

## Chapter 2

### The brain

#### Learning outcomes

By the end of this chapter you should:

- have a good understanding of the developmental processes of the brain;
- have the ability to identify some of the major structures and views of the brain;
- understand some of the basic psychological functions in the four main lobes;
- have developed your organisational skills.

#### Introduction

The nervous system is quite complex, with many sub-divisions within it, though the mechanisms of each of these divisions is very similar; all rely on neurons of one form or another, and the interaction between these neurons is crucial for the system to function. This similarity becomes even more obvious when nervous system development is explored – and having a good understanding of this process can help in understanding the complex interactions that are ‘hard-wired’ into our biological systems, so this is a helpful place to start.

#### The development of the central nervous system (CNS) and the brain

As any psychology student knows, humans are complex organisms, but they begin relatively simply as a single cell, the **zygote**. A zygote is the first incarnation of a new individual and is formed when fertilisation occurs (an ovum or egg joins with a sperm) through sexual reproduction. Ova and sperm are often referred to by the term **gamete**, which is simply a term for describing cells that join together during fertilisation. One of the important characteristics of gametes is that they contain only half the **DNA** of the parents – they are said to be **haploid cells**. So the sperm will contain half of your father’s DNA and the ovum will contain half of your mother’s DNA. On their own, then, gametes are fairly useless as they contain only half the DNA needed to make a person, but fusing them together during fertilisation results in a single cell with the full complement of DNA needed to make a person – this is referred to as a **diploid cell**. In essence, an individual begins with a single cell that is a combination of biological material from their mother and from their father. Everyone starts from this point, and a combination of the genetic material that this cell

contains and the life experiences that the organism goes through over time will determine the characteristics of the organism that is produced – Hitler and Einstein both started out this way, and so did you! What is important to note here is that individuals develop according to the effects of biology and environment together – not one or the other.

One big question is how complex life forms develop from one relatively simple cell, and the answer lies in cell division. Just 12 hours after fertilisation – the creation of the zygote – the zygote divides into two cells; this cell division continues exponentially from here, so the original single-celled zygote eventually becomes a mass of many cells and is termed a **blastocyst**, which will develop into the foetus.

The nervous system contains many different types of cell, such as different types of neurons, specialised to perform certain functions. The blastocyst contains embryonic stem cells, which are a single type of cell capable of developing into any type of specialised cell that is required – from specialised neurons to specialised heart cells and so on. This aspect of embryonic stem cells makes them incredibly important as they have the potential to grow into any specialised type of cell and could therefore be used for treating almost any disorder – you would not have to transplant a specific organ into a failing body and risk organ rejection if you could use stem cells to ‘grow’ that particular organ.

This property of stem cells in blastocysts means that a fully functioning human with very many specialised parts can grow out of it. As the zygote develops into a blastocyst and then into a foetus, this is exactly what happens (see Figure 2.1). At around 20–21 days after fertilisation, the cell division of the zygote results in the formation of a neural tube structure. One end of this structure will develop into the brain, and the other will develop into the spinal cord. Over the next few days some distinct structures of the brain emerge, growing out of the neural tube and future spinal cord. Initially these distinct brain structures are gross differences that roughly divide the brain into three divisions (see Table 2.1). At this point it is worth bearing in mind that some sources state that there are three major divisions, while others will state that there are four, as they divide the prosencephalon into two (the telencephalon and the diencephalon).

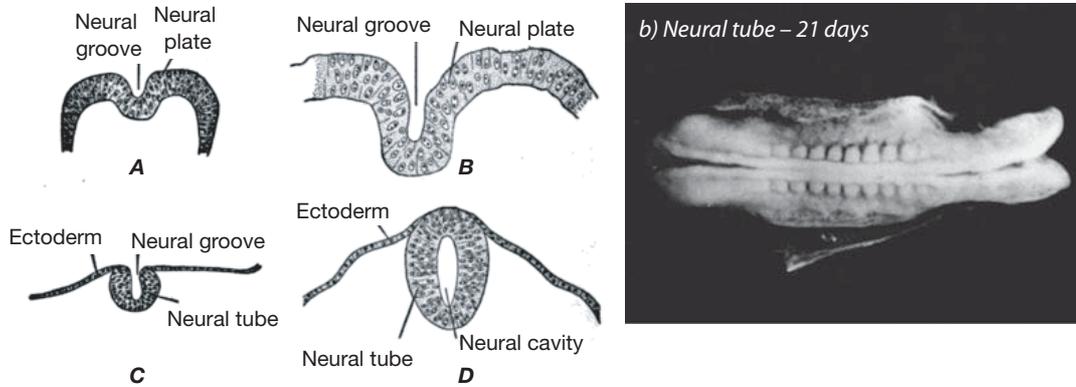
Each major division (however many you use) contains many smaller and more specific structures that are associated with particular brain functions, and these are discussed throughout this book. For now, though, we will look at how the brain develops its more intricate structures.

**Table 2.1:** Major divisions of the brain

<i>Also known as:</i>	
Prosencephalon (includes telencephalon and diencephalon)	Forebrain
Mesencephalon	Midbrain
Rhombencephalon (includes cerebellum, pons and medulla)	Hindbrain

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**Figure 2.1: Embryonic brain development**



*a) Cross-section of neural tube*



## Developmental stages

The gross morphology of the developing brain needs to be populated by neurons of different types and functions, and this population of the gross morphology takes place in several stages.

1. Neural proliferation.
2. Neural migration.
3. Neural differentiation.
4. Synaptogenesis.
5. Neuronal survival.
6. Synaptic survival.

It is important to note that these stages are not performed serially. Each stage does not need to be complete before the next one begins; all of these stages may be in process at any one time. Understanding the processes involved at each of these stages can give us a good insight into some of the power and flexibility of the brain, and it is worth spending some time understanding these.

### Neural proliferation

Through cell division, the foetus develops and grows by increasing the number of cells that it has. For the brain and nervous system, the cells of the neural tube divide and proliferate, giving rise to a large number of stem cells that will become different types of neuron or **glial cells** (see Chapter 3 for a more detailed description of glial cells). Once these cells have developed into neurons or glia, they no longer divide – only the neural tube stem cells can divide and proliferate. This fact led to an early idea that humans were born with a fixed number of neurons that could not be replaced or added to through cell division (Watson and Sterling, 1998) – essentially, you got what you were born with and no more. This is no longer considered to be accurate, as Shankle et al. (1998) demonstrated significant post-natal neuronal cell growth between the ages of 15 months and 6 years. So the neuronal proliferation stage, once thought to end around the time of birth, has been noted to continue up until the age of at least 6 years. In fact, a longer period of neuronal generation throughout brain development may have significant evolutionary advantages by encouraging diversity of functioning (Hoffman, 2010). That is to say, the longer that the individual can generate new neurons during their development the more likely they are to develop more specialised and more diverse neuronal systems, which can help the individual survive or adapt to their environment.

At this point you should also be able to appreciate some of the potential of harvesting or using embryonic stem cells for treatment purposes, particularly for diseases that involve neuronal

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degeneration, such as Parkinson's disease. If the disease process results in the 'death' of neurons, and neuronal generation ends relatively early in the developmental process – perhaps around the age of 6 – then it would be advantageous to be able to transplant stem cells into a diseased brain in the hope of these generating new, healthy neurons. The therapeutic effects of such therapy have, however, proved less than convincing after two decades of experimental treatments (Vidaltamavo et al., 2010).

### Neural migration

Once cells have been generated at the neural tube they have to move into place throughout the developing nervous system or brain. This is accomplished using a 'scaffolding' system of glial cells known as radial glial cells (Rakic, 2003). These glial cells radiate out from the centre of the neural tube (the ventricle) to the surface of the neural tube in a structured way and provide a 'scaffold' along which cells can migrate throughout the nervous system, 'crawling' along the scaffold toward the surface. As more cells migrate along the scaffold and arrive at the surface, the surface becomes crowded with more and more cells eventually resembling a complex mass of interconnected cells, which can be seen in a more mature brain. At this point the cells that have migrated along the radial glia remain undifferentiated – that is to say, they do not yet possess any of the specialised qualities of neurons found in certain regions of the brain or nervous system. These relatively basic cells that have completed migration need to undergo the next stage of development, which is differentiation.

### Neural differentiation

Neural differentiation is the process by which basic neuronal-type cells develop into the specialised types of neuron required by the area of the nervous system they have migrated to. So cells that have migrated to an area of sensory input into the nervous system (e.g. touch) may develop into unipolar (sensory) neurons; cells that have migrated to an area interacting with muscles may develop into motor neurons; and so on.

The interesting question is how such basic non-specialised cells develop into the 'right' kind of specialised neuron for the area of the nervous system they have migrated to. The answer lies in the genetic material in each cell. Gene expression moderates the development of the neuron, controlling the growth of axons (nerve fibres that project from neurons) and dendrites (nerve fibres that axons of adjacent neurons send signals to) from the initial cell. Gene expression is the term given to whether a particular gene has been activated or not.

Each cell at this stage is diploid, that is, it has a full set of DNA containing many thousands of genes, each of which has a specific function. If one of these genes is activated, it will set in motion a range of chemical processes that produce a protein, and this protein is used to create cell structures such

as axons and dendrites. So if a gene is expressed, or switched on, a protein will be created and perhaps a dendrite formed; if the gene is not expressed, no dendrite will be formed. In this way the complex morphology of a neuron is controlled.

So what determines if a gene is expressed or not? A complex number of factors are involved, and they occur both within and without the cell – the intrinsic and extrinsic cellular environments that play a role in gene expression.

- Intrinsic cellular environments are specific to each cell and usually relate to the levels of chemicals within that cell, the presence of glucose or oxygen, which may trigger gene expression.
- Extrinsic cellular environments refers to the environments outside the cell, which may be the presence of particular chemicals in connecting cells or, more broadly, the nutrient status of the mother, which can provide rich material for development or toxic materials such as alcohol, which may impede development.

In essence, then, for neuronal differentiation there is an interplay between intrinsic and extrinsic cellular environments. The correct chemicals need to be present in order to 'switch' the gene on, and, following this, the correct chemicals need to be present in order for the gene to be able to build a protein from these building blocks.

## Synaptogenesis

At this stage in the developmental process, cells have been created, they have migrated into position, and they have become differentiated in their morphology by growing axons and dendrites. The next stage is for these neurons to connect up to each other, and to other structures, and form synapses. In order to do that, the axons and dendrites of various neurons need to grow towards each other.

The tip of the developing axon or dendrite is referred to as the growth cone (Gordon-Weeks, 2000), and this tip has fine outgrowths on it. Some of these are hair-like structures known as filopodia, and some are sheet-like structures known as lamellipodia. Both filopodia and lamellipodia react to the extrinsic cellular environments around them, moving towards certain chemicals and 'dragging' the growing axon or dendrite behind it.

The source of attraction for the filopodia and lamellipodia are chemicals released from target nerve cells or structures (such as muscles) that may attract or repel the growth cones. As the growth cones detect the presence of a chemical attractant in the extrinsic cell environment, the growth cone will orient towards it and 'follow' the signal. The signal becomes more concentrated nearer to its source, so by following the concentration of the chemical attractant the growth cone will drag the axon or dendrite to its source, and once there a synapse will be formed. This is termed chemotropic guidance and it is in this way that many synapses may be formed.

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## Neuronal survival

During the development of the nervous system, large numbers of neurons are created, though not all of them survive. In fact, it has been estimated that between 20 per cent and 80 per cent of neurons may die in various locations in the nervous system (Toates, 2006).

In order to survive, a neuron must make a connection, or synapse, with another cell or structure. Once this connection is made, the target cell releases a neurotrophic factor that is taken into the neuron; neurons that receive such neurotrophic factors survive while neurons that do not receive the neurotrophic factor die.

The term neurotrophic factor is a term that refers generally to the life-giving chemical that is given to the neuron by the target cell, though this can take several specific forms. Perhaps the most common form of neurotrophic factor is nerve growth factor, also known as NGF (Levi-Montalcini, 1982). NGF is produced by the target cells but is taken up along the axon of the neuron into its cell body where it prevents the neuron from dying.

Cells which do not receive such neurotrophic factors die through a process of programmed cell death (Burek and Oppenheim, 1996), which is a genetically driven process of cell 'suicide', determined, in part, by the failure to make any synaptic connections or receive any neurotrophic factor.

At this point you should be able to see that the developmental process of the nervous system and the brain is a competitive one – many neurons are created, but not all survive. Competition is rife between the neurons to make synapses with other cells, so that they may receive a neurotrophic factor such as NGF and therefore survive in the first instance. Of course, neurons need to survive over a longer term too, which is where the next stage of development comes in.

## Synaptic survival

Making a synaptic connection with another cell and receiving an initial boost of a neurotrophic factor in order to avoid programmed cell death is fine in the short term, but the synapse needs to be maintained over the longer term if it is to be effective; it is no use having a synaptic connection that lasts for only a matter of days. So while the synapse needs to be made in order for the neuron to survive, the synapse must be an effective working connection if the synapse is to survive. An active working synapse will maintain a flow of the neurotrophic factor, stabilising the survival of the neuron and its connection, while an inactive non-working synapse will not allow enough of the neurotrophic factor into the neuron for it to survive.

In order for the synapse to survive it must be used by the organism on a fairly regular basis. Regular use reinforces the synaptic connection and ensures the survival of that connection and of the neuron itself.

While this is relatively simple to understand, it does have some important consequences. Imagine that the synapse in question is between the brain and a leg muscle, so it involves a motor neuron. If the organism can walk around activating the leg muscle repeatedly, the synapse will survive. However, if the individual is in an environment where walking is impossible (confined in a small cage, say), then any synapse between the brain and leg that occurs will not be reinforced through use and will eventually die. In this sense the environment can have a significant impact upon neuronal survival. The futility of arguing whether nature or nurture is responsible for any behaviour becomes obvious – nature and nurture work together in developing the nervous system and neuronal synapses.

The survival of synapses, and neurons, can be seen in terms of evolutionary natural selection – the survival of the fittest (Edelman, 1987). Only strong and useful synapses continue to exist, while the rest are 'pruned' as neurons succumb to programmed cell death and inactive synapses wither. In fact, the number of neurons in the brain doubles between the ages of 15 months and 6 years (Shankle et al., 1998) while the number of synapses increases from birth to a peak at the age of 3 years before being pruned again by the time of puberty (Bruer, 1998). In this way our development begins with an 'over-production' of neurons seeking to make synapses; however, many of these synapses will not be reinforced through interactions with the environment so they will 'die'. This ability to over-produce what is needed and then 'weed out' neurons or synapses that are not needed reflects just such an evolutionary process of development.

**Task** — Take some time now to go over the stages of development outlined above and review your understanding of them. Once you have an understanding of the mechanisms, consider what consequences this has for understanding how psychological functions develop.

Up to this point the chapter has focused quite intensely on the *mechanics* of the nervous system and brain, and it is helpful to pause here and reflect on the impact they may have on the *functions* of the nervous system or brain. You should be able to see that complex functions are not fully developed from the moment of conception or birth but rather develop over time from much simpler functions; for example, you cannot produce spoken language without first having motor-neuronal control of your lips, tongue, breathing and so on.

Genetics plays a role in providing the raw instructions for the development of the neurons involved, but the environment can help to generate, maintain or kill off the neurons and synapses that are produced. So two people may have motor neurone disease but for very different reasons; one individual may have an abnormality in a particular gene (SOD1, superoxide dismutase 1) that has produced an enzyme that is toxic to motor neurons; or the disease may be due to some environmental factor. Only around 10 per cent of cases of motor neurone disease are thought to have an inherited genetic cause (Siddique and Deng, 1996), and it may be that the toxic factor has been ingested via diet for some cases.

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## The adult brain

Having discussed how the brain starts out, let us turn to the brain that you may be more familiar with from its wrinkled image. It was tempting to call this section 'the fully formed brain', but that is very misleading as the brain is, in many ways, in continuous development, developing new synapses in response to the environment, repairing damaged tissue, and even possibly growing new neurons over time. This makes the study of the brain incredibly complex, as individuals' different life environments result in individual reinforcement of synapses and neurons, giving each brain an individualised content.

In many ways brains are like faces: most faces are the same in that they have two eyes, a nose, a mouth, ears, and so on, but each face is also unique. Just as most faces have commonalities in their structure so too do brains and we will discuss them here. It is worth noting at this point that in order to discuss the various aspects of the brain you will need to become familiar with some quite odd terminology that has come from a variety of sources (Greek, Latin, medicine, biology, to name a few). This also means that some parts of the brain have more than one name – one that was the 'original' and one that may be a more modern term – so when you go away to read around the subject be ready for the terminology to change from one book to the next, even though they are talking about the same thing.

## Views of the brain

One of the other things you will need to do is to learn to visualise the brain, so that you can interpret diagrams and photos that you will see. As the brain is a three-dimensional object you can look at it from different angles and directions, and these have been named to make interpretation of diagrams and photos a little easier. Table 2.2 lists some of these.

Not only do these terms refer to viewing angles of the whole brain, they are often used to refer to specific parts of larger brain structures. So the dorsal aspect of some structure will be 'the bit towards the top', while the posterior aspect of some structure will be referring to 'the bit towards

**Table 2.2:** Viewpoints of the brain

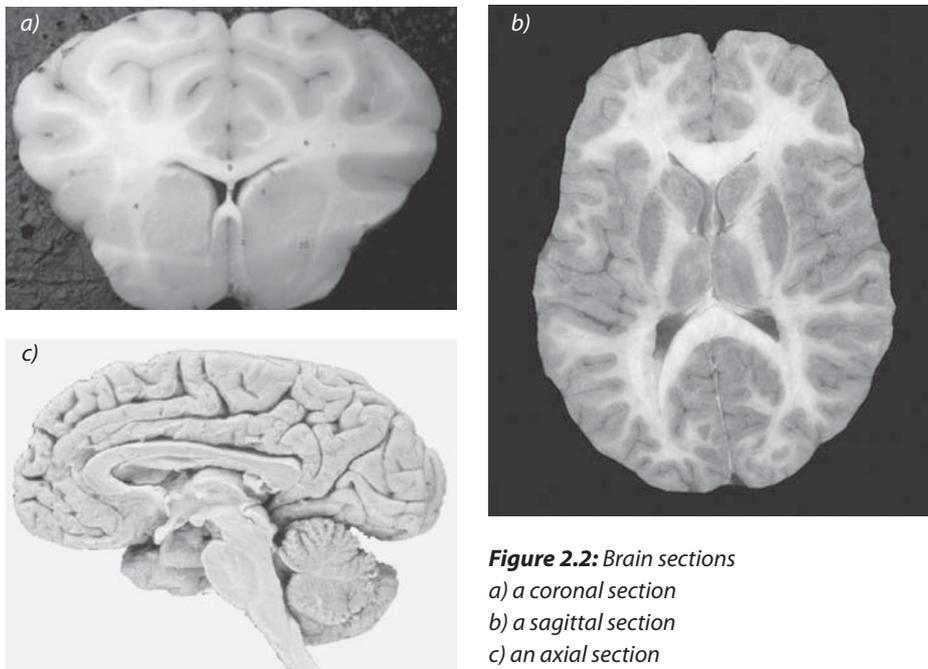
<i>Viewpoint</i>	<i>Terminology</i>	<i>Alternative terminology</i>
Looking down on to the brain	Dorsal view	Superior view
Looking from the front (face on)	Rostral view	Anterior view
Looking from underneath	Ventral view	Inferior view
Looking from the back	Caudal view	Posterior view

the back'. In addition, there are a couple of other terms that are often used: 'lateral', which means 'towards the sides', and 'medial', which means 'towards the middle'.

There are different ways of slicing brains open to look inside, and these have specific names too. Brain 'slices' sounds a little too gruesome, so they are properly referred to as brain sections. A **sagittal section** is sliced vertically between the eyes; an **axial section** is sliced horizontally (as if taking the tops of both ears and the top of the head off); and the third and final section is the **coronal section**, which is a vertical slice through both shoulders (as if taking the face off). The resulting images of the brain are much less gruesome than all this sounds, and some examples are given in Figure 2.2.

### Main structures of the brain

There is no doubt that the brain is an extremely complex structure, so to get a handle on how it works we need to divide it into a number of different parts. There are a number of different systems of classification of the various regions of the brain, but one of the best ways of simplifying the parts of the brain is to divide it into four parts: the brainstem, the limbic system, the cerebellum and the cerebral cortex. We will briefly look at the functions of these parts next, concentrating mainly on the cerebral cortex where many of the so-called 'higher' functions – such as perception and planning – reside.



**Figure 2.2:** Brain sections  
a) a coronal section  
b) a sagittal section  
c) an axial section

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### The brainstem, limbic system and cerebellum

These three parts of the brain are known as 'sub-cortical' regions, both because they are located under the cerebral cortex and because they are older in an evolutionary sense. The brainstem, which sits at the top of the spinal cord, controls many of the most basic functions that are necessary for the survival of the organism, including heartbeat, breathing and blood pressure. The limbic system contains regions that are responsible for our emotional reactions to events, and it can initiate hormonal responses. Regions in the limbic system include the amygdala, the hippocampus, the hypothalamus and the thalamus. The **amygdala** is crucial for emotional processing, particularly for negative emotions such as fear and threat, while the **hypothalamus** controls thirst, mood, hunger and temperature. Activity in the hippocampus is related to memory and learning, among other functions, while parts of the thalamus receive incoming signals from the various senses such as vision and hearing. The cerebellum (often known as 'the little brain') is located at the back of the brain, and plays an important role in motor movements and balance.

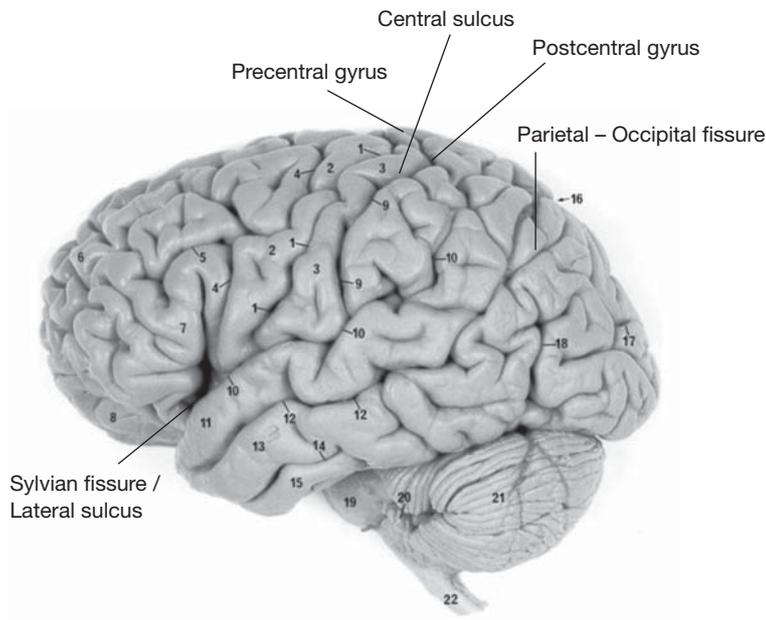
### The cerebral cortex

One of the things you should notice from the coronal and axial sections of Figure 2.2 is that the brain seems to appear darker around the edges. The dark edges you see in these sections are the outer layer of the brain known as the cerebral **cortex**, which is a six-layered sheet of tissue densely packed with neurons; because of its dark colour it is known as **grey matter**. Inside this outer layer is the white matter – mainly myelinated axons connecting cortical neurons to the rest of the nervous system or to cortical neurons in other regions of the cortex.

As the cortex is one large sheet of tissue, it needs to be 'folded up' to fit into your head. These folds are what give the brain its wrinkled appearance. Happily for us, almost all cortices are folded up in the same way, giving rise to very specific patterns of folds that, like eyes and ears, we all have but in a unique configuration.

It has been at least a couple of paragraphs since introducing some terminology so it is about time for some more. The folds or creases in the cortex are referred to as sulci (the singular is **sulcus**) or fissures; sometimes these terms are used interchangeably, but you may find some people referring to shallow folds as sulci and deeper folds as fissures. The surface of the cortex that is not disappearing into a crease is known as a **gyrus** (the plural is gyri). Almost all brains have the same sulci and gyri in roughly the same places, and these can act as the landmarks by which we can navigate around the cortex. Some important landmarks that use sulci and gyri are shown in Figure 2.3.

The central sulcus, Sylvian fissure and parietal-occipital fissure can be used to create theoretical divisions of the brain. The anterior area of the cortex enclosed by joining the central sulcus to the Sylvian fissure is known as the frontal cortex; the inferior area defined by joining the Sylvian fissure



**Figure 2.3:** Landmarks of the cortex

to the parietal-occipital fissure is known as the temporal cortex; the superior area defined by the Sylvian fissure, the central sulcus and the parietal-occipital fissure is known as the parietal cortex; and the remaining posterior area is known as the occipital cortex. You will see articles referring to frontal, temporal, parietal and occipital lobes – this refers to the whole section of brain covered by these areas of the cortex, as the cortex is just the outer layer. Essentially, then, the frontal cortex is the outer layer of the brain towards the front of the brain, while the frontal lobe is all of the brain in that area – the outer layer *and* the inner layers.

These four areas of the cortex are associated with specific behaviours and psychological processes that we will discuss in more detail.

## The frontal lobes

Perhaps the most famous case used to illustrate the characteristics of frontal lobe functioning is that of Phineas Gage in 1848 (Macmillan, 2008). Phineas Gage was a foreman working for the Rutland and Burlington Railroad company laying tracks across Vermont in the USA. In order to lay railway tracks the ground has to be reasonably level, so any large rocky obstacles either have to be avoided or blown up to make a level route. Part of Phineas's job was overseeing the demolition of such obstacles – drilling holes in the rock, filling them with gunpowder and setting a charge to create an explosion and so on. In particular, Phineas used to 'tamp-down' the explosive in the hole by hitting the loose powder with an iron bar known as a tamping iron. By hitting the powder with the bar, the powder becomes more tightly packed giving more energy and destructive force to the subsequent explosion.

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However, one Wednesday afternoon in September 1848, an accident occurred as Phineas was tamping down the explosives. As he hit the powder with the tamping iron, the explosive went off, sending the tamping iron flying back through his hands, into his skull beneath his left eye and out of the top of his skull, eventually landing about 20 metres behind him. Remarkably, he survived. He was carried to a cart, which took him home, where he climbed down from the cart himself, sat on his veranda and told the story to passers-by until a doctor arrived. The medical care he received was probably a key factor in Phineas's continued survival until his death in 1860, some 12 years after the accident.

What makes this case so interesting for students of bio-psychology is Phineas's behaviour in the 12 years following the accident. Though there is some debate as to the actual areas of his brain that were damaged by this accident, it is clear to see that the frontal lobes would have been affected to some degree by the tamping iron passing through his head. The observations made at that time about the changes in Phineas's behaviour can provide an insight into the implications of frontal lobe damage.

Phineas recovered reasonably well but never regained his original job, and his post-accident behaviour was described by his doctor as irreverent, grossly profane, impatient and capricious about his plans for his future, with the balance between his intellect and 'animal propensities' destroyed. This was clearly at odds with his behaviour prior to the accident, which was said to be efficient, shrewd and capable. Clearly, then, the frontal lobes may be seen either to mediate 'animalistic urges' or to control 'civilised, intellectual behaviour'. Phineas's case has often been retrospectively, perhaps even incorrectly, reported as an example of what can happen when the civilising effects of the frontal lobes are removed, resulting in an animal-like 'frontal lobe syndrome'.

One of the difficulties of Phineas's case is the lack of solid evidence about his behaviour in the historical record. However, evidence is available from more contemporary sources to suggest such a complex, 'executive' role for frontal lobe functions. Luria et al. (1964) noted a patient, 'Zav', who had frontal lobe damage and was unable to copy sequences of movement or reproduce a series of rhythmical taps even though she could understand the instructions to do so. From observations of Zav, Luria and colleagues concluded that frontal lobe damage resulted in being unable to inhibit impulsive reactions, seemingly responding instead to the demands of the environment, which may account for Phineas's capricious and profane manner.

This pattern of behaviour has been found in a number of patients with frontal lobe damage (Konow and Pribram, 1970; Wallis, 2007) and led to the idea of a frontal lobe 'syndrome' consisting of disinhibited and impulsive behaviour. This 'syndrome', though, is by no means found in all patients with frontal lobe damage; the pattern of impairment observed in such patients is often varied and can include emotional blunting, poor memory performance or an inability to shift between tasks.

One attempt to explain this variety of deficits over and above the use of a purely descriptive phrase such as 'frontal lobe syndrome' refers to executive functions or goal-directed behaviour. Aspects of the (intact) frontal lobes are thought to play a role in complex behaviours required to complete certain goals, and goal completion is thought to require some organisational function that controls and manages simpler psychological functions that need to be strung together to complete more complex goals. For example, going out to eat may be a relatively straightforward goal, but in order to complete this task, you need to manage and combine several simpler behaviours (using a telephone, driving a car, reading a menu, communicating with your guests and the waiter). Executive functions take on the task of ordering these simpler tasks and initiating them as and when appropriate; it is this executive functioning that is thought to be affected by frontal lobe damage.

If these executive functions are damaged in some way, then individuals may not be able to self-initiate sequences of actions, and consequently they either perform the same action repeatedly (known as perseveration) or they act only in response to the environment rather than under their own initiative. An individual with such deficits could be said to be capricious or animalistic, just as Phineas Gage was described.

## The parietal lobes

The range of psychological functioning mainly associated with the parietal lobes is visuo-motor guidance – tasks that involve making use of visual information or coordinating motor movements of the body to visual information from the environment. In its simplest form this may be a simple 'find the letter' task; for example, when you are looking through a word search puzzle to find specific letters or words, it is your parietal lobe that is active. This activation of the parietal lobe can be shown by examining fMRI or PET activity when participants are asked to visually search for, and locate, specific targets (see Corbetta, 1998, for a review). What such studies show is that there is a network of activity that becomes active when individuals are asked to perform such visual searches – a network that spans both the frontal and the parietal lobes.

Not only does activity in the parietal lobe increase in response to visual searches, the area is also active when individuals are asked to visually imagine objects and then manipulate (usually rotate) them (Kosslyn et al., 1998). In order to activate your parietal lobe right now, imagine the letter 'A', and then rotate it in your mind – it is this kind of mental rotation that requires parietal lobe functioning. Kosslyn and colleagues discovered that there seemed to be two distinct 'circuits' of activity activated by different stimuli; if the imagery was a geometric shape, then the parietal and occipital lobes were activated; however, when the imagery was a human hand, then parietal, occipital and frontal lobes were activated. This suggests there are different systems at work – one for mental rotation and one for preparatory motor movements (as participants were imagining rotating their own hands when using the hand stimuli).

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The parietal lobe has also been implicated in the function of corollary discharge, which is essentially a signal from the brain to itself about what it is doing. That sounds complicated, so an example may help at this point. Put the book down for a second and jump up and down. As you move, you will perceive yourself moving through the environment, moving up and down or side to side. What you do not perceive is that the environment is suddenly moving, as it might in an earthquake. The reason for this is that in order to jump, your brain is sending lots of signals to your muscles, but at the same time it is sending messages back to itself to let you know that it is not an earthquake – it is actually you causing the disturbance. This feedback signal is known as corollary discharge and it lets us know what we are doing and what we are not doing so that we can accurately perceive the world around us – it is also why we cannot tickle ourselves. Corollary discharge originates in the frontal lobes but terminates in the parietal lobes in an area known as the somatosensory cortex, a strip of cortex that represents sensation throughout our bodies – we will discuss this in more detail in Chapter 4.

Up to this point the evidence for parietal lobe function (visuo-motor guidance) is based on observing activation levels in the brain during certain tasks, but what happens if the parietal lobe is damaged in some way? As you may imagine, damage in this area results in a visuo-motor disturbance and this is known as visual neglect (McFie and Zangwill, 1960). Visual neglect is a rather rare and strange condition where individuals are unable to 'see' parts of the world around them, often behaving as if they do not exist. Typically, neglect stems from stroke damage to the right parietal lobe and shows contra-lateral effects; that is, if the damage is in the right hemisphere, the neglected area of the world is on the patient's left side. In some cases, the damage is so great that an entire side of space is neglected by the individual and such encompassing one-sided neglect is referred to as hemi-neglect.

If asked to copy a drawing, individuals with neglect will often do so but miss out aspects of the original drawing that fall in the neglected area; they may not attempt to dress the left side of their bodies; or they may misread hyphenated words such as 'visuo-motor' simply as 'motor'. Clearly such a deficit would be frustrating and at times dangerous to live with. It is thought that such damage to the right parietal lobe results in a deficit in visual attention rather than in the visual system itself, so those with neglect are able to see a whole scene but are unable to attend to all of it.

## The occipital lobes

The function most associated with the occipital lobes is the sensory processes of vision, and while the majority of the occipital lobes are devoted to vision, visual processing is not confined to the occipital lobes alone. The occipital lobes are home to the primary visual cortex, which processes the visual information it receives via a certain route from the eyes. If damage occurs to cells at any point in the visual pathway from the eye to the visual cortex in the occipital lobe, then the result

can be blindness or impaired vision, depending on the extent of the damage. Blindness or, more commonly, impaired vision from occipital lobe damage is referred to as cortical blindness, as the eyes still retain their full functioning but the signals they send are not processed.

The 'map' of visual information in the primary visual cortex is said to be topographic. This means that the relationship between specific points of the visual field are retained by the cortex – if you damage one area of the cortex, then the corresponding area of the visual field will be impaired. Although this cortical representation is topographic, it is inverted, with the top of the visual field being represented by the bottom of the visual cortex and the left side of the visual field being represented by the right side of the visual cortex. So damage to the left ventral visual cortex results in an area of impaired vision for the upper right of the visual field. Such an area of impaired vision is known as a scotoma. Scotomas are not areas of blindness or blackness in your vision but rather areas that are just not perceived, rather like the blind spot we all have in each eye but do not notice.

Other visual problems can be related to damage of the occipital lobe, and because of the complex organisation and pathways of the visual system, such damage can result in some quite different experiences. One such experience is **visual agnosia** (a deficit in object recognition).

Perhaps the most widely reported instance of this is the man who mistook his wife for a hat (Sacks, 1985) – Dr P, to give him his proper pseudonym. Dr P was a music teacher who developed a large growing tumour in his occipital lobe, resulting in his rather odd visually related behaviour that is indicative of visual agnosia. Although Dr P's sight was fine, his processing of visual information was disrupted in that he was unable to recognise objects or faces from vision alone; thus he attempted to grasp his wife's head, lift it off and place it on his own, believing her to be a hat. More poignantly, he was unable to recognise his family, friends, colleagues or students from their photographs alone; instead, he had developed a method of recognising distinctive features and using these to trigger his memory for the person. He was therefore able to recognise his brother from a photograph because of his brother's big teeth and square jaw, and he was able to recognise a student from the way they moved, but without these cues Dr P was at a loss to recognise them.

## The temporal lobes

In order to illustrate some of the functions attributed to the temporal lobe we will return to case studies again, but this time to one that is a little more modern. In 1957 Scoville and Milner reported the case of HM, who from an early age had experienced epileptic seizures. They were increasing in severity – so much so that at the age of 27 it was decided to treat his condition through neurosurgery, bilaterally removing his medial temporal lobes as this was where the epileptic foci were. Now you know your terminology, you should be able to work out that bilateral medial temporal lobe removal involves removing the middle (medial) parts of the temporal lobe from both (bilateral) sides of the brain.

## The brain

This surgery led to a successful cessation of seizures, but it had a noticeable impact on HM's psychological functioning – it impacted quite drastically on his memory. Following the operation HM would quite happily sit and read the same magazine over and over, would complete the same jigsaw puzzle repeatedly without any recognition or familiarity. He would even be unable to remember what he had had for lunch or even that he had had lunch as little as half an hour after eating it.

HM eventually worked in protected employment in a state rehabilitation centre in the USA, performing quite repetitive work (for example, putting cigarette lighters into cardboard display cases), and he successfully lived alone in his own bungalow. However, six months after starting this work he was unable to recall his place of work, his job, or the route to and from his workplace. Since his surgery HM clearly had difficulties in forming new memories (known as **anterograde amnesia**), and his case, along with many others, has led to the idea that the temporal lobes are important in memory functioning.

Mr B developed a tumour in his left temporal lobe at around the age of 38 when he had become a successful and well-respected oil company executive. Following the tumour his memory performance markedly decreased and he had difficulty maintaining performance in his job. In fact, his memory was particularly poor for verbal narratives but seemed unimpaired for recalling digits or shapes. This curious pattern of memory deficit may be due to the tumour affecting only his left temporal lobe, leaving the right temporal lobe functioning quite typically, which suggests that the right and left temporal lobes may play different roles in memory functioning.

The memory deficits of Ms C further this argument as she developed a tumour in her right temporal lobe at around the age of 22. Ms C was a promising student prior to the tumour, but afterwards she developed specific memory deficits, in that she had problems with recalling shapes but had no difficulties with her memory for verbal narratives. It is possible, then, that the left temporal lobe is required for verbally based memories while the right temporal lobe is required for visually based memories.

You might at this point start to question whether we therefore have different kinds of memories, performed by different regions of the temporal lobe or brain, and you would be right to do so. Memory is not one psychological function but a whole array of different and interacting processes, and we will explore this in more detail in Chapter 6. As well as not underestimating the complexity of the psychological processes discussed here, it is also important not to underestimate the complexity of the brain. The temporal lobes are a great example of this, for not only are they implicated in memory functioning, they are implicated in other processes too. For example, the temporal lobes are the end-point for all incoming information from our auditory (hearing) system, sometimes referred to as the auditory cortex, and damage to this area can result in impairments in understanding language. So while reviewing the functions of the lobes it is worth remembering that the lobes which we have been discussing are large brain areas that include many structures

carrying out different functions, so any attempt to define just one function that a lobe carries out is inherently over-generalised. So you need to be aware that the functions of the lobes we have discussed really are just the tip of the iceberg – we will explore some of these issues further in later chapters.

## Critical thinking activity

### The role of the cerebral cortex in controlling sexual impulses – a case study

Critical thinking focus: analysing and evaluating

**Key question:** *What can case studies tell us about the importance of the brain in controlling impulses?*

For this activity, you should locate and read the following paper:

Burns, JM and Swerdlow, RH (2003) Right orbitofrontal tumor with pedophilia symptom and constructional apraxia sign. *Archives of Neurology*, 60, 437.

If there are any words or ideas that you do not understand, please use an online dictionary to check what they mean.

The paper reviews a case study of a 40-year-old male who suddenly develops an interest in child pornography, and has no control over his sexual urges. Read the paper, and think about the following questions.

1. What does the section on personal history tell us about this man? Does his paedophilia and lack of sexual impulse control seem out of character?
2. The man was diagnosed with a tumour in the orbito frontal cortex (OFC). What does his behaviour tell about the function of the OFC? What does the report name as the functions of the OFC?
3. How can the researchers justify their idea that the tumour is linked to inappropriate sexual behaviour?
4. Are case studies useful in understanding how the brain works? What criticism can you think for case studies?

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## Critical thinking review

1. The fairly detailed personal history that is reported in the paper is interesting, and reveals a fair amount of information about the previous behaviour of the patient. It seems that paedophilia and lack of control for sexual impulses are out of character. The report reveals that he started developing an interest in child pornography in a very short space of time, and started molesting his step-daughter after living with her for some years without having sexual interest in her. Thus it seems that something 'triggered' the sudden paedophilia and inappropriate sexual behaviour, and that the behaviour escalated and spiralled out of control very rapidly.
2. OFC is important in the development of social and moral behaviour. Especially in adults, damage in the OFC can result in the inability to control impulses that are not socially acceptable. Patients with OFC lesions have their moral judgements intact (i.e. they know what is right and what is wrong), but they are not able to control their behaviour, even though they would like to be able to control it. Thus, OFC has a crucial role in keeping our behaviour in check, and controlling socially unacceptable behaviours.
3. The case study report reveals close links between the OFC tumour and sexually inappropriate behaviour. The paedophilic tendencies coincided with the migraines and the growth of the tumour. When the tumour was removed, the patient reported that the sexual urges were also gone. Further, the year following the operation, the patient started developing migraines and inappropriate sexual interests again. An MRI scan revealed that the tumour had reappeared. It is clear that the repeated appearance of the tumour, coupled with the migraines and the poor sexual impulse control, are closely linked to each other. It is safe to say that the OFC tumour made a major contribution to the paedophilic interests of the patient.
4. Case studies can be useful because they reveal richer information about the patient that can be used in understanding how brain tumours are related to behaviour. Case studies should always be discussed in relation to existing research, as the authors of the article being studied here have done successfully. Case studies can be used only to support existing quantitative research, as we cannot generalise from a single person to the whole population. So, for example, if we find that in one person OFC damage is linked to paedophilia, we cannot conclude that this is the case for all OFC patients unless other research has found a statistically significant relationship in studies with multiple patients.

## Skill builder activity

### Mapping the main structures of the brain

Transferable skill focus: communication

**Key question:** *What are the main structures of the brain?*

The communication of concepts, theories and facts is an important task of a scientific researcher. While the primary form of communication is generally the written or spoken word, a valuable and often efficient way of communication is by visual image. Pictures and diagrams can often convey large amounts of information in an economical way, proving true the old saying that 'a picture is worth a thousand words'. This task is an opportunity for you to develop your skills of communication by visual image. At the same time, the process of creating images or pictures of the structures of the brain should help you to remember the different areas of the brain.

The activity should be completed with pens and a large piece of paper. The aim is to draw three pictures of the human brain – your pictures can be as creative and colourful as you like. The first picture should be a diagram of the different views of the brain (see Table 2.2). The second picture should be of the lateral surface of the left hemisphere, and should include the four major cortical lobes and some details about the major functional roles of each lobe. The third picture should be a medial view of the brain, showing the cortex, the parts of the limbic system, the cerebellum and the brainstem. Again, you should include some details of the important functions of these brain regions. For the last two pictures, it may help to do an internet search of images of the brain.

## Skill builder review

This activity helps you to develop your visual communication skills, which can be a very effective way to communicate complex information. Sometimes people can grasp ideas or facts more easily when they see a diagram or an image. Also, by making a drawing of the various regions of the brain, you may improve your chances of remembering these details in the future. Lastly, you spent some time drawing, which is always a fun skill to practise.

The brain

## Assignments

1. Can the brain change in adults as a result of learning? Review this with regard to spatial knowledge and grey matter volume in the hippocampus.
2. How does the brain grow? Discuss the different processes of neural growth during brain development in infancy.
3. Are females and males different? Find out evidence for sex differences in the neuronal wiring and structure of the brain.

## Summary: what you have learned

- You should have a good understanding of the six main developmental stages during the maturation of the brain.
- You should have a solid understanding of how the brain is structured.
- You should be able to locate the different lobes in the cerebral cortex, and understand what their basic functions are.
- You should be able to discuss normal brain functioning, and what happens when there are tumours or lesions.

## Further reading

**<http://thebrain.mcgill.ca/>**

*A good educational website for exploring the brain:*

**[www.sciencemuseum.org.uk/WhoAml/FindOutMore.aspx](http://www.sciencemuseum.org.uk/WhoAml/FindOutMore.aspx)**

*Another good website, maintained by the Science Museum in London.*

Blakemore, SJ (2012) Development of the social brain in adolescence. *JRSM*, 105, 111–116. (You can download a copy on academic search engines.)

*Interesting article on adolescence brain development.*

Huttenlocher, PR (2009) *Neural plasticity: the effects of environment on the development of the cerebral cortex*. Cambridge, MA: Harvard University Press.

*A good book on neural plasticity of the human brain.*