GRESHAM

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EXPLORING THE BRAIN

Lecture 8

SEEING AND HEARING

by

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EXPLORING THE BRAIN: SEEING AND HEARING

For us humans, most of the information about the outside world comes from what we see and hear. In simpler nervous systems with much simpler lifestyles there is no need for a rich tapestry of visual scenes which we enjoy. A frog would benefit little from being able to distinguish the fine detail of the Mona Lisa, for example. In a frog world, all a frog wants to know is if there are predators or prey: its retina has accordingly become adapted to be sensitive only to shadows, which would be cast by predators flying over, or prey that it can eat, namely flies moving backwards and forwards. Fine detail of objects is irrelevant and therefore not registered by the frog's eyes: when presented with a piece of cork dangling on a string, crudely resembling a fly flitting past, then a frog will make all the predatory and gustatory movements not only of sticking out its tongue to catch the fly, but of licking it lips as well.

As a general rule in the animal kingdom, the more complex or relatively big the eye in relation to the body, then the smaller the rest of the brain. More processing will go on at the earliest stages, in the peripheral organ, than in more sophisticated brains where the input will not have already been heavily biased. Insects have 'compound' eyes, which look a little like geodesic domes arranged on each side of the head. Each eye consists of ten thousand distinct modules which amounts to ten thousand facets all angled in different directions. Some insects have up to thirty thousand such facets. Light is funneled through each module, so that there is a huge magnification. However the results, in human terms, would be far from ideal, as the lenses of these facets cannot be focused. The huge advantage for the insect however is that a large visual field is projected on to a small number of cells without the need for the insect to move its head. The more facets, the more detailed the picture. This type of eye has the advantages of being very sensitive to any change in the visual scene, and to the planes of light polarisation: however the compound eye cannot give a high degree of resolution, of accuracy.

The human eye is very different: it is ball-shaped and consists of two main sections, separated by the lens. The lens is a transparent, elastic convex structure that is suspended by ligaments to control its shape, and this shape can change from one moment to the next according to whether you want to look a long way off or alternatively, only a short distance. Together with the cornea at the very front of the eye, the lens helps us to focus. The coloured iris, which varies so much between individuals, regulates light by constricting or dilating the pupil. The gap between the cornea and the lens in this front part of the eye is filled with a watery fluid. By contrast, in the second compartment comprising the main body of the eye, the cavity is filled with a jelly like substance.

At the very back of the eye is the retina, which is the image-detecting zone. If you look at the retina under a microscope, there are a tangle of cells seemingly forming a jumbled mass that looks a little like a net: hence the name 'retina', from the Latin for net, 'retus'. These cells in the retina respond to changes of light by a change in an electrical signal. This change in electrical signal is passed on to two more relays of cells before being transmitted on into the brain proper, via the bundle of fibres known as the 'optic nerve'.

The exit point where this nerve leaves the retina and burrows into the brain is the so called 'blind spot': this is the site where the nerve is leaving the retina, so there is obviously no room for any light-sensitive cells, nothing can be sensitive to light, and thus nothing can be seen. This blind spot is just to the side of the middle of the eye, near where the nose is. On the other side of the middle of the eye, to the side where the ear is, is an area of the retina called the 'fovea'. The fovea is a small indent where there is a high concentration of a certain type of cells that are sensitive to light. So if light strikes this area then it will be here where vision is optimal as there are more cells to do the job. Birds of prey can have up to five times more concentrated cells in their fovea than humans. In addition, unlike us humans, eagles have two foveas. One, the 'search fovea' is for vision sideways, whereas the other, the pursuit fovea, functions in judging depth , which is done with both eyes.

Another difference is that unlike human eyes, all bird eyes are fixed in their sockets. Thus in order to turn their eyes, birds have to turn their entire head and neck. Our lifestyles would be drastically compromised if we were unable to move our eyes back and forth without moving our heads: imagine reading for example! For both the eagle and us however, light (electromagnetic waves) travels through the eye ball and penetrates the outer two layers of the retina to be first processed by the light sensitive cells. The particular cells sensitive to colour are known as 'cones', whilst the other type of light-sensitive cells are referred to as 'rods'. These rods are for vision in darkened conditions, leaving three types of cone to respond principally to one of three primary colours: red, green and blue. Within the electromagnetic spectrum, our human eyes detect only a very small section as visible light, between 400 and 700 nanometers on a scale varying between 10 metres, the wavelength of an AM radio, to less than a nanometer, the wavelength range in which we find X-rays and gamma rays.

But how is light actually registered in the brain? It must first be converted, by the retina, into electrical impulses. In the dark there is a steady release of a chemical messenger from the rods onto the next relay of cells along in the retina. When light first strikes, it is absorbed by a special chemical ('rhodopsin') within the rod. The ensuing change in this chemical caused by the absorption of light then triggers a cascade of chemical reactions inside the cell. The end result of these reactions within the rod is a change in its electrical properties.

It is this change in electrical properties, namely the voltage normally generated by the rod, that will change the message it has been transmitting as long as there was darkness. In the case of the other type of light sensitive cells, the cones, we start to process colour by the selectivity of different cones responding to certain ranges of light with peak sensitivities for red, green or blue wavelengths. Different colours will excite different combinations of these cones in different proportions. For example a certain wavelength will excite red and green cones in equal numbers and be perceived as yellow.

So far then we have seen that light, electromagnetic waves, are converted by cells in the retina into electrical signals. However the retina does not just signal uniformly and equally about everything in your visual field. The image is relayed on into the brain having been processed with an enormous bias. If for example, there is a large uniform area within an object, then only weak signals are passed on. Whereas if there is contrast, edges, then the visual signals will be most vigorous. So unlike a camera, the retina is really only concerned with change. But change does not just occur in space, with contrasting edges, there is also change in time, namely movement. The retina can adapt so that it no longer responds to stationary objects, whilst still retaining the ability to signal for movement. Only think of why a flashing light is more noticeable and hard to ignore than a static, steady light, to appreciate the preference of the nervous system for states of change. After all, our survival may well depend on a change in the situation around us far more than if everything has remained the same.

But the eyeball itself is not a self-contained centre for vision: rather it is the gateway by which the all-important signals gain access to the brain for further processing still, before we can actually 'see'. From the retina, cells send out electrical signals along the fibres exiting via the blind spot, deep into the brain, to a region named after the Greek for room, the thalamus. This brain structure, which occupies a substantial part of the middle section of the brain (the 'diencephalon'), then relays the signals on to a committed region of the cortex, the outer layer at the back of the head, the 'visual cortex'. There are certain cases of people that unfortunately have had loss of certain specific parts of the visual cortex, thereby giving neuroscientists some very helpful and intriguing insights into understanding what might be happening there to enable us to see.

For example a lady in her forties, due to a stroke, had cells damaged in a highly localised region within the visual cortex: but she could still see normally. The interesting aspect of her condition was that although she could see all stationery objects as well as anyone else, she was unable to see objects in motion. If, for example, she poured out tea, it apparently seemed frozen like a glacier. Indeed she was unable to engage in this activity because she could not stop pouring: she could not see the level of the fluid in the cup rising sufficiently well to know when to stop. This patient also said that when she spoke to people, conversation proved a problem because she was unaware of the movements of the mouth of the speaker. Even worse, and more dangerous, was when it came to crossing the road. She was unable to monitor the progress of a car: first it was one place and then suddenly it was almost on top of her. On the other hand, its not as if this lady had a problem in detecting movement generally, because when movement was presented to her through her sense of sound or touch, then she could indeed detect it as moving.

So it seems that although this particular patient could see colour and form, she was unaware of movement. Comparable situations have been reported since the First World War, when many more people came to display the consequences of head injuries, due to the wounds of battle. A physician of the time, George Riddoch, made a study of these patients: he reported that there were people who could see movement, unlike the lady we have just been discussing, but no other attribute at all. So in these cases, patients could see movement but no shape or colour. Often anyone with normal vision can experience this phenomena: if something moves in one's extreme peripheral vision, one is aware of movement, but you then need to turn your head in order to 'see' exactly what it was that moved.

Similarly there are those who can see form and can see movement, but who cannot experience colour. The whole environment to them seems a monochromatic backdrop. A world composed entirely of shades of grey occurs when people have damage to critical regions on both sides of their head. But if, on the other hand, the brain is only damaged on one side, then half the world appears in colour and, bizarrely, half the world in black and white.

Finally, some patients with damage to the visual system can see movement and colour, but no form. Here, it is possible to 'see' objects but not recognize what they are, a condition known as 'agnosia' (from the

Greek, literally, a 'failure to recognize'). Agnosia can vary in its severity from patient to patient, and even the same patient will have better 'form vision' at some times compared to others. One reason why this particular condition can be so variable has been suggested by the vision expert Semir Zeki: if complex forms were to be gradually assembled in our brains from less complex patterns, then perhaps this gradual process of construction could be arrested at different stages in different people. Some patients would thus have a more extensive visual repertoire than others. Zeki suggests that understanding and seeing are not two separate processes, but rather that the two are inextricably linked: if you see something you will automatically understand what you are looking at. On the other hand, if you do not 'see' an object in front of you, Zeki argues that it is because there has been a collapse of the higher integrative processes for complex form recognition in the visual cortex. Obviously, you will not 'recognize' the object. You will be to a greater or lesser extent 'form blind'.

Thanks to such cases as these, it is evident that vision of form, movement and colour can occur independently of each other. Current thinking then is that we process vision at least partly 'in parallel', that is to say we are processing visual signals simultaneously but in different parts of the brain. Different aspects of our vision, form, colour, and movement seem to us a cohesive whole, but are actually processed, at least in part, by different systems connecting up, through relays, from the retina to the back of the head. Exactly as we saw for movement then, we can see that different regions of the brain are working together to contribute to what we regard as a single 'function', in this case seeing. But the big mystery is how does it all come together again? Where in the brain do we make all the parallel visual signals converge into a single entity?

Some people have suggested that there is a convergence of these different pathways in certain parts of the brain, like railway tracks leading into Grand Central Station. This scenario, almost an effective 'Vision Centre' is, in a sense, almost a late twentieth century version of the doctrine of Phrenology which we met in the previous chapter. But just imagine that we possessed the brain equivalent of one or two Grand Central Stations in our heads: if such an area were damaged, then it follows that vision would be completely lost. But this scenario never occurs. So here is yet a further example of how the brain is not organised as a simple bundle of mini-brains. The connections between brain regions are not directed to converge into an executive centre, but are more likely to take the form of balanced dialogues between key brain regions, just as can occur also for the control of movement.

What about hearing? Instead of light, the incoming signal referred to as ‘sound’ is caused by vibrations in the air, ‘sound waves’, which are funneled into the ear and ultimately converted into electrical signals. The outer ear, or auricle, ensures that sound waves are compressed into an outer canal about an inch long. At the opening of this canal are wax and hairs which trap foreign matter and thus keep the inner parts of the ear clean. The auditory canal is separated from the middle ear by a thin membrane, the eardrum. In the middle section of the ear, behind the eardrum, there is an air filled cavity with 3 small bones, named after their appearance: hammer (malleus), incus (anvil) and stapes (stirrup). Vibrations of the eardrum cause these bones to move and push against another membrane, the 'oval window' which in turn causes motion of fluid in the inner ear. This motion of fluid activates hairs on cells in that 'cochlea', which then activates the cells to pass on electrical signals into brain.

Whereas in all animals, the principles of converting changes in air pressure to changes in electrical energy, always hold, there are crucial differences to accommodate the range of very different lifestyles across the animal kingdom. For example, dolphins have two kinds of receiver, one in the jaw through which travels in a thin oil to the eardrum, and the other in an oil filled 'melon' in the forehead. The difference in density between the flesh and water is so slight, that sound is not stopped by surface of the body. Another important difference for marine creatures and ourselves is that water is denser than air. Hence sound disturbances compress and relax more readily: sound travels five times faster and much further in the sea. For example, whale sounds have been heard 15 miles away from the nearest visible whale.

Another well known example of a special type of hearing is supplied by the bat. Bats use a sonar system to hunt and avoid objects in the night. Normally, a bat emits four to five pulses a second, but when it detects an obstacle or target, this rate is stepped up to 200 pulses a second. The sound bounces off the object and reflects back to bat. The frequency range for bat hearing (25,000 to 100,000 Hz) is well beyond our own modest 15,000Hz. Indeed, measurements of men in their forties show that the sensitivity of the ear falls 80Hz every month!

From the cochlea, electrical impulses are relayed up to a special part of the outer layer of the brain, the 'auditory cortex'. However we still do not know why electrical signals arriving in this part of brain are experienced as sound, whereas those arriving in other parts of cortex are perceived as vision.

For all the senses, there is also the enigma of the nature of the first person subjective conscious element. There is much more to hearing for example than mere vibrations. We do not hear a symphony as vibrations, any more than we see a face as lines and contrast. Rather, our perceptions are unified wholes, shot through with memories, hopes, prejudices and other internalised 'cognitive' idiosyncrasies.

Another tantalising and related mystery of the brain is why electrical signals arriving in the visual cortex should be experienced as vision, whilst exactly the same kind of electrical signals, arriving in another part of the brain such as the auditory cortex, should be perceived as hearing. No one has yet given a satisfactory explanation: one idea, however, is that we learn through experience to distinguish sound from sight, whilst another idea is that each sensory system is linked preferentially in some way to certain types of movement, which emphasises the distinction.

On the other hand, there are well known examples of where this distinction between the senses falls down, a mixing of the senses known as 'synaesthesia': people displaying synaesthesia may claim for example to 'see' certain musical notes in certain colours. Virtually any combination of two of the five senses is possible, although it is the experience of different colours on hearing different sounds, that is the most common. Synaesthesia tends to occur more in childhood, but can often be triggered in adults with psychotic disorders such as schizophrenia or by hallucinatory drugs. The division of the senses then is clearly attributable to some aspect of normal brain organisation, but an aspect that is not immune to individual perturbation. One possibility is that there are additional connections in the brain of the synaesthetic that extend not only from the sense organ in question to the cortex appropriate for that modality, but which also innervate another cortical sense area as well. This idea however is not very likely as it would not account for the variability in synaesthetic experiences, namely that such states only occur under certain conditions. More likely is that the areas of cortex not allocated to the primary processing of each of the individual senses, namely the association cortex, somehow plays a part.

We saw in Chapter 1 that even compared to the brain of our nearest relative the chimpanzee, the areas of human brain classified as association cortex, are vast. It is possible that inputs from association cortex into the areas of cortex devoted to particular senses might in some way, at some times, be aberrant. Certainly, such a scenario would account for the greater predominance of synaesthesia in children, before distinctions between the senses are 'learnt', and where the neurons of the brain are less 'hard wired' and thus more flexible and versatile in their operations. A malfunctioning of physiology (the working of neurons) rather than anatomy (their physical connections) would also explain why synaesthesia can suddenly appear say, in the brain of a schizophrenic. On the other hand, any real explanation of synaesthesia is impossible as it hinges on a subjective perspective, the first hand experience of an individual which we cannot share, but at most be told about. Synaesthesia is a facet of consciousness, that ultimate riddle of the brain.

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