ACUTE VISUAL DISTURBANCE

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Symptoms and signs

In assessing a patient who has experienced an acute disturbance of vision in an uninfamed eye an accurate history is crucial, because it may be that a longstanding visual defect has only just been noticed by the patient. It is important to make this differentiation as this alters the range of diagnoses and the urgency of treatment. Acute visual disturbance of unknown cause requires urgent referral.

In many cases the diagnosis may be made from the history. Symptoms of floaters or flashing lights suggest a vitreous detachment, a vitreous haemorrhage, or a retinal detachment. Horizontal field loss usually indicates a retinal vascular problem, whereas a vertical defect suggests an abnormality posterior to the optic chiasm. If there is central field loss (“I can’t see things in the centre”) there may be a disorder at the macula. Associated symptoms such as headache may indicate giant cell arteritis or migraine.

The visual acuity gives a strong clue to the diagnosis. A total lack of perception of light indicates complete occlusion of either the central retinal artery or the arteries supplying the head of the optic nerve. The nature of the field defect gives clues as outlined above. Obstruction of the red reflex on ophthalmoscopy suggests a vitreous haemorrhage, although the patient may have a pre-existing cataract. The appearances of the macula, remaining retina, and head of the optic nerve will indicate the diagnosis if there has been a haemorrhage or arterial or venous occlusion in these areas.
Posterior vitreous detachment

Posterior vitreous detachment is the commonest cause of the acute onset of floaters, particularly with advancing age, and is one of the commonest causes of acute visual disturbance.

**History**—The patient presents complaining of floaters. In posterior vitreous detachment the vitreous body collapses and detaches from the retina. If there are associated flashing lights it suggests that there may be traction on the retina, which may result in a retinal hole and a subsequent retinal detachment.

**Examination**—The visual acuity is characteristically normal, and there should be no loss of visual field.

**Management**—If there is any doubt about the precise diagnosis the patient should be referred to an ophthalmologist on the same day so that an associated retinal hole or detachment may be excluded.

Vitreous haemorrhage

**History**—The patient complains of a sudden onset of floaters, or “blobs,” in the vision. The visual acuity may be normal or, if the haemorrhage is dense, it may be reduced. Flashing lights indicate retinal traction and are a dangerous symptom. Haemorrhage may occur from spontaneous rupture of vessels, avulsion of vessels during retinal traction, or bleeding from abnormal new vessels. If the patient is shortsighted, retinal detachment is more likely. If there is associated diabetes mellitus the patient may have bled from new vessels.

**Examination**—The visual acuity depends on the extent of the haemorrhage. Projection of light is accurate unless the haemorrhage is extremely severe. Ophthalmoscopy shows the red reflex to be reduced; there may be clots of blood that move with the vitreous.

**Management**—The patient should be referred to an ophthalmologist to exclude a retinal detachment. Underlying causes such as diabetes must also be excluded. If a vitreous haemorrhage fails to clear spontaneously the patient may benefit from having the vitreous removed (vitrectomy).

Retinal detachment

Retinal detachment should be suspected from the history. It is only when the detachment is advanced that the vision and the visual fields are affected and the detachment becomes readily visible on direct ophthalmoscopy.

**History**—The patient may complain of a sudden onset of floaters, indicating pigment or blood in the vitreous, and flashing lights caused by traction on the retina. These, however, are not invariable and the patient may not present until there is field loss when the area of detachment is sufficiently large, or a deterioration in visual acuity if the macula is detached. Retinal detachment is more likely to occur if the retina is thin (in the shortsighted patient), damaged (by trauma), or if the ocular dynamics have been disturbed (by a previous cataract operation). Traction from a contracting vitreous haemorrhage in a diabetic patient can also cause a retinal detachment.

**Examination**—The visual acuity is normal if the macula is still attached, but the acuity is reduced to counting fingers or hand movements if the macula is detached. Field loss (not complete in the early stages) is dependent on the size and location of the detachment. Direct ophthalmoscopy will yield normal results if the detachment is small; detached retinal folds may be seen in larger detachments.
Management—The patient should be referred urgently. Only small retinal holes with no associated fluid under the retina can be treated with the laser, which causes an inflammatory reaction that seals the hole. True detachments usually require an operation to seal any holes, reduce vitreous traction, and if necessary drain fluid from beneath the neuroretina.

Arterial occlusion

History—The patient complains of a sudden onset of visual disturbance. This may be temporary (amaurosis fugax) if the obstruction dislodges, or it may be permanent. It is often described as a “curtain” descending over the vision.

Examination—The visual acuity depends on whether the macula or its fibres are affected. There may be no direct pupillary reaction if there is a complete occlusion of the central retinal artery. The extent of field loss depends on the area of the retina affected. The inner two thirds of the neuroretina is supplied by the retinal artery and its branches, and the outer third is supplied by the choroid. The arteries may be blocked by atherosclerosis, thrombosis, or emboli, and the attacks may be associated with a history of transient ischaemic attacks if the aetiology is embolic. When the retina infarcts it becomes oedematous and pale and masks the choroidal circulation except at the macula, which is extremely thin—hence the “cherry red spot” appearance. Ophthalmoscopy may be normal initially, before the oedema is established. Plaques of cholesterol or calcium may be seen in the vessels.

Management—Giant cell arteritis must be excluded by the history, examination, and performing an erythrocyte sedimentation rate. Emboli from the carotid arteries and the heart should also be excluded. Attempts may be made to open up the arterial circulation in acute cases by ocular massage, or by carbon dioxide rebreathing to cause arterial dilatation. Factors predisposing to vascular disease (for example, smoking, diabetes, and hyperlipidaemia) should be identified and treated.

Occlusion of the posterior ciliary arteries may cause ischaemia and infarction of the head of the optic nerve (ischaemic optic neuropathy). The nerve head swells and this may be mistaken for papilloedema. Papilloedema, however, is usually bilateral and the visual acuity is not affected until late in its development. In addition, the optic disc in ischaemic optic neuropathy is pale because of the lack of blood perfusion. Giant cell arteritis must be excluded in these cases as the other eye may also go blind if intravenous and oral steroid treatment is not started promptly.

Venous occlusion

Raised blood pressure causes thickening of arteries leading to compression of vein

Branch retinal vein occlusion.
Disciform macular degeneration

History—The visual acuity will be disturbed only if the occlusion affects the temporal arcades and damages the macula. Patients may otherwise complain only of a vague visual disturbance or of field loss. The arteries and veins share a common sheath in the eye, and venous occlusion most commonly occurs where arteries and veins cross, and in the head of the nerve. Thus raised arterial pressure can give rise to venous occlusion. Hyperviscosity—for example, in myeloma—and increased “stickiness” of the blood—as in diabetes mellitus—will also predispose to venous occlusion. This leads to haemorrhages and oedema of the retina. Occlusion of the central retinal vein within the head of the nerve leads to swelling of the optic disc.

Examination—Visual acuity will not be affected unless the macula is damaged. There may only be some peripheral field loss if a branch occlusion has occurred. Ophthalmoscopy shows characteristic flame haemorrhages in the affected areas, with a swollen disc if there is occlusion of the central vein. Cotton wool spots imply an ischaemic retina and are a bad prognostic sign.

Management—Hypertension, diabetes mellitus, hyperviscosity, and glaucoma must be identified and treated. If the retina becomes ischaemic it stimulates the formation of new vessels on the iris (rubeosis), and neovascularisation of the angle may cause glaucoma. Laser treatment is used to ablate the ischaemic retina to prevent this happening.

Retrobulbar neuritis

History—The patient notices a sudden disturbance of central vision. Straight lines may seem wavy and objects may be distorted, even seeming larger or smaller than normal. Eventually central vision may be completely lost. This central area of visual distortion or loss moves as the patient attempts to look around it. The layer under the retina is the black retinal pigment epithelium. Most commonly with increasing age (the patient is normally over 60) and in certain conditions (for example, high myopia) neovascular membranes may develop under this layer in the macular region. These membranes may leak fluid or bleed causing an acute disturbance of vision.

Examination—The visual acuity depends on the extent of macular involvement. If the patient looks at a grid pattern (Amsler chart) the lines may look distorted centrally. The peripheral fields are normal. On fundal examination the macula may look normal, or there may be a raised area within it. Haemorrhage in the retina is red but it appears black if it is under the retinal pigment epithelium. There may be associated deposits of yellow degenerative retinal products (Drusen).

Management—Some cases are treatable with a laser, which occludes these neovascular membranes. If a patient has had a subretinal neovascular membrane in one eye that has destroyed central vision, they are at risk of the same thing occurring in the other eye.
Migraine

**History**—Migraine may initially present with symptoms of visual loss. The features are well known and include:

- A family history of migraine
- Attacks set off by certain stimuli—for example, particular foods
- Fortification spectra in both eyes that include zigzag lines and multicoloured flashes of light
- Associated headaches and nausea (though these symptoms may not be present).

**Examination**—The patient may have a bilateral field defect but this usually resolves within a few hours.

**Management**—Conventional treatment with analgesics and antiemetics may be necessary. Long term prophylaxis may be required if attacks occur frequently.

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Cardiovascular disease

Damage to visual pathways leading to contralateral homonymous hemianopia

**History**—The patient may have a hemiparesis on the same side as the visual field loss. Patients sometimes complain of “the beginning or end of a line of print disappearing,” and some may complain of a decrease in acuity. The visual pathways pass through a large area of the cerebral hemispheres, and any vascular occlusion in these areas will affect these pathways. Lesions behind the chiasma are the most common and lead to contralateral field loss in both eyes. More posteriorly placed lesions tend to spare the macular vision in the affected fields.

**Examination**—The visual acuity should be preserved though patients may say half the Snellen chart is missing. The appropriate bilateral visual field loss is present.

**Management**—It is important to make the diagnosis and exclude any underlying cause for vascular disease. The field defects sometimes improve with time, and patients should be taught to compensate for their field defect with appropriate head and eye movements.

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MATERIA NON MEDICA

Solomon’s seal

I have no musical talent and being unable to read a note of music I rate as a musical illiterate. For almost as long as I can remember, however, I have enjoyed listening to music, and one who gave me a great deal of pleasure was the pianist Solomon, whose wartime concerts and recitals brought music to servicemen and women in many parts of the world. It was sad to hear recently of Solomon’s death, but somehow the news brought back many happy memories. It also served to remind me of one of the more embarrassing moments in my life.

My first house job, some years after the second world war, was at a satellite of my teaching hospital. This small unit was located in a converted house in a rather pleasant Georgian square of a type not uncommon in central London. As a hospital it was not well designed, but the residents’ quarters on the ground floor were quite unusually luxurious and I was the only resident. A spacious living room looked out over the square and in front of one large window was a grand piano. Although this was rather battered, it was probably still quite serviceable and I never did find out how it came to be there. Early one evening, after a long operating session during which I had held on to various things with small distinction, I came back down to my quarters and, being tired, switched on the radio. I realised that I had missed much of a Beethoven recital by Solomon, but a little Beethoven is better than none and I lay back in my chair to enjoy the *Pathétique*. The twilight had that quality of dusk and amber so characteristic of autumn evenings in London and I did not switch on the room lights. The sonata is a delight, and without thinking I moved to the piano and sat fingering my way through the second and third movements. With better luck than judgment I usually managed to have my hands at the right end of the keyboard at the right time, and with only a minor flourish finished my recital at exactly the correct moment. It was a little disturbing to realise that the ripple of applause was coming not from the radio but from about a dozen people gathered outside the open window. Confused, I managed to switch off the radio; no deceit was intended as I got up from the piano, an indistinct figure in a white coat, bowed to my audience, and made off at great speed for another part of the hospital before some enthusiast could request an encore.

My embarrassment was not lessened when some weeks later an elderly patient asked me if the young doctor who played Beethoven so beautifully still worked in the hospital. I replied to her sadly that, as far as I knew, no doctor who could play Beethoven had worked at the hospital in recent years.

So if ever you hear of the ghostly piano player of a rather pleasant London square in the Georgian style, you will know how the story started.—J T Weeks, occupational physician, Pinawa, Manitoba, Canada