THERE ARE MANY CAUSES OF PAINLESS LOSS of vision, however, it is difficult to differentiate between causes that need immediate referral to an ophthalmologist, those which can be delayed referral or those which need referral to other specialists. This article will describe some of the most common causes of painless loss of vision and the decision-making processes surrounding referral, and assist A&E nurses in deciding or advising upon effective management. The article will use the classification of loss of vision illustrated in Figure 1 in order to group together and describe some of its many causes.

HISTORY
In describing a loss of vision, patients will often use terms that are vague and non-specific. It is up to the nursing or medical staff to determine what has actually happened to the patient and what has prompted him or her to attend the A&E.

From the point of view of vision loss, it is important to ascertain the parameters of the problem.

- Are there patches or areas of actual vision loss or is the vision blurred?
- Was it sudden or gradual loss of vision? If sudden, is it possible that it has been there for a while but only just noticed? For example, did the patient notice the loss of vision when he or she covered an eye – if that was the case, it may have been present for some considerable time? If the loss was gradual, over what period of time has it occurred (days, weeks or even months)?
- Does the loss involve some or all of the vision? Are there sectors of the field of vision that are missing?
- Is the loss worse in the middle of what the patient sees or around the edges of the field?
- Was the loss transient – has it come back now or is it recovering. How long was vision affected for or does it seem to be permanent?
- Is the vision now getting better, worse, or is it about the same?
- Are there any other symptoms that the patient is experiencing? Often the patient may not consider other symptoms as the eye problem is the issue that worries them. If questioned, however, other symptoms may be ascertained which the patient does not readily associate with the eye problem such as headache, weakness, or pain elsewhere.

MONOCULAR VERSUS BINOCULAR LOSS OF VISION
Ocular pathology, or optic nerve problems will cause monocular loss of vision. A problem at, or posterior to, the optic chiasma in the brain will cause binocular loss of vision. It is most unusual for a patient to suffer from bilateral simultaneous eye disease and, should this occur, the whole field of both eyes will be affected rather than half of field, which is usual in neurological eye problems. The only exception to this occurs in the case of bilateral blurring of vision, which has appeared over a small number of days. This is characteristic of papilloedema.

A generalisation, but one that works in practice, is that if a patient complains of binocular loss of vision, the problem is likely to be of neurological rather than ophthalmic origin and a neurological opinion should be sought.
One of the most common causes of transient, bilateral loss of vision is classic migraine. The patient is likely to complain of the loss of large parts of the visual field. This aura usually lasts for 20 - 30 minutes and then resolves and is followed by a severe headache. In a first episode of migraine, the patient may be frightened by the visual symptoms and may not associate the headache with the loss of vision. Another migraine type is known as acephalgic migraine. The patient experiences the aura but does not go on to develop the headache. It may be difficult to convince the patient that these symptoms do not constitute an eye problem.

Homonymous hemianopia (hemianopia – loss of half of the visual field, homonymous – on the same side) (Fig. 2).

Patients may complain they are unaware of things approaching from the side of the field defect. They may also have trouble with reading, as they may not be able to follow a line of print. Visual acuity may be only mildly reduced in each eye, as part of the macular function on each side is likely to be intact. Distance visual acuity testing may demonstrate that the patient is unable to see the letters on the Snellen chart on the side of the field defect.

The most common areas of damage are in the optic radiation and the occipital cortex. The hemianopia may be incomplete and temporal lobe lesions cause predominantly upper field loss. Causes of homonymous hemianopia include vascular lesions such as embolus or haemorrhage, tumours, and inflammatory lesions in these specific areas of the brain. This type of field defect may accompany obvious systemic symptoms such as hemiparesis or hemiplegia. The patient should be referred to a neurologist for further assessment.

Bitemporal hemianopia (Loss of the field of vision on the temporal side in each eye) (Fig. 3). Bitemporal field loss usually indicates a lesion in or around the optic chiasma. Most chiasmal lesions result from compression by tumours arising from structures around the chiasma such as pituitary adenoma, meningioma, craniopharyngiomas or aneurysms (Cheng et al 1997). The patient may complain of blurring of the temporal field or of difficulty undertaking tasks such as driving. Cranial nerve palsies may also occur due to compression by a tumour and the patient should be asked about symptoms of double vision. Evaluation by a neurologist is the most appropriate course of action for the patient.

MONOCULAR LOSS OF VISION

Profound loss of vision This is characterised by complete or severely diminished vision affecting the whole of the visual field. This may occur suddenly or gradually over a period of days. Sudden, profound loss of vision suggests a vascular cause and the most likely of these are central retinal artery occlusion and vitreous haemorrhage.

Vitreous haemorrhage is the most likely cause if there is an associated history of diabetes. The patient may not be aware of eye changes related to the diabetes, especially if no regular eye screening takes place. The patient may be aware of the haemorrhage taking place and may describe a cloud of floaters (the first blood) which becomes more dense over a short period, resulting in a profound loss of vision. Any attempt by the clinician to visualise the back of the eye will be unsuccessful due to the blood in the vitreous cavity. The patient should be referred to an ophthalmologist although it is unlikely that (laser) treatment will take place until the vitreous haemorrhage has cleared sufficiently for the retina to be visualised.

In central retinal artery occlusion, the patient may describe the vision disappearing ‘like someone switching the light off’. The loss may be absolute and is, at best, likely to be ‘count fingers’ or less. Some patients retain a degree of central vision due to the presence of a cilioretinal artery, an anatomical anomaly. The retina is likely to be pale due to swelling within the retina and the foveal...
(macular) area is seen as a ‘cherry red spot’ as the retina is very thin and the choroid is seen underneath the retina, without swelling to mask the colour. An embolus in the central retinal artery may be seen. This condition is an ophthalmic emergency and while investigations as to the cause of the condition are necessary. (These include urgent ESR as giant cell arteritis may be a factor, lipid profiles, full blood count – to rule out coagulopathies and ultrasound scan of the coronary arteries and echocardiography – in order to identify the site of the embolus.) Immediate treatment must start in A&E, even before the patient sees an ophthalmologist.

Treatment is aimed at allowing increased perfusion of the retina by reducing the intraocular pressure. It includes the administration of intravenous acetazolamide 500mg to reduce intraocular pressure, ocular massage, to encourage the outflow of aqueous and often in ophthalmic units, the patient is asked to rebreathe exhaled air by breathing into a paper bag. This increases the carbon dioxide concentration in the body, thus dilating blood vessels and possibly allowing the embolus to move further into the retinal circulation. If this occurs, a sector of visual loss, rather than profound loss may be a good outcome for the patient. An anoxic retina is irreversibly damaged in 90 minutes (Pavan-Langston 1996) and for patients who wake up with this condition, or for patients who do not attend A&E immediately, the visual outcome is poor.

In some conditions, vision loss may become progressively profound over the whole field of vision over a number of days. The most likely cause of this is optic neuritis, described under blurring of vision, which is its more likely presentation. Profound loss of vision which appears gradually, starting with a segment of the visual field and enlarging to cover the whole of it, is likely to be due to a retinal detachment. This is described under segmental loss of vision.

**SEGMENTAL LOSS OF VISION**

The most likely causes of the loss of an area of the visual field in one eye are vascular causes such as occlusions of branches of the retinal artery or vein (branch retinal artery or vein occlusions) or retinal detachments. If the onset is sudden and stays the same, the cause is likely to be vascular. If the area of visual loss changes over time, the cause is likely to be a retinal detachment.

**Branch retinal artery and vein occlusions**

These may be seen with an ophthalmoscope; the branch artery occlusion will lead to a segment of retina being paler than the rest. All the vessels will appear in the correct location and an embolus may be seen in one of the vessels. There may be multiple retinal haemorrhages seen if the cause of the loss of vision is a branch retinal vein occlusion. The haemorrhages will be in the area of the retina which is served by the blocked vein. Retinal oedema may be seen and an occlusion may be visible. There is no immediate treatment for either condition, although follow up by an ophthalmologist will be necessary. Local ophthalmologists will give advice about the appropriate timing of outpatient appointments.

**RETINAL DETACHMENT**

Spontaneous retinal detachment affects one in 10,000 of the population each year (Kanski 1990). It is more common in males and in short-sighted (myopic) eyes (Pavan-Langston 1996). It usually occurs due to collapse of the vitreous gel in middle age causing traction on a weak area of retina and causing a hole to form in it. Other causes include traction on the retina in conditions where fibrovascular tissue has developed between the retina and vitreous such as in diabetic retinopathy and sub-retinal disorders such as tumours or inflammation that allow passage of fluid between the retina which pushes it off its basement membrane.

Symptoms characteristic of retinal detachment include:

- **FLASHERS** – due to traction on the retina or to areas of the retina moving. The only way that the brain can interpret movement of the retina is in terms of light so as the retina moves, the brain interprets and the pa-
The patient "sees" flashes of light.

**Floaters** – the appearance of a large circular floater is due to the detachment of the vitreous gel from its ring shaped attachment at the optic disc. A shower of tiny floaters is due to haemorrhage into the vitreous as a small retinal blood vessel is involved in the retinal tear.

A sector of loss of vision may be noticed which tends to enlarge over a period of hours or days. The patient may complain of seeing a "shadow" which tends to move, or a curtain descending over the eye. This is due to an area of retina which is detached and may be enlarging or moving within the patient’s field of vision.

Central vision may be lost due to macular detachment.

The detached retina will appear grey and may seem slightly wrinkled. Patients with retinal detachment need an urgent ophthalmic opinion. If central vision is present, the macula is still attached and it is likely that surgery will be immediate in order to preserve this situation. If the macula has been detached for some time, it is likely that surgical delay of a few days will not adversely affect the outcome for vision as macular function is not likely to be restored (Cheng et al 1997).

**Loss of central vision** Common causes of loss of central vision include age related macular degeneration (ARMD), optic neuritis, central serous retinopathy and macular burns.

ARMD refers to a gradual degeneration of the macula. It is the most common cause of visual loss in the over 75s and affects around 20 per cent of individuals. There is usually a very gradual loss of central vision. The patient may have noticed that they have to use a bright light to read by and that words fade after a few minutes. Although this is not an acute problem, elderly patients may present in A&E because they have reached a point where they can no longer manage the problems alone. Referral to an ophthalmologist is essential although there is little effective treatment for this condition. Patients retain navigating vision - their peripheral visual field is not affected.

**Optic neuritis** refers to inflammation of the optic nerve. Episodes are usually monocular, although they may be binocular. It is most common in adults between the ages of 20 and 40 and is more common in females. Optic neuritis is the presenting feature in 25 per cent of patients with multiple sclerosis (MS) and occurs in 70 per cent of established cases. Many patients with idiopathic optic neuritis will go on to develop MS. Various texts suggest figures of 50 per cent (Pavan-Langston), 60 per cent (Ootrey et al 1998) and "most" (Cheng et al 1997)!

The patient is likely to present with loss of central vision, which may progress to a generalised loss of vision and can become severe. It is maximal after approximately two weeks and tends to recover after 4-6 weeks. Over a period of months, most patients recover 6/12 vision or better. Other symptoms include, pain around or behind the eye which is worse on ocular movement due to the inflamed optic nerve moving as the eye moves. Perception of colour in the affected eye is...
likely to be reduced. This can be tested using the top of a red pen and comparing the perception of red in each eye. The pupil reactions will be abnormal and the optic nerve head may appear normal or may be swollen. Referral to a neurologist or neuro-ophthalmologist for further assessment and possible treatment is the preferred course of action. A possible diagnosis of MS should not be discussed in A&E as, even with a confirmed diagnosis of optic neuritis, MS is still only a possibility and the A&E department has neither the time nor the resources for the counselling which may be necessary in this situation.

Central serous retinopathy (CSR) may occur in young adult males and has an unknown cause. Symptoms usually include a unilateral blurring of central vision and a generalised darkening of the visual field with some distortion. Visual acuity is usually only mildly reduced. It is rare for it to be less than 6/18 but it may reduce to 6/60 (Cheng et al 1997). Although referral to an ophthalmologist is necessary, most episodes of CSR resolve within three-six months. Treatment is not usually indicated though laser treatment has been shown to assist resolution in some cases where the CSR episode is persisting.

Macular burns may be caused by MIG welding equipment. The light produced is high intensity white light rather than ultra-violet light as in most other welding equipment. The eye transmits this light rather than absorbing it and it can cause macular burns, which will result in some loss of central vision. The patient may notice a black mark in the centre of their vision, which stays in the same place when they move their eye. Macular burns may be caused by the patient looking at the sun without adequate eye protection. It may be suggested that this is an unlikely event but, as we are to experience a very rare, total eclipse of the sun in August 1999, it is a phenomenon which nurses must start to consider. Even in areas in the zone of totality, such as Cornwall and Devon, as the sun starts to emerge again, people without adequate eye protection may easily suffer eye injury. Previous solar eclipses have resulted in a number of patients with macular burns, which involve permanent loss of vision.

**BLURRING OF VISION**

Blurring of vision may be due to problems anywhere from the cornea to the optic nerve and the brain. Many patients will have problems in differentiating between generalised blurring and loss of central vision; careful questioning is needed to obtain a full picture of the problem. Vitreous haemorrhage or vascular occlusions may cause sudden onset blurring of vision. These have been dealt with elsewhere. Other causes of blurring of vision tend to develop more gradually and may include CSR and optic neuritis. Again, these have been dealt with elsewhere. Patients with papilloedema often present with blurring of vision. This may be worse in one eye and may be exacerbated by, for example, standing up (Cheng 1997). Concurrent symptoms may be ignored by the patient in favour of the eye problem. Patients with bilateral swollen optic discs need urgent neurological referral.

Patients occasionally present with refractive errors, which they have not noticed previously. It may be they have covered one eye and noticed that their vision in the remaining eye is not good. This may provoke much anxiety and encourage them to self-re-
fer to A&E. Visual acuity should be checked using pinholes to negate the effect of any refractive error. If vision improves dramatically with pinholes, a significant proportion of the blurring is likely to be due to refractive error and, in the absence of any other findings, the patient may be referred to an optometrist.

Opacities in any of the clear structures of the eye will result in blurring of vision as less light is allowed to reach the retina. The most common opacity is due to cataract. Again, the patient may have noticed the loss of vision by closing one eye, or worry about their symptoms may have prompted self referral. Cataract causes glare and reduction in vision. There is likely to be reduction of the red reflex and lens opacities may be seen on examination with a slit lamp or, if severe, with a pen torch. Cataract is not an urgent condition and it may be most appropriate, in the absence of any other cause for the loss of vision to reassure the patient that cataract does not progress very quickly and to refer them back to their general practitioner for referral to an ophthalmologist. If the lens opacity had occurred after trauma, or is in a younger person, more urgent referral to an ophthalmologist should be considered.

Corneal problems resulting in blurring of vision such as opacities or irregularities should be referred to an ophthalmologist as a matter of some urgency.

Transient loss of vision

Transient loss of vision may be due to a vast range of conditions. A number of these such as papilloedema and migraine have been dealt with earlier. Other, common causes of transient loss include carotid artery disease and giant cell arteritis. Intermittent angle closure glaucoma is a rare but possible cause of these symptoms. Retinal emboli from carotid artery disease often produce transient visual loss known as amaurosis fugax. This may be described as a curtain being lowered and then lifting over the vision. It is likely to last seconds to minutes rather than hours. It may be a sign of impending cerebro vascular accident and therefore, cardio-vascular investigations are appropriate. Turning the head may precipitate an attack and this is characteristic of carotid artery disease.

Patients with giant cell arteritis often complain of headache and tenderness over the scalp. This may be obvious when they comb their hair. They may also notice jaw claudication and pain on chewing. An urgent ESR is indicated and may be more than 80 mm. Urgent referral is required.

Patients may present with symptoms of pain in and around the eye and blurring of vision which begins, usually at night, when the pupil becomes larger due to the reduced light levels and may last a number of hours. It may have resolved by the time the patient attends A&E. If the anterior chamber appears shallow, and the symptoms are as described, intermittent angle closure glaucoma may be suspected and urgent referral to an ophthalmologist is required so that the eye can be evaluated and if necessary, prophylactic laser treatment can be undertaken to prevent further attacks.

CONCLUSION

The patient presenting to A&E with loss of vision may cause some diagnostic and management problems. It is hoped this article has clarified some of the issues surrounding this area of practice, enabling A&E nurses to continue to facilitate the best possible care for this client group.

REFERENCES


