

## CHAPTER 1

# The first steps in seeing

Which animals can you identify from their eye alone (answers below)? Eyes appear to have evolved on many occasions and come in a variety of forms (the most obvious distinction being between the single eyes such as our own and the compound eyes of such creatures as insects). However, you might also note that even within a type of eye there are many variations. For example, in single eyes the shape of the pupil varies greatly. Our human pupils are round, but those of the cat are more oval to allow it to close to a smaller size than ours. In other animals (e.g. geckos) the pupil is a slit that when closed becomes just three small holes in a line and thus can shut out far more light than our own.

Answers (from left to right, top to bottom): (a) robber fly, (b) jumping spider, (c) ogre-faced spider, (d) Gigantocypris (deep-sea crustacean), (e) horsefly, (f) scallop, (g) Histiotteuthis (squid), (h) Dilophus (fly), (i) Tridacna (giant clam).



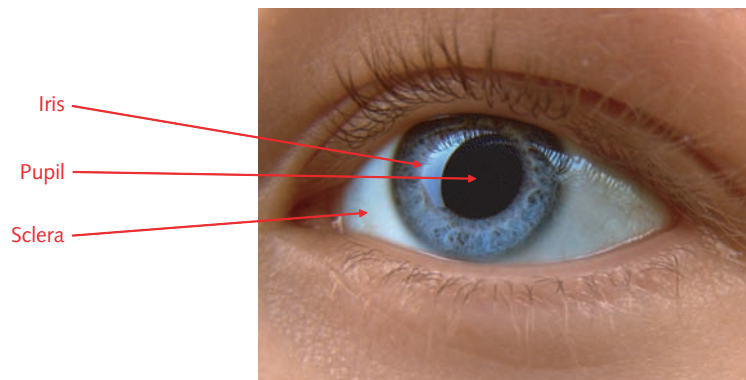
## CHAPTER OVERVIEW

Even without having read any of this book, it seems likely that you have realized that the eyes are rather important for seeing. Close them and vision disappears. But what actually goes on inside these squishy little spheres? Early scientists cut a hole in the back of a bull's eye and were astonished to find that the eye produced a small upside-down image of the world. They concluded that they had solved 'vision'. Unfortunately they had not (which means lots more reading for you!). After all, who is looking at this upside-down image? If we have a little man in our head looking at the image, how does he see? Does it matter that the image on the back of our eye is upside-down? In this chapter we explain what the eye actually does and what happens when things go wrong with it. It turns out that the back of the eye, called the **retina**, is a far outpost of the brain; it is an intricate web of nerve cells that turns the light entering the eye into a set of electrical signals. These messages begin a remarkable journey that turns light from the world into the phenomenon we experience as vision.

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## The eye

There is good evidence that the eyes are important for vision. Close your eyes and you can see rather little. Poke your eyes out with a pointed stick and you will see even less. So if we want to trace the visual pathway then the eyes seem a good place to start. Figure 1.1 shows a front view of a human eye and Figure 1.2 a cross-section through it. The first thing to notice is the **cornea**, the transparent window through which light enters the eye. The cornea is curved and acts as a lens; in fact it is the main lens of the eye. (Three-quarters of the eye's focusing power comes from the cornea, and only a quarter from the bit of the eye we call the 'lens'.) The purpose of these pieces of optical equipment called



**Fig. 1.1.** Front view of the human eye.

lenses is to focus light on to the retina (the cells at the back of the eye – we’ll tell you about these later).

Lenses have two important characteristics; they have at least one curved surface, and they are made of stuff through which light travels more slowly than through air. This means that light gets bent when it hit the surface (just as a stick appears bent when it is half in water). Figure 1.3 shows why. Imagine you are driving a car along a tarmac road beside a sandy beach (position A). All four tyres have an even grip on the road and the car travels in a straight line. When the right-hand front wheel hits the sand (position B) the car will turn towards the right as the left-hand wheel will still be gripping the tarmac while the wheel in the sand will lose traction and move forward more slowly. Once the car is completely in sand (position C) it will once again travel in a straight line, much more slowly, but its direction of motion will have been bent by the tarmac/sand edge. Exactly the same thing happens when light hits the cornea. Light travels at different speeds through different materials and it goes faster through air than through your cornea. How much bending takes place depends on the nature of the materials on each side of the boundary. When you open your eyes under water, everything looks very blurry. Light

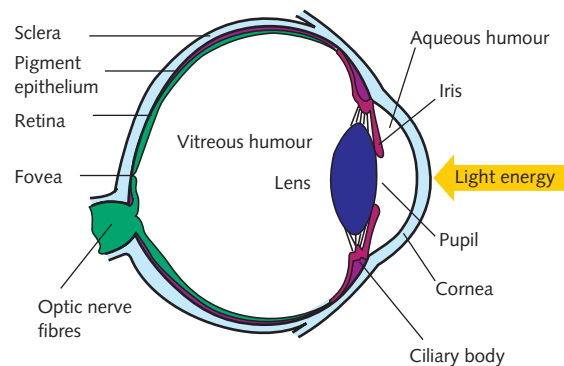


Fig. 1.2. Horizontal cross-section through the human eye.

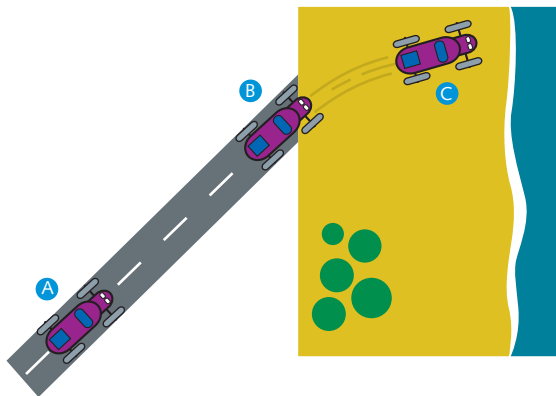
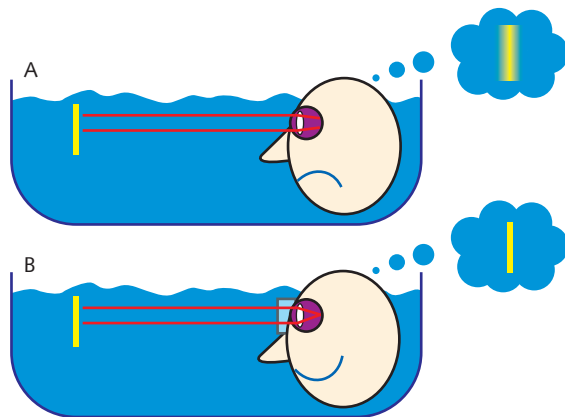


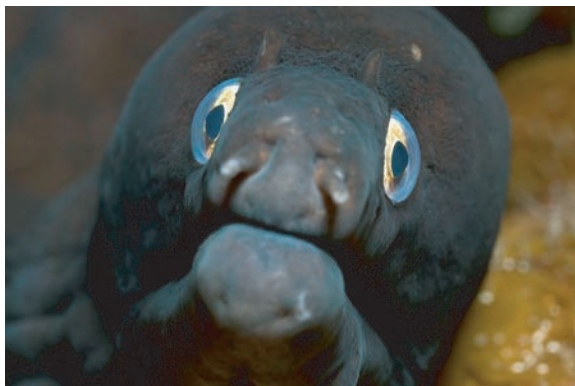
Fig. 1.3. Why light gets bent when it passes from one material into another. See text for details.

travels at similar speeds through water and cornea, so little deflection of the light takes place and you are unable to focus the world on to your retina (Figure 1.4a). Wearing goggles re-establishes an air/cornea boundary and, sure enough, we can see clearly again (Figure 1.4b). Fish, which don't usually wear goggles, tend to have much more bulging eyes to compensate for the small degree of deflection between water and the lens (Figure 1.5).

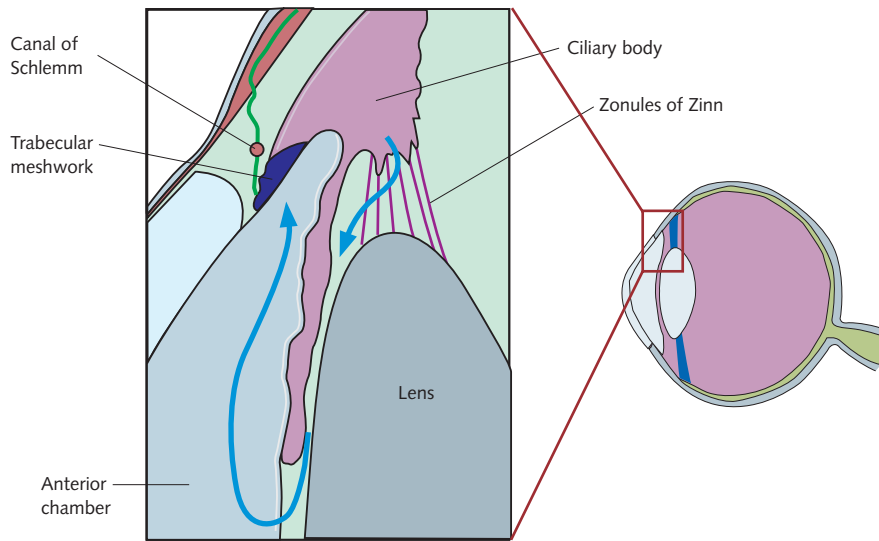
Behind the cornea is the anterior chamber filled with **aqueous humour**, a watery liquid that flows from the ciliary body, through the delightfully named zonules of Zinn, through the pupil, and into the anterior chamber. From here it passes through the trabecular meshwork and leaves the eye down the (we're not joking, honest) canal of Schlemm (Figure 1.6) – anyone trying to think of a name for a rock band need look no further. The canal of Schlemm actually runs in a circle just beneath the border of your iris (the coloured bit) and the white of your eye (called the sclera). The pressure of the aqueous humour is very important. In someone with glaucoma, a disease in which the pressure becomes too high, retinal damage and blindness can result (Figure 1.7). When you go the optician to have an eye examination you will probably have the pressure of your aqueous humour



**Fig. 1.4.** Why things look blurry when you open your eyes under water. Light travels at a similar speed in water and through the cornea, so the cornea fails to act as good lens. The solution is to wear goggles, which re-establish an air/cornea boundary.



**Fig. 1.5.** Why don't things look blurry to fish? The answer is they have really bulging corneas that can bend light more than ours. This fish is a moray eel. Don't get this close to one and certainly don't annoy one. They have been known to attack humans, though apparently they can be quite friendly once they are used to you.



**Fig. 1.6.** In the commonest form of glaucoma the aqueous humour drains through the trabecular meshwork but gets clogged in the canal of Schlemm. This is called 'open angle' glaucoma because the angle is clear. In 'closed angle' glaucoma, a much rarer condition, the blockage seems to be in the trabecular meshwork.



**Fig. 1.7.** How the world might look to someone with glaucoma. Much of peripheral vision may be lost without the patient realizing it.

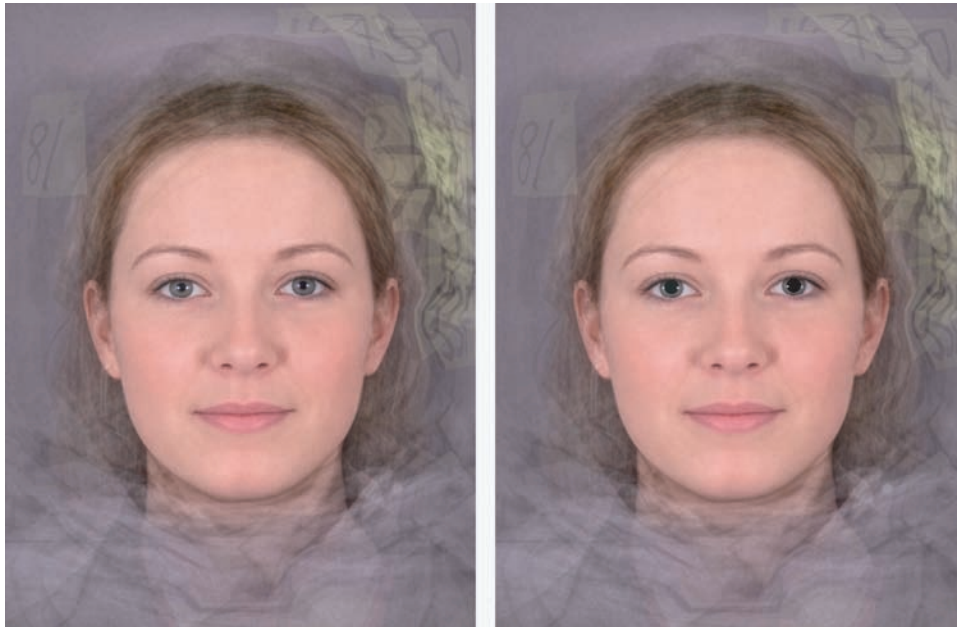
checked by having a puff of air blown against the cornea. This air puff is calibrated to distort the surface of the cornea by a set amount. If the cornea doesn't move enough, it suggests the pressure is too high. In the same way, when you blow up a balloon you can check the pressure by gently squeezing the balloon to feel how much 'give' there is.

Next we come to the **iris**, a structure that provides an adjustable aperture and is the coloured part of the eye. When light levels are high the iris constricts and the **pupil** – the hole in the middle of the iris – gets smaller, limiting the amount of light passing through; when the light is dim, then the iris relaxes, allowing more light through. This seems like a neat way of regulating the amount of light reaching the retina – and is used in cameras to the same purpose, but as the area of the pupil when fully dilated is only 16 times bigger than when it is fully constricted, this isn't a lot of use. The range of luminance that the

visual system can deal with, from the dimmest lights that can be detected up to intensities that will start to damage the retina, is about 10 000 000 000 : 1. How the visual system actually deals with such a wide range of luminance is discussed in Chapter 2.

It turns out that sometimes our pupils constrict and dilate for reasons other than in response to the incoming light level. It has long been known that women with large pupils are considered more attractive by men (Figure 1.8). This explains why deadly nightshade, which causes the pupils to dilate when applied to the eyes, is called belladonna (which mean ‘beautiful woman’ in Italian). The pupils dilate when one is excited, and it is claimed that showing pictures of naked women to heterosexual men – or pictures of naked men to heterosexual women – produces pupil dilation. Of course you get the responses you would expect from homosexual men and women as well. There is, apparently, one curious sex difference; pictures of naked sharks induce pupil dilation in men and pupil constriction in women.

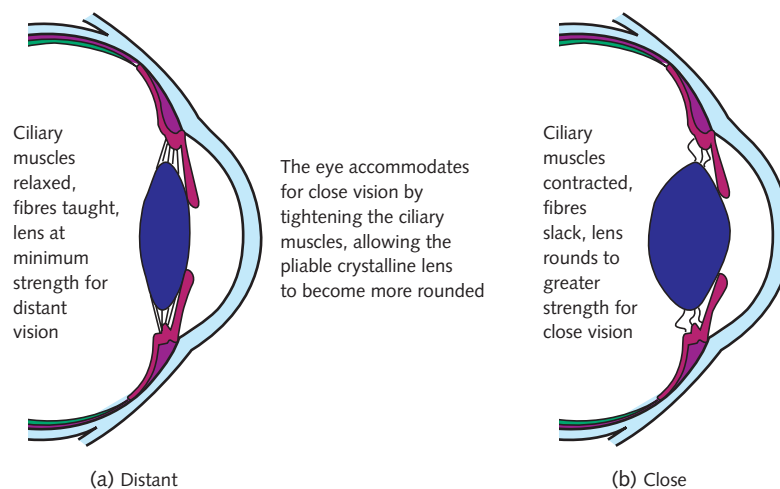
Pupil dilation can of course reveal your excitement when you might wish to conceal it – hence the gambler’s eye-shade to prevent his opponents gaining a clue the poker face intends to conceal. It is claimed that jade dealers have long examined the pupils of prospective customers – pupil dilation will push up the asking price, in the knowledge that the customer is interested. (The solution to this is to take off your sunglasses just as the jade dealer presents you with a piece of jade; your pupils will constrict in the bright light and a bargain is assured.)



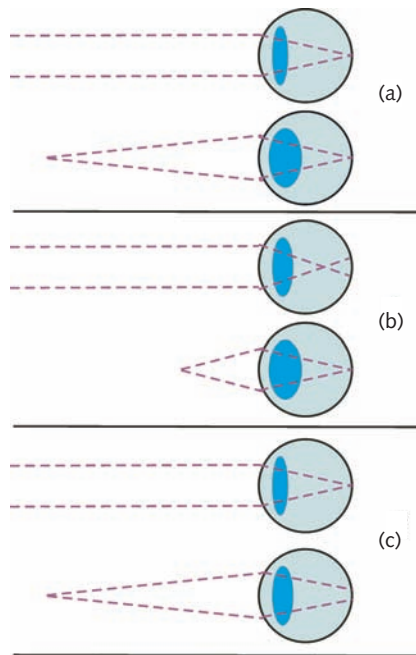
**Fig. 1.8.** Do big pupils make a woman look more attractive? Some drugs, for example deadly nightshade (belladonna), dilate the pupils.

Keen photographers will know that pictures taken with a small aperture have a greater depth of focus than pictures taken with a large aperture. (One of the neat things about a pinhole camera is that everything is in focus, regardless of how far away it is.) The same is true for us – when our pupils constrict, our depth of focus increases. This is quite important as we get older, as you’ll discover on the next page.

Beyond the iris we reach the **lens**. Although the lens has less power than the cornea, it has one great advantage: it is adjustable. The lens is held between the zonules of Zinn. Contraction of the circular ring of ciliary muscles relaxes the zonules, so the lens gets fatter and we have more refractive power. This is what is required when we need to focus on a close object. When we want to focus on things further away, we need the lens to be stretched into a skinnier shape and this is achieved by relaxing the ciliary muscles (Figure 1.9). For this system to work correctly you need to get a few things right. First, you need to have your cornea of the appropriate curviness to focus light on the retina. Another way of looking at this is to say that you need to have your eyeball the right length for your optics. The point is that the power of the lens system must be appropriate for the size of your eyeball. Figure 1.10a shows just such an eye; the light rays from distant objects that reach the eye are near parallel and need little bending to bring them to a focus on the retina, so the lens gets pulled into a skinny shape by the ciliary muscles. Close objects send diverging rays to the eye, which need to be bent more to bring them into focus, so the ciliary muscles relax, the lens goes fat, and the light is bent more. If your eye works like this then your vision is said to be **emmetropic** and you’re very lucky. If, however, your optics are too strong for the length of your eye – or your eye is too long for your optics, it comes to the same thing – then you are **myopic**. This is a fancy way of saying you’re short-sighted (Figure 1.10b). Distant things are blurred because they get bent too much by the



**Fig. 1.9.** Accommodation allows the lens of the eye to become more rounded when looking at close objects, by contracting the circular ciliary muscles and relaxing the zonules of Zinn: (a) distant vision, (b) close vision.



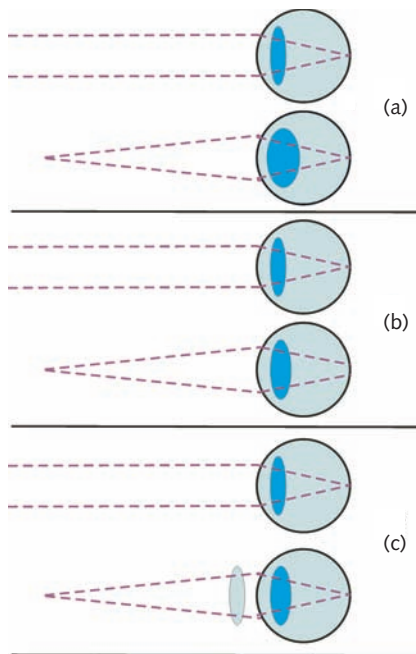
**Fig. 1.10.** Matching the lens system to the size of your eyeball is critical: (a) emmetropic vision – both near and far objects can be focused on the retina; (b) short sight – the strong lens focuses close objects on the retina but distant objects are brought to a focus in front of the retina; (c) long sight – the weak lens is adequate to focus distant objects but is not strong enough for close objects, which are brought to a focus behind the retina.

optics but very close objects, which only come into focus after a lot of bending of the light, will look sharp. The opposite condition, in which the lens is too weak (or the eye too short) is called **hypermetropia**, or long-sightedness (Figure 1.10c). Distant objects that need little bending to be focused can be dealt with OK, but close objects can only be focused behind the retina.

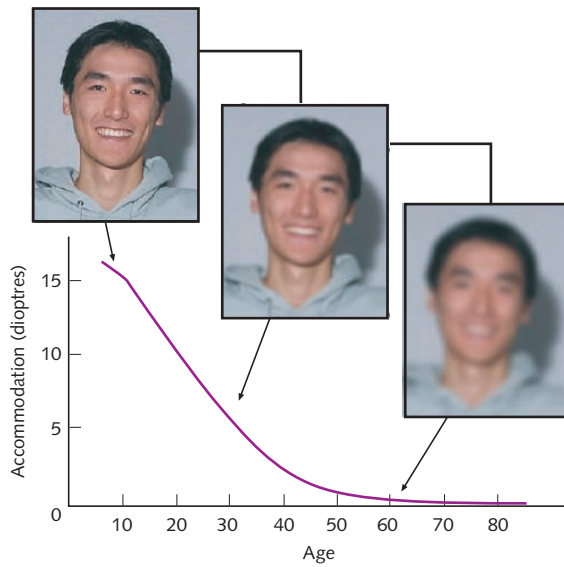
You don't need to be a genius to see that both myopia and hypermetropia can be helped by putting lenses in front of the eyes. A hypermetrope has a weak lens and needs extra power, so needs a converging lens to add to the existing optical system. A myope needs a diverging lens to reduce the power of the optical system. These lenses can be put into frames that balance on the bridge of the nose and are held in place by struts that hook around the ears. Crude, but effective.

As we get older the lens loses its natural elasticity (and so does much else in the body, believe us). This means that when we relax our ciliary muscles the lens does not move as much as it should and our **near-point**, the closest point we can focus on, moves away from us. This is called **presbyopia**, and it's time for reading glasses.

In Figure 1.11a we see the eye of a normal young person, able to change the shape of the lens to accommodate distant and close objects. In Figure 1.11b we see what happens with age; the lens now shows little or no accommodation and, if distant objects are still reasonably well focused on the eye, near objects will look blurry as the ability of the lens to change shape has been lost. In Figure 1.11c we see the solution to the problem, a lens to help seeing close objects – reading glasses. The extent of the loss of accommodation is



**Fig. 1.11.** Presbyopia, the effects of ageing on our lens: (a) the emmetropic vision of Figure 1.10a; (b) inability to focus on close objects as the lens becomes inelastic. The solution (c) is to wear reading glasses.

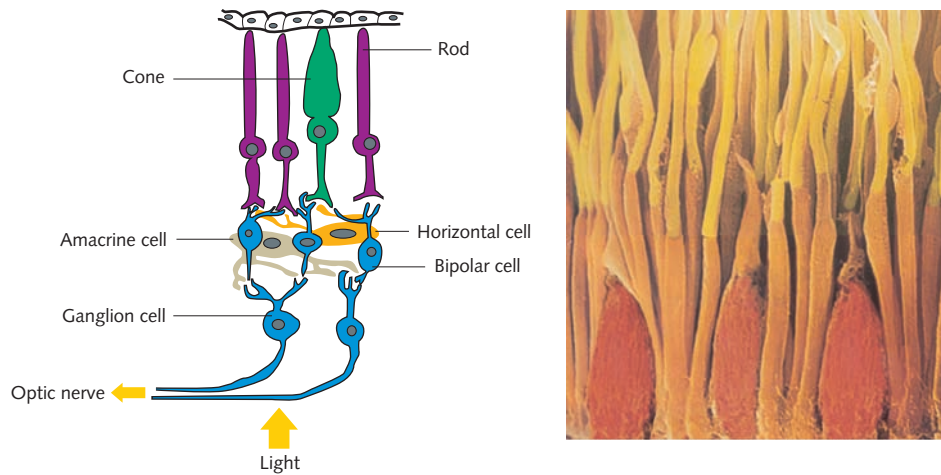


**Fig. 1.12.** The accommodative power of the lens changes with age. The elasticity of the lens declines from birth onwards. Even fit young students are already well down the perilous slope towards reading glasses.

quite frightening – by the age of 30 you have lost around half of your focusing power and by 55 there is nothing much left at all (Figure 1.12).

The main cavity of the eye, behind the lens, is filled with a gelatinous substance called the **vitreous humour**. This keeps the eyeball in shape and the retina pinned to the back of the eye. If the vitreous humour shrinks (and it does in old people) then the retina can become detached and may need to be spot-welded back with a laser.

Eventually we reach the **retina**, the light-sensitive layer at the back of the eye. This is the important bit and the start of real visual processing. Figure 1.13 shows a cross-section of the retina. Light approaches from below in this view, and the light-sensitive photopigments are to be found in the outer segments of the **photoreceptors** near the top of the picture. So the light must travel through a good deal of neural gunge before reaching the receptors. This seems an odd arrangement, as if we designed a camera and then put the film in the wrong way round. One reason for this strange arrangement may be that the process of transduction – the turning of light energy into electrochemical energy within the nervous system – is an operation that requires a good deal of energy, and that energy must be supplied by the blood supply. Clearly, blood can be delivered more easily to the back of the eye than to the surface. Interestingly, the octopus has a retina in which the receptors are at the front rather than at the back of the retina. Perhaps this shows that our own arrangement is an accident but, as we manage to see quite well despite all the gunge in front of our receptors, it was an accident not worth correcting. Actually, we still have a certain number of blood vessels that lie on the surface of the retina and, although we are normally unaware of them, we can make them strikingly visible (see Box 1.1). Notice that another penalty for having an ‘inverted’ retina is the presence of a blind spot in each eye



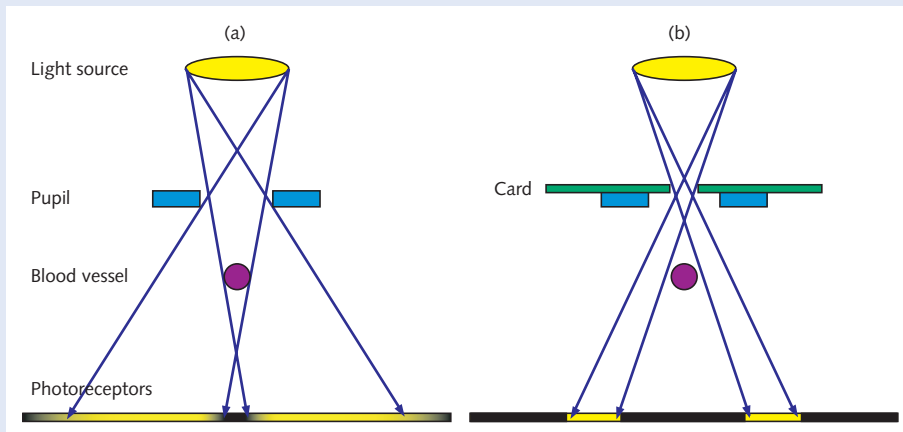
**Fig. 1.13.** A cross-section through the retina. Note that light would come from the bottom of the figure and would have to pass through the retina before reaching the photoreceptors. The photo, a scanning electron micrograph of primate retina, shows what the rods and cones really look like.

### BOX 1.1 Seeing your own blood vessels

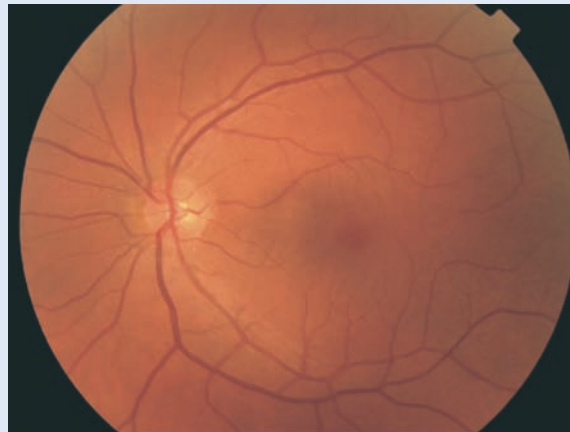
If our retinas are covered with blood vessels that lie between our receptors and the outside world, why can't we see them all the time? There are two key reasons; first, the blood vessels don't move and the visual system is notoriously poor at detecting anything that is stationary (or 'stabilized') on the retina. Second, the light reaching the retina is sufficiently diffuse for shadows not to form on the receptors (see Figure 1.1.1). We can overcome these problems quite easily and then we can see the shadows of our own blood vessels. Follow these instructions carefully and make sure you carry out step 2 before step 3:

- 1 Take a piece of stiff paper or card
- 2 Make a small hole in the card with a drawing pin
- 3 Close one eye and raise the card to the other
- 4 Jiggle the card slowly around close to the surface of the cornea while looking at a well-illuminated blank surface.

You should see a pattern of shadows that resemble the veins on a leaf (see Figure 1.1.2); these are the shadows of your retinal blood vessels. Note that there is one area where there are no shadows – the point where your vision is focused. This is because there are no blood vessels covering the retina at the fovea, the area of our vision where we have our highest acuity.



**Fig. 1.1.1.** (a) As the blood vessels are small in comparison to the size of the pupil, and are quite distant from the photoreceptors, they cast little shadow. (b) The introduction of a small aperture – the pinhole in the card – will result in a shadow of the blood vessel falling on the photoreceptors.



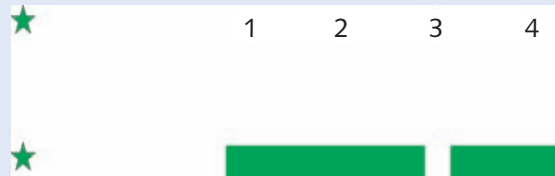
**Fig. 1.1.2.** The pattern of blood vessels over the retina, as seen through an ophthalmoscope. Note that the fovea (centre right) is free of blood vessels and the optic nerve head or 'blind spot' (centre left) is clearly visible where the blood vessels converge and leave the eye.

where the neural bundle passes through the rest of the retina (see Box 1.2). If a creature only had one eye this would be bad news as predators could learn to creep up on it from the blind direction. Having two eyes with blind spots pointing different ways solves this problem. Perhaps this is why, as a rule, animals have two eyes, exceptions being copopods (which are small plankton-like things and not a breakfast cereal) and the cyclops (the

**BOX**  
1.2**The blind spot**

Each of our eyes has a blind spot – the region where all the stuff gets in and out of the retina and therefore has no photoreceptors – and therefore no vision.

Figure 1.2.1 should help you to see your blind spot (or not see if you see (or not see?) what we mean). Close your left eye, and look at the upper green star with your right eye. One of the numbers should completely disappear! If you move your head back and forth (no sniggering) you should find that a number nearer to the star disappears when you are closer to the page. King Charles II of England (the one who hung out with Nell Gwynne, the girl with the oranges) used to play a little parlour game where he lined up the heads of people in his court with his blind spot, and thus saw them apparently beheaded. He was clearly interested in such matters since his father, Charles I, had been beheaded in a rather more permanent way.



**Fig. 1.2.1.** Seeing the blind spot.

Now move your head until the number 3 disappears and then look at the lower green star. You should find that the gap in the line disappears – but what is more it is replaced with a green line! So what is going on? It seems that some ‘filling in’ process must occur. The blind spot takes on the characteristics (such as average colour and brightness) of the area around it, and can even extrapolate the existence of lines. Two hypotheses may explain what is going on. The easiest explanation is that as we can’t see in this spot we automatically see what is around it by default. Alternatively there may be some active process that extrapolates what is seen around this spot and expands it to cover the blind spot.

The same lack of awareness of a blind spot occurs if for some reason some part of a retina stops working (as can happen as a result of retinal detachment). Although there is now an extra ‘blind spot’ (known as a scotoma), the person is often unaware that there is no vision in that part of the visual field. In fact, this can be rather bad news. Patients with glaucoma (see p. 23) can lose most of their peripheral visual field, but are often unaware of this until the loss is pretty much total. How come? Nobody is quite sure but it does seem that brain processes try so hard to ‘fill in’ missing information that the person with glaucoma is unaware that their visual world is disappearing.

most famous of these, Polyphemus, ended up with a somewhat larger blind spot that he bargained for.)

Once the light reaches the outer segments of the receptors, neural processing can begin. The receptors are connected to **bipolar cells** and these in their turn synapse with

**retinal ganglion cells.** The ganglion cells are important, for it is their axons that carry information from the eye on its journey towards the visual cortex. Connecting across the retina laterally are two further cell types – **horizontal cells** at the point where the receptors synapse with the bipolar cells and **amacrine cells** where bipolar cells synapse with the ganglion cells. For our purposes we need to know little about bipolar, horizontal, or amacrine cells, but we do need to know a little more about receptors and ganglion cells.

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## The photoreceptors

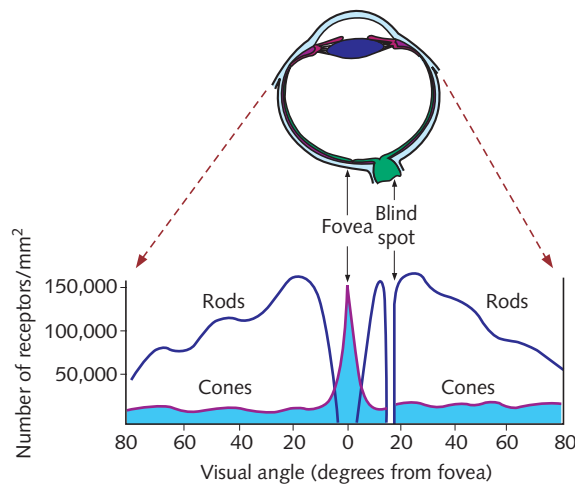
There are ten things you should know about the photoreceptors:

- 1 There are two types of photoreceptor in the human eye, **rods** and **cones**. The outer segments of the rods are generally rod-shaped and the outer segments of the cones are generally cone-shaped (Figure 1.13).

- 2 All our rods are basically the same; they all contain the same photopigment (the stuff that absorbs the light) in their outer segments. This is called **rhodopsin**. Rhodopsin is purple in colour and is known (not unreasonably) as ‘visual purple’. Of course the colour of an object tells us about the light reflected from it rather than the light absorbed by it. Rhodopsin reflects much red and blue light (hence the purple), but it absorbs green light preferentially.

- 3 Cones come in three main sorts, often (wrongly) called red, green, and blue. So-called ‘red’ cones contain a photopigment that is most sensitive to long wavelengths of light, ‘green’ cones are most sensitive to middle wavelengths of light, and the ‘blue’ cones to shorter wavelengths. So we should call the cones ‘long-wave’, ‘middle-wave’ and ‘short-wave’, but this only complicates things so we’ll call them red, green, and blue. It is because we have three cone types that we have colour vision. This is covered more fully in Chapter 5.

- 4 Rods respond very well to extremely dim light and are therefore very useful in dim conditions – i.e. at night. As the light level increases, so does activity in the rods. Because rods are so sensitive, they will be responding as much as they can when the light is still quite dim, so they are completely useless in full daylight. Cones, on the other hand (well, not really on the other *hand*), are much less sensitive, hence are not used under dim conditions but are the ones responsible for most of our daytime vision. We have all had the experience of going into a dark cinema from a bright day and found ourselves groping in the dark before sitting in a stranger’s lap. Some minutes later we can look around and see quite clearly. When we enter the dark cinema there is not enough light to get our cones going, and we have to rely on our rods. Unfortunately our rods have just been outside in bright sunlight and are completely bleached. It takes some minutes in the dark for the rods to recover their sensitivity. When only rods are active we call our vision **scotopic**,



**Fig. 1.14.** The distribution of rods and cones across the retina. Note that, although cones are most densely packed in the fovea (around 150 000/mm<sup>2</sup>), even in the periphery there are still several thousand per square millimetre.

when it is so bright that the rods cannot function we call our vision **photopic**, and the region in between when both receptor types play a role is called **mesopic**. The issue of light and dark adaptation is covered further in Chapter 2.

5 Rods are most sensitive to green light, whereas the cone system overall is most sensitive to yellow light. Next time you are given a red rose in moonlight (and this will be sooner for some of you than for others) note the relative brightness of the red of the flower and the green of the foliage. You should notice that the red of the petals looks very dark and the green of the leaves looks relatively light. Next morning compare the flower and the foliage again as the rose lies in the morning sunshine filtering on to your pillow; now the red flower will look lighter than the green foliage. This is known as the **Purkinje shift**. Don't mention this fact to your loved one if you want to receive red roses in the future. If you think the Purkinje shift is just slushy nonsense, then perhaps you will be more interested to know that in World War II night pilots were briefed in rooms illuminated by red light. This ensured that their rods, insensitive to the long wavelengths, were not being bleached and were, therefore, ready to spring into action the minute Biggles and his chums left the briefing room for the dark of the airstrip and their waiting crates.

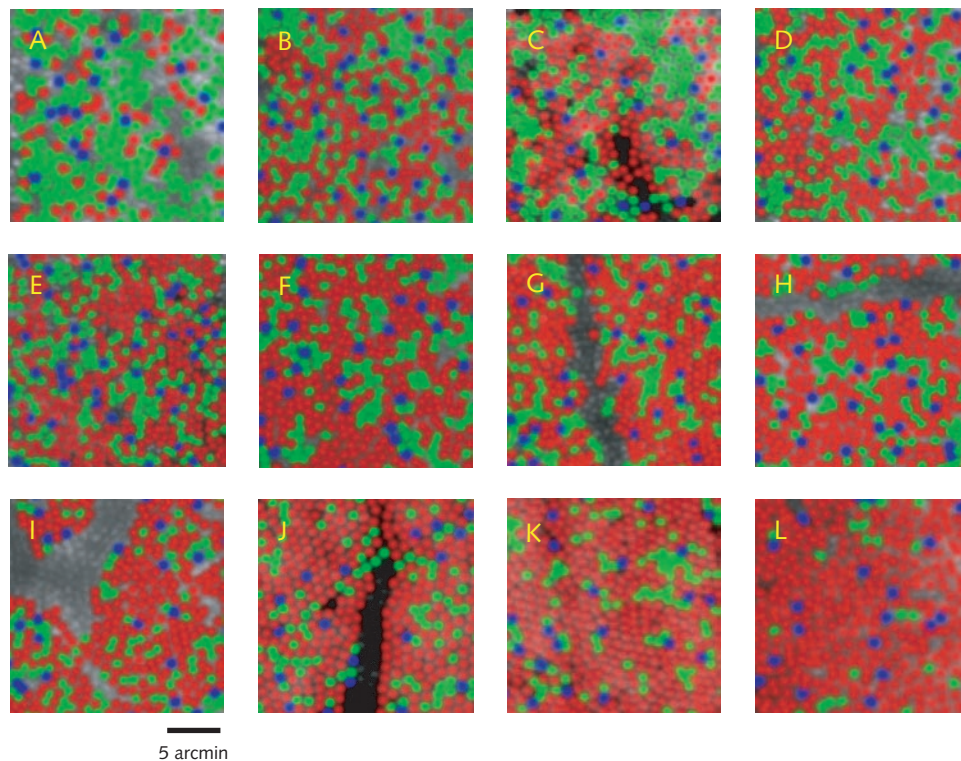
6 Photoreceptors are not evenly distributed across the retina. Cones are heavily concentrated in a central area known as the fovea (Figure 1.14). When you look straight at something, it is imaged on the fovea. Rods are completely absent from the central fovea and are most densely packed some 12–15° into the periphery (Figure 1.14). This explains why, when you look at extremely faint stars, they disappear when on the fovea but can be seen in the near periphery.

7 There are no blue cones at all in the central fovea – blue cones are rather strange things (and are only understood by rather strange people) and somewhat unlike either red or green cones. Interestingly, people with normal colour vision can have very different numbers of red and green cones (Figure 1.15).

8 Ganglion cell axons and blood vessels leave the eye at a point known as the **blind spot** or **optic disc**. This region, situated about  $12\text{--}15^\circ$  into the nasal retina, is devoid of all receptors and consequently we are blind in this area (see Box 1.2).

9 Congenitally colour-blind people have a problem with either their red or green cones. One of these cone types may be completely missing, or it may be weaker than is normally the case. Colour blindness is dealt with in detail in Chapter 5.

10 er...that's it.



**Fig. 1.15.** Images of the cone mosaics of 10 subjects with normal colour vision, obtained with the combined methods of adaptive optics imaging and retinal densitometry. The images are false-coloured so that blue, green, and red are used to represent the blue, green and red cones respectively. (The true colours of these cones are yellow, purple, and bluish-purple.) The mosaics illustrate the enormous variability in red/green cone ratio: the ratios are A, 0.37; B, 1.11; C, 1.14; D, 1.24; E, 1.77; F, 1.88; G, 2.32; H, 2.36; I, 2.46; J, 3.67; K, 3.90; L, 16.54. The proportion of blue cones is relatively constant across eyes, ranging from 3.9 to 6.6% of the total population. Images were taken either  $1$  or  $1.25^\circ$  from the foveal centre. For 2 of the 10 subjects, two different retinal locations are shown. Panels D and E show images from nasal and temporal retinas respectively for one subject; J and K show images from nasal and temporal retinas for another subject.

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## The retinal ganglion cells

The last layer of cells in the retina is known as the **retinal ganglion cells** (see Figure 1.13). We shall deal with some of the properties of these cells at length in Chapter 2. Here we shall simply note that they come in two varieties – large and small. We shall call the large ones **M cells** (M for magnocellular) and the small ones **P cells** (P for parvocellular). These cells differ not only in their size but also in their properties. Careful study has revealed that P cells distinguish between signals coming from the red cones and those coming from the green cones. So, for instance, some P cells will be excited by the red cones and inhibited by the green cones. The M cells don't seem to care about this distinction, and mix them up. Therefore only the P cells appear to carry the information about colour. On the other hand, the M cells appear much more suited to carrying information about the dynamic aspects of the world such as movements and flicker. Studying these cells is quite difficult, as they are all intermingled in the retina. But one thing we do know is that both types of cells send their signals to the next visual structure, the **lateral geniculate nucleus (LGN)**, but do so in such a way that all the M cells project to one bit of the LGN and the P cells project to other bits. The grouping of these cells allows us to lesion them selectively and discover what they do, something that we can't do in the retina. We shall therefore pick up this story again shortly when we get to the LGN.

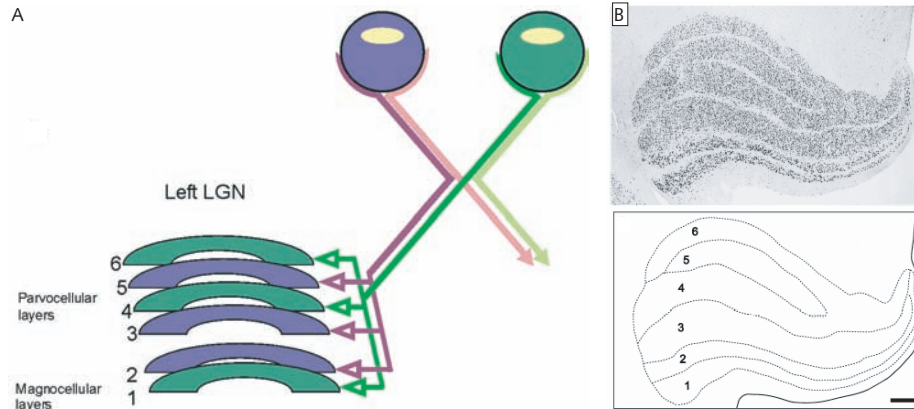
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## Beyond the eye – the optic nerve

The optic nerve leaves the eye at the blind spot and begins its journey to the cortex which, curiously, is about as far from the eyes as it's possible to get and still remain inside the head. The main pathways are shown in Figure 1.16. First the optic nerves from the two eyes converge at a point called the **optic chiasm**. Here a partial decussation occurs; this means that some of the fibres cross over to the other side of the brain and some don't.

The axons from ganglion cells on the nasal side of each retina cross and those from the temporal (i.e. where your temples are) side of each retina don't. The result of this is that now the left-hand bunch of nerve fibres carries information about the right-hand part of the world (the right visual field) and the right-hand bunch of nerve fibres carries information about the left visual field. At this stage the optic nerve changes its name to **optic tract**. You can see that chopping through your optic *nerve* will blind you in one eye, whereas chopping through your optic *tract* will leave you unable to see half the world – this is known as a **hemianopia** (see Chapter 11). This crossing-over of sensory pathways is fairly general; you may know that someone who has a stroke on one side of the brain may experience problems with feeling and moving their limbs on the other side of the body.





**Fig. 1.17.** The LGN has six layers, three from each eye. Layers 1 and 2 are the magnocellular layers, so called because they have bigger cell bodies than the upper layers.

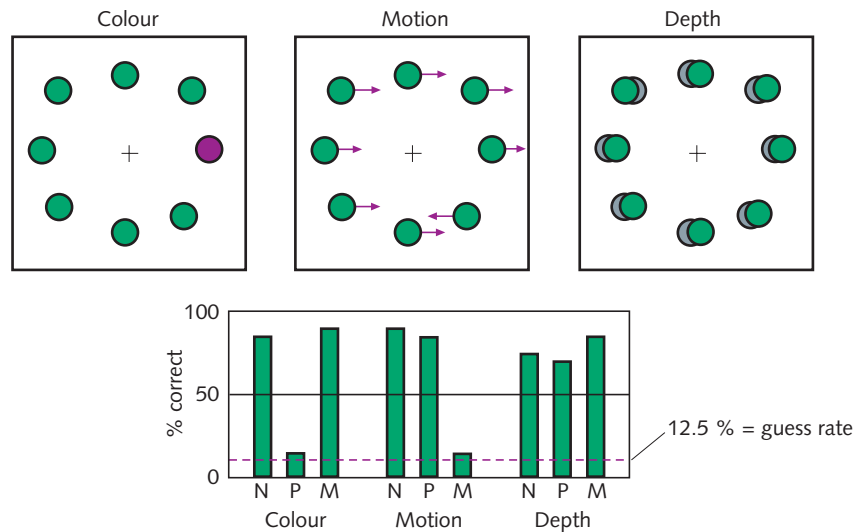
6 (contralateral) appear to be just the same as layers 3 and 4 respectively, so they seem to be redundant. So moving from layer 1 to 6 the order is contra, ipsi, ipsi, contra, ipsi, contra. This seems a bit odd, and certainly hard to remember. At least two very well-known authors get the order wrong in their books, but it would be ungentlemanly to identify them (Hubel, 1988, *Eye, brain and vision*, p. 66; Wandell, 1996, *Foundations of vision*, Figure 6.3).

In each of the LGN's six layers the cells retain what is called **retinotopic mapping**. This means that ganglion cells adjacent in the retina (and therefore also from adjacent directions in the visual world) will project to cells adjacent in the LGN. Hence it forms an orderly map of the visual world, where adjacent cells receive information from adjacent part of the image until all the image is covered. So each LGN has six maps of the world (one in each layer), one on top of the other. Of course, each map is of only half the visual world, the left LGN representing the right visual field and the right LGN the left visual field. The layers of the LGN have been described by one of the greatest visual neurophysiologists, David Hubel, as being like a club sandwich. This is a very useful analogy for many British vision scientists who previously had little idea of what a club sandwich looked like.

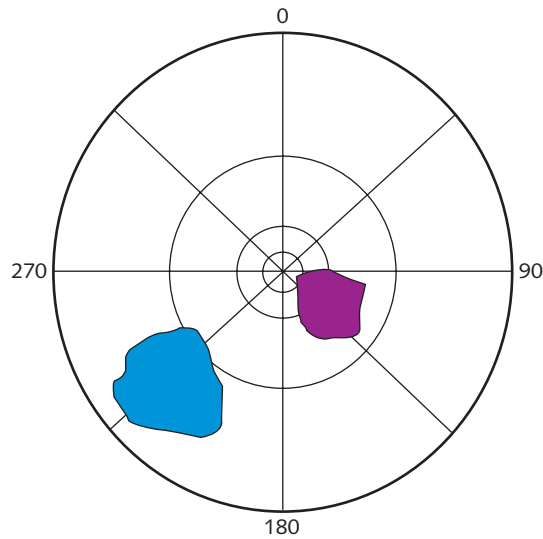
As we discussed earlier, the M and P divisions of the LGN receive their input from very different cell types in the retina. Remember that the M retinal cells seemed to carry information about movement and flicker, and the P retinal cells carry colour information. Do the M and P cells of the LGN have similar properties to the M and P cells of the retina? The answer seems to be yes. The experiments that have given us this answer selectively damaged (known as lesioning – see Chapter 12) either the M divisions or the P divisions alone and tested what happened to the vision of the animal. In one experiment the scientists trained animals to fixate a central point and then move their eyes to the 'odd one

out' on a screen (see Figure 1.18). They then took advantage of the retinotopic coding in the LGN by placing very small lesions in either the M or the P division. These small lesions produce only a small area of visual field where one might expect a loss of vision (according to where the map was damaged – see Figure 1.19) – the rest of the visual field was unaffected. They could now see how well the animal performed when a target stimulus was placed in the affected field and compared the performance with how well it did when the target was in an unaffected field.

They found that the lesions affected different targets in different ways. Lesions to the P stream destroyed the animal's ability to detect targets defined by colour, by fine detailed texture, or by a subtle change in shape; whereas targets defined by movement or flicker were impaired by lesions in the M division. Interestingly, other targets, such as those defined by a gross change in shape or by stereopsis (depth derived from having two eyes – see Chapter 7), survived either lesion – suggesting that information needed for these tasks could be carried by either division. From these and related experiments, we get the picture that the M stream carries information about coarse features and movement, whereas the P stream can carry information about finer features and colour. A nice way to remember this, or even visualize it, is that if we only had our M stream the world would look as if we were watching a rather badly tuned black-and-white TV – the detail and colour are lost. If we only had our P stream it would be like seeing a picture postcard – the colour and detail are there, but the movement is lost.



**Fig. 1.18.** Schiller *et al.*'s experiment. Monkeys lesioned in the parvocellular layers have impaired colour discrimination, but motion and depth discrimination remain intact. Magnocellular lesions impair motion, but not colour or depth discrimination.



**Fig. 1.19.** The lesions in Schiller *et al.*'s experiment were restricted to either parvocellular (purple) or magnocellular (blue) areas of the LGN. The areas of the visual field affected by the lesions revealed the effects of losing one pathway or the other.

Until fairly recently, this story about the M and P streams seemed complete. However, it turns out that there are other cells, mainly situated between the layers, that are also important for vision. These small cells are known as the **koniocellular cells** (meaning 'sand-like') or **K cells**. There are about 100 000 of them in your LGN (about the same number as the M cells) and their input comes from a special type of ganglion cell in the retina. This type of retinal ganglion cell has a major input from the blue cones, and hence it is believed that the K pathway is heavily involved in a form of colour vision that is based on a blue–yellow comparison, whereas the colour information carried in the P pathway is based on a green–red comparison. However, we shall save the details of this exciting story for the chapter on colour vision.

So the properties of the LGN cells look very much like the properties of the retinal ganglion cells. The obvious question to ask now is: what is the LGN doing, then? Unfortunately there is, as yet, no clear-cut answer, but intriguingly the strongest input into the LGN is not from the retina but from the cortex itself – the very area to which the LGN sends its output. That is, the biggest input to the LGN comes 'top-down' rather than 'bottom-up'. This has led to the idea that the LGN might be important in filtering what information gets through to the cortex – in this analogy the LGN serves as the spotlight of attention, highlighting information coming from certain bits of the visual field. However, there is little actual evidence for this, and for the moment the LGN remains, in the words of one eminent vision scientist, the 'Club Sandwich Mystery'. Information from the LGN is sent on to the cortex itself, but that's another story, one that will have to wait until Chapter 3.

**BOX 1.3** Dyslexia and the magnocellular pathway

People with dyslexia have failed to learn to read efficiently, although other functions are preserved. For instance dyslexics often jumble the letters in a word, and could therefore spell 'dyslexia' as 'dailysex' (this may also cause dyslexia, but the authors have insufficient experience on this matter). If you have ever gone to a toga party dressed as a goat, there's a good chance you are dyslexic.



Recently it has been suggested that some of these reading problems might occur because of damage to the magnocellular pathway. The LGNs of dead dyslexics (rest assured they departed from natural causes rather than from an over-zealous vision scientist) have been found to have magnocellular layers much smaller than those from normal readers (who were also dead). As the magnocellular pathway seems to be heavily involved in our perception of motion (see also Chapter 6) this suggests that dyslexics should also be poor at motion perception tasks. Recent work has measured various aspects of motion perception in dyslexics, and has shown such a deficit. For instance dyslexics need a much greater difference between the speeds of two patterns in order to spot this difference – and their motion area of the brain (area MT – see Chapter 6) shows reduced activation in brain imaging studies. Perhaps you should be wary of having a dyslexic as your designated driver!

Although this theory seems attractive, it is not without its problems. Why should a problem in the magnocellular system produce a selective deficit in reading? One idea is that the magnocellular system is important in refreshing the image each time your eyes move, and if this replacement of one image by the next is not working properly then successive images from one fixation to the next become confused – and reading is difficult. But then why should dyslexics find spelling difficult?

A second problem is that for every experiment that has found a visual deficit in dyslexics there seems to be another reporting no difference (whether this is due to M cell function or not), hence the presence of such deficits is not yet fully established. Thirdly, if a test only measures one aspect of vision (e.g. motion perception) and finds a deficit, how do we know that other aspects of vision are not also affected? Perhaps dyslexics are just not good at vision tasks in general.

## ● READINGS AND REFERENCES

Most ophthalmology books will give you more detailed information about the eye itself and about problems such as short-sightedness, but we find Davson (1990) nice and straightforward. The retina will be covered in most textbooks but the book by Dowling (1987) is a delight and well worth a look if you need to know what all those other cells that we ignored do, or how the receptive fields of the ganglion cells are constructed. The LGN seems to receive scant coverage in most books – perhaps reflecting our puzzlement as to just what it is doing. For many fascinating insights into the eyes of many other creatures, see Land and Nilsson (2001).

### *Papers on specific issues*

**Evolution of the eye** How our eyes (and those of other animals with very different eyes) came to be the way they are is covered by Gregory (1967, 1997). The evolution of the eye has often been held up by creationists to be impossible. For a view on just how easy it could have been, see Nilsson and Pelger (1994) and Dawkins (1995).

**Pupil dilation, etc.** For a series of fascinating insights into when and how our pupils change size, see the classic work of Hess (1965). More recently, pupil size has been used to try to detect deception too – see Lubow and Fein (1996).

**Rods and cones** For the actions of rods and cones, Dowling's book mentioned above is excellent. For a little more detail see also Daw *et al.* (1990).

**M, P, and K cells** The lesioning studies of the M and P divisions of the LGN were performed by Schiller *et al.* (1990). However, what appear to be very contradictory results can be found in Livingstone and Hubel (1988). See also Chapter 11, where we follow these pathways as they reach deep into the cortex. For information on the koniocellular pathway, see Hendry and Reid (2000).

**What LGN cells might actually be doing** There have been many theories that have tried to give a specific function to the LGN (rather than just passing on information from the retina to the cortex). One of the most recent and interesting is by Sherman (2001).

**Dyslexia and visual problems** There has been an explosion of interest in the idea that a specific visual problem might underlie the problems of a person with dyslexia. Many papers exist on this still controversial area. To get a flavour of the evidence for and against, see Stein *et al.* (2000) and Skottun (2000) respectively.

### ● POSSIBLE ESSAY TITLES, TUTORIALS, AND QUESTIONS OF INTEREST

- 1 Discuss the differences between rods and cones – what makes each suited to vision during night and day?
- 2 What are the functions of the M and P subdivisions of the LGN? How do we know this?
- 3 How and why do the retinas of animals change with their lifestyles?
- 4 Can dyslexia be explained by a selective loss of M cell function?

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