# Optic Tract Syndrome

### Neuro-ophthalmologic Considerations

Steven A. Newman, MD, Neil R. Miller, MD

• A review of the case histories of ten patients with optic tract lesions, an uncommon clinical entity, showed the primary characteristic to be a homonymous visual field defect that may be complete or incomplete. When the defect is incomplete, there is relative incongruity. When it is complete, there is an associated contralateral relative afferent pupillary defect. Visual acuity and color vision are preserved, unless there is bilateral involvement or anterior extension to involve the optic nerve or chiasm. When of sufficient duration, the contralateral fundus demonstrates band or "bow tie" atrophy of the disc and nerve fiber layer.

(Arch Ophthalmol 1983;101:1241-1250)

Homonymous visual field defects from optic tract lesions are rare,<sup>1,2</sup> but are becoming increasingly recognized.<sup>34</sup> Certain features of optic tract involvement appear to allow definitive diagnosis by virtue of the specific anatomy involved. We present our experience with ten patients having lesions involving the optic tracts.

#### **REPORT OF CASES**

CASE 1.—A 9-year-old girl experienced the sudden onset of severe headache and vomiting with transient, horizontal diplopia. During the next several weeks, her headaches increased in frequency and severity. Visual acuity at the time of admission to The Johns Hopkins Hospital, Baltimore, was 20/15 OD and 20/20 OS. Color vision was not tested. Visual fields were full, with bilateral enlargement of the blind spots. Pupils were equal and normally reactive. There was no afferent defect. Ophthalmoscopic examination revealed bilateral papilledema. A computed tomographic (CT) scan revealed a cystic suprasellar mass.

A right frontal craniotomy was performed, at which time a large spongioblastoma was found to be adherent to the hypothalamus, displacing the chiasm to the left. The tumor was partially resected, and the patient received postoperative radiotherapy (5,000 rad). Six months later, the patient was readmitted with recurrent headaches. Visual acuity and color vision remained normal; however, visual field testing revealed an incomplete, incongruous, left homonymous hemianopia (Fig 1). Pupils were equal and normally reactive. There was no afferent defect. The optic discs were normal. Neuroradiologic evaluation demonstrated severe hydrocephalus, and the patient was treated with bilateral ventriculoperitoneal shunts. During the next five months, the patient's visual acuity and color vision remained normal; however, the left homonymous hemianopia progressed until it became complete (Fig 2). During this period a left afferent pupillary defect became evident, and ophthalmoscopy revealed hemianopic atrophy (Fig 3)

In this child, despite retention of normal visual acuity and color vision, gradual progression of a left homonymous hemianopia was associated with the appearance of an ipsilateral (contralateral to the lesion) afferent pupillary defect. This finding suggests that an afferent pupillary defect is not seen in optic tract lesions until there is substantial asymmetry in the degree of visual field loss between the two eyes.

CASE 2.- A 12-year-old boy was first examined in March 1977, suffering from progressive, intractable, left-sided, grand mal, and psychomotor seizures. Visual acuity was 20/20, color vision was normal, and visual fields were completely full. Pupils were normally reactive to light. There was no afferent defect. Optic discs and nerve fiber layer were normal. Two and a half years later, the patient underwent a right hemispherectomy with removal of subcortical material and sectioning of the right optic tract. Postoperatively the patient's seizures resolved, although he had a residual left hemiparesis. When he was reexamined six months postoperatively, his visual acuity and color vision remained normal. Visual fields demonstrated a complete left homonymous hemianopia (Fig 4). Pupils were equal; however, there was a marked, relative left afferent defect. Ophthalmoscopic examination revealed superior and inferior arcuate nerve fiber layer defects with diffuse pallor of the optic disc on the right. The left optic disc showed band atrophy with generalized loss of fibers from ganglion cells nasal to the fovea.

Surgical interruption of the optic tract produced a complete homonymous hemianopia and a classic pattern of hemianopic, retrograde nerve fiber layer degeneration. This condition was associated with the development of an afferent pupillary defect contralateral to the site of the surgical lesion despite the continued presence of normal visual acuity and color vision.

CASE 3.—A 17-year-old boy was referred

Accepted for publication Aug 30, 1982.

From the Neuro-ophthalmologic Unit, The Wilmer Ophthalmological Institute, The Johns Hopkins Hospital, Baltimore.

Reprint requests to the Neuro-ophthalmological Unit, The Wilmer Ophthalmological Institute, The Johns Hopkins Hospital, 601 N Wolfe St, Baltimore, MD 21205 (Dr Miller).

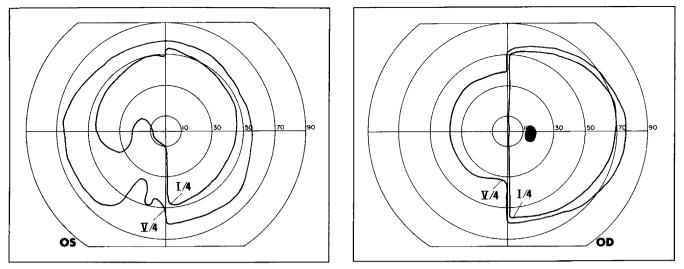


Fig 1.-Initial visual fields in patient 1. There is an incomplete, incongruous, left homonymous hemianopia.

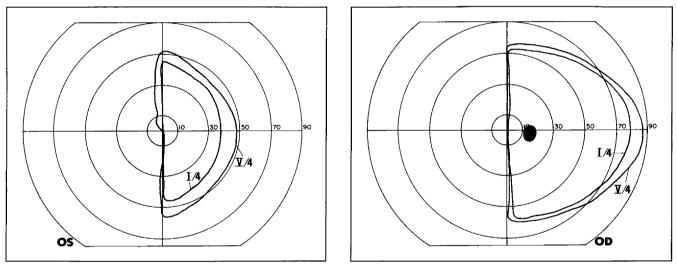


Fig 2.—Visual fields in patient 1 five months after initial examination. There is now a complete, left homonymous hemianopia.

to the Neuro-ophthalmologic Unit with a four-month history of progressive leftsided numbness following a viral illness. This condition was associated with mild weakness of the left side. Two months later, the patient experienced increasing incoordination, stiffness, and dysarthria. He also noted difficulty seeing to the left side.

Neurologic examination on admission to the hospital revealed a spastic left hemiparesis, dysarthria, and left hemihypesthesia. Visual acuity was 20/25 OU, and color vision was normal. There was a virtually complete left homonymous hemianopia (Fig 5). Pupils were equal, but there was a marked left afferent pupillary defect. Convergence was poor, and there were bilateral, mild, horizontal gaze pareses with saccadic dysmetria, especially with the patient looking to the left. Ophthalmoscopic findings were normal. Serologic tests for connective tissue disease and syphilis were negative, but there were several oligoclonal bands seen on a specimen of CSF. The patient was believed to have demyelinating disease.

In this patient, multifocal demyelinating white-matter plaques were believed to have included right optic tract involvement. Although the patient's fundi were believed to be normal, the presence of a complete homonymous hemianopia with an ipsilateral (contralateral to the lesion) afferent defect make a diagnosis of an optic tract syndrome inescapable. Because the subjective field defect preceded the evaluation by approximately six weeks, it is possible that retrograde degenerative changes had not yet occurred.

CASE 4.- A 22-year-old woman experi-

enced transient episodes of left-sided numbness at 8 and 10 years of age. At the age of 22, she experienced the sudden onset of a left hemiparesis and hemihypesthesia. A CT scan demonstrated a large vascular mass in the region of the right optic tract, and an arteriogram demonstrated an extensive arteriovenous malformation in the right ambient cistern. On initial neuroophthalmologic examination, the patient's visual acuity was 20/20 OU, and color vision was normal. There was a complete left homonymous hemianopia, except for a small island of vision in the left hemifield of the left eye (Fig 6). Pupils were equal, but there was a definite left afferent pupillary defect. Ophthalmoscopy showed classic hemianopic nerve fiber laver and optic atrophy. The patient underwent craniotomy and complete removal of the arteriovenous malformation. Postoperatively she continued to have a left hemiparesis and complete left homonymous hemianopia.



Fig 3.—Ocular fundi in patient 1 show hemianopic optic atrophy corresponding to complete, left homonymous hemianopia seen in Fig 2.

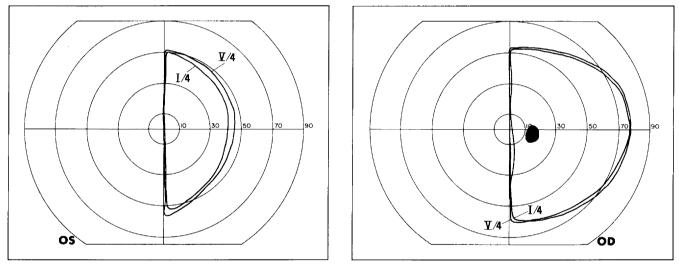


Fig 4.-Visual fields in patient 2. There is complete, left homonymous hemianopia.

In this patient, a portion of the arteriovenous malformation was fed directly through branches of the anterior choroidal artery. It is likely that this patient's optic tract syndrome was vascular in nature.

CASE 5.—A 29-year-old right-handed woman began to have generalized seizures involving the right hand at the age of 7. At the age of 26, she experienced psychomotor seizures consisting in staring spells and automatisms. An EEG demonstrated intermittent sharp waves that were more pronounced over the left temporal region. A brain scan was normal, as was a cerebral arteriogram. A pneumoencephalogram demonstrated some asymmetry of the left temporal horn. A neuro-ophthalmologic examination, performed one year later, revealed normal visual acuity and color vision. There was an incomplete, right homonymous hemianopia that was denser in the lower portion (Fig 7). No afferent pupillary defect could be demonstrated. Ophthalmoscopy revealed nerve fiber layer dropout in both eyes, with "band" atrophy of the right optic disc. Subsequently a neurogenic bladder and cerebellar symptoms developed. Serologic studies and lumbar puncture were unrevealing.

Although the etiology of this patient's neurologic symptoms and signs remains unclear, the presence of nerve fiber layer dropout and hemianopic optic atrophy suggests the diagnosis of a left tract lesion. Noteworthy is the absence of an afferent pupillary defect in the setting of an incomplete homonymous hemianopia (see also case 6).

CASE 6.—A 33-year-old man was referred to The Johns Hopkins Hospital in September 1980, with a complaint of increasing headache and a sensation of "darkness" on the right side of the visual field. Results of a neurologic examination were normal. Visual acuity was 20/20 OU, and color vision was normal. Visual fields showed an incomplete, incongruous, right homonymous hemianopia that was scotomatous in the right eye (Fig 8). Pupils were equal and normally reactive. There was no afferent defect. Ophthalmoscopic findings were normal. A CT scan demonstrated a left,

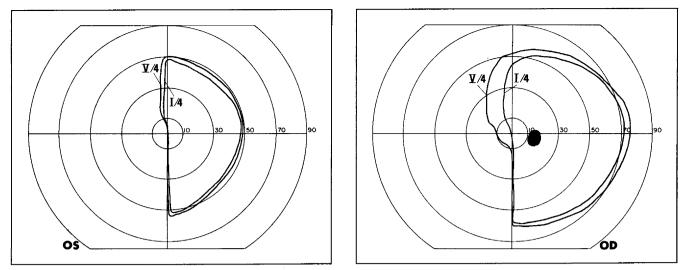


Fig 5.—Visual fields in patient 3. There is a virtually complete, left homonymous hemianopia.

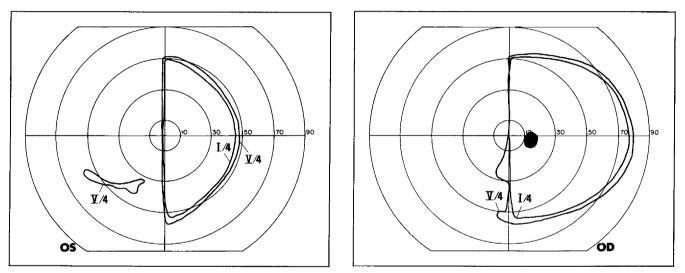


Fig 6.—Visual fields in patient 4. There is complete, left homonymous hemianopia except for small island of field in inferior left field of left eye.

cystic parasellar mass. At craniotomy, a cystic craniopharyngioma was found to be elevating and compressing the left optic tract. The lesion was incised and drained but could not be completely removed. Postoperatively the patient's visual acuity and color vision remained normal, and there was complete resolution of the visual field defect (Fig 9). The fundi remained normal.

This partial, left optic tract lesion produced an incongruous, homonymous hemianopia with preservation of acuity and color vision. Decompression of the tract produced rapid resolution of the field defect. This incomplete syndrome was unassociated with an afferent pupillary defect.

CASE 7.—A 35-year-old man was first examined after he experienced diplopia associated with a severe headache. A complete neuroradiologic examination demon-

strated a large arteriovenous malformation in the ambient cistern. At the time of initial examination, the patient's visual acuity was 20/20 OU, color vision was normal, visual fields were full, and the fundi were normal. Pupils were equal and normally reactive. There was no afferent defect. Ocular motility testing demonstrated a mild, left, fourth nerve paresis. During the next four years, the patient's diplopia recovered; however, he experienced several episodes of subarachnoid hemorrhage, and it was elected to attempt removal of the malformation. Postoperatively the patient had a mild dysphasia and loss of the right visual field. When he was examined ten days after surgery, the patient's visual acuity remained 20/20 OU, and color vision was normal; however, there was a complete right homonymous hemianopia (Fig 10). Pupils were equal, but there was a moderate right afferent pupillary defect. Ophthalmoscopic results were normal.

This patient experienced a complete left optic tract syndrome after removal of an arteriovenous malformation that involved branches of the anterior choroidal artery. As in case 4, this patient's optic tract syndrome was undoubtedly vascular in nature.

CASE 8.—A 50-year-old right-handed woman suffered a grand mal seizure in 1978. Neuroradiologic examination demonstrated a right temporoparietal mass. A right temporal lobectomy was performed, and a grade 2 astrocytoma was disclosed; the patient subsequently received 4,000 rad to the right temporoparietal region. Immediately postoperatively the patient had a left hemiparesis and complained of difficulty with vision to the left side. Neuroophthalmologic examination performed two months postoperatively demonstrated a visual acuity of 20/20 OU and normal color vision. There was a complete left

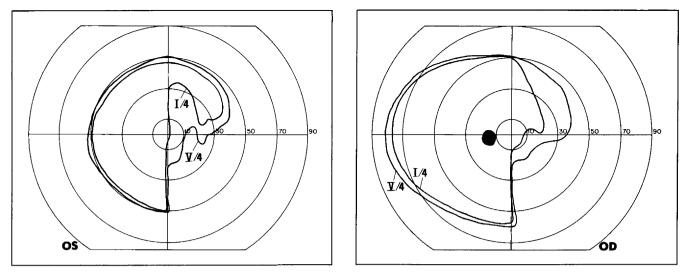


Fig 7.-Visual fields in patient 5. There is incomplete, right homonymous hemianopia, denser below.

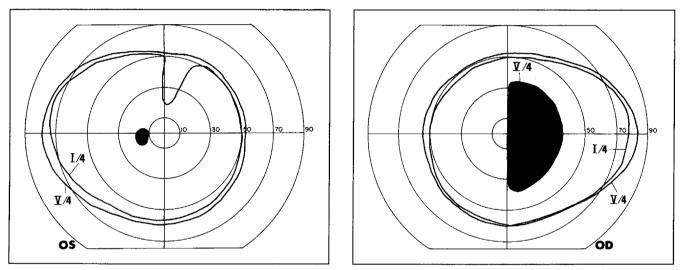


Fig 8.—Preoperative visual fields in patient 6. There is incomplete, right homonymous hemianopia that is densely scotomatous in right eye.

homonymous hemianopia (Fig 11). Pupils were equal, but there was a marked left afferent pupillary defect. Optokinetic testing demonstrated symmetric, normal responses to horizontal stimuli. Ophthalmoscopy revealed hemianopic nerve fiber layer and optic atrophy.

Although the patient's tumor undoubtedly involved the optic radiation, the finding of a contralateral afferent pupillary defect and hemianopic atrophy suggests that either the tumor extended to involve the right optic tract or the right optic tract was damaged at the time of surgery.

CASE 9.—A 55-year-old man was admitted to the hospital with a two-year history of personality change and one month of difficulty seeing to the right side. Neuroradiologic evaluation, including CT scan and cerebral arteriography, revealed a deep, left-sided mass involving the temporal and parietal lobes. The patient had normal neurologic findings, except for some degree of confusion and disorientation. Visual acuity and color vision were normal. There was a complete right homonymous hemianopia (Fig 12). Pupils were equal, and there was a mild right afferent defect. Optokinetic testing demonstrated an asymmetric response, with saccades to the right being markedly decreased. Ophthalmoscopy showed no abnormality.

A left craniotomy was performed; a grade 3 astrocytoma was encountered and subtotally removed. The anterior 5 cm of the left temporal lobe was also removed. Postoperatively, the patient was treated with tapering doses of systemic decadron and radiotherapy (5,100 rad).

Three months later the patient was reexamined. Visual acuity and color vision remained normal. Visual fields showed appreciable improvement, with an extremely incongruous, right homonymous hemianopia that was localized to the superior quadrant in the right eye (Fig 13). Pupillary reactions were normal, and the afferent pupillary defect that had previously been noted had now disappeared. Ophthalmoscopic results remained normal.

It is likely that this patient's optic tract syndrome occurred from either direct tomor compression of the left optic tract or secondary edema involving the tract. Decompression and radiotherapy subsequently restored tract function. As the patient's visual field improved, his afferent pupillary defect disappeared, while visual acuity and color vision remained unchanged and normal. The residual visual field defect probably represents damage to the optic radiation in the

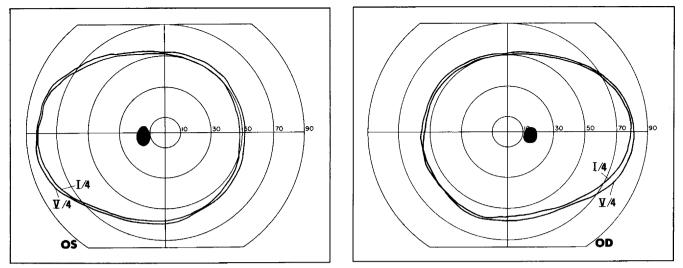


Fig 9.—Visual fields in patient 6 two weeks after craniotomy with incision and drainage of cystic craniopharyngioma. Visual fields have returned to normal.

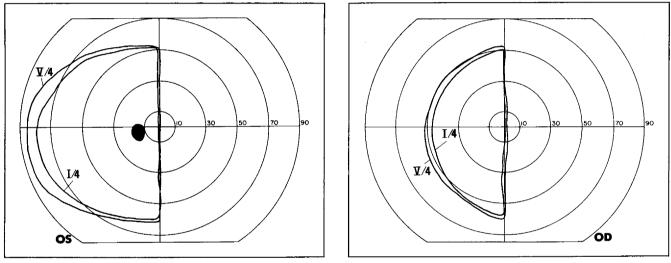


Fig 10.-Visual fields in patient 7. There is complete, right homonymous hemianopia.

## left temporal lobe, thus explaining the continued lack of optic atrophy.

CASE 10.—A 58-year-old man noted progressive loss of visual acuity in his right eye. Visual acuity OS was 20/600 and he was unable to identify any of the pseudoisochromatic plates. Visual acuity OD was 20/20 and color vision was normal. Visual fields demonstrated an incongruous, right homonymous hemianopia that was denser in the lower portion of the left eye (Fig 14). Pupils were equal, but there was a marked right afferent defect. Ophthalmoscopy revealed hemianopic atrophy, and a CT scan revealed an enhancing, suprasellar lesion.

A frontotemporal craniotomy was performed, and a large meningioma, presumably arising from the planum sphenoidale, was identified. It compressed the right optic nerve and chiasm, and extended posteriorly to compress the left optic tract. The patient was not examined postoperatively.

In this patient, a right homonymous hemianopia associated with hemianopic optic atrophy indicated a left optic tract lesion; however, loss of visual acuity and color vision contralateral to the lesion suggested involvement of the contralateral optic nerve. This phenomenon was demonstrated at the time of surgery. Parasellar masses often give a combined picture of nerve, chiasm, and tract defects.

#### RESULTS

All patients in this series were examined by one of us (N.R.M.). Visual acuity testing was performed with standard Snellen's letters at distance and Jaeger's cards at near. The results were recorded as best corrected if the acuity was less than 20/25. Color vision screening was performed using the Hardy-Rand-Rittler pseudoisochromatic plates. Visual field testing included confrontation technique, Bjerrum's (tangent) screen using both white and red targets, and kinetic perimetry with Goldmann's perimeter. Pupillary reactions were evaluated with an indirect ophthalmoscope as a light source. Ophthalmoscopy was performed with both direct and indirect ophthalmoscopes, and both color and red-free photographs were obtained for most patients. The results of our examinations are seen in the Table and Figs 1 through 4.

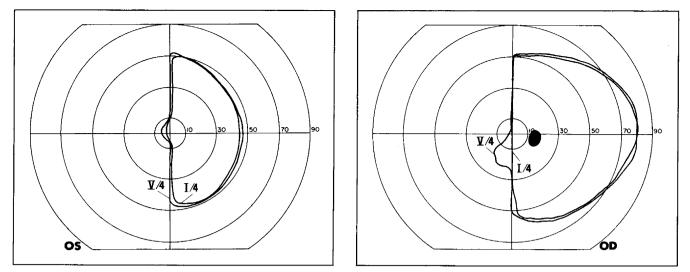


Fig 11.-Visual fields in patient 8. There is complete, left homonymous hemianopia.

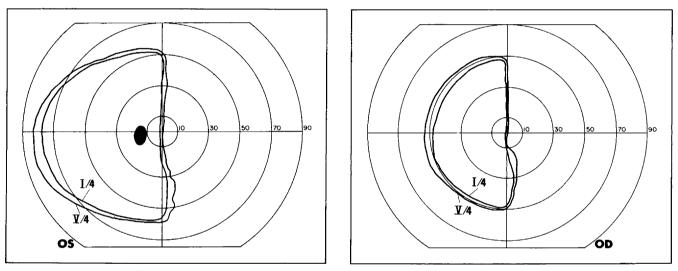


Fig 12.-Initial visual fields in patient 9. There is complete, right homonymous hemianopia.

There were six men and four women in our series. The mean age at diagnosis was 33 years (range, 9 to 58 years). All patients underwent CT scanning and cerebral angiography. Seven patients had lumbar puncture with CSF analysis. Optic tract abnormalities involved the right side in five cases and the left side in five cases. Eight of the ten patients ultimately underwent craniotomy with visualization of the optic tract abnormalities in all cases.

Five patients had an underlying neoplasm, although in one of these patients (case 8), a subsequent complete tract lesion was believed to have been surgically induced. Two patients had tract lesions that were clearly surgically induced. In one patient (case 7), removal of an arteriovenous malformation resulted in a complete homonymous hemianopia. In a second patient (case 2), a hemispherectomy was performed for intractable seizures. Of the remaining three patients, one had an arteriovenous malformation, a second had demyelinating disease, and in a third patient the origin of the optic tract syndrome remains unknown.

Six patients complained of hemianopic visual field difficulties; only one patient complained of decreased vision. Visual acuity and color vision were completely normal in all but one patient. When first examined, six patients had complete homonymous hemianopias and four patients had incomplete, incongruous hemianopias. In one of the patients (case 9), an initially complete defect partially resolved; in a second patient (case 6), an incomplete hemianopia completely resolved; and in the third patient (case 1), an incomplete defect gradually progressed to become complete.

A relative afferent pupillary defect was present in eight of the patients at some stage of their disease. In one of these patients (case 9), the afferent defect subsequently resolved as the visual field defect improved. In another patient (case 1), the pupillary defect developed as the patient's visual field defect progressed. Both patients maintained normal visual acuity and color vision. None of the patients demonstrated anisocoria.

Six of the patients demonstrated hemianopic retinal fiber and optic nerve atrophy with band or "bow tie" atrophy in the eye opposite the lesion.

In addition to ophthalmologic signs, five patients had neurologic signs,

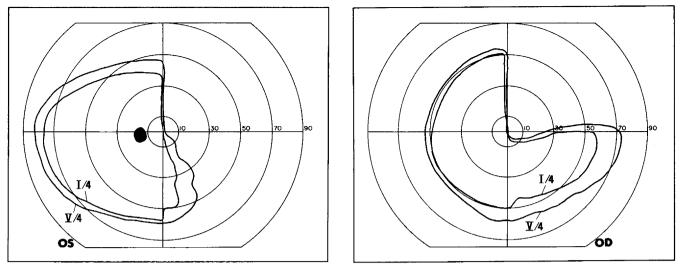


Fig 13.—Visual fields in patient 9 three months following subtotal resection of left temporal lobe astrocytoma and postoperative radiotherapy. There is incomplete, incongruous, right homonymous hemianopia that is localized to superior quadrant in right eye.

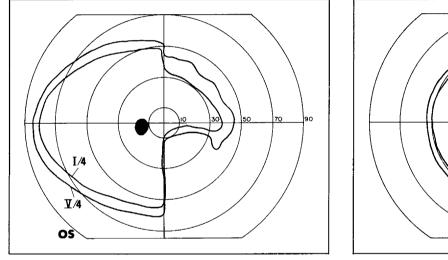


Fig 14.-Visual fields in patient 10. There is incongruous, right homonymous hemianopia, denser below, in left eye.

including seizures in three and hemiparesis ipsilateral to the lesion in two. Two additional patients experienced hemiparesis after craniotomy, and one patient had a mild dysphasia.

#### COMMENT

Our experience with the optic tract syndrome suggests that several generalizations may be made regarding various visual findings in such patients.

#### Visual Acuity

Cogan<sup>5</sup> emphasized that lesions of the retrochiasmal visual system do not produce loss of visual acuity unless they are bilateral. Nevertheless, two reports describing the ophthalmic features of patients with optic tract lesions found a high prevalence of monocular or binocular visual loss (82%).<sup>34</sup> Although this finding would seem to contradict Cogan's statements, Savino et al<sup>3</sup> suggested that most of the patients in their series had extension to involve the chiasm or optic nerve, thereby resulting in decreased visual acuity. The work of Frisén<sup>6</sup> seems to support this contention. In fact, Frisén stated the following:

Until evidence to the contrary is accumulated, it appears more rational to localize anterior visual pathway lesions with impaired acuity in one or both eyes to optic nerve(s) or the chiasm rather than to the optic tract.

All of Frisén's patients with optic tract lesions reportedly had normal

acuity. Of our patients, only one (case 10) had reduced central vision contralateral to the lesion. In this patient, a meningioma involved the optic nerve on one side and the optic chiasm and optic tract on the other side.

ÓD

#### **Visual Field Defects**

It would seem that nonsurgical lesions involving the optic tract generally produce incomplete homonymous hemianopia.<sup>3,4,6</sup> Savino et al described only one patient with a complete homonymous hemianopia, and Bender and Bodis-Wollner<sup>4</sup> also reported that only one of their patients had a complete homonymous hemianopia that was traumatic in origin. Bell and Thompson<sup>7</sup> found only four of 24 patients with complete field

Case/ Age, yr/Sex	VA	Initial Examination			Clinico- pathologic	Final Examination				
		VF	Pupil	Fundi	Findings	VA	VF	Pupil	Fundi	Comment
1/9/ <b>F</b>	20/20 OU	NL	NL	PAP	Spongioblastoma	20/20 QU	ILHH, CLHH	NL, LAPD	NL, HA	Development of LAPD with development of CLHH
2/12/M	20/20 OU	NL	NL	NL	Seizures	20/20 OU	CLHH	LAPD	HA	Right OTS after hemispherectomy
3/17/M	20/25 OU	CLHH	LAPD	NL	Multiple sclerosis					
4/22/F	20/20 OU	CLHH	LAPD	HA	AVM	20/20 OU	CLHH	LAPD	HA	Right OTS probably due to optic tract infarction
5/29/F						20/25 OU	IRHH	NL	HA	Cause unknown
6/33/M	20/20 OU	IRHH	NL	NL	Cystic craniopharyngioma	20/20 OU	NL	NL	NL	No afferent pupillary defect with scotomatous hemianopia
7/35/M	20/20 OU	NL	NL	NL	AVM	20/20 OU	CRHH	RAPD	NL	Complete OTS after removal of AVM
8/50/F					Tempoparietal astrocytoma	20/20 OU	CLHH	LAPD	НА	Questionable extension o tumor to optic tract v surgical damage
9/55/M	20/20 OU	CRHH	RAPD	NL	Temporoparietal astrocytoma	20/20 OU	IRHH	NL	NL	Disappearance of afferen pupillary defect with partial resolution of hemianopia
10/58/M	20/20 OD, 20/600 OS	IRHH	RAPD	НА	Meningioma					Optic nerve, chiasm, and tract involved

\*Abbreviations and symbols are as follows: VA, visual acuity; VF, visual field; NL, normal; CRHH, complete right homonymous hemianopia; CLHH, complete left homonymous hemianopia; IRHH, incomplete right homonymous hemianopia; ILHH, incomplete left homonymous hemianopia; PAP, papilledema; AVM, arteriovenous malformation; OTS, optic tract syndrome; APD, afferent pupillary defect; ellipses, no examination.

defects and normal visual acuity. Conversely, Frisén suggested that visual field defects due to optic tract lesions are usually complete. He attributed his findings to the small size of the tract. Five of our patients had complete homonymous hemianopias, two of which (cases 2 and 7) were surgically induced. One additional patient (case 9) had a transiently complete hemianopia, and in another patient (case 1) the initially incomplete defect gradually completed.

It seems clear that when visual field defects from optic tract lesions are incomplete, they tend to be incongruous. The value of incongruity in differentiating an optic tract lesion from a lesion of the lateral geniculate body or optic irradiation seems less clear. Savino et al<sup>3</sup> suggested that "the presence of gross incongruity is almost certainly a sign of optic tract involvement." Although our patients with incomplete homonymous field defects had obvious incongruity, the degree of incongruity present did not seem to be useful in distinguishing optic tract abnormalities from abnormalities of the lateral geniculate body or optic irradiation.

#### Pupillary Abnormalities

Pupillary defects associated with homonymous hemianopias have been recognized since 1883, when Wernicke<sup>8</sup> summarized his own findings and those of earlier observers and suggested that stimulation of the nonseeing portion of the retina resulted in less reaction than that seen when the seeing portion was illuminated. This pupillary hemikinesia has always been extremely difficult to observe because of the tendency for light scatter. and many investigators have questioned its existence. In addition, Cibis et al<sup>9</sup> used infrared pupillography and demonstrated hemihypokinesia in five patients with postgeniculate lesions, all of whom had normal visual acuity and ophthalmoscopic examination results. This finding would seem to greatly limit the diagnostic usefulness of "Wernicke's pupil" in the diagnosis of pregeniculate lesions.

A second pupillary sign associated with hemianopia was described by Behr,<sup>10</sup> who noted that the pupil ipsilateral to the homonymous field defect tended to be slightly larger than that contralateral to the field defect. Although three of the patients in the series reported by Savino et al<sup>3</sup> had slightly enlarged pupils on the side of the hemianopia, none of the patients reported by Bender and Bodis-Wollner<sup>4</sup> had such abnormalities, and we found no such abnormalities in our patients.

The most useful pupillary abnormality that is seen in patients with optic tract lesions seems to be a relative afferent pupillary defect contralateral to the lesion. The presence of such a defect seems to relate directly to the degree of difference in the amount of visual field loss between the two eyes.

Although approximately 65% of visual fibers cross in the optic chiasm of the cat, the percentage is substantially lower in primates. Ramon y Cajal" estimated that the ratio of crossing to noncrossing fibers was 2:1, but more recent studies suggest a ratio of 53:47.12 These findings correlate well with observations of more receptor density on the nasal aspect of the retina than on the temporal aspect<sup>13,14</sup> and are in keeping with the larger size of the temporal field, which is 60% to 70% larger than the nasal field. This asymmetry led Burde<sup>15</sup> to predict the occurrence of a relative afferent pupillary defect contralateral to an optic tract lesion. When Bell and Thompson' reexamined their four patients with complete optic tract lesions and normal visual acuities, all four were found to have relative afferent defects. Previous series<sup>3,4</sup> have found afferent pupillary defects in patients with ipsilaterally decreased visual acuity; however, as has already been noted, these patients undoubtedly had involvement of the ipsilateral optic nerve. It is the afferent pupillary defect that occurs in patients with complete or extensive homonymous hemianopia but without evidence of optic neuropathy that establishes the diagnosis of an optic tract syndrome without question.

All of the patients in our study who had normal visual acuities and a complete homonymous hemianopia demonstrated a relative, contralateral afferent pupillary defect. Similarly, those patients with incongruous, incomplete field defects were all free of afferent defects except the patient with decreased acuity. The presence of a contralateral afferent pupillary defect in the setting of a complete homonymous hemianopia distinguishes a tract lesion from involvement of the radiations. In addition, we have produced contralateral afferent pupillary defects in two monkeys in which the optic tract was sectioned without damage to the optic nerve. Similar studies have been reported by O'Connor et al.<sup>16</sup> That the afferent pupillary defect seems to be dependent solely on the degree of visual field abnormality can be seen by the

1. Smith JL: Homonymous hemianopia: A review of 100 cases. Am J Ophthalmol 1962;54:616-622.

2. Trobe JD, Lorber ML, Schlezinger NS: Isolated homonymous hemianopia: A review of 104 cases. Arch Ophthalmol 1973;89:377-381.

3. Savino PJ, Paris M, Schatz NJ, et al: Optic tract syndrome: Review of 21 patients. Arch Ophthalmol 1978;96:656-663.

4. Bender MB, Bodis-Wollner I: Visual dysfunction in optic tract lesions. Ann Neurol 1978;3:187-193.

5. Cogan DG: Neurology of the Visual System. Springfield, Ill, Charles C Thomas Publisher, 1967, p 250.

6. Frisén L: The neurology of visual acuity. Brain 1980;103:639-670.

7. Bell RA, Thompson HS: Relative afferent pupillary defect in optic tract hemianopias. Am J Ophthalmol 1978;85:538-540.

8. Wernicke C: Ueber hemiopische pupillenreaction. Fortschr Med 1883;1:49-53. fact that an afferent pupillary defect developed in patient 1 in our series only after an incomplete homonymous hemianopia progressed to become complete, and patient 9 showed disappearance of an afferent pupillary defect as a complete homonymous hemianopia resolved into an incomplete defect. In both patients, visual acuity and color vision remained normal in both eyes, despite the changes in pupillary reactivity. These findings are also in keeping with recent studies by Thompson et al.<sup>17</sup> suggesting that afferent pupillary defects correlate more closely with visual field defects than with visual acuity loss.

#### **Ophthalmoscopic Appearance**

The earliest recognition of retrograde degeneration and resultant hemianopic nerve fiber layer and optic atrophy has been attributed by Hoyt and Kommerell<sup>18</sup> to Lauber.<sup>19</sup>

#### References

9. Cibis GW, Campos EC, Aulhorn E: Pupillary hemiakinesia in suprageniculate lesions. Arch Ophthalmol 1975;93:1322-1327.

10. Behr C: Die Untersuchungsmethoden: II. Die Lehre von Pupillenbewegungen, in Graefe A, Saemische I (eds): Handbuch der gesamtem Augenheilkunde. Leipzig, East Germany, W Engelmann, 1924, vol 2, pp 115-117. 11. Ramon y Cajal S: Die Structur des Chiasma

11. Ramon y Cajal S: Die Structur des Chiasma opticum, nebst einer allgemeinen Theorie der Kreuzung der Nervenbahnen. Leipzig, East Germany, Johann Ambrosius Barth, 1899.

12. Kupfer C, Chumbley L, Downer J: Quantitative histology of optic nerve, optic tract and lateral geniculate nucleus in man. J Anat 1967;101:393-401.

13. Osterberg G: Topography of the layer of rods and cones in the human retina. Acta Oph-thalmol 1935;6(suppl):8.

14. Van Buren JM: The Retinal Ganglion Cell Layer. Springfield, Ill, Charles C Thomas Publisher, 1963, p 130. The period necessary for retrograde degeneration following a pregeniculate lesion is about six weeks. When such hemianopic changes are present, they signify a pregeniculate lesion or one that developed early in infancy. There is good evidence that transsynaptic degeneration does not occur in adults.<sup>20</sup>

#### Conclusions

Our series reemphasizes the tendency for optic tract syndromes to occur in man. The study suggests that visual acuity is normal in such patients unless the optic nerves are also involved. Visual field defects are homonymous, often incomplete, and usually incongruous. When complete, they invariably are associated with an afferent pupillary defect contralateral to the lesion. Hemianopic retinal and optic atrophy may occur six weeks following damage to the tract.

15. Burde RM: The pupil. Int Ophthalmol Clin 1967;7:849.

16. O'Connor PS, Kasdon D, Tredici TJ, et al: The Marcus Gunn pupil in experimental optic tract lesions. *Ophthalmology* 1982;89:160-164.

17. Thompson HS, Montague P, Cox TA, et al: The relationship between visual acuity, pupillary defect, and visual field defect. *Am J Ophthalmol* 1982;93:681-688.

18. Hoyt WF, Kommerell G: Der fundus oculi bei homonymer hemianopie. Klin Monatsbl Augenheilkd 1973;162:456-464.

19. Lauber H: Die ophthalmoskopische Differentraldiasnose der infra-und supranuclearen Hemianopsie, zugleich ein Beitrag zur Topographie des Faserverteilung in der Netzaut. Berud Versammml Deutsch Ophth Gesellsch 1927;46:89-94.

20. Miller NR, Newman SA: Transsynaptic degeneration. Arch Ophthalmol 1981;99:1654.