superior Homonymous Quadrantopia (S-HQ): This involves the loss of the upper quadrant of the visual field on the side opposite the cerebral lesion. For example, a right temporal lobe lesion (affecting the inferior optic radiations/Meyer's loop) results in a left superior quadrantopia. The temporal lobe is the critical site for S-HQ, often secondary to ischemic events or surgical intervention in this area. S-HQ is sometimes referred to by the mnemonic "pie in the sky," describing the loss of the upper wedge of vision.

Inferior Homonymous Quadrantopia (I-HQ): This involves the loss of the lower quadrant of the visual field contralateral to the lesion. A lesion located in the right parietal lobe (affecting the superior optic radiations) results in a left inferior quadrantopia. I-HQ is frequently associated with lesions higher up in the visual pathway, often resulting from vascular events impacting the white matter of the parietal lobe. Clinically, I-HQ can be particularly debilitating as it affects the ability to see the ground immediately ahead, impacting ambulation and leading to frequent trips or falls.

Macular Sparing: A phenomenon frequently observed in cases of homonymous quadrantopia, particularly those caused by lesions in the visual cortex. Macular sparing occurs when the extreme central region of the visual field remains intact, typically a circular area of 5 to 10 degrees. This sparing is attributed to the fact that the macula, responsible for high-acuity central vision, is represented over a disproportionately large area of the visual cortex and may receive collateral blood supply from both the posterior cerebral artery (PCA) and the middle cerebral artery (MCA), protecting it from isolated PCA occlusion which often causes peripheral field loss.

Incongruity: While perfect quadrantopia is typically homonymous (identical in shape and size in both eyes), some degree of incongruity (differences between the visual field defects of the two eyes) can occur, particularly with lesions located more anteriorly in the optic radiations. Highly congruent defects, meaning the field loss is nearly identical between the two eyes, generally localize the lesion closer to the occipital cortex.

4. Etiology and Pathogenesis

The causes of quadranopia are diverse but fundamentally involve pathologies that selectively damage the optic radiations or the area of the primary visual cortex responsible for representing only one quadrant of the visual field. The most common underlying cause across all forms of homonymous quadranopia is a cerebrovascular accident, or stroke. Ischemic strokes affecting the territory supplied by the posterior cerebral artery (PCA) are highly implicated, as the PCA is the primary supplier of the occipital cortex and the deeper structures containing the optic radiations. Depending on the size and location of the infarct, the damage can be confined enough to produce a clean quadranopia rather than a full hemianopia. For instance, a localized small vessel occlusion affecting the temporal horn white matter can precisely disrupt Meyer's loop, leading to S-HQ.

Beyond vascular causes, space-occupying lesions such as brain tumors constitute another major etiology. Both benign and malignant tumors--including gliomas, meningiomas, and metastatic lesions--can grow slowly, leading to the gradual compression and destruction of the optic radiation fibers passing through the temporal or parietal lobes. Because tumors often respect anatomical boundaries initially and exert focal pressure, they are frequently responsible for producing subtle, slow-onset quadranopia. The specific characteristics of the tumor's growth pattern (e.g., a tumor originating near the temporal pole compressing Meyer's loop from below) directly translate into the type of quadranopia observed.

Traumatic brain injury (TBI) can also result in quadranopia, typically due to shearing forces or localized contusions affecting the white matter tracts, although TBI is often associated with more diffuse visual pathway damage. Furthermore, surgical procedures, particularly those involving the resection of temporal lobe structures (e.g., for epilepsy treatment), carry a calculated risk of inducing iatrogenic S-HQ due to the necessity of navigating near Meyer's loop. Other less common causes include demyelinating diseases like multiple sclerosis, localized infections (abscesses), or arteriovenous malformations (AVMs) that bleed into or compress the visual pathways.

5. Clinical Significance and Impact

The clinical significance of quadranopia extends beyond mere visual loss; it profoundly impacts a patient's daily functional capacity, spatial awareness, and quality of life. The impact is highly dependent on which quadrant is lost. For example, a right homonymous quadranopia (loss of the right quarter of vision in both eyes) affects the visual field required for scanning text and tracking objects, making reading particularly arduous, as the patient continually loses the next word or must constantly shift their gaze to compensate for the blind spot. This difficulty in reading often leads to significant frustration and occupational challenges.

In cases of inferior quadranopia (I-HQ), where the lower visual field is lost, patients commonly report difficulties with ambulation and navigation. They are prone to stumbling over obstacles, misjudging steps, or bumping into low objects because the visual input detailing the immediate

environment at their feet is missing. Conversely, superior quadranopia (S-HQ), while still impactful, may be less debilitating for walking but can interfere significantly with tasks that require viewing objects overhead or tracking information presented above eye level, such as recognizing traffic lights or signage.

Furthermore, the presence of quadranopia often indicates a significant underlying neurological event that necessitates immediate and comprehensive investigation. The diagnosis of quadranopia is not an endpoint but rather a critical step in the diagnostic process, immediately triggering neuroimaging (MRI or CT) to identify the specific nature of the lesion (e.g., acute stroke, chronic tumor, or hemorrhage). The prognosis and required management strategy are inextricably linked to the underlying cause; early identification of quadranopia can thus be lifesaving if the cause is an aggressively treatable pathology like an acute stroke or a rapidly growing tumor.

6. Treatment and Rehabilitation Strategies

The treatment of quadranopia is two-pronged: addressing the acute underlying cause and providing long-term rehabilitation for the resulting visual deficit. Acute management focuses on the etiology--for an ischemic stroke, this involves clot lysis or removal; for a tumor, it involves surgical resection, chemotherapy, or radiation; and for trauma, it involves managing swelling and pressure. Once the primary pathology is stabilized, the quadranopia itself is often permanent, necessitating strategies to help the patient adapt to the lifelong visual field loss.

Rehabilitation strategies focus on optimizing the remaining vision and teaching compensatory techniques. One common approach involves **Visual Restoration Therapy (VRT)**, which attempts to stimulate the border zone between the intact and damaged visual fields, though its effectiveness remains a subject of ongoing debate in the ophthalmological community. More practically, occupational therapists and vision specialists employ **Compensatory Training**, which teaches systematic saccadic eye movements. Patients learn to habitually scan into the blind quadrant, effectively bringing the critical information into the remaining intact visual field. For instance, a patient with a left homonymous quadranopia is trained to make broad, deliberate sweeps of the eyes to the left.

Optical aids, such as specialized spectacle-mounted prisms, are sometimes employed to shift images from the blind quadrant into the seeing field. These devices can be particularly helpful for peripheral awareness during ambulation, although they often introduce distortions and are not universally tolerated for central tasks like reading. Ultimately, successful management requires a multidisciplinary approach involving neurologists, ophthalmologists, neuro-rehabilitation specialists, and occupational therapists to maximize the patient's independence and minimize the disruption caused by the persistent visual field defect.

7. Further Reading

[Optic Chiasm \(Wikipedia\)](#)

[Meyer's Loop \(Wikipedia\)](#)

[Brodmann Area 17 \(Wikipedia\)](#)

[Stroke \(Wikipedia\)](#)

[Perimetry \(Wikipedia\)](#)

[Calcarine Sulcus \(Wikipedia\)](#)

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