PERMANENT VISUAL LOSS

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Introduction

The neurologist is not infrequently confronted with a patient complaining of visual loss. On some occasions, the patient has already been seen by an ophthalmologist (M.D.) or, more often, an optometrist (O.D.). However, it is not unusual for the neurologist to be the first health care provider to examine the patient. As an expert on the central nervous system, of which the eye is a part, the neurologist is expected to be able to evaluate a patient's complaint of visual loss and provide at least a cursory examination of the ocular apparatus and visual pathways.

Traditionally, the only part of the "eye" considered the domain of the neurologist is the optic nerve and its connections into the brain. At the very least, therefore, the neurologist should be able to recognize when visual loss is caused by an optic nerve problem *and when it is not*. The neurologist needs to know the classic defining features of an optic neuropathy. When these defining characteristics are not met, other abnormalities involving the ocular media and the retina should be considered. To appropriately localize the lesion within the eye and to generate a diagnosis, the neurologist must at least be aware of the other clinical entities that can cause visual loss, especially sudden visual loss, besides optic nerve damage. No one realistically expects the neurologist to be an expert on the eye. However, there are certain "red flags" on clinical evaluation which should make the neurologist think about these other diagnoses and seek prompt ophthalmologic referral. In some situations, it is the neurologist, with his or her particular knowledge of the brain and its adnexae, who makes the correct diagnosis and initiates the appropriate management, even when the problem is ocular and should have been the domain of the ophthalmologist.

Clinical Evaluation

History: Any patient with recent visual loss should be evaluated promptly, as some of the ocular causes of visual loss do indeed represent medical emergencies with implications for treatment. Obviously, as in any neurologic evaluation, a good history is of paramount importance. Assess whether there was a previous history of lazy eye, ocular trauma, or family history of visual loss. Does the patient have any systemic illnesses (e.g., diabetes mellitus, Marfan's syndrome), or neurologic diseases (e.g., multiple sclerosis, carotid stenosis) that might place them at risk for various causes of visual loss.

Patients with visual loss may complain of a decreased ability to see things at a distance or near, blurring of images, or loss of pieces of their visual field. To determine the location, nature, and ultimately the cause of the visual problem, the most important determination is whether visual loss is in one eye or both (not always such an easy question and you may have to specifically ask whether the patient has checked by covering each eye separately) (Table 1). Truly monocular visual loss signifies an abnormality in the eye itself or in the optic nerve anterior to the chiasm. The next most important issue is whether the visual loss is transient or permanent. The tempo of visual loss is also important, as is the presence or absence of associated symptoms, especially pain (Table 2).

Examination: The first step is to determine if there is indeed visual loss and rule out if it is simply refractive (ie., that the patient only needs a new set of spectacles). The best means of determining if there is visual loss is to measure the *visual acuity* of a patient. It is the only sensory function that can be truly quantitated with minimal equipment and, yet, paradoxically, it is one of the elements of a complete neurological examination that is often omitted, even when the patient has visual complaints. The best method is a well-illuminated distance chart at the appropriate distance, but a near card (also at the appropriate distance of 14 inches) can be used. The patient should wear his or her spectacle correction. If a near card is being used, reading glasses must be worn if the patient is in the presbyopic age group (beginning in the 40s). Test each eye individually and "push" the patient to read the smallest line they can. Unless you encourage them, many patients give up too soon and lead you to the false conclusion that they have subnormal vision. If the acuity is less than 20/20, you need to know if refractive error is responsible, since uncorrected visual acuity is of no interest to the neurologist. You are in no position to manipulate lenses, but there is an easy "short cut", the so-called "poor man's refraction", and that is through the use of a pinhole. If an uncorrected refractive error is the cause of subnormal visual acuity, the patient's vision should be improved by looking through a

pinhole (which can be placed over their glasses if necessary). *Visual fields*, at least by the confrontation method, should be performed on every patient. As with visual acuity measurements, each eye should be tested separately. Detection of visual stimuli within the central four quadrants of vision should be assessed, with special attention to respect of the vertical and horizontal meridia.

The *pupils* should be examined for size, shape, and reactivity to light and near. The most important part of the pupil evaluation of the patient with visual loss is the search for a so-called "Marcus Gunn" or "swinging flashlight" sign, evidence of a relative afferent pupillary defect. The basic features of a relative afferent pupillary defect and its detection are well known and require only brief review. Normally, when light is directed into either eye, both pupils react equally. The brighter the light source, the greater the degree of bilateral pupillary constriction. The amount of pupillary constriction to the same light source directed to either eye should be identical. If there is unilateral optic nerve (or retinal ganglion cell) dysfunction, the light signal received by the brainstem efferent centers will be relatively less than when the same light source is presented to the unaffected eye. Hence, both pupils will constrict less than when the involved eye is stimulated, and more when the normal eye is stimulated. Swinging the light source back and forth emphasizes this difference in transmission of the afferent signal, since both pupils will reset at the size appropriate for the amount of light transmitted by the illuminated optic nerve. When the light source swings from the affected eye to the unaffected eye, further constriction of both pupils will be demonstrated; when the light swings back to the involved eye, relative dilation of both pupils will occur. Even though the examiner may only look at the pupil upon which the light is shining, it is important to be aware that *both* pupils are changing size equally during this maneuver. By placing neutral density filters over the normal eye, the examiner can neutralize the relative afferent defect and quantitate its severity.

Ophthalmoscopic examination of the eye is an essential component of the neurologic examination. Actually *seeing* the fundus is a necessary first step, and not always an easy task in the office or at the bedside. Common obstacles to obtaining an adequate view of the fundus include too small a pupil, suboptimal use of available equipment, and media opacities (which may be the cause of visual loss in that patient).

Every neurologist should have easy access to dilating drops and should feel comfortable in their use. Typically, one instills a short-acting parasympathetic antagonist such as tropicamide 1% (Mydriacyl, Mydriafair, Ocu-Tropic, Tropicacyl) and a short-acting sympathomimetic such as phenylephrine 2.5% (AK-Dilate, Dilatair, Mydfrin, Neo-Synephrine, Ocu-Phrin). Dilating drop bottles all have red caps. Dilation occurs after initial instillation within 20-30 minutes and usually recovers within 4-6 hours. The drops may "sting" initially and the ensuing pupillary dilation and cyclopegia may cause photophobia, glare and accomodative difficulties.

In our practice, the only relative contraindication to dilation is the neurology or neurosurgery ICU patient with a fluctuating or precarious neurologic status. Communication with nursing and physician staff (as well as the patient and family) will help avoid any unnecessary panic when large, nonreactive pupils are discovered. Always dilate both pupils, as a unilateral, dilated, nonreactive pupil is particularly fear-provoking.

A frequently voiced concern is that of inducing "glaucoma" with the use of dilating drops. Most patients with "glaucoma" have chronic open angle glaucoma, a disease of chronic raised intraocular pressure that is not induced or worsened by the use of dilating drops. There is no contraindication to using dilating drops in patients with chronic open angle glaucoma. Dilating drops may cause *acute angle closure glaucoma* (an entity related to chronic open angle glaucoma only in as much as both conditions manifest elevated intraocular pressure). In patients with a narrow angle between the iris and the cornea (ie. a shallow anterior chamber), a dilating pupil may cause a blockage of the normal outflow of aqueous humor and an acute elevation of intraocular pressure. There may be pain, blurred vision, halos around lights, nausea, redness of the eye, and clouding of the cornea. It should be treated with topical pilocarpine (1-2%) to constrict the pupil, a systemic carbonic anhydrase inhibitor, and an intravenous osmotic agent, until the definitive therapy of laser or surgical iridectomy can be performed.

The direct ophthalmoscope has many features useful in the observation and interpretation of the ocular media and fundus. Focusing on the more anterior portions of the eye (ie., standing back from the patient and using more plus diopter settings -- the black numbers on the ophthalmoscope dial) will help reveal media abnormalities that may be the cause of visual loss, may relate to the underlying problem, or may preclude an adequate fundus examination. The direct ophthalmoscope will give an excellent two-dimensional view of the posterior pole, specifically the optic disc, the macular region, and the vessels of the major arcades. We are a very "disc-o-centric" specialty, but every neurologist should get into the habit of moving temporally to also get a good look at the macula. The single most common mistake in the use of the direct ophthalmoscope when viewing the fundus is not getting close enough to the ophthalmoscope and to the patient. Examiner glasses can be removed and the examiner's hand holding the ophthalmoscope should be touching the patient's cheek. By changing the focus in and out, one can estimate the relative depth and elevation of fundus features, such as the optic cup or the optic disc. The green light (red-free filter)

can be used to see details of the nerve fiber layer. If the examination must be performed in the undilated patient, it is particularly important to have the ophthalmoscope as close to the eye as possible, have the patient fixate on a distant target (to minimize the near triad and further miosis), and dim the room lights.

Other techniques of ocular and funduscopic examination are not readily available to the neurologist and referral to an ophthalmologist may be appropriate. Slit lamp biomicroscopy provides a three-dimensional, cross-sectional, magnified view of the cornea, anterior chamber, lens and vitreous. Abnormalities of the media are readily clarified and localized in depth. Slit lamp examination with a 90 diopter, 60 diopter or Ruby lens allows for a three-dimensional view of the posterior pole. Similarly, indirect ophthalmoscopy with a 20 diopter lens gives a three-dimensional view of the fundus, with a wider view including the peripheral retina. Fundus photography may allow for more leisurely study of optic disc and retina details. Fluorescein angiography has the added advantage of providing information regarding the functional vascular status of the retina. Optical coherence tomography (OCT) has the advantage of providing two-dimensional cross-sectional anatomic views of the layers of the retina, including the nerve fiber layer.

Recognizing an Optic Neuropathy

It has been estimated that the optic nerves contain 38% of all the axons entering or leaving the brain. Not surprisingly, therefore, neurologists often find themselves confronted by patients with complaints of visual impairment. Optic neuropathies account for most instances of neurogenic visual loss.

The classic features of an optic neuropathy are:

1) central visual loss
2) clear view through to the optic nerve
3) relative afferent pupillary defect
4) swollen or pale appearing optic nerve

If all these features are met, there is little question as to localization of the lesion. Of course, it is not always so clear-cut. Some optic neuropathies may spare central visual acuity. In up to 50% of patients with nonarteritic anterior ischemic optic neuropathy, for example, visual acuity is good despite altitudinal visual field loss. In other acute optic neuropathies, such as the majority of cases of retrobulbar idiopathic optic neuritis, the optic nerve appears normal for at least 4 to 6 weeks before optic nerve head pallor ensues. However, in both these examples, the presence of other features, especially a relative afferent pupillary defect, facilitates recognition of the optic nerve as the locus of pathology. A more difficult situation occurs when the optic neuropathy is bilateral and symmetrical, and, therefore, a *relative* afferent pupillary defect may not be present.

Clinical Entities That Are Not Optic Neuropathies

There are many causes of visual loss that are not optic neuropathies with which the neurologist should be familiar. They will be briefly reviewed here and categorized by how many of the criteria for the classic diagnosis of an optic neuropathy they meet.

Ocular Media Abnormalities: Clinical entities that cause visual loss and do not allow a clear view back to the fundus are almost always problems with the ocular media: the cornea, the anterior chamber, the lens, or the vitreous. Many of these problems can be recognized with penlight observation or use of the direct ophthalmoscope focused on the more anterior eye (ie. with more plus diopters dialed in). Corneal surface changes, scarring, edema, or structural abnormalities (such as keratoconus) will make the view in difficult. A hyphema (blood in the anterior chamber) may be visible to the naked eye. Cataracts will cause blurring, darkening, or an orange-brown discoloration of the fundus details, a glaring reflection of the ophthalmoscope's light, or complete obscuration of view. Vitreous hemorrhage, inflammation (uveitis), or debris may also completely obscure or blur the view of the optic nerve and retina.

Angle closure glaucoma (see above) is a sudden rise in intraocular pressure usually occurring in patients anatomically predisposed for a narrow drainage angle. The patient typically has nausea as part of the vagal reflex, a painful eye from the elevated pressure, a red eye from increased vascular congestion, a large and non-reactive pupil from ischemia to the iris, and subnormal vision from corneal haze. Corneal haze can be recognized with the direct ophthalmoscope looking for a reduction in the normal luster of the corneal surface, especially as compared to the fellow eye. The presence of a fixed dilated pupil and pain may mislead the physician to conclude that there is aneurysmal compression of the third nerve. However, the other ocular findings and the absence of ptosis or motility disturbances should lead to appropriate diagnosis and management. On rare occasions, angle closure may be

intermittent, thereby causing transient blurring of vision (usually with associated eye pain), mimicking so-called amaurosis fugax. If left untreated, optic nerve damage may ensue, contributing to visual loss and resulting in a relative afferent pupillary defect, potentially leading to further confusion in diagnosis. However, by the time optic nerve injury has occurred, the ocular media findings should be quite apparent.

Vitreous hemorrhage most commonly occurs in the diabetic with diabetic retinopathy and neovascularization, but also occurs after trauma and after subarachnoid hemorrhage (Terson's syndrome). Smaller vitreal hemorrhages are usually described as large "floaters" which move when the eye is moved. Larger vitreal hemorrhages persistently disrupt central vision. Examination will reveal a diminished red reflex and the view of the fundus will be obscured on direct ophthalmoscopy.

Abnormalities of the Macula and Outer Retina: The most common problem faced by the neurologist in the differential diagnosis of visual loss is deciding whether the visual loss is the result of a lesion of the optic nerve or a lesion of the macula. Both optic nerve lesions and macular lesions can reduce central acuity and both can cause central scotomas on visual fields. Both may also affect color vision, although the amount of color vision deficit for any given visual acuity deficit is usually much greater for an optic neuropathy than a maculopathy. Maculopathies are rarely painful, as opposed to some causes of optic neuropathy, especially idiopathic optic neuritis in which pain, particularly pain exacerbated by eye movement, is a common feature. Classically, maculopathies cause visual distortions and vision is slow to recover after bright light, features not usually found among optic neuropathies. If the macula definitely looks abnormal, the answer is clear, but some retinal lesions are quite subtle and difficult to detect. However, it is the *absence of the relative afferent pupillary defect* that should lead the examiner to suspect a problem removed from the optic nerve.

Central serous retinopathy (CSR) is a relatively common cause of visual loss that occurs when serous fluid accumulates in the subretinal space underneath the macula causing a relative detachment of the layers of the retina. It makes the macula look like a blister. Presumably fluid has leaked from the choroid through a break in the retinal pigment epithelium. It occurs preferentially in males (male to female ratio of 10:1) in their fourth and fifth decades of life, especially so-called type-A personalities under increased stress. The symptoms are fairly sudden in onset and consist of painless blurred and dim central vision and usually metamorphopsia. Most eyes improve spontaneously within one to six months. Fifty percent of patients experience recurrences. The clinical picture may be mistaken for optic neuritis, but the male gender, the metamorphopsia, the lack of pain, and the usual absence of a relative afferent pupillary defect should raise suspicion for CSR.

Macular degeneration is typically a progressive, bilateral acquired degeneration of the outer retina in the region of the macula. With age, some patients develop chronic degenerative changes, so-called age-related macular degeneration. An early sign of the process is the appearance of yellow-white deposits with irregular borders known as drusen (a completely different entity from the drusen found in optic nerves). These drusen result from thickening of Bruch's membrane or from the retinal pigment epithelial cells' inability to get rid of lipofuscin and other waste products. Hypo- or hyperpigmentation of the retinal pigment epithelium may also be present. There are many other causes of maculopathy, many hereditary, occasionally toxic, almost always bilateral. A specific diagnosis depends on the location of the deposits, the results of fluorescein angiography, the age of the patient and the family history. Most patients with severe visual loss from macular degenerations develop choroidal neovascularization which places them at increased risk for subretinal hemorrhage, subretinal exudate and macular edema.

A *macular hole* is just that. Idiopathic macular holes and cysts occur primarily in women in the sixth through eighth decades of life, probably as a result of progressive vitreoretinal traction. A fully formed hole is visible as a sharply delineated defect in the middle of the macula. The other eye may become similarly involved in up to 30% of patients.

Acquired enlargement of the physiologic blind spot, both symptomatic and asymptomatic, is usually the result of swelling of the optic nerve head. Occasionally, however, blind spot enlargement may occur with a normal appearing optic nerve and signify peripapillary outer retinal dysfunction, the so-called *acute idiopathic blind spot enlargement* (AIBSE) syndrome. AIBSE is a syndrome characterized by the sudden onset of a monocular temporal blind area centered on the physiologic blind spot, often with associated photopsias in the scotomatous field. Women are affected at least twice as frequently as men, and most patients are between the ages of 20 and 40 years. Visual acuity and color vision are typically spared and there may or may not be a relative afferent pupillary defect (present less than 50% of the time). Ophthalmoscopic and fluoroangiographic findings are often normal or consist of nonspecific pigmentary changes or subtle grayish discoloration of the peripapillary retina. The electroretinogram, especially a multifocal electroretinogram is frequently abnormal. AIBSE generally resolves over several weeks or months but occasionally will recur in the same or opposite eye.

Retinal Detachment and Retinovascular Occlusion: The fibers that form the optic nerve originate in the ganglion cells, one of the most inner layers of the retina. The axons of the ganglion cells lie superficial to the ganglion cell layer and are designated as the nerve fiber layer prior to their coalescence into the optic nerve. Damage to the ganglion cell body or the nerve fiber layer is tantamount to damage to the optic nerve; ie., there will be visual loss, a relative afferent pupillary defect if unilateral, and ultimately optic nerve atrophy. Since the central retinal arterial and venous circulations subserve the inner layers of the retina (including the ganglion cell and nerve fiber layers), retinal vascular occlusive events will result in inner retinal damage, visual loss, and a relative afferent pupillary defect.

Acute vascular events involving the inner retina have a dramatic and distinct funduscopic appearance, allowing for immediate correct diagnosis. When a retinal artery becomes occluded the normally transparent retina supplied by that artery becomes white and edematous. There may be segmentation of the arteriolar blood column (boxcarring) and a reduction of the arteriolar lumen. A visible embolus is present in 10-20% of patients with a central retinal artery occlusion and 60-70% of patients with a branch retinal artery occlusion.

Occlusion of the central retinal artery (CRAO) is a medical emergency. Central and severe visual loss typically occurs painlessly and without warning. Occasionally, patients may experience episodes of transient monocular blindness prior to complete visual loss, especially in those cases related to ipsilateral carotid disease or giant cell arteritis. The classic funduscopic appearance of CRAO is central retinal whitening surrounding a cherry red spot in the macular region. This occurs because the retina is very thin at the level of the macula and the underlying choroidal circulation shows distinctly red against the white, swollen, ischemic surrounding overlying retina. In the acute phase, the optic nerve head appears normal since its blood supply is not the central retinal artery, but rather branches from the ciliary circulation. Chronically, however, with death of the inner retinal layers, including the ganglion cell layer and the nerve fiber layer, the optic nerve becomes pale. The retinal vessels ultimately become narrowed and sheathed. Most cases of CRAO are caused by an embolus obstructing either the central retinal artery or the ophthalmic artery. The standard of care acutely is to initiate a host of measures designed to improve circulation to the retina, including ocular massage to dislodge the embolus, breathing a mixture of 95% oxygen and 5% carbon dioxide to promote vasodilation, systemic administration of acetazolamide to reduce intraocular pressure, and anterior chamber paracentesis to dramatically reduce intraocular pressure. Unfortunately, only rarely is vision restored. In specialized centers, intra-arterial and intravenous thrombolysis has been attempted with anecdotal success.

In an *ophthalmic artery occlusion*, both the central retinal artery circulation and the ciliary circulation are compromised, resulting in ischemia to the inner and outer retina and the optic nerve. The funduscopic appearance is that of both retinal and optic nerve swelling, with no cherry red spot (the choroidal circulation is also compromised). In *branch retinal artery occlusions*, the area of retinal whitening and edema, as well as the vision and visual field loss, are determined by the location and amount of retina subserved by that particular vessel.

Occlusion of the central retinal vein (CRVO) produces a dramatic funduscopic appearance, although visual loss may be minimal. The veins are markedly dilated with diffuse hemorrhage involving the superficial and deep layers of the retina. There are usually cotton wool spots (small infarctions of the nerve fiber layer) and swelling of the optic nerve head. Descriptions of CRVO include "blood and thunder" and "pizza pie". Underlying hypertension and hypercoaguability need to be considered in these patients.

A *retinal detachment* occurs when the connections between the overlying retina and the underlying retinal pigment epithelium (and the nourishing choroidal blood supply) are severed. If the detachment involves the retina centrally, this results in poor central vision and a relative afferent pupillary defect. Ophthalmoscopy will reveal the detached retina ballooning forward or simply a red reflex with an obscured view of the fundus. Myopic patients are especially vulnerable to retinal detachments, as are patients who have recently had intraocular surgery or ocular trauma, or who have a family history of retinal detachment. Detachments are often heralded by photopsias (flashes of light). If the detachment originates peripherally, there may first be a period of time when the patient notices a veil or shadow over a portion of the visual field prior to central visual involvement. Prompt evaluation by an ophthalmologist may preempt further detachment and allow for the appropriate reattachment surgery.

Retinal Degenerations: As noted above in the section on recognizing an optic neuropathy, when optic neuropathies are bilateral and symmetric, all criteria for diagnosis of optic neuropathy may be met except for the presence of a *relative* afferent pupillary defect. These primary bilateral optic neuropathies may be difficult to distinguish from a group of retinal disorders, commonly designated retinal degenerations or dystrophies, in which secondary optic disc pallor occurs bilaterally. Retinal disorders may “masquerade” as bilateral optic neuropathies, including vitamin A deficiency retinopathies, toxic retinopathies, and carcinoma-associated and melanoma-associated paraneoplastic retinopathies. Optic nerve pallor may be present, but there is usually also some degree of retinal arterial attenuation. The cone dystrophies in particular are characterized by bilateral loss of central vision, profound

color vision deficits, and frequently a relatively normal appearing fundus examination except for bilateral temporal disc pallor. The cone dystrophies are commonly sporadic, although inherited forms have been reported. Visual acuities typically deteriorate to the 20/200 to 20/400 level. Clues to suggest retinal cone dysfunction include the profound dyschromatopsia, photophobia and an inability to see as well in bright as in dim light (hemeralopia), retinal arterial attenuation, and, eventually, changes in the appearance of the macula, often resembling a bull's-eye. An electroretinogram is diagnostic in these disorders.

It is an optic neuropathy!

Once you have determined by history and examination that a patient does indeed have an optic neuropathy, then a differential diagnosis as to the underlying cause of that optic neuropathy must follow. Essentially all categories of disease processes must be considered (Table 3). Unfortunately, the optic nerve has a very limited repertoire of how it can express itself when it is damaged or perturbed. If the pathology involves the optic nerve head, you may see swelling of the nerve, so-called disc edema. If the locus of the pathology is behind the eyeball, termed retro-bulbar, then it is likely that the optic nerve will appear normal at the time of acute visual loss. Ultimately, after 4-6 weeks, pallor will ensue if permanent damage has occurred. Important clues in establishing the etiology of an optic neuropathy include the age of the patient, the tempo of onset and progression of visual loss, the presence or absence of pain, the presence or absence of bilateral involvement, the level of visual acuity, the pattern of visual field loss, the appearance of the optic nerve head, and the presence or absence of associated signs.

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Table 1: Neuro-ophthalmologic evaluation of the patient complaining of visual loss

Neuro-ophthalmic history:		
	Loss of vision:	- Monocular or binocular ?
		- Transient or permanent ?
		- Tempo of onset
		- Pattern of fluctuation
		- Associated symptoms
	Diplopia ?	
	Oscillopsia ?	
	Proptosis ?	
	Pain ?	
Neuro-ophthalmic examination:		
	Afferent system:	- Visual acuity
		- Color vision
		- Visual fields
		- Slit lamp examination (or at least look at the eye)
		- Funduscopic examination (after pupillary dilation)
	Efferent system:	- Ductions and versions
		- Alternate cover test
		- Nystagmus
	Pupils:	- Size (in dark and in light)
		- Relative afferent pupillary defect ?
		- Reactivity to light and near
	Lids and orbits:	- Observation
		- Ptosis ?
		- Exophthalmometry
		- Retropulsion
		- Bruits
	Neurological examination	

Table 2: Causes of monocular visual loss (permanent or transient) associated with pain

Vascular visual loss from internal carotid artery dissection
Carotid occlusion
Ocular ischemic syndrome
Giant cell arteritis
Optic neuritis
Angle closure glaucoma
Ocular trauma
Migraine (retinal migraine)

Table 3: Categories of disease processes that can cause an optic neuropathy

Inflammatory
Infectious
Non-infectious
Vascular
Compressive/Infiltrative
Neoplastic
Non-neoplastic
Hereditary
Toxic/Metabolic
Traumatic
Mechanical
Elevated intracranial pressure
Elevated intraocular pressure