Unilateral Visual Loss
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Black-and-white
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<Opening titles>

<Narrated by Sanders to camera>

Of all the fibres that enter the human brain about half originate within the eyes. This representation places the visual system in a pre-eminent role and establishes vision, as Plato suggested, as the most piercing of the senses.

The concentration of important fibres in small tracts, however, makes them vulnerable to lesions and it needs all the skill and resources of modern medicine to make accurate diagnoses. The ophthalmologist plays a particularly important role in detecting these early lesions because they may be the first sign of systemic or neurological disease.

In this first programme I would like to discuss some causes of visual loss in one eye and describe some of the modern methods of investigation. As an introduction, however, I would like to review some special anatomical and physiological features of vision and explain the importance of a thorough clinical examination.

<Sanders refers to projected diagrams to illustrate>

When visual information enters the eye, an impulse is generated which traverses the optic nerve to the chiasm where the nasal fibres from each retina decussate and then pass to a synapse in the lateral geniculate body. From here a further fibre takes the information to the occipital cortex. Thus an object seen in the right side of the visual field is appreciated in the left retinae on both sides and the information is organised in the left side of the brain.

Today I plan to discuss only the anterior visual system and I will concentrate on painless causes of visual loss in the retina, the optic disc and the optic nerve.

When light enters the eye it is concentrated by the focusing system of the eye on the retina and traverses this transparent structure to induce a photo-chemical reaction in the rods and cones. There are about 130 million rods and cones in each retina constructed of thin discs and nourished by a thick layer of pigment, the pigment epithelium. Activation by light produces a neural impulse which passes via the bipolar cells to the ganglion cell. A ganglion cell therefore receives information from many photo receptors and the area of sensory input is called the receptive field of the ganglion cell. Impulses from the ganglion cell pass in the optic nerve fibre in the superficial retina to the optic disc and they retain a relationship to each other which continues to the occipital cortex.

The majority of fibres are concerned with central macular vision – the area concerned with visual acuity, and pass to the temporal side of the optic disc. Whilst fibres from the peripheral retina pass round these to enter the optic disc above, below, or nasally. Thus visual loss occurs when the central, macular or optic nerve fibres are involved, whereas arcuate field defects (so named because of their shape) are usually due to lesions of the optic disc or optic nerve.

Here we see fibres depicted and superimposed – you can see the pattern of nerve loss in an arcuate defect, the commonest cause of which is glaucoma although this will not be discussed in this programme.

As the fibres pass out of the retina along the optic nerve, they traverse the lamina cribrosa which is a continuation of the sclera and composed of connective tissue. It is unique for neurones to pass through a tight, connective tissue framework – this makes them vulnerable to disease in this area. The blood supply to the retina comes from the central retinal artery but the optic disc is mainly supplied by other branches of the ophthalmic artery, the posterior ciliary arteries on each side.

00:05:37:00

< **Sanders refers to projected diagrams to illustrate** >

In the optic nerve, the macular fibres are initially temporal as they emerge from the macula region and the peripheral fibres are situated above, below and nasally; but as the fibres pass posteriorly in the optic nerve the macular fibres take a more central position and are surround by peripheral fibres and the posterior portion of the optic nerve, the nasal and temporal fibres, begin to separate in preparation for decussation in the chiasm.

<**Sanders to camera**>

An understanding of this simple visual organisation of the visual system is extremely important in obtaining maximum information from clinical examination and I would now like to describe some of the important principles of clinical examination.

<**Sanders displays a Snellen chart**>

Visual acuity is first tested for distance using the Snellen chart which is placed at six metres from the patient. At this distance the letters are constructed to subtend an angle of five minutes of arc at the eye from the appropriate distance, and the width of

each line forming a letter subtends one minute. Thus the eye must be able to resolve one minute in order to see the letter. The largest letter would present five minutes at the eye from a distance of sixty metres and successive lines represent 36 metres, 24, 18, 12, 9 and 6 metres. Visual acuity is recorded as six over the lowest line that can be read by the patient. 6/6 is the accepted normal and if vision is worse than 6/60 then counting fingers at one metre are tested, hand movements and finally light perception, is tested.

<Sanders displays the Jaegar chart>

Next, near vision is assessed using either the Jaegar chart or the Times Roman Faculty of Ophthalmologists reading chart. This is depicted in N5, N6, and N8. And N8, for practical purposes, corresponds to the print of a normal newspaper.

<Sanders to camera>

Both tests should be used in all cases because the myopic or short-sighted person cannot see in the distance but can read for near whereas the hypermetropic or long sighted person can see in the distance but cannot read for near.

<Sanders displays a small test card>

It is convenient for practical purposes and when testing vision in unusual situations to use a small test card which can be carried and is easily manipulable.

<Sanders to camera and displays the pinhole test>

For non-ophthalmologists, refractive errors can be eliminated by using the pinhole test. When a patient views through a small pinhole refractive errors in the eye are eliminated and visual acuity due to refractive errors is eliminated. Normal vision, if it occurs with a pinhole test, suggests a refractive error but organic disease cannot always be diagnosed if it does not return to normal.

<Sanders displays the colour chart>

Now there are two further tests for central vision. The first of these is the colour chart which has a series of different coloured circles and either symbols or letters are represented. Have no fear however if you cannot see any symbols on this diagram because black and white television is colour blind.

<**Sanders displays the Amsler chart**>

The other central method of assessing vision is the Amsler chart which delineates the central ten degrees of vision. It is therefore possible to ask a patient to record any area of distortion or loss of vision on the chart.

00:10:13:00

< Table >

Peripheral Vision

Visual fields

a) confrontation
b) Bjerrum screen
c) Goldmann perimeter
d) static perimetry

<**Sanders narrates over above table**>

Peripheral vision is next tested by assessment of the visual fields. This can be done in a number of different ways.

Firstly, confrontation testing can be performed which is easy to do, convenient and can be done on patients who cannot co-operate fully. More precise methods of testing the visual fields include the Bjerrum screen, the Goldmann perimeter and more refined methods of static perimetry. I would like to demonstrate in detail groups a, b, and c.

<Sanders narrates over a demonstration with a seated female>

Confrontation testing is first performed with both eyes open with a hand in each hemi-field and this is to detect a homonymous hemianopia. Next, finger counting is assessed to see whether the patients can count fingers in the left and right fields. Defects of the parietal lobe can be assessed by an inattention defect when the patient will only notice one finger moving. Next, one eye is covered and the test repeated. This testing will detect a bi-temporal hemianopia and is done for each eye in turn.
Next, a small red object is used which is brought in from the periphery and usually disappears in the region of the blind spot and the patient records this and then arcuate fibre defects are detected by bringing the spot in from the centre when small para-central lesions can also be noted.

<Sanders to camera>

Though confrontation tests are valuable for detecting gross lesions and may also detect subtle lesions, we need slightly more sophisticated quantitative tests to make accurate assessment.

< Sanders refers to projected diagrams to illustrate >

These include first of all the Bjerrum's screen (or tangent screen) in which the patient observes a small central object and different sized objects are brought in from the periphery. The technique used in most ophthalmic hospitals is that of the Goldmann perimeter and this consists of a patient situated in a bowl with the observer having close observation and constant conditions of illumination are obtained.

<Sanders narrates over diagrams>

Careful testing of the pupillary light response is important because it provides objective evidence of optic nerve function. It is therefore particularly valuable in children, people with communication difficulties and comatose patients.

When a light is shone in a normal eye, the impulse passes down the optic nerve into the tract and it is here that the pupillary fibres bifurcate from the visual fibres to pass to the mid-brain region. These fibres innovate the Edinger-Westfall nucleus equally and symmetrically. The pupillo-constrictor fibres pass to the third nerves equally so that the pupillary constriction is equal and symmetrical on both sides.

If the same intensity of light is then shone in the other eye, again an equal and symmetrical pupillary response will occur. The light stimulus to this eye produces a direct pupillary response on the same eye and a consensual, which is equal, on the other side. If one optic nerve is damaged, less afferent information will pass into the pupillary system so that the pupillary response on both sides will be less. By passing the light from the abnormal to the normal side subtle differences in optic nerve function may be detected. This test is called the swinging flashlight test and records a defect of the afferent system in one eye relative to the other.

00:14:45:00

<**Sanders narrates over a demonstration with a seated female**>

Next, I'd like to show the importance of testing the pupillary responses. For this test it is first possible to see that there is a direct light response when the light is shone in one eye. There is a similar and equal response in the left eye. More subtle information can be detected by passing the light from one side to the other and a pupillary response of equal amplitude occurs in each eye.

Notice there is a small light response and then the pupil takes up a constant size. On reverting to the other eye there is a small contraction before the pupil retains again the same constant size.

< Table >

Investigations

Fundus examination and photography

a) Red free photography

b) Fluorescein photography

<**Sanders narrates over above table**>

Next we come to investigations and simple investigations include fundus examination and photography. For this, we can use two techniques a) Red free photography and b) Fluorescein photography.

< **Sanders refers to projected diagrams to illustrate** >

Here we see a fundus camera through which the patient's retina is photographed with the pupil dilated. Red free photographs are taken by the manipulation of filters and using this we can see in this patient the normal pattern of nerve fibres coming into the optic disc.

In diseased processes there are defects in the nerve fibre layer which are shown up well by red free photography. And in this example you see these bands which are arcuate bands (we've previously depicted diagrammatically) showing loss of neurones in the retina. By injecting a florescent dye into the anticubital vein of a patient, the dye enters the retinal vessels and we can record its progress through these vessels. It first enters the retinal artery, and you see here the retinal artery depicted white with the entrance of dye. There is background white florescence due to dye entering at the same time the Choroidal blood vessels. Having passed through the retinal arteries, the dye passes through the capillaries and enters the veins and the veins have a very characteristic Fluorescein appearance and you see there is a tramline appearance due to the florescent dye at the margins of the veins and this occurs in all the major veins.

When they are filled, the retinal circuit is then complete and this circuit is being recorded by pictures taken approximately every second. We conclude the run by taking a picture ten minutes after the injection to see whether there has been any

abnormal leakage of dye from the retinal vessels. Here is a picture taken ten minutes after injection showing some re-circulation of dye but no abnormal leakage.

This technique allows us to study dynamically conditions of the retina, pigment epithelium and optic disc which previously we could only observe ophthalmoscopically.

< **Sanders refers to projected diagrams to illustrate** >

Next I would like to describe a final investigation – that of visual evoked responses and this is performed with the patient seated with electrodes around his occipital cortex and he views either a flashing light or, as in this instance, a reversing pattern. Reversal of the pattern produces occipital impulses which can be measured. This test, as conducted at the National Hospital, is of use in measuring conduction down the optic nerve and also the amplitude of the impulse. It is an important objective test of visual function.

Painless visual loss in one eye may be due to either disorders of the retina or the optic nerve on that side.

Conditions that involve the retina include retinal detachment, disorders at the macular which may be either retinal or choroidal and also retinal vascular disease - either involving the arteries or the veins. These conditions can be diagnosed with the aid of the ophthalmoscope. And I am going to restrict the rest of this talk to the disorders of the optic nerve and the first condition is that involving the optic disc.

00:20:15:00

<**Sanders (off camera) interviews an unidentified man**>

<Sanders>
You've had some visual disturbance in your right eye Sir, can you just indicate how this occurred and what you noticed?

<Man>
Yes, it occurred very early in October this year. It became apparent I was having trouble with my right eye. I appeared to have a bit of mist in the top of the eye so I went to the medical establishment where I work and got some treatment and they said 'well we can't see anything.' They put some drops in and there was nothing to report as far as they were concerned. I wasn't satisfied with that so I went to see my doctor and my doctor arranged for me to go to a London hospital to have further treatment. Having got to a London hospital, the sight by that time had disappeared by half in the right eye - across here.

<Sanders>
Which half had disappeared?

<Man>
The top half

<Sanders>
The top half of your vision. Did that happen suddenly?

<Man>
Rather suddenly, yes. It went from a sort of a mist to a defined line right across the eye.

<Sanders>
Did you have any pain in your eye?

<Man>
Not of any significance, no.

[Probably accidental flash of earlier ID BOARD briefly interrupts the discussion]

<Sanders>

...Brighter above or below..

<Man>
It's brighter below.

<Sanders>
Good. Now I am going to bring a red object down and I want you to tell me when it becomes red.

<Man>
Now.

<Sanders>
Good. Tell me when it becomes red?

<Man>
Now.

<Sanders>
Tell me when it becomes red?

<Man>
Now.

<Sanders>
Good. I would just like to show your pupillary responses. If you'd like to look straight into the camera again.

<**Sanders demonstrates with a close-up of the man's eyes**>

Now, in this instance, on shining a light in his right, or defective eye, there is still a pupillary response. But when I transfer the light from the right eye to the left eye, you will notice that the right eye dilates, the left eye constricts, the right eye dilates the left eye constricts.

<Sanders to camera>

This patient therefore has rapid visual loss in his right eye. Vision is reduced to hand movements. There is an upper altitudinal field defect and he has a relative afferent pupillary defect on the right.

< Sanders refers to projected diagrams to illustrate >

Fundus examination showed a normal optic disc on the left and the left eye was entirely normal, but on the right the lower part of the disc is pale, consistent with his upper altitudinal field defect and the upper part of the disc is swollen and hyperaemic.

Floursecein studies were performed on the right eye. This is in the arterio-venous phase and here is the optic disc. Notice that the lower part of the optic disc shows poor capillary filling. The upper part in contrast shows dilated vessels and there is a solitary haemorrhage. Perfusion of the lower part of the disc has therefore been grossly impaired whereas the upper part is dilated as a compensatory mechanism.

Residual pictures show that these vessels at the upper part of the disc are abnormal and that there is leakage of florescent dye into the surrounding retina in this area. This is in contrast to the left disc which is within normal limits.

Angiography on this patient of the cerebral vessels showed normal carotid patency and a magnified view - which is possible with special techniques - showed the commencement of the ophthalmic artery. By the technique of subtraction it is possible to remove the bone shadow so that the ophthalmic artery can be seen more clearly and it is often even possible to find even small branches to the eye.

In this patient therefore, the carotid and ophthalmic arteries are patent and it is probable that his disturbance is due to small vessel disease affecting the optic disc. As you remember, the blood supply to the optic disc is from the short posterior ciliary

arteries which pass in to perfuse the disc. Occlusion of these vessels may produce either segmental or total infarction of the optic disc.

In this patient he's infarcted the lower portion of his optic disc with compensatory dilation of vessels and retinal vessels in the upper portion.

< Table >

Ischaemic popillopathy
Vessel wall-
Artertitis (giant cell)
Arteriosclerosis

Blood constituents -
Polycytheamia
Emboli

Blood flow -
Hypotension

<Sanders narrates over above table >

Now there are many medial causes of this condition and the term that is used is ischaemic popillopathy or ischaemi**c** optic neuropathy. It may be due to disorders of the vessel wall such as artertitis or arteriosclerosis.

It maybe due to disturbances of the blood constituents such as polycytheamia or Emboli and it may be due to disorders of blood flow such as hypotension and carotid occlusion. It is important to diagnose this condition because in over 50% of cases the second eye is similarly involved.

<Sanders to camera>

The only abnormality detected in the patient you have just seen is that of grossly elevated plasma triglyceride levels. It is hoped that by dietary control of this, involvement of the second eye will be prevented.

00:26:00:00

The next case I would like to demonstrate is a patient with involvement of the optic nerve.

<**Sanders (off camera) interviews an unidentified man**>

<Sanders>
Now Sir, you've had some impairment of vision in the right eye. Could you tell me how this came on?

<Man>
Yes, this happened fairly suddenly over a period of about 10 or 11 days – I noticed a rapid deterioration of vision in the right eye. Various other symptoms occurred as well during the month or so before that, I had a bit of difficulty with the legs, with co-ordinating the movement of the legs and also the left leg in particular was rather numb to any kind of sensation. Pin pricks and things didn't register as much as with the right leg.

<Sanders>
With your visual loss, did you have any pain on moving your eye or any tenderness of the eye?

<Man>
There was pain on moving it, on looking to the side or up and down.

<Sanders>
Has your vision got better since the initial episode?

<Man>
It has improved quite a bit. I still feel I'm not using my right eye very much but certainly from going into hospital until now it has improved.

<Sanders>
Could you look straight at the camera? I've got a red dot here. Do you see it now? Is it red?

<Man>
With both eyes?

<Sanders>
Yes, cover your left eye. Now, tell me do you see the red dot?

<Man>
No, I can't see it.

<Sanders>
Tell me when you see it?

<Man>
Now.

<Sanders>
Good. Tell me when you see it?

<Man>
Now.

<Sanders>
Tell me when you see it?

<Man>
Now.

<Sanders>
Tell me when you see it?

<Man>
Now.

<Sanders>
Good.

<Sanders narrates over a demonstration with the man's eyes>

I just want to test your papillary responses. If you look straight towards the camera. Could you slip off your glasses off and look straight towards the camera. Quite relaxed. That's very good. I'm just going to shine a light in your right eye and your left eye and you will notice again that when the light is shone in the right eye it dilates and in the left eye it constricts. Right eye dilates, left eye constricts, right eye dilates, left eye constricts. This again indicates a defect in the right optic nerve. Thank you very much.

< Table >

Retrobulbar Neuritis
Rapid central visual loss
Pain on ocular movement

<Sanders narrates over above table >

This patient has retrobulbar neuritis on the right side. This is characterised by rapid central visual loss and in this instance the patient's vision was reduced to hand movements in the central region. He had a central scatoma on field testing as you saw. He had a relevant afferent papillary defect and his visual evoke responses showed a reduced impulse with delayed conduction down the optic nerve. He also

shows the other classic symptom of retrobulbar neuritis, that of pain on ocular movements.

Now, retrobulbar neuritis in about half the cases progresses to multiple sclerosis and it is probable that this patient, with his other neurological signs, has this diagnosis.

00:30:00:00

< **Sanders refers to projected diagrams to illustrate** >

Now the final case that I would like to show is a patient with compression of the optic nerve. The optic nerve can be compressed by lesions within the orbit, by lesions within the optic canal and finally by lesions within the intracranial cavity.

<**Sanders (off camera) interviews an unidentified man**>

<Sanders>
Sir, you've had poor vision all your life, is that right?

<Man>
Yes doctor.

<Sanders>
And recently you've noticed visual loss in the left eye?

<Man>
Also yes.

<Sanders>
Could you tell me how that has come about?

<Man>
It crept in and abruptly by the 4th of September this eye lost everything.

<Sanders>
So it suddenly got worse.

<Man>
Yes.

<Sanders>
Has it got worse since then?

<Man>
I don't think it could get worse. It's improving. I can see outlines now.

<Sanders>
You've had an operation. Has it improved since the operation?

<Man>
I would say yes.

<Sanders>
It has improved slightly since the operation. Good. Now look straight at me, Mr. Roberts. Can you tell me how many fingers I have?

<Man>
Two. One.

<Sanders>
Look straight at me. How many fingers?

<Man>
Two.

<Sanders>
Do you see my hand moving?

<Man>
Yes.

<Sanders>
Do you see my hand moving here?

<Man>
Yes.

<Sanders>
Good. Thank you very much.

< **Sanders refers to projected diagrams to illustrate** >

This patient presented a difficult diagnostic problem because fundus examination showed over long-standing choroidal retinal inflammatory changes. And this eye, the right eye, had always had impaired vision. Vision in this eye was reduced to perception of light only. In the other eye also he had some early macular changes which you see depicted here by small white dots. Vision in this eye deteriorated from 6/9 to counting fingers over the period of 2 months. Now these fundus changes were not sufficient to produce his severe visual loss. It was therefore important to investigate him neuro-radiologically and preliminary studies suggested a tumour and therefore angiography was performed. This is a carotid angiogram showing the carotid artery and the anterior cerebral artery is grossly elevated due to a large tumour situated in this region. This is seen more clearly on a subtraction study, that there is a large tumour lying in this area and that it is also fed from branches from an enlarged ophthalmic artery. A craniotomy was therefore performed and a large olfactory groove meningioma was removed. His vision, as you saw from the film, has slightly improved.

< Table >

Chronic progressive visual loss in one eye is due to compression until proved otherwise.

<Sanders narrates over above table >

It is therefore important to remember that chronic progressive visual loss is due to a compressive lesion until proved otherwise.

< Table >

Orbital tumours

Tumours in relation to Optic Canal

Intracranial tumours

<Sanders narrates over above table >

Now, there are many lesions that can compress the optic nerves. These include orbital tumours, tumours in relation to the optic canal and intracranial tumours. These may be of many sorts, you have seen this patient with a meningioma. But aneurysms, lesions from the sinuses and lesions in relation to the pituitary fossa can also damage one or both optic nerves.

<Sanders narrates over a diagram>

In conclusion, therefore, unilateral visual loss may be due to retinal disease. This includes macular disease, retinal detachment and retinal vascular disease. I have demonstrated a patient with ischaemic papillopathy or infarction of the optic disc. And sudden visual loss may also be caused by papillitis when it occurs at the optic disc.

We've also seen a case of retrobulbar neuritis in which the disc was normal but in which there was sudden loss of central vision.

And finally you saw a patient with compression of the optic nerve and this may occur in the orbit, in the canal or intracranially and compression may be due to meningioma, aneurysms, pituitary lesions or lesions in relation to the sinuses. In the

last patient that you saw progressive visual loss was found to be due to an intracranial tumour and though unilateral visual loss has been the theme of this talk that conditions that involve one optic nerve may also involve the second eye.

<**End credits**>