The end in sight: a look at the occipital lobe

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Abstract

The occipital lobe is the endpoint of the visual pathway and the part which is most fully concerned with vision. This paper will first describe the anatomy of the occipital lobe including functions of cell types, topographical retinal representation, and vascular supply. A detailed discussion of the clinical manifestations of occipital lobe lesions including patient symptoms, visual field defects, and visual recognition defects will follow. Finally, the evaluation of patients with suspected occipital lobe lesions will be discussed with a case example presented to illustrate the relationship between occipital lobe function and clinical practice. © 1998 Elsevier Science Ireland Ltd. All rights reserved.

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1. Introduction

Neural fibers containing visual information terminate and are most largely represented in the occipital lobe. Thus, the sense of vision depends on its integrity. The occipital lobe represents the orderly endpoint of a neurological network that begins with the retina. Knowledge of its anatomy and workings is important in understanding visual function, identifying lesions, and interpreting visual field findings. But the occipital lobe is not just concerned with sight per se. Connections between the occipital lobe and its adjacent parietal and temporal lobes also serve higher visual functions, such as reading, writing, and recognition. Systemically, the occipital lobe may be considered in the context of its anatomical and physiological relationship to the entire human body, allowing for increased understanding of patient health and systemic disease.

2. Anatomy

2.1. Gross anatomy of the occipital lobe

The occipital lobe comprises the posterior portion of the cerebral hemispheres. It rests on the tentorium cerebelli, a double fold of dura mater separating the occipital lobe from the cerebellum. The rostral boundary of the lobe is formed by the parieto-occipital sulcus on the medial part of the cerebral hemispheres [1]. The lateral boundary is formed by an imaginary line joining the parieto-occipital sulcus and the pre-occipital notch [2] (Fig. 1). Along the lateral surface, the lateral occipital sulcus runs transversely dividing the occipital lobe into the superior and inferior gyrus. Medially, the calcarine sulcus divides the lobe into the cuneus and lingual gyrus [3]. It is along the superior and inferior banks of the calcarine sulcus that the primary visual cortex rests. Visual fibers from the lateral geniculate nucleus (LGN) terminate in this area.

2.2. Anatomy of the striate cortex

The primary visual cortex is also known as area 17 of Brodmann, V1, or the striate cortex. The last term refers to the pattern of geniculo-calcareous nerve fibers known as Stria of Gennari [4]. The striate cortex extends medially within the calcarine sulcus to just beneath the corpus callosum. Laterally, it extends 1–2 cm onto the outer surface of the occipital lobe [5]. The striate cortex is divided into six layers or laminae.
with layer I being the most superficial. Neurons have been divided into two main types based on the arrangement of their dendritic branches: pyramidal cells and stellate cells. The optic radiations terminate primarily in lamina IV, though a few end in lamina I and VI. Lamina IV has been further subdivided into IVa, b, and c [4]. Parvocellular axons from the LGN terminate in sublayer IVc-β while LGN magnocellular axons terminate in sublayer IVc-α [5]. Some cells in lamina VI project back to the LGN. A prominent bright white band composed primarily of visual projection fibers in sublayer IVb comprises the line, or stria of Gennari [4].

2.3. Functions of striate cortical cells

Much of the pioneering work in describing the functions and interactions of striate cortical cells has been done by Hubel and Wiesel. They have identified four types of cortical cells based on their receptive field properties: circularly symmetric, simple, complex, and hypercomplex. These types are hierarchically arranged, with each group receiving input primarily from those at the lower level and the circularly symmetric group receiving input predominately from the LGN [4].

Circularly symmetric cells are similar in function to those of the LGN. They respond to spots of light regardless of orientation, movement, or speed. The receptive fields of simple cells have on regions and off regions. That is, if a visual stimulus falls into the on region, the cell will fire; if it falls within the off region, it will not. Receptive fields of simple cells are usually arranged in parallel bands of alternating on and off regions. They respond to stimulus orientation, direction of movement, and speed of movement. For example, a given simple cell with a vertical receptive field will respond vigorously to a vertical stimulus that falls within its receptive field. If instead the stimulus is obliquely-oriented, or if it falls outside its receptive field, there will be no response or the response will be dampened. Thus, for the first time in the visual pathway, patterns and contours are able to be perceived. Simple cells are almost exclusively located in layer IV of the striate cortex.

Complex cells have many similar properties to simple cells. One difference is that simple cells tend to respond to a line with a particular orientation only in a specific position whereas complex cells tend to respond to such a line regardless of its position. Complex cells are generally found outside area IV.

Hypercomplex cells possess all of the properties of the previous three with the additional ability to respond to stimulus length. Hypercomplex cells are also located in areas outside area IV. One distinguishing feature of both complex and hypercomplex cells is that they each receive input from fibers carrying information from both eyes. A given cell will have an identical receptive field when each eye is tested individually. However, when both eyes are tested together, a particular cell's response will be much stronger than when it is tested with one eye occluded.

Striate cortical cells are arranged in columns of ocular dominance. All cells in a slice made perpendicular to the cortical surface, extending throughout the entire six layers will respond preferentially to the same eye. Adjacent columns will show ocular dominance corresponding to the other eye. Hubel and Weisel have determined that the width of an ocular dominance column is 0.4 mm in the macaque monkey. In addition, these columns tend to respond to the same stimulus orientation (the one exception being the circularly symmetric cells in layer IVc that have no orientation preference). Each column responds best to a slightly different orientation compared to the one next to it. All 180° are represented in approx. 1 mm of cortex [4].

Ocular dominance columns are not fully developed at birth. Experiments with newborn monkeys or kittens have shown that when an eye is occluded, cells corresponding to that eye become shrunken and their counterparts in the other eye hypertrophy. These findings may have some significance in the development of amblyopia in humans [5].

2.4. Parastriate and peristriate areas

The parastriate and peristriate areas comprise what is known as the visual association cortex. They function to integrate raw sensory information received in the striate cortex with other areas of the brain. Thus lesions in these areas may produce deficits in visual recognition, visual orientation, and areas combining visual and other sensory information.

The parastriate cortex (area 18 of Brodmann) lies adjacent to the striate cortex in the occipital lobe on
each side of the brain. It is also six-layered, but lacks the line of Gennari. The peristriate cortex (area 19) surrounds the parasitriate cortex. It lies mostly in the posterior parietal lobe but is within the temporal lobe inferiorly. It is histologically similar to parietal lobe.

Area 18 receives afferent fibers from the ipsilateral area 17. It also has connections to ipsilateral area 19 as well as the contralateral areas 18 and 19. Fibers cross the corpus callosum projecting into the contralateral hemisphere integrating opposite halves of the visual field. Another important function of area 18 is its role in the formation of voluntary eye movements. Fascicles are sent to the frontal lobe which coordinate incoming visual sensory information with voluntary deviations of the eyes. It has also been shown that fibers from area 18 project back to the LGN providing a cortical–geniculate feedback loop in cats, though this has not yet been shown in humans.

Area 19 has substantial connections with the pulvinar and pretectum. It plays a role in visual memory, visual associations and integrating visual information with other modalities [5].

3. Visual field representation in the striate cortex

As noted previously, most of the primary visual cortex lies buried within the calcarine sulcus. Probably the most important early model depicting retinal representation in the visual cortex was developed by Holmes [6]. In the Holmes map, the ipsilateral hemiretina and the contralateral hemifield are represented on each side of the calcarine sulcus. Thus, the right calcarine cortex will correspond to the right hemiretina of both eyes and the left hemifields of vision. The upper bank of the calcarine sulcus will correspond to the superior retinas and the inferior visual field. By this model, a lesion to the right supero-temporal bank of the calcarine sulcus will affect the left inferior visual field.

In the visual field, the horizontal meridian is represented along the base of the calcarine sulcus, while the vertical meridian is usually represented along the exposed medial surface of the occipital lobe [7]. The macula is represented at the posterior pole of the occipital lobe, while the fovea corresponds to its extreme tip. The peripheral retinas are projected in a sequential fashion to the anterior portion of the visual cortex. The far peripheral nasal retina from the contralateral eye only projects to the medial far anterior portion of the cortex [5]. This area of unpaired nerve fibers corresponds to the unioocular “temporal crescent” in visual space, representing 60–100° from fixation in the horizontal meridian [8].

The central retina, with its density of neurons and importance in visual acuity, has long been recognized to encompass an expanded representation in the visual cortex. Based on the Holmes model, the central 15° of retina are considered to be served by 25% of the striate cortex [6]. Recent work by Horton and Hoyt has called these numbers into question. Based on studies of MRIs of patients with occipital lobe lesions, the researchers conclude that Holmes underestimated the area of cortex devoted to central vision. According to Horton and Hoyt, the area of visual cortex corresponding to the central 15° of vision is closer to that of the macaque monkey, 70%. The ‘cortical magnification factor’, a measure of the amount of cortex devoted to a given area of retina, is inversely proportional to its eccentricity from the fovea. That is, the closer a point on the retina is to the fovea, the more visual cortex space will serve it [7].

The Horton and Hoyt model has important implications for visual field testing and the interpretations of visual field defects. For example, according to the model, a central 30° visual field tests as much as 83% of the striate cortex. A central 24° visual field tests over 80%. It also explains why visual field defects limited to the temporal crescent are so rare. Because the temporal crescent is so far peripheral in the visual field, its representation in the striate cortex is very small. A lesion encompassing this part of the cortex is likely to extend to other areas of the cortex causing a larger field defect [7].

The Horton and Hoyt model also relates to the concept of macular sparing. This refers to the preservation of central vision, considered to be at least the central 5°, in the context of peripheral field defects. Macular sparing, when it occurs, is usually restricted to occipital lobe lesions. However, if the macula is not spared, it does not necessarily imply that the lesion is located outside the occipital lobe. Inouye (1909), found macular sparing his study of wounded soldiers and attributed it to bilateral macular representation in each occipital lobe. Later models have supplanted this notion, attributing the phenomenon of macular sparing to the dual blood supply to the posterior pole of the occipital lobe by the posterior cerebral artery and the middle cerebral artery. Because the amount of cortex devoted to the macula is so large, there is a greater chance that perfusion will be maintained and central vision spared, in the event of infarct by one of the arteries [7].

4. Blood supply to the visual cortex

The vertebrobasilar system provides most of the blood supply to the visual cortex. The posterior cerebral artery (PCA), emerging from the circle of Willis, is the most important vessel in this area. Proximal branches of the PCA supply the mesial areas of visual cortex and those corresponding to the mid-peripheral
and peripheral visual field. Laterally, there are anastomoses between branches of the PCA and those of the middle cerebral artery (MCA). Additionally, there are accessory blood supplies from the parieto-occipital and posterior temporal arteries approx. 50% of the time [5].

The posterior pole of the occipital lobe, concerned with central vision, is supplied by an anastomosis of terminal branches of the posterior and middle cerebral arteries. These are the parieto-occipital (superiorly), calcarine (centrally) and the posterior temporal (inferiorly) branches of the PCA and the occipital branches of the middle cerebral artery [4].

5. Clinical manifestations of occipital lobe lesions

5.1. Visual field defects

Since fibers from corresponding points of both retinas run so close together in the occipital lobe, visual field defects are almost always homonymous. The more posterior the lesion, generally the more congruous the field defect. The most common visual field defect is a homonymous hemianopsia [8,9] (Fig. 2). However, it should be noted that a complete, hemianopsia with ‘splitting of the macula’ (the vertical borders of the hemianopsia passing through the center of fixation) does not localize any specific postchiasmatic point in the visual pathway. Quadrantanopsias or various types of other homonymous congruous defects may also occur [9]. In contrast to lesions in other parts of the visual pathway, which often result in other sensory or motor deficits, occipital lobe lesions tend to give isolated visual symptoms [8].

5.2. Etiologies and risk factors

Lesions in the occipital lobe may be caused by infarction, inflammation, trauma and neoplasia [10], with infarction being the most common etiology [8]. Trobe et al. [11] studied 104 cases of isolated hemianopsias and attributed 89% of them to strokes. Similarly, in a study of 140 cases of homonymous hemianopsias Fujino et al. [12] found that 71% of them were vascular in origin. Of these, 86% were localized to the occipital cortex. The occipital lobe was the location of the lesion in slightly over half the cases of hemianopsia in Fujino’s study.

Most occipital infarctions are due to emboli from the heart or the vertebrobasilar system. Such emboli may be the result of valvular heart disease, subacute bacterial endocarditis, atrial fibrillation, or ulcerated arterial plaques. Arterial thrombosis of the posterior cerebral artery is uncommon. Other, less common sources of infarctions that have been implicated in occipital lesions include hyperviscosity and hypercoagulable states, migraine, oral contraceptive use, polycythemia vera, sickle cell anemia, cardiac catheterization, and chiropractic cervical manipulation. Cerebral hemorrhage and neoplasm must also be ruled out [9] (Fig. 3). Bogousslavsky et al. [13] studied the risk of developing contralateral infarcts in patients who had prior unilateral occipital infarcts. Of the 58 patients who were followed for an average of 39.6 months following their initial stroke, 13 developed an infarct on the contralateral side. The average time for this to occur was 4.2 months. A complete loss of vision occurred in six patients. Two or more of the following risk factors were strongly associated with developing bilateral infarcts: smoking, hyperlipidemia, cardiac disease, diabetes, and hypertension [13].

5.3. Symptoms

On occasion patients may be asymptomatic. It is not highly unusual for the clinician to discover a previous infarct resulting in a visual field defect that the patient was not aware of. In Trobe’s study of 92 cases of occipital lobe infarcts, 15% were asymptomatic at the time of discovery [11].

Many patients suffering from occipital lobe visual field defects will have vague complaints of not seeing well. They may have difficulty locating objects in space or have a tendency to bump into things. Patients are often unable to localize the field defect or to relate its position in space to the individual eye. In a study by Pessin et al. of 35 patients with ischemic occipital lobe infarcts, nearly one half presented with only visual symptoms [14].

Because of the role of the vertebrobasilar system in many ischemic occipital lobe lesions, patients may experience phenomena — transient or persistent — characteristic of its hypoperfusion at the onset of the stroke. Symptoms that have been reported include, but are not limited to photopsias, diplopia, bilateral ‘gray-outs’, and oscillopsia. In addition, systemic symptoms such as vertigo, hemiparesis, paresthesia, and nausea may occur. Ocular signs including nystagmus, gaze palsies, and skew deviation may also be
observed. Cerebral ischemia also may cause headache on the side of the lesion. This is thought to be due to ischemia to the dural structures in the area of the lesion that receive sensory innervation from the recurrent meningeal branch of the ophthalmic division of the trigeminal nerve. The pain may thus also be referred to the eye [15]. In Pessin’s study of patients with occipital lobe infarcts, 60% were found to have an accompanying headache [14].

While lesions due to vascular events usually result in symptoms of rapid onset, those resulting from neoplasms often have a gradual onset of symptoms [10]. When a tumor causes symptoms of rapid onset, they may be due to a hemorrhage in the tumor itself, thus mimicking a vascular event. The most common occipital lobe neoplasms are gliomas, meningiomas, and metastases. Because of its relatively small size, tumors are rarely confined to the occipital lobe. Thus, occipital tumors rarely cause exclusively visual defects. Primary occipital lobe tumors are likely to extend forward, invading other lobes, as the structures surrounding the lobe (skull, falx cerebri, tentorium cerebelli) are unyielding. Symptoms of occipital lobe tumors such as headache, nausea, hallucinations, seizures and vomiting are likely due to increased intracranial pressure and thus do not serve to localize the lesion [10]. Symptoms of cerebeller compression such as ataxia may result if the tumor extends downward [4].

5.4. Bilateral lesions

Cortical blindness is a term usually used to refer to the complete loss of vision due to bilateral occipital lobe lesions. They may occur either simultaneously or consecutively. There is no light perception and loss of reflex lid closures to bright light although normal pupillary reactions remain intact. The extraocular motilities are also normal. The primary etiology is hypoxia to the visual cortex. Trauma and tumors are less common causes. Studies involving visually evoked responses in cortically blind patients have yielded mixed results. The optokinetic nystagmus (OKN) response is usually absent [4].

Crossed quadrant hemianopsia occurs when there is a homonymous quadrantanopsia in diametrically opposed quadrants of the visual field [8]. It results from lesions of the superior and inferior banks of the calcarine sulcus, with each lesion occurring on a different side of the visual cortex [4]. It is sometimes referred to as a ‘checkerboard defect’.

Bilateral altitudinal defects may occur following trauma to both occipital lobes. Bullet wounds have been shown to lead to this field defect. Tumors and vascular events are rare etiologies [4].

Anton’s syndrome is used to describe the state of denial of (typically, cortical) blindness. Patients claim that they are sighted and are apt to form fabrications in support of this. The reason for this phenomenon is not known [4].

5.5. Visual recognition defects

Visual agnosia describes the inability to identify familiar objects by sight despite having normal visual acuity. These patients are able to identify objects by other senses. For example, they may not be able to name an object as a pen upon seeing it, but identify it immediately when it is placed in their hand. This phenomenon results from damage to the visual association cortex, as well as to the parietal and temporal lobes [10].

Prosopagnosia refers to the inability to recognize familiar faces. It usually results from bilateral occipital lobe lesions. It involves damage to the connections between the visual association and the temporal cortex. The latter serves memory, so these lesions prevent visual perceptions from being stored in memory [10].

Color agnosia is the ability to perceive color, but the
inability to give it a name. It often occurs in association with alexia (see below). There is a disruption in the connection between the visual association cortex and the language center [10].

Cerebral dyschromatopsia manifests as the ability to name colors, but the inability to perform color vision tests such as pseudoisochromatic plates. There is often a concurrent bilateral superior homonymous hemianopsia suggesting damage to the inferior portions of both occipital lobes. Cerebral dyschromatopsia may also be found in patients with prosopagnosia, since the area of the occipital-temporal junction has been damaged [9].

Optic ataxia is characterized by an inability to locate objects in visual space. These patients have difficulty when they attempt to reach for or point to a particular object. It may be limited to one side of the body or one hemifield. The area of damage lies between the occipital and parietal lobes [10].

Alexia is a term which describes the inability to read despite having normal vision. The pathway involved in reading is as follows: Information from the right hemifield is transmitted from the left occipital lobe to the left (or dominant) angular gyrus in the parietal lobe. The angular gyrus is concerned with the formation and interpretation of written words. Information from the left hemifield is transmitted from the right occipital lobe to the left angular gyrus across the splenium of the corpus callosum to the left angular gyrus. Damage to the left angular gyrus results in alexia with agraphia, a condition in which the patient can neither read nor write [9].

In the case of alexia without agraphia, the ability to write is preserved. Therefore the person can write but not read his own writing. It is caused by a lesion in the left occipital lobe which disrupts both the primary left visual cortex and the association fibers from the right occipital lobe that have crossed the splenium of the corpus callosum. Because of the lesion in the left visual cortex, a right hemianopsia results. Although there is vision in the left hemifield, visual information cannot reach the angular gyrus due to the lesion in the area of the crossing fibers. However, because the angular gyrus remains intact, the patient can still write. This condition is usually caused by an infarct in the proximal area of the left posterior cerebral artery [9].

5.6. Other defects

Palinopsia refers to a condition in which images remain perceived that are no longer in view. These images commonly occur only in the blind visual field. They are typically paroxysmal and tend to occur in association with other neuro-ophthalmologic symp-

toms. Parietal and occipital lobe lesions have been implicated [9]. Visual hallucinations, when the occipital lobe is involved, are typically unformed. The 'scintillating scotoma' seen in the classic migraine is an example. Other conditions such as occipital arteriovenous malformations, meningiomas, or incipient infarcts may lead to similar symptoms [9].

6. Clinical evaluation of patients with suspected visual pathway lesions

6.1. History

Since occipital lobe lesions frequently result in exclusively visual symptoms, the eye doctor is often the first health care provider that the patient encounters. Because of the potentially severe consequences of visual pathway lesions, an informed, comprehensive clinical work-up is crucial. In addition to the basic eye exam, emphasis must be placed on particular questions and tests that are instrumental in diagnosing visual pathway lesions.

In taking the history, patients should be asked if they have difficulty seeing in any part of their visual field, if they tend to bump into things, have accidents or have difficulty reaching for objects. Ocular symptoms include diplopia, scintillations, hallucinations, and transient visual loss. The patient should also be questioned regarding systemic symptoms such as headaches, nausea or vomiting, paralysis or paresthesia as well as any memory or recognition deficits. Attempts should be made to determine which eye and/or visual field is affected. Rapid vs. gradual onset should also be determined as this often aides in diagnosis.

A complete medical history should be elicited. An investigation regarding the patient’s cardiac history is important if an infarct is suspected. Special attention should also be paid to any history of transient ischemic attacks, head trauma, vascular occlusive disease, blood disorders or cancer [10]. Other risk factors such as smoking should also be noted.

6.2. Testing

In performing the fundus exam, the clinician should be careful to note any emboli in the retinal vessels. These are indicative of cardiac or carotid disease which also may account for a vascular-induced visual pathway lesion. Extraocular motilities and pupillary responses are always an important part of the work-up any time neurological disease is suspected. Automated static or Goldman kinetic visual field testing should be done. This often helps in localizing lesions.
Quadrantanopsias and hemianopsias can often be discovered through confrontation visual field testing. OKN testing may also be helpful in that asymmetric responses may be seen with parietal lobe lesions. Lesions to other parts of the visual pathway (provided they are limited to one side of the brain) generally do not affect OKN [10].

A CT scan or MRI should be ordered any time a lesion in the visual pathway is suspected. If there is any possibility of cerebral hemorrhage, a CT should be chosen as the initial imaging test over an MRI. This is because a CT is better at imaging an acute cerebral hemorrhage than an MRI and in differentiating a hemorrhage from an infarct. This is important because anticoagulant or platelet inhibiting drugs are often utilized in cases of infarcts, but are contraindicated in acute hemorrhagic conditions [8].

6.3. Treatment options

Bilateral yoked Fresnel prisms may be helpful to patients with hemianopsias. The base is placed in the direction of the visual field defect so that objects within the area of the scotoma are now imaged on the seeing hemifield. Thus, a patient with a right hemianopsia would have prisms placed base right OU. The amount given is typically between 15 and 30 prism diopters. Mirrors attached to the patient’s spectacle frames have also provided some success for patients with visual field defects. Devices to aide in reading such as line markers or typoscopes, as well as rotating the text 90° to avoid crossing the vertical midline have been used [4,8].

7. Case report

The following case serves to illustrate an example of an occipital lobe lesion in the context of the patient’s medical and ocular history.

A 57-year-old white male presented to the Hudson Valley Healthcare System Optometry Service with a 1-week history of binocular blurry vision coupled with a headache behind his right eye which continued back across the right side of his head. At the onset of the headache he experienced weakness in his left leg for 20 min. His ocular history was notable for a constant left exotropia and amblyopia. He had bilateral cataract extractions, right eye in 1989 with an anterior chamber IOL, left eye in 1990 with a posterior chamber IOL. He had a YAG capsulotomy to the left eye in 1994. His medical history was significant for rheumatic fever at age 8. He also reported being told by a previous doctor that he had suffered a mild heart attack at one point. The only medications he took were Zantac and Advil prn. He smoked two packs of cigarettes per day and was a long-term drinker before stopping 1 year ago.

Best corrected vision was 20/25 OD and 20/40 OS. Motilities, pupils and color vision were normal. Cover test showed a constant left XT of approx. 30 prism diopters at distance and near. Slit lamp exam was unremarkable except for the IOLs which were clear and centered. IOPs by applanation tonometry were 21 mmHg. in each eye. The C/D ratios were 0.30 in each eye with healthy fundi.

Confrontation visual fields revealed a left superior quadrant defect in each visual field. A 120-point screening visual field test was performed (Fig. 4) which showed a left superior homonymous quadrantopia. In the right eye the scotoma extended inferiorly below the horizontal meridian. There were also some superior nasal defects in the left eye.

The patient was referred for a CT scan of the head to rule out an occipital lobe lesion. The results showed an area of low attenuation with mild edema in the right occipital lobe extending slightly into the parietal lobe. This was consistent with an infarct of approx. 1 week origin (Fig. 5). The patient was referred to his primary care physician at the hospital where he was placed on an aspirin per day as well as decadron for 24 h. A referral was made for a neurological consultation. The neurologist discontinued the aspirin and placed the patient instead on Ticlopidine, an antiplatelet agent. The patient was placed on a 24-h Holter monitor and sent for an echocardiogram and duplex scan. All were normal. No other neurological findings were noted.

The patient was seen in the eye clinic 2 months later for follow-up. His headaches were no longer present. He also reported that his vision had improved, but still didn’t seem exactly the same as it was before the stroke. He had no visual recognition or neurological defects. A central 30-2 threshold visual field was performed at this visit (Fig. 6). The results were similar to the first visual field. However, the IOP was measured at 30 mmHg. OU at this exam. The elevated IOP raised several questions regarding the previous visual field results. Was the relative defect below the horizontal on the right nasal side an extension of the quadrantanopia or due to glaucoma? Was the superior nasal arcuate defect on the left side also due to glaucoma?

In order to investigate these possibilities, at the next visit a peripheral 60-4 visual field test was done (Fig. 7). Since early glaucoma typically does not affect the far periphery, but a lesion to the visual pathway can, it was felt this visual field would provide further pertinent information. If the visual field defect extended into this area then the defect below the horizontal would be more likely due to the stroke rather
than glaucoma. The relative defect did extend into the far periphery. The intraocular pressure was 27 mmHg in each eye at this visit. The patient's wife also informed us that the patient's mother had glaucoma.

Although the inferior nasal field defect in the right eye was probably due to the stroke, the nasal arcuate defect in the left eye, the patient's high intraocular pressures, and family history were all indications that the diagnosis of glaucoma was likely. The patient was placed on a trial of brimonidine 0.2% q12h OD.

7.1. Case discussion

This case highlights a number of points described previously. As is not infrequent, the eye doctor was the first practitioner seen in the case of a neurological event. The patient's symptoms were largely visual,
indicating occipital lobe involvement. However, he did have some initial contralateral weakness which was consistent with the finding that the infarct extended into the parietal lobe, which is concerned with motor movements. His headache was presumed due to ischemia to the dural structures innervated by the recurrent meningial branch of VI and referred to the eye, as noted above. The most likely vessel involved in the infarction was the right posterior cerebral artery. In addition to serving the visual cortex, this artery provides some of the blood supply to the ipsilateral dura. Occlusion of this vessel could also account for the patient's ocular and head pain.

As noted above, a homonymous quadrantanopsia is a not uncommon result of an occipital lobe infarct. In this case it was relatively congruous, indicating a lesion in the posterior region of the right inferior bank of the calcarine sulcus. The fact that it appears to have involved some of the parietal fibers may account for its extension into the inferior visual field in the right eye.

The patient was at risk for a stroke by several factors. A cardiac embolus is the most common source of an occipital lobe infarct. A study of patients with occipital lobe infarcts by Bruno et al. found that 42% were due to cardiac emboli, 67% were smokers, 8.5% had rheumatic valvular heart disease and 38% had an undetermined source. This patient had a history of rheumatic fever and was a long-time smoker. The CT scan ruled out a hemorrhagic stroke, so the patient was able to be placed on anticoagulants and encouraged to quit smoking.

An added twist to this case was the diagnosis of glaucoma. This illustrates the need for diligence when evaluating what may seem like relatively straightforward visual field results. In addition, it demonstrates the usefulness of performing different visual fields to sort out multiple defects along the visual pathway.

8. Summary

The occipital lobe is the most posterior of the four lobes comprising the cerebral hemispheres. Visual fibers from the LGN terminate in the striate, or primary visual cortex on the banks of the calcarine sulcus, with the adjacent association cortexes serving to integrate visual information with other areas of the brain. Striate cortical cells respond to stimulus characteristics such as shape, movement, speed, orientation, and direction. Lesions in the occipital lobe may be caused by a variety of systemic conditions including events such as infarction, hemorrhage, or neoplasm. Visual field and other perceptual defects may result, which are characterized by the location of the lesion. The understanding of occipital lobe anatomy and function is instrumental in arming the eye care practitioner with a knowledge base for which to provide comprehensive patient care.

References