Monocular temporal hemianopia

Seymour A Hershenfeld, James A Sharpe

Abstract

Monocular temporal hemianopia was identified in 24 patients. The field of the fellow eye was normal. Computed tomography or magnetic resonance imaging showed juxtasellar lesions in 19 patients. Fifteen had pituitary adenomas, two had tuberculum sella meningiomas, one a craniopharyngioma, and one an astrocytoma. One patient had optic neuritis. A relative afferent pupillary defect (RAPD) was detected in most patients. Field loss was functional in two. Two had congenital optic disc dysversion with hemianopia which did not respect the vertical meridian. Monocular temporal hemianopia is attributed to involvement of the ipsilateral optic nerve close enough to the chiasm to selectively impair conduction in crossing nasal retinal fibres from the ipsilateral eye, but too anterior to affect crossing nasal retinal fibres from the contralateral eye. The combination of an RAPD, with or without optic disc pallor, on the side of monocular temporal field loss implicates compression of the optic nerve at its junction with the chiasm.

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Neuro-Ophthalmology Unit, Division of Neurology and Department of Ophthalmology, The Toronto Hospital, University of Toronto, Canada
S A Hershenfeld
J A Sharpe

Correspondence to:
Dr J A Sharpe, The Toronto Hospital, 399 Bathurst Street, Suite ECW 6-022, Toronto, Ontario, Canada, M5T 2S8.

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At the optic chiasm axons from ganglion cells of the nasal and temporal retina separate, the nasal retinal fibres crossing in the chiasm to the contralateral optic tract and the temporal retinal fibres coursing through the lateral chiasm to the ipsilateral optic tract. Compression or infiltration of the chiasm causes several characteristic patterns of visual field loss. By interrupting the crossing nasal fibres chiasmal lesions typically

cause binocular temporal hemianopic visual field defects aligned along the vertical meridian.¹⁻³ Anterior parachiasmal masses that compress one optic nerve and the chiasm cause a junctional scotoma, consisting of an ipsilateral central scotoma and a superotemporal field defect in the opposite eye. Masses beneath the chiasm cause field defects that are usually denser superiorly. Paracentral bitemporal hemianopia has been attributed to posterior chiasmal compression.¹⁻⁴ An incongruous homonymous hemianopia signifies optic tract involvement, while decrease of visual acuity associated with homonymous hemianopia implicates posterior chiasmal involvement.¹⁻⁴ Occasionally arcuate hemianopic scotoma in the temporal field are associated with disease of the chiasm.^{4,5}

Monocular temporal hemianopia⁶⁻¹¹ can be a functional disorder⁹⁻¹¹ or a manifestation of chiasmal compression.⁶⁻⁸ We report monocular temporal hemianopic defects in 24 patients. The field of the fellow eyes was normal in each case. The clinical features and pathophysiology of this distinctive pattern of field loss are discussed.

Patients and methods

Twenty four patients examined over 15 years in the neuro-ophthalmology unit of The Toronto Hospital, had monocular temporal hemianopic visual field defects. They had computed tomography (CT) or magnetic resonance imaging (MRI) of the suprasellar region and optic nerves. Appropriate endocrine studies were carried out. Visual fields were tested by confrontation, tangent screen examination, Perimetron automated projection perimeter, Goldmann perimeter, or automated perimetry with an Allergan Humphrey machine. Fields were tested serially for periods ranging from 1 month to 15 years (mean 4.1 years).

Results

All monocular hemianopias were superior temporal visual field defects or involved both the upper and lower temporal field. Clinical features in the 24 patients are listed in Table 1. Sixteen of the patients had monocular hemianopia at the time of presentation. In eight patients monocular temporal hemianopia was detected during follow up examinations 1 week to 15 years (mean 4.6 years) after treatment of their suprasellar disease.

Visual acuities in the eyes with the monocular temporal defect were typically good, but varied from 20/20 to finger counting, indicating involvement of the nasal central field (Table 1). Sixteen patients had visual acuity 20/40 or better. Twelve of the 24 patients had right monocular defects. Goldmann or Humphrey perimetry was performed on 23 patients. One patient with optic

Table 1 Summary of clinical information

Patient and diagnosis*	Age	Sex	Interval between presentation and detection of monocular hemianopia	Visual acuity		Side of hemianopia
				Right	Left	
1 Pit	31	M	1 month	20/20	20/20	L
2 Pit	50	M	0	20/80	20/20	R
3 Pit	66	M	0	20/20	20/20	R
4 Pit	28	M	0	20/20	20/20	L
5 Cr	40	F	1 month post-op	20/30	20/400	L
6 Pit	61	F	12 months post-op	20/40	20/50	R
7 Dys	60	F	0	20/20	20/20	L
8 Pit	46	M	0	20/20	CF	L
9 Pit	48	M	15 yrs post-op	20/20-3	20/20-3	L
10 Pit	56	F	8 yrs post-op	CF	20/20	R
11 Pit	44	M	0	20/20	20/20	L
12 Pit	56	F	11 yrs post-op	20/20	20/25	L
13 Pit	47	F	0	20/20	20/20	L
14 Pit	47	F	0	20/25	20/30	R
15 Pit	42	F	0	20/25	20/70	L
16 Fct	43	F	0	20/20	20/20	R
17 Dys	49	F	0	20/400	20/20-3	R
18 Pit	54	F	24 months post-op	20/30	20/30	R
19 Pit	41	F	1 week post-op	20/25	20/25	L
20 Men	33	F	0	20/50	20/20	R
21 Men	57	F	0	20/60	20/20	R
22 Fct	41	F	0	20/20	20/20	R
23 Ast	17	M	0	20/25	20/20	R
24 On	19	F	0	20/20	20/25	L

*Pit=pituitary adenoma; Cr=craniopharyngioma; On=optic neuritis; Men=tuberculum sella meningioma; Fct=functional; Dys=optic disc dysversion; Ast=astrocytoma.

Figure 1 Monocular field defect on tangent screen in a patient with a tuberculum sella meningioma. HM, hand movements; WTO, white test object.

disc dysversion had tangent screen examination only. Twenty two patients showed monocular defects on tangent screen examination (Fig 1). Six of these with a monocular defect on careful tangent screen examination performed by one of us, proved to have binocular defects on automated perimetry. Of these six patients, three had bitemporal defects, two showed arcuate hemianopic defects in the fellow eye, and one had a junctional scotoma on Humphrey field testing. Two of these six patients showed monocular defects on both Goldmann perimetry (Fig 2) and tangent screen examination that later proved to be bitemporal on Humphrey field testing.

Twelve patients had follow up perimetry on the Humphrey machine; four had defects in the fellow eye, indicating that automated static perimetry identified defects undetected by kinetic techniques on tangent or Goldmann perimetry. On the other hand detection of monocular temporal field loss, by Humphrey perimetry (Fig 3) as the only defect in eight patients, confirmed the persistence of genuine monocular temporal hemianopia and the sensitivity of kinetic perimetry.

Two patients had optic disc dysversion associated with a monocular defect that did not respect the vertical midline; it sloped into the upper

nasal field, resulting in a monocular pseudo-temporal hemianopia (Fig 4).

Funduscopy showed optic disc pallor in the eye with the monocular defect in 13 of the 24 patients, normal discs in nine, and disc dysversion in two (Fig 5). Three patients had normal appearing optic discs, but they had a relative afferent pupillary defect (RAPD), estimated clinically, on the side of the monocular field loss. Three other patients had bilateral optic disc pallor and they had RAPD on the same side as the monocular temporal defect. There was an RAPD in 18 patients, all on the side of the monocular visual field defect. Six patients had normal pupils. Of these six cases one had bitemporal field defects before surgical resection of a pituitary adenoma, but only monocular loss on Goldmann perimetry after surgery; two had field loss in the fellow eye which was only detected later on automated perimetry; one patient had optic disc dysversion; and two were functional.

Pituitary adenoma was the cause of monocular field loss in 15 of our 24 patients. Two had tuberculum sella meningiomas (Fig 6), one a craniopharyngioma, and one had an astrocytoma. Two had optic disc dysversion and one had optic neuritis (Table 2). CT or MRI showed unilateral extension towards the side of the hemianopia in nine cases, and extension away from the monocular temporal field defect in three patients. In the others, imaging did not reveal asymmetry of the suprasellar masses. Two patients had monocular defect without objective evidence of visual pathway damage that we attributed to a functional (psychoneurotic) disturbance. The functional visual field defects involved both the upper and lower hemifield, whereas the genuine monocular field loss involved the upper, or both upper and lower quadrants.

Discussion

These patients illustrate an uncommon manifestation of damage to the visual pathway. At least one example of monocular temporal hemianopia was recognised in the nineteenth century, and was probably on a functional basis.¹² Traquair concluded that with early chiasmal lesions, the upper temporal defect may only be evident in one field.¹ According to Scott, 'a lesion at the anterior chiasmal angle extending laterally from its medial aspect affects first of all the crossed fibres of the affected side with resultant temporal hemianopia on the side of the lesion'.¹ A genuine junctional scotoma of Traquair consists of a monocular scotoma with a normal field in the fellow eye.¹ A junctional scotoma more often consists of a monocular temporal hemianopia with a central scotoma in the other eye on the side of the lesion. Traquair¹ also believed that lesions of the optic tract cause incongruity that may be extreme, one field showing a well developed quadrant defect while the other field was normal, but this has not been confirmed. He offered the explanation that, 'Owing to the fact that at the level of the optic tract, the fibres from corresponding retinal points had not yet completely come together, a sufficiently small lesion in one tract, especially in

Figure 2 Goldmann visual field in patient 15 years after transcranial removal of chromophobe adenoma of pituitary gland, shows temporal hemianopic scotoma and upper temporal step defect in left eye.

Figure 3 Humphrey visual field of patient with tuberculum sella meningioma, showing monocular temporal defect in eye in the same patient as illustrated in Figure 1. (A) left eye, (B) right eye.

its anterior portion, may affect one field only and incongruity is common^{7,1}.

Complete monocular temporal hemianopia is typically a functional deficit.^{9-11 13 14} Absence of a relative afferent pupil defect and the persistence of the hemianopia during binocular field testing easily distinguishes functional hemianopia from genuine hemifield loss. A temporal hemianopia in one eye with a normal field in the fellow eye is incompatible with normal pupillary reactions. Our patients with exclusively monocular defects and tumours all had an RAPD on the side of field loss.

Fifteen out of the 24 had a pituitary adenoma as the cause of the monocular hemianopia, two had tuberculum sella meningioma, two had optic disc dysversion, one case had craniopharyngioma,

one had a low grade glioma (astrocytoma), one had optic neuritis, and two cases were functional. Tilted optic discs may simulate monocular or bitemporal hemianopia; the field defects have sloping margins and do not respect the vertical midline; the temporal defects in all of our other patients were aligned along the vertical meridian. Imaging by CT scan or MRI showed unilateral expansion of tumour towards the side of hemianopia in nine cases, and towards the side opposite the monocular temporal field defect in three cases. Precise localisation of tumour impingement on the chiasm or optic nerve was not possible.

One patient with optic neuritis presented with a left monocular temporal scotoma. The Optic Neuritis Study Group detected monocular temporal hemianopia in 4.5% of 415 patients.¹⁵

Elkington⁶ reported on the preoperative symptomatology in 260 patients with pituitary adenoma. Visual fields, obtained by perimetry, showed unilateral temporal defects in 24 patients (9.2%) of the series. Hoyt³ noted that monocular temporal scotoma can indicate the presence of a parasellar tumour, but concluded that the perimetric sign defied logical correlation with established patterns of intrinsic chiasmal anatomy. He stated that monocular hemianopia involving the temporal field probably represented effects of occlusion or stasis in nutrient chiasmal vessels. In a series of 1000 cases of pituitary adenoma,⁷ a superior temporal defect in one eye was recorded in 33 patients. A paracentral temporal scotoma occurred in one eye of 12 patients, and a monocular inferior temporal defect occurred in four

Figure 4 Tangent screen in patient with optic disc dysversion showing monocular pseudotemporal hemianopia.

Figure 5 Left fundus of patient whose field is shown in Figure 4, illustrates dysversion of the optic disc.

Figure 6 T1 weighted MRI shows tuberculum sellae meningioma in a woman who presented with right monocular superotemporal visual field defect.

patients. In another series⁴ of 49 patients with tumours compressing the intracranial optic nerve, optic chiasm, or optic tract, most patients had visual loss predominantly affecting one eye, but authentic monocular temporal hemianopia was found in only two patients.

Monocular temporal hemianopia may be explained by involvement of the ipsilateral optic nerve close enough to the chiasm to selectively impair conduction in ipsilateral crossing fibres but too anterior to affect the crossing nasal retinal fibres from the contralateral eye. Nasal compression or infiltration of the proximal intracranial optic nerve ipsilateral to the field defect, where nasal and temporal retinal fibres have begun to separate, is probably responsible. Retrograde degeneration studies in the monkey chiasm showed that arcuate nerve fibre bundles remain intact well into the chiasm.⁴ Nasal and temporal arcuate bundles may segregate proximal to that segment of optic nerve where the nasal retinal crossing fibres enter the chiasm.^{4,16}

Table 2 Causes of monocular hemianopia in 24 patients

Pituitary adenoma	64%
Tuberculum sellae meningioma	8%
Astrocytoma	4%
Craniopharyngioma	4%
Optic disc dysversion	8%
Functional	8%
Optic neuritis	4%

Tumour compression of the prechiasmal optic nerve segment before the nasal retinal fibres from one eye have begun to cross into the chiasm could explain the monocular hemianopia identified in our patients. Monocular field loss may be caused by a differential mechanical pressure effect due to asymmetric tumour growth,^{17,18} or by differential occlusion of chiasmal nutrient blood vessels.^{19,20}

Our experience indicates that when temporal hemianopia is identified in only one eye, juxta-sellar lesions such as pituitary adenoma or meningioma are likely to be responsible. The optic disc may be normal or pale. The presence of an RAPD with or without optic disc pallor on the side of the monocular field loss signifies compression infiltration, or demyelination of the optic nerve junction with the chiasm.

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