DEFINITION: A field defect where visual field shows loss of all or part of the superior half or the inferior half of the visual field of one or both eyes, and which respects the horizontal meridian. In case the field defect crosses the horizontal meridian than the field defect could no longer be called an altitudinal field defect. This is in contrast to the field defects which respects the vertical meridian due to neurological lesions of the visual pathways. Altitudinal field defect may be unilateral or bilateral. The main causes are as follows:

RETI NAL CAUSES
1. Branch Retinal Artery Occlusion
2. Branch Retinal Vein Occlusion
3. Retinal Coloboma

OPTIC NERVE LESION
1. Ischaemic optic neuropathy (both arteritic and non-arteritic types)
2. Papilloedema
3. Optic disc coloboma

LESION IN CEREBRAL CORTEX
1. Superior or Inferior calcarine cortex lesion
2. Temporal lobe lesion
3. Parietal lobe lesion
4. Tumours affecting both occipital lobe may produce bilateral superior or inferior altitudinal field defect. Although vascular damage can produce either superior or inferior defects; traumatic injury (most commonly from bullet wounds) usually causes only bilateral inferior altitudinal field defects. This is because damage to the lower portion of the occipital lobes; which would produce bilateral superior altitudinal defects; often results in laceration of the dural sinuses; with almost fatal results.
5. Bilateral posterior cerebral artery infarction involving primarily the inferior occipital lobes leads to bilateral superior altitudinal visual field defect.

MECHANISM OF ALTITUDINAL VISUAL FIELD DEFECT
The prelaminar and laminar portions of the optic nerve are supplied by an elliptical arterial “circle” (i.e., Zinn’s corona or Haller’s circle) formed by anastomoses around the optic nerve between medial and lateral parapapillary short posterior ciliary arteries. Branches originating from the arterial circle run anteriorly to the peripapillary choroid transversely to optic nerve (prelaminar and laminar region), and posteriorly to the pial plexus system. The location of the circle is approximately 200-300 micrometer posterior to the subarachnoid space. The arterial circle cannot be visualized with fluorescein angiography because of blockage of fluorescein by the sclera. The Zinn-Haller arterial circle actually is two half circles that are separated at the horizontal meridian by the entry points of the medial and lateral short posterior ciliary arteries, providing an altitudinal blood supply to the anterior optic nerve. Reduced perfusion pressure within the territory of the paraoptic branches of the short posterior ciliary arteries may result in optic disc hypoperfusion and infarction that results in an altitudinal pattern of visual field involvement.

BILATERAL SUPERIOR OR INFERIOR (ALTITUDINAL) HEMIANOPIA
A unilateral visual field defect in all or most of the upper or lower portion of the visual field is always caused by a lesion of the retina or optic nerve. Similarly, bilateral visual field defects of this type usually represent bilateral lesions damaging the retinas or optic nerves. In many of the cases one eye is affected before the other. In such cases the pathology is ischaemia, and most patients have an underlying systemic vasculopathy, such as GCA (Giant Cell Arteritis), diabetes mellitus or systemic hypertension that has caused non-simultaneous bilateral ischaemic optic neuropathy. Rarely, a large prechiasmal lesion compresses both optic nerves producing bilateral-altitudinal field defects. In most of these cases the etiology is a pituitary adenoma that compresses the inferior aspects of both the optic nerves, producing bilateral superior altitudinal field defects. In other cases however, compression of the optic nerves from below elevates them against the dural shelves extending out from the intracranial end of the optic canals. Pressure from the dura against the superior aspects of the nerves subsequently produces bilateral inferior altitudinal defects.

BILATERAL CHECKER BOARD ALTITUDINAL HEMIANOPIA AND THE VERTICAL “HEMIFIELD SLIDE PHENOMENON”
Patients with bilateral altitudinal field defects do not necessarily lose the superior or inferior field in both eyes. Some patients with bilateral optic neuropathies particularly those who develop bilateral simultaneous or non-simultaneous anterior ischaemic optic neuropathy (AION) develop a superior altitudinal field defect in one eye and an inferior altitudinal field defect in the other. In addition to the expected visual difficulties with
visual functions that result from loss of visual acuity, colour vision and field defects such patients may also experience binocular diplopia or difficulty reading caused by decompensation of a pre-existing vertical or horizontal phoria. The problem encountered by these patients result from loss of the normal partial overlap of the superior or inferior fields of the two eyes, this overlap normally permits fusion of images and helps stabilize ocular alignment in patients with vertical or horizontal phorias. Because their remaining visual fields represent only the superior projection from one eye and the inferior projections from the other, patients with a superior hemianopia in one eye and an inferior hemianopia in the other eye do not have a physiological linkage between the two remaining altitudinal hemifields. In such patients a preexisting asymptomatic phoria becomes a symptomatic tropia because of vertical or horizontal separation or overlap of the two remaining hemifields. Patients thus complaints of diplopia and may have difficulty reading because of doubling or inability to see printed letters or words. This condition is called the HEMIFIELD SLIDE PHENOMENON.

And was initially described in patients with bitemporal hemianopic field defects, and it is in such patients that it most often occurs. However the hemifield sliding phenomenon can also occur in patients with heteronymous altitudinal or broad arcuate field defects.

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