KEY KNOWLEDGE
- infancy and adolescence as periods of rapid development and changes in brain structure and function, including development of myelin, synaptic pruning and frontal lobe development
- the impact of injury to the cerebral cortex on a person’s biological, psychological and social functioning and the ability of the brain to undergo adaptive plasticity, illustrated by rehabilitation of people with brain injuries
- the use of animal studies and neuroimaging techniques to develop understanding of human neurological disorders including Parkinson’s disease.

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Before neuroimaging techniques became available, it was widely believed that the brain stopped developing at around the age of 12. One reason for this belief is that the brain’s overall structure is almost complete at birth and it grows very little in size during childhood.

Although the sizes and shapes of our brains look very alike, no two human brains are actually identical. Genetic information directs the course of our brain’s development and the experiences we have throughout life actively shape its structure. In this sense, our brain never reaches a point where we can describe it as ‘fully developed’.

From birth through to the end of life, neurons and the connections between them change in response to our experiences. They change to represent and store this information so that we can learn and remember. Sometimes neurons die and connections are eliminated, especially if not needed or used. But the more we repeat a thought, feeling or action, the more connections that are dedicated to it and the stronger those connections become. In some cases, the brain can repair itself and a healthy part can take over the function of a damaged area. However, despite its remarkable adaptability and capacity for change, the brain cannot be transplanted or replaced.

In this chapter we examine the onset and process of brain development in infancy through to late adolescence, by which time most areas have reached maturity. We then examine the adaptability of the brain in response to experience. In particular, we consider how experience can change the brain’s structure, especially when damage through injury interferes with its normal functioning.

**BRAIN DEVELOPMENT IN INFANCY AND ADOLESCENCE**

Infancy and adolescence are periods of rapid development and change in brain structure and function. These occur in a genetically programmed, orderly way. At birth, the infant’s brain has just about all the neurons it will ever have despite being only about one quarter the size of an adult’s brain. By six months of age the brain will reach about half its adult size, almost three-quarters the size by two years of age and about 90–95% of its adult size by the age of 6. By the mid-20s or so the brain will have reached adult size by most estimates, but some parts are still maturing, particularly cortical areas.

Brain growth and development are orderly processes, but this does not mean that they occur at the one pace within each individual. There are bursts and spurts, most notably in early childhood and adolescence.

Areas deep within the brain that are responsible for vital survival functions develop first. It is essential that breathing, heartbeat, circulation, sleeping, sucking and swallowing are possible when the infant leaves the uterus (Epstein, 1986; Kolb & Wishaw, 2014).

Although the brain quadruples in size from birth to adulthood, it is not due to an increase in the number of neurons. A substantial amount of its growth is due to two processes — the development of myelin and the growth of new neural connections through synaptogenesis and the branching of dendrites.

![FIGURE 4.1 Changes in brain and body proportions as we grow older. The brain reaches full adult size by the mid-20s but some areas are still developing.](image)

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- Weblink
  - Video on brain development 8m 7s
Development of myelin

The growth and development of white, fatty myelin around many axons through myelination contributes to the increase in brain size. This important process allows neurons to be more efficient in sending messages to other neurons (although not all axons are ever myelinated).

Myelination starts before birth during fetal development and continues through childhood, adolescence and into adulthood. The most intense period of myelination occurs shortly after birth. By this stage, axons have also grown in size. They are longer, with denser branching at their ends because there are more axon terminals. There is also a burst of myelination in adolescence.

Myelination typically emerges in the hindbrain then spreads over time into the midbrain and forebrain. Within the cerebral cortex, sensory areas are myelinated before motor areas. This progression of myelination through the brain is consistent with the overall course of brain growth and development.

Myelination does not occur in a uniform way across the cortex. Sensory and motor areas are myelinated by age 3 or 4, whereas association areas in the frontal and temporal lobes responsible for more complex functions are myelinated last (see figure 4.2).

Synaptogenesis and synaptic pruning

Synaptogenesis accounts for most of the brain’s growth in size. Synaptogenesis involves the formation of new synapses between the brain’s neurons. A synapse is the place where neighbouring neurons connect and communicate — where messages are passed from axon terminals to dendrites. After birth, the neurons continue to develop new dendrites, the dendrites can grow more branches and each branch can grow spines, making the dendrites extremely ‘bushy’ in appearance (see figure 4.3). Each of these dendritic spines provides a site where a neuron can connect with an adjacent neuron and collect information.

Generally, synaptogenesis occurs more quickly in sensory (and motor) areas of the cortex than in association areas. It is believed that this allows the brain to have the capability to respond to the constant stream of new environmental input, for example, to deal with touch sensations and all the new sights, sounds, smells and so on that bombard the sense organs.

Synapses in the brain begin to form long before birth. After birth, synaptogenesis occurs so rapidly within the first 15 months that the total number of synapses increases tenfold. The infant brain forms far more neural connections through synaptogenesis than it will ever use. So, weak or unused connections are ‘pruned’. This process of eliminating synaptic connections is called synaptic pruning. Synaptic pruning is considered to be the means by which the brain ‘fine tunes’ its neural connections. It is a long-term process, continuing for many years, but in different areas of the brain at different times.

Synaptic pruning also tends to occur first in sensory areas and last in association areas. It is complete in the visual cortex by about age ten, but the elimination of dendrites can continue in association areas of the frontal lobe until well beyond age 20, eventually stabilising in adulthood around age 30. There is a burst of synaptic pruning during early adolescence, with almost half the synaptic connections eliminated at this time (Kolb & Wishaw, 2014; Spear, 2010).

What is constant across different areas of the brain, however, is that the number of synapses in an adult is about 40% less than the number in a three-year-old. Which connections will be kept and which will be pruned is based on experience. The connections that have proven to be valuable and have strengthened through repeated use are retained. Those that have become weak or obsolete decay and disappear. The entire process occurs as if the rule ‘use it or lose it’ is being followed. It is also a process that closely ties experience to brain development (Gazzaniga, Ivry & Mungun, 2014; Kolb & Wishaw, 2014; Spear, 2010).
Synaptic pruning and myelination also occur last in the motor cortex and other areas towards the back of the lobes. This is consistent with the general, overall pattern of brain growth and development. Areas at the bottom grow and develop before those at the top, and areas at the back before those at the front.

The progression of myelination, synaptogenesis and synaptic pruning through the frontal lobe (and other lobes) follows this overall pattern. Within the frontal lobe, synaptogenesis occurs more quickly in the motor cortex and other areas towards the back of the lobe than it does in the prefrontal cortex at the front. Synaptic pruning and myelination also occur last in the prefrontal cortex.

During the early childhood years, between the ages of 3–6 years, there is a significant increase in the number of neural connections established in the frontal lobes through synaptogenesis. As this occurs, children become increasingly sophisticated in cognitive abilities, especially when compared to capabilities at birth.

During the ages of about 7 to 15 years, the rapid synaptic growth shifts to the temporal and parietal lobes. This is believed to be associated with significant increases in language development.

During the ‘teenage’ years of around ages 16 to 20, there is a heightened level of synaptic pruning in the frontal lobes whereby unneeded connections are actively eliminated. This pruning assists more efficient functioning of neuronal activity, which is why it is believed to occur at this time (Huffman, 2012).

**FIGURE 4.3** The infant brain forms far more neural connections than it will ever use. The brain then ‘prunes’ any synaptic connections that are not used in a process based on a ‘use it or lose it’ rule.

**Frontal lobe development**

Brain development continues into adulthood until about the mid-twenties or so, with the frontal lobes last to fully mature. The prefrontal cortex — the association area just behind the forehead — is the very last part of the brain to mature. This is consistent with the general, overall pattern of brain growth and development. Areas at the bottom grow and develop before those at the top, and areas at the back before those at the front.

The progression of myelination, synaptogenesis and synaptic pruning through the frontal lobe follows this overall pattern. Within the frontal lobe, synaptogenesis occurs more quickly in the motor cortex and other areas towards the back of the lobe than it does in the prefrontal cortex at the front. Synaptic pruning and myelination also occur last in the prefrontal cortex.

**FIGURE 4.4** Periods of more rapid synaptogenesis and synaptic pruning occur during brain development from early childhood to later adolescence. The prefrontal cortex is the last brain area to mature and the heightened level of synaptic pruning between 16–20 years assists more efficient functioning of neuronal activity.

Some psychologists regard it as significant that the prefrontal cortex is the last brain area to reach maturity. It is considered significant because of the role of the prefrontal cortex in the more advanced, ‘higher level’ mental functions, such as our ability to reason, plan ahead, organise, solve problems, make decisions and so on. It has been suggested that the occasional impulsive, unpredictable, unstable or immature behaviour displayed by many adolescents is at least partly a reflection of their brain still being a ‘work in progress’ because it still has yet to reach full adult maturity.

The underdeveloped prefrontal cortex has been proposed as one explanation for the immaturity of decision making and higher incidence of ‘unstable’ behaviour among many adolescents. During adolescence, emotions and impulses can be intense and compelling, especially as the adolescent's limbic system — the motivational and emotional centre of the brain — tends to be quite well-developed and mature. But the part of the brain that is responsible for reasoning, decision making, exercising judgment and emotional regulation is still maturing. As a result, although adolescents are able to think critically, they may sometimes have difficulty doing so. They are also more likely to act irrationally than during periods of later brain development. The adolescent also engages in risky behaviours more than adults. Some of the most life-threatening risky behaviour is especially common during the ‘teenage years’ (Dove et al, 2016; Grison, Heatherton & Gazzaniga, 2015; Horstman, 2012; Spear, 2010).

**LEARNING ACTIVITY 4.1**

**Review questions**

1. Construct a timeline to outline growth in brain size from birth to adulthood.
2. Explain why the human brain never reaches a point where it is ‘fully developed’.
3. (a) What is myelination?
   (b) When does it start and end?
4. (a) What is synaptogenesis?
   (b) When does it start and end?
5. (a) What is synaptic pruning?
   (b) Suggest why there is an excess of synapses at birth and why so much pruning occurs.
6. (a) Explain the meaning of ‘use it or lose it’ in relation to brain development.
   (b) Some people believe that the ‘use it or lose it’ principle means that a great deal of learning is not possible after the age of 3. How accurate is this view?
7. Explain how myelination and synaptogenesis collectively account for a substantial increase in brain size after birth.
8. Draw a picture of a brain and use numbers and/or arrows that become narrower to indicate the overall pattern of brain growth and development.
9. Give an example of a risky, life-threatening behaviour commonly associated with adolescence. What is a possible explanation in terms of lobe development?

**LEARNING ACTIVITY 4.2**

**Reflection**

An underdeveloped prefrontal cortex in early adolescence may be one reason why some teenagers have difficulties with self-control, and lack efficient and adult decision-making abilities.

Comment on whether this reason justifies inappropriate behaviour by teenagers and explain your view.
IMPACT OF INJURY TO THE CEREBRAL CORTEX AND ADAPTIVE PLASTICITY

At most times, our brain serves us well. When intact and undamaged, it usually enables us to successfully adapt to our environment and meet the demands of everyday life. When the cerebral cortex or any other part of the brain is injured, people can experience one or more impairments that may affect them physically and/or how they think, feel and behave. Many people with cortical damage can function quite effectively in everyday life, but the impact will depend largely on the individual involved and the nature and severity of the injury itself. Appropriate treatment also plays a vital role in the level of recovery.

Brain injury refers to any brain damage that impairs, or interferes with, the normal functioning of the brain, either temporarily or permanently. Most cases of brain damage occur after birth and in such instances are referred to as acquired brain injury. That type of damage can be caused by an accident, an intentional blow, violent shaking of the head, stroke, alcohol and other drugs, brain surgery, infection (such as meningitis), brain inflammation (such as encephalitis) or a brain disease (such as Parkinson’s disease) (Brain Injury Australia, 2017).

Brain injury can have sudden onset, when caused, for example, by a blow, infection, stroke or drug use. Alternatively, brain injury can have insidious onset, for example, from prolonged use of alcohol or another substance, a tumour or a neurodegenerative disease.

A neurodegenerative disease is characterised by the progressive decline in the structure, activity and function of brain tissue. Parkinson’s disease and Alzheimer’s disease are both neurodegenerative diseases. Essentially, neurons within the brain (‘neuro’) gradually become damaged or deteriorate (‘degenerate’) and lose their function, hence the term ‘neurodegenerative’.

Brain injury is common. It is estimated that about 1 in 4 Australian adults have a brain injury that impairs everyday life in some way. Three out every four of them are aged under 65. As many as two out of every three of these people acquired their brain injury before they turned 25. And three out of every four people with acquired brain injury are male (Australian Institute of Health and Welfare [AIHW], 2017; Brain Injury Australia, 2017; Synapse, 2016).

The most common cause of acquired brain injury is stroke. A stroke occurs when a blood vessel bringing oxygen and nutrients to the brain bursts or is clogged by a blood clot. As a result, brain tissue is deprived of blood, causing the brain cells to die within minutes. This affects any mental process or behaviour controlled by those brain cells, such as thinking, speech and movement. The next largest cause of acquired brain injury is an accident or trauma, known as traumatic brain injury.

Traumatic brain injury

Traumatic brain injury is a type of acquired brain injury caused by a blow to the head or by the head being forced to move rapidly forward or backward, usually with some loss of consciousness. There may be a momentary loss of consciousness (which, for example, can happen from a punch to the face) through to a long-term bout of unconsciousness or coma. Sometimes a traumatic brain injury results in very serious and often life-threatening problems.

Traumatic brain injury may be the result of a motor vehicle accident, fall, assault, sporting accident, gunshot wound or violent shaking. Traumatic brain injury is not the same as head injury, since a person...
can sustain damage to the face, scalp and skull without necessarily injuring their brain. When the head is struck hard, the brain slams against the inside of the skull. As a result of this blow or rapid movement, brain tissue may bleed, bruise, tear, twist or become swollen.

Some traumatic brain injuries are classified as mild, like concussion, which is most commonly acquired when playing contact sports such as AFL football and rugby. Problems in most cases tend to be short-lived and people return to normal functioning fairly quickly.

In some cases, however, people continue to experience problems that can last for weeks, months or even years. This is known as post-concussion syndrome and persistent symptoms often include headaches, dizziness, balance problems, fatigue, anxiety, depression, insomnia, light and sound sensitivity, and problems with attention, concentration and memory.

**Severity of brain injury**

- **Mid**
  - e.g. good recovery, limited concentration, able to return to work.

- **Moderate**
  - e.g. improvement over time, difficulties with coordinating movements, inability to organise, may require different line of work.

- **Severe**
  - e.g. decreased movement control, decreased ability to communicate, requires support with daily living, unable to return to work.

- **Very severe**
  - e.g. unable to control movement, unable to communicate, requires 24-hour support, unable to return to work.

**FIGURE 4.7** A brain injury can range from mild to very severe.

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**BOX 4.1 The Glasgow Coma Scale**

A range of clinical tests can be performed to assess the severity of a traumatic brain injury. One test involves measuring the degree of decrease in observable responsiveness to external stimuli. The Glasgow Coma Scale is commonly used for this purpose. The test is simple and considered a reliable and objective way of recording the initial and subsequent level of consciousness in a person after a brain injury. It may be used by trained staff at the site of an injury like a car crash or sports ground, for example, and in a hospital’s emergency department or intensive care unit (Institute of Neurological Sciences, 2017).

The Glasgow Coma Scale assesses three aspects of a brain injured person’s responsiveness — eye opening, verbal responses and motor responses. The assessment criteria are shown in the chart below. In each category, the top item is the ‘normal response’ and no response (None) is the ‘lowest response’.

For example, for eye opening, if the person’s eyes open spontaneously in the presence of the assessor, then a score of 4 is assigned. If spontaneous eye opening is not demonstrated, a verbal stimulus is used. The assessor introduces themselves with speech and requests eye opening. If the person opens their eyes, then a score of 3 is assigned. The assessor then determines verbal and motor responses. The scores for each category are then added, yielding a total score between 3 (a person showing no response) and 15 (a person who is alert and well-oriented). A total score of 8 or less is classified as ‘severe severity’. A person given this score may be in a coma.

**Glasgow Coma Scale**

<table>
<thead>
<tr>
<th>CRITERIA</th>
<th>POINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EYE OPENING</strong></td>
<td></td>
</tr>
<tr>
<td>Spontaneously</td>
<td>4</td>
</tr>
<tr>
<td>To sound</td>
<td>3</td>
</tr>
<tr>
<td>To pressure</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>VERBAL RESPONSE</strong></td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Confused</td>
<td>4</td>
</tr>
<tr>
<td>Words</td>
<td>3</td>
</tr>
<tr>
<td>Sounds</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>MOTOR RESPONSE</strong></td>
<td></td>
</tr>
<tr>
<td>Obey commands</td>
<td>6</td>
</tr>
<tr>
<td>Localising</td>
<td>5</td>
</tr>
<tr>
<td>Normal flexion</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal flexion</td>
<td>3</td>
</tr>
<tr>
<td>Extension</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
</tbody>
</table>

A total score of 13 to 15 is classed as mild severity. A total score of 9 to 12 is classed as moderate severity. A total score of 3 to 8 is classed as severe severity.

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Conducting a Glasgow Coma Scale assessment

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Mind Matters: A documentary on brain injury (55m 19s)
Impact of injury to the cerebral cortex

Brain injury to the cerebral cortex has different effects on different people, both in the short term and long term. When the cerebral cortex is damaged, some other part of ourselves will also be affected. Even a mild injury can result in a serious disability that will interfere with a person’s daily functioning and personal activities, often for the rest of their life. The extent of some of these changes may only become apparent as time progresses (Brain Injury Australia, 2015).

In this section, we examine research findings on individuals with an injury to particular areas of their cerebral cortex, how this affects their everyday experience and what their injury has revealed about cerebral cortex functions. We start with one of the earliest and best-known cases of an injury to the cerebral cortex in the frontal lobes. Then we consider a spatial disorder associated with damage to a specific area of cortex in the temporal lobe.

Biological, psychological and social changes due to frontal lobe injury

Phineas Gage, a construction foreman working on a new railway line in the US state of Vermont, was only 25 years old when he suffered a massive head injury that seriously injured his frontal lobes. Gage was supervising a crew of workmen in September 1848.

To remove a large rock in the way of the track, Gage poured gunpowder into a deep, narrow hole drilled into the rock. The gunpowder was packed in tightly with an iron rod before a fuse was lit to ignite it. The rod was more than a metre long, 3.5 centimetres in diameter and weighed 6 kilograms.

As Gage was packing down the gunpowder, a spark from the rod ignited the gunpowder and blew the rod into his cheek and out through the top of his skull. After going through his skull, the rod is said to have landed somewhere between 20 and 50 metres away, depending on which report is read. Gage was pushed backwards and fell to the ground. His body began to shake uncontrollably, but he was still alive. Within minutes of the accident he is reported as sitting up and talking to people near him (Blakemore, 1977). The doctor attending him was able to stop the bleeding, and cleaned out loose bits of brain tissue and bone before dressing the wound.

There was no immediate indication that Gage’s mental or physical abilities had been affected by the accident, despite injury to both his frontal lobes, including the prefrontal cortex. However, the once friendly, considerate and quietly spoken Phineas Gage is reported to have become impatient, crudely spoken, aggressive, irresponsible and hard to get along with. His friends and acquaintances said that he had changed so much he was no longer the person they...
had known. Twelve years later, at the age of 36 or 37, Phineas Gage died (Macmillan, 2017).

Gage’s doctor, John Harlow (1848), wrote a detailed account of the accident as well as of Gage’s condition and symptoms. Years later, when he learned of Gage’s death, he petitioned Gage’s family to exhume the body and allow him to keep Gage’s skull and the rod as a ‘medical record’. These are on display in a museum at Harvard University in America.

Nearly 150 years later, American psychologist Hanna Damasio and her colleagues (1994) examined the metal rod and damage to Gage’s skull. Using skull measurements and computer imaging techniques, they reconstructed the pathway of the rod to more accurately pinpoint the brain injury (see figure 4.8). Then, they searched for case studies of patients with a known injury in the same area of cortex as Gage’s injury. They examined these isolated case studies and found that Gage’s symptoms were consistent with those reported by other patients.

Common among patients with an injury to the forward part of the frontal lobes (in the prefrontal cortex) is an unusual collection of biological, psychological and social changes.

### Biological changes

Biological changes are primarily physical in nature. Patients with a severe injury to the prefrontal cortex often have a range of problems with motor activities. In particular, their overall level of motor activity and ordinary voluntary, spontaneous movements are markedly reduced. For example, facial expressions tend to become blank and head and eye movements are minimal. Some reflexes that are evident only during early infancy, such as the grasping reflex of the hand, may also reappear.

### Psychological changes

Psychological changes primarily involve emotion, personality and cognition, which in turn impact on behaviour.

Emotional changes often include a persistent apathy (lack of concern about anything) and lack of emotional responsiveness, including lack of concern for the past or present. However, the patients experience episodes in which this apathy is dramatically broken by bouts of euphoria (extreme excitement); impulsive behaviour; disregard for social conventions; verbal and physical aggressiveness, boastfulness, silliness and, sometimes, unrestrained sexual activity. Collectively, these types of changes are often perceived by others as changes in the individual’s personality.

A reduced responsiveness to pain is also commonly reported. In relation to cognitive abilities, general intelligence — as measured by IQ scores — shows only slight changes. However, creative thinking and problem solving tend to be affected, and forgetfulness is shown in many tasks requiring continual attention. Some researchers believe that the apparently insignificant effect of frontal lobe damage on measured intelligence may be explained by limitations of traditional intelligence tests; for example, they tend to measure ‘convergent thinking’ rather than ‘divergent thinking’. Convergent thinking is used when looking for a single correct answer and, unlike divergent thinking, does not require a significant amount of creativity (Centre for Neuro Skills, 2017).

Many case study reports of patients with a severe frontal lobe injury involving the prefrontal cortex have also emphasised problems with goal-directed behaviour, especially an inability to plan activities and use foresight. Daily activities of these patients seem disorganised and without a clear direction of where these activities are leading or why they are being undertaken. For example, a patient given a simple set of errands may be unable to complete them (if able to do any at all) without numerous false starts, backtracking and confusion (Breedlove, Watson & Rosenzweig, 2010).
rates of contact with police, more court appearances and more convictions, longer periods of detention or imprisonment, and are more likely to be victims of crime. However, their offences tend to be relatively minor (e.g. 40% of offences involve theft or road traffic infringements) and are committed under the influence of alcohol (Brown & Kelly, 2012; Centre for Neuro Skills, 2017).

**Social changes**

Biological and psychological changes associated with frontal lobe injury, particularly personality changes and onset of socially inappropriate behaviour, can be difficult for partners, family members, friends, work colleagues and others in the individual’s social network. This can lead to a breakdown in personal relationships and loss of social support. In addition, the individual may experience difficulty establishing new social relationships.

According to Brain Injury Australia (2017), injury to the cerebral cortex puts the individual at an increased risk for unemployment, lack of affordable housing, homelessness and social isolation.

In addition, individuals with this type of brain damage are significantly overrepresented in the criminal justice system. One Australian study has found that over 40% of prisoners have an acquired brain injury. In addition, compared to the general population, people with a brain injury have higher
In the late 1930s Portuguese neurologist Egaz Moniz devised the lobotomy, a surgical procedure that severed nerve fibres to cut off the foremost portion of the frontal lobes from the rest of the brain. The operation was initially used to treat people diagnosed with a mental disorder, especially patients who could not control their emotions. Moniz received the Nobel Prize in Physiology or Medicine in 1949 for his advancement of the lobotomy.

In America, many doctors embraced the lobotomy and performed it on tens of thousands of men and women with mental disorders such as schizophrenia, major depression and obsessive–compulsive disorder. Often called a prefrontal or transorbital lobotomy, it was a crude but simple operation, sometimes performed in a doctor’s office using a local anaesthetic, with an ice pick inserted into the brain via an area under the patient’s upper eyelid. The ice pick was then moved around until the connecting neural tissue was severed, in part or whole, depending on the diagnosis of the patient’s disorder. The entire procedure took 3–4 minutes.

The operation generally had the effect of decreasing the patients’ emotional responsiveness. Many became emotionally docile, remaining extremely and consistently calm even when in frustrating circumstances. As a result, the patients were much easier to manage in psychiatric hospitals. But it also left them disconnected from their social surroundings and had adverse effects on cognitive functions.

The prefrontal cortex is not only involved in regulating emotional responses, but also in the execution of many higher order mental abilities. Consequently, the lobotomy left most patients unable to plan and organise their lives effectively as well as other problems associated with thinking. This eventually raised concerns about the lobotomy, although it continued to be practised until the mid 1950s, when new medications were developed to treat psychological disorders.
**Spatial neglect due to parietal lobe injury**

A patient in a rehabilitation facility wakes in the morning and proceeds to shave his face. When he puts the shaver down to go to breakfast, it is apparent that he shaved only the right side of his face. While eating breakfast, the patient starts to look for his coffee cup until someone points out that it is just slightly to the left of his dish. At lunch or dinner, he may leave the food on the left half of his plate untouched while asking for more, only to be reminded that there is still food on the plate. If asked to read compound words such as football or birthday, he will read ball and day, overlooking the first half of the word. If questioned, he states that he read the words correctly. If asked to draw a clock, he will draw a circle correctly but then crowd all the numbers into the right half. If asked to draw a person, he will draw only the right side of the body, leaving out the left arm and leg. If questioned, he states that the drawings look alright to him (Springer & Deutsch, 1998).

This unusual behaviour is associated with brain injury resulting in a disorder called spatial neglect which causes problems with attention. Generally, **spatial neglect**, also called **hemispatial neglect** and **visual neglect**, is a neurological disorder whereby individuals are unable to notice anything either on their left or right side even though there may be no sensory loss. They tend to behave as if that one side of their world does not exist.

Pen-and-paper tasks are commonly used as part of the behavioural assessment of spatial neglect. These may include copying drawings (as shown in Figure 4.13), drawing objects and cancellation tasks (as shown in Figure 4.12). Cancellation tasks tend to be the most sensitive of the behavioural tests. These require patients to find targets (sometimes embedded amongst distractors) on a centrally placed sheet of paper.

Spatial neglect is most commonly observed in stroke or accident victims who have fairly extensive injury to the cerebral cortex in the rear area of the parietal lobe of the right hemisphere. Consequently, these patients mostly neglect the left side of their world. Spatial neglect of the right side sometimes occurs after similar damage to the left hemisphere (or in subcortical areas), but much less frequently and in a milder form. In either case, the side of the world opposite to the damaged hemisphere tends to be neglected, rather than the same side (Kolb & Whishaw, 2014; Li & Malhotra, 2015; Parton, Malhotra & Husain, 2004).

Spatial neglect is a complex disorder with many different types and subtypes. Although neglect is mostly experienced with the visual sense, it may occur for other senses, such as hearing or touch, or with movement. Furthermore, it may be isolated to one or a combination of these senses. When tested, some patients acknowledge the presence of something on the neglected side and mistakenly report its presence as if it appeared on the non-neglected side.

*Figure 4.12* A star cancellation task from a behavioural test for spatial neglect. The stimulus is positioned centrally and the patient is asked to find and mark all the small stars without marking the large stars or letters. This patient was only able to locate the small stars at the far right of the stimulus despite having unlimited time to complete the task.

Thus, for example, a patient may be given an auditory stimulation on their left neglected side and claim that the sound came from the right. Or, in the case of neglect involving movement, the patient may be asked to raise their left hand and, if they respond at all, they may raise their right hand.

FIGURE 4.13 Drawings of common symmetrical objects by an individual with spatial neglect. Note that the left side of each object has been ignored.

The extent of neglect among different individuals varies and depends on the severity and specific location of their brain injury. It may range from indifference towards objects on one side to denial of the very existence of that side of the body. For example, one patient called a nurse in the middle of the night to ask her to help him throw his own left leg out of bed, thinking that ‘the alien leg’ had been put there as a cruel joke by other patients. Less severely affected individuals may simply ignore things in their left or right visual field and not necessarily all parts of their body on that side (Li & Malhotra, 2015; Parton, Malhotra & Husain, 2004; Stirling, 2002).

The higher incidence of spatial neglect when there is injury to the right rather than left parietal lobe demonstrates the importance of the cerebral cortex in the right parietal lobe in attention and in conscious awareness of objects and the self. Like many other mental processes, however, other brain areas are also involved in attention and consciousness. Interestingly, many individuals with spatial neglect insist that there is nothing wrong with how they perceive and act in the world.

As shown in figure 4.14 below, some people with spatial neglect make a gradual recovery from the disorder. Most make some degree of spontaneous recovery but tend to have significant cognitive impairments, particularly relating to attention. Treatments tend to be unlikely to be successful unless they are tailored to the underlying cognitive impairments in individual patients (Li & Malhotra, 215).

Psychologists are unclear about why it occurs following damage to the parietal lobe, nor is there any widely accepted explanation of the disorder.

FIGURE 4.14 German artist Anton Raderscheidt suffered a stroke that damaged the cerebral cortex in the right parietal lobe. Note his gradual recovery from spatial neglect, as evidenced by the progressive addition of details to the left side of his self-portraits. Whether or not someone recovers from spatial neglect, how well they recover and the speed of recovery depends on the individual and the severity of their brain injury.

< Permission clearance pending >
**BOX 4.3 Oliver Sacks’ case studies of patients with cerebral cortex injuries**

British neurologist Oliver Sacks (1933–2015) reported many case studies that describe the effects of injury to the cerebral cortex in his books that included *The Man Who Mistook His Wife for a Hat* (1985) and *Seeing Voices* (1990). There have also been movies based on some of his books.

In one case study, Sacks (1990) described the experiences of a patient whose brain injury involved an association area in the occipital lobe. The man could still see the basic features of objects, such as colour, edges and movement. He was also able to recognise basic geometric shapes.

When Sacks showed the man a rose and asked him to identify it, the man responded: ‘About six inches in length. A convoluted red form with a linear green attachment. It lacks the simple symmetry of the Platonic solids, although it may have a higher symmetry of its own . . . ’ After some time spent continuing to reason about its parts, the man finally guessed that it might be some sort of flower.

Sacks then held the rose under the patient’s nose and asked him to smell it. ‘Beautiful!’ the man exclaimed. ‘An early rose. What a heavenly smell!’ The man could easily identify the rose by smell but not by sight, even though he could see every feature and describe most of them in considerable detail.

According to Sacks, the man was unable to integrate the information because of damage to an association area that would have helped him make the connection between the visual and olfactory (smell) parts of the relevant information stored in his memory.

In another case study, Sacks (1985) described the case of ‘Christina’, who had lost the ability to feel the position of her own body. She reported feeling disembodied, like a ghost. For example, on one occasion when she was a patient in a hospital, she became annoyed at a visitor whom she thought was tapping her fingers on a tabletop. But it was actually Christina, not the visitor who was doing it. It was as if her hands were acting on their own and her body was doing things of which she was unaware.

Sacks diagnosed Christina as having lost all her sensory feedback about joints, muscles and positions of her limbs. For unknown reasons, the sensory neurons that would normally carry this information to the primary somatosensory cortex in the parietal lobe were malfunctioning. This case study provided important insights into *kinesthesias*, the sense of knowing where our body parts are in space.

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**LEARNING ACTIVITY 4.3**

**Review questions**

1. (a) Explain the meaning of acquired brain injury.
   
   (b) Distinguish between brain injuries with sudden or insidious onset with reference to two examples of each type.

   (c) Explain why boxing is a dangerous sport, especially professional boxing for which protective headwear is not required during bouts.

   (d) Distinguish between meningitis and encephalitis.

2. (a) What specific brain injury did Phineas Gage have?
   
   (b) What did his injury indicate about the role of this brain area in mental processes and behaviour?

   (c) How accurately and completely do Gage’s symptoms represent symptoms of most people who damage the same brain area?

3. (a) Injury to which area of the cerebral cortex is most commonly associated with spatial neglect?
   
   (b) Explain what spatial neglect is with reference to examples of mental processes and behaviour associated with the disorder.

   (c) Give an example of a visual scene that may be reported by an individual with spatial neglect while watching a sports event. Your example should identify the location of the brain injury, the event and the position from where the scene is viewed.

   (d) Suggest an example of a biological, a psychological and a social change that could occur together with spatial neglect. Explain your choice of examples.
LEARNING ACTIVITY 4.4

Data analysis on recovery from spatial neglect

Spatial neglect is quite common among patients with brain damage caused by a stroke. One of the earliest studies on recovery from right or left neglect induced by a stroke was conducted by British neurologist Derick Wade and his colleagues (1988). Patients admitted to hospital and surviving for 6 months were regularly tested on three cognitive tasks to measure their recovery.

One of the tasks required each patient to cross out all ‘1’s and ‘4’s presented in different strings of numbers (i.e. a cancellation task). Examples of strings of numbers used in the study are:

<table>
<thead>
<tr>
<th>String of numbers</th>
<th>Percentage correct</th>
</tr>
</thead>
<tbody>
<tr>
<td>983.7:0356350....</td>
<td>758.390238756....</td>
</tr>
<tr>
<td>3829.7:65960758</td>
<td>537260:987.8285</td>
</tr>
<tr>
<td>.7382059385837.</td>
<td>38:958.9:62:578</td>
</tr>
</tbody>
</table>

The results for 15 patients are plotted in the graph on the right. Of the 15 patients, nine had left visual neglect and six had right neglect.

1. What do the X and Y axes of the graph describe?
2. How many patients showed recovery from spatial neglect?
3. Explain, with reference to the data, whether there was a difference in rate of recovery by each patient.
4. (a) After how many days did one of the patients experience a sudden and dramatic relapse from recovery? 
   (b) Suggest a possible reason for the relapse.


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LEARNING ACTIVITY 4.5

Analysis of research on brain function

A psychologist wanted to test research findings that specific areas of the primary motor cortex in the left and right frontal lobes are involved in specific voluntary motor movements on opposite sides of the body.

She tested her hypothesis through an experiment conducted as part of a case study involving one male research participant. The participant was suffering from severe, unpredictable epileptic seizures and had been referred to the psychologist by the participant’s doctor, as the psychologist had expertise in diagnosing the source of epileptic seizures.

Taking advantage of the opportunity to test her hypothesis, the psychologist obtained permission from the participant to study voluntary motor movements during brain scanning that had been organised to locate the source of the epileptic seizures. Approval for the experiment was also obtained from an ethics committee.

Working as a member of a team that included a qualified radiologist and neurosurgeon, she asked the participant to raise his right foot when a yellow light was flashed in the scanning chamber and to raise his left foot when a blue light was flashed. There were five trials involving each foot, but the different coloured lights were flashed randomly.

She found that a specific area of primary motor cortex in the left frontal lobe was active whenever a voluntary movement of the right foot was made and a corresponding area of cortex in the right frontal lobe was active whenever a voluntary movement of the left foot was made. These results supported the hypothesis and were consistent with those of similar studies previously conducted by other researchers.

1. Formulate a research hypothesis for this experiment.
2. What is the operationalised independent variable?
3. What is the operationalised dependent variable?
4. Explain why the psychologist flashed the lights randomly.
5. Suggest a limitation of this study.
6. Has the psychologist breached any ethical standards? Explain your answer.
7. Explain the meaning of participant confidentiality, voluntary participation and informed consent in relation to this experiment.
Brain plasticity

Throughout our lives our brains constantly change. This reflects the brain’s plasticity. Plasticity (also called neural plasticity and neuroplasticity) is the ability of the brain to change in response to experience.

Change occurs primarily at the synapse and therefore at the microscopic level. Individual neurons and their connections can be modified for different reasons — during its development when we are young, during learning throughout our entire lives and sometimes in response to brain injury.

The brain as a whole does not change its shape. New neural pathways can form and link up with existing pathways and existing pathways may interconnect with other pathways. These types of changes involve neurons as well as glial cells. Their activities result in changes to the brain’s physical structure and function. For example, the brain can reorganise and reassign its neural connections and pathways based on which parts of it are overused, underused or injured. Its structure is constantly remodelled by everyday life experience and environmental demands as it adapts to meet our needs.

Lifelong plasticity accounts for many of the learning experiences we have throughout life, such as learning our native (‘first’) language as a child, learning to play a musical instrument as an adolescent, learning to text message as an adult, learning to use a computer in old age, and so on. Our genes govern the overall architecture of our brain, but experience guides, sustains and maintains the details.

Plasticity is a characteristic of probably all animal brains but the larger brains of mammals have more capacity for change. For example, if a monkey is trained to push a lever with a finger several thousand times a day, the brain tissue that controls the finger changes to reflect the experience. More motor cortex neurons are active in the same area than were active before the training (Breedlove, Watson & Rosenzweig, 2010).

Human brains function in a similar way. Whether learning to use a keyboard or a skateboard, we perform with increasing skill as our brain incorporates the learning within its structure. The neural activity underlying this process occurs in a systematic way and not haphazardly.

Although some areas of the brain such as the sensory and motor cortices have a higher level of plasticity than others, it is unclear as to whether all brain areas have plasticity. However, the brain of a developing individual is even more plastic than that of an adult, particularly at specific times in development when it seems that the brain is more responsive to certain types of experiences. This is one reason why infants tend to learn a new language more quickly than do adults. Similarly, infants recover more quickly from brain injury than do adults due to the greater plasticity of their brain (Breedlove, Watson & Rosenzweig, 2010; Myers, 2007; Sweatt, 2016).

Adaptive plasticity

The term adaptive plasticity is commonly used in relation to brain injury. Adaptive plasticity refers to the ability of the brain to compensate for lost function and/or to maximise remaining functions in the event of brain injury. For example, in the months following a brain injury, an individual may show very noticeable improvements that occur naturally with little or no intervention. These are associated with what is believed to be a period of physiological stabilisation of the injured area. Language recovery by adults after a stroke can be relatively extensive, but the full extent of the recovery may not be evident for 1 or 2 years. Recovery from traumatic brain injury by children in particular can be remarkable. However, there are also cases among children and adults where any measurable recovery does not occur.

How the brain changes in response to injury, and the effectiveness of its response, depends on...
Mechanisms underlying recovery
At the neuronal level, two important processes for recovery are rerouting and sprouting. In **rerouting**, an undamaged neuron that has lost a connection with an active neuron may seek a new active neuron and connect with it instead. **Sprouting** is the growth of additional branches on axons or dendrites to enable new connections. When sprouting occurs from a damaged neuron, the new growth projects to an area that has been ‘deactivated’ by damage to other neurons. Thus, sprouting involves rerouting as well.

Sprouting and rerouting enable the formation of entirely new neural connections to compensate for loss of function due to brain injury. This essentially means that the brain’s adaptive plasticity enables it to take over or shift functions from damaged to undamaged areas. Such plasticity can occur at all levels of the central nervous system, from the cerebral cortex down to the spinal cord.

In order for neurons to reconnect or form new connections, they need to be stimulated through activity. Relevant types of experience during recovery from brain injury are therefore important influences on the speed of recovery. For example, depending on the location and degree of brain damage, stroke or accident victims often need to ‘relearn’ tasks they previously performed routinely such as reaching, walking, speaking or reading. The younger the individual, the greater the likelihood of successful ‘relearning’ and subsequent new learning.

**Recovered functions**
Through adaptive plasticity, functions that were assigned to certain areas of the brain can sometimes be reassigned to other undamaged areas of the brain to compensate for changing input from the environment. For example, an extraordinary amount of stimulation of one finger can result in that finger ‘taking over’ a part of the somatosensory cortex that usually represents other adjacent fingers. If you lost your middle finger in an accident, the area of the somatosensory cortex that represents that finger will initially be unresponsive because there is no longer any sensory input received from the location of the missing finger. You might expect the ‘left middle finger neurons’ of the somatosensory cortex to degenerate and eventually disappear. Instead, over time, that area of the somatosensory cortex will begin to receive input from the adjacent fingers and become responsive to stimulation of these fingers.

This has been demonstrated experimentally in studies in which researchers have surgically destroyed areas of the somatosensory cortex of monkeys. The results of such studies typically show that the somatosensory cortical ‘map’ representing the destroyed areas gradually shifts to undamaged adjacent areas of the parietal lobes, restoring the ability to experience bodily sensations (Thompson, 2000).

A dramatic example of the brain’s reassignment of functions to other areas is evident when a function is taken over by the opposite cerebral hemisphere if injury destroys the part of the hemisphere where the function is primarily located. Recovery from other types of extensive brain injury by adults through adaptive plasticity can also be quite dramatic. Some patients with a paralysed hand or arm, for example, can recover its use within a few months.

The case of a 25-year-old female who was an accomplished pianist highlights this fact. She had a stroke that damaged left hemisphere areas of her brain and was unable to speak and lost complete use of her right hand. It was a devastating loss at a young age, and her inability to play the piano...
only added to her distress. The woman was placed in a rehabilitation program that involved repeated attempts to engage the right side of her body, including speech therapy and piano playing. After several months of rehabilitation, she regained nearly full use of her right hand, and she was again able to speak. She also demonstrated exceptionally rapid finger movements in both hands, displaying speed and coordination beyond those of the average (non-stroke-affected) person. Today she has resumed her piano playing and has fully recovered her abilities to the virtuoso levels attained before the stroke (Azari & Seitz, 2009).

FIGURE 4.17 Neuroplasticity enabled a stroke patient to fully recover her exceptional motor skills used as a concert pianist. Rehabilitation was an important part of her recovery.

An even more dramatic example of the brain's plasticity involves recovery of language following loss of a hemisphere. A 5-year-old boy had almost all the cerebral cortex of the left hemisphere surgically removed to treat his uncontrollable and life-threatening epileptic seizures. The boy had been experiencing as many as 10 to 12 seizures a day since he was 3. The results of various tests conducted before the radical surgery led to a psychological diagnosis that 'following onset and continuation of seizures, speech and learning steadily regressed and the patient was classified as retarded'. At first, the boy's language abilities worsened, but then improved rapidly.

Long-term follow-up tests over the next 21 years revealed above average language abilities and intelligence. It appeared that loss of most of the left hemisphere during early childhood had not impaired language development. The patient had gone on to complete a university degree and was assessed as also having an excellent memory and highly developed motor and spatial skills.

Whereas surgical removal of the left hemisphere of an adult's brain usually results in severe impairment of language, affecting both speech and writing, surgical removal of the left hemisphere during early childhood does not necessarily have permanent consequences for cognitive and behavioural functions (Breedlove, Watson & Rosenzweig, 2010; Devlin et al, 2003; Smith, Walker & Myers, 1988).

Adaptive plasticity does not only occur to compensate for damage. It can also occur as a consequence of everyday experience. For example, neuroimaging studies using PET and MRI show that in musicians who play string instruments, the area of the somatosensory cortex that represents the fingers of the left hand (the hand requiring greater motor learning for fine finger control) is larger than the area that represents the right hand (which is used to manipulate the bow), and larger than the left hand area in nonmusicians. Similarly, concert pianists have larger than usual cortical areas for finger control and professional quilters have highly developed areas for the thumb and forefinger, which are critical to their craft (Nelson, 1999).

There is also evidence that other brain areas can increase in size through extensive use. For example, to become a taxi driver in London, individuals have to go through a comprehensive training course (averaging about 34 months) and then pass a strict test of their ability to find the shortest route between any two locations. As a result of this type of training and assessment, London taxi drivers have become renowned for their ability to efficiently navigate their
way throughout one of the most complex and largest metropolitan areas in the world without using a street directory (or GPS).

When MRI scans of London taxi drivers (who find new routes daily) are compared with London bus drivers (who follow a limited number of set routes daily), they show that the rear part of the hippocampus of taxi drivers, which is involved in spatial navigation (and memory formation), is significantly larger.

And, the more years an individual has driven a taxi, the larger the hippocampal area, and vice versa (Maguire et al, 2000; Maguire et al, 2003).

**BOX 4.4 Rehabilitation with constraint-induced movement therapy**

Although there is often some spontaneous recovery from an acquired brain injury when the injury ‘settles’ with the passing of time, participation in a rehabilitation program is an important part of the recovery process, especially in moderate and severe cases.

One treatment that has been found to help stroke patients regain considerable use of a limb forces them to constantly use the limb. Constraint-induced movement therapy requires patients to use, for example, an affected arm, by immobilising the good arm for up to 90% of the time they are awake. In addition, the patient is required to practise moving the affected limb repeatedly for up to six hours a day.

In one study, 13 patients aged between 33 to 73 years regained as much as 75% of normal use of the paralysed arm within 12 days. There was also evidence of 'rewiring' (remapping) of the motor cortex. The researchers called this ‘treatment-induced plastic changes in the human brain’. In follow-up examinations up to 6 months after treatment, arm movement remained at a high level, and the size of the relevant motor cortex areas in the two hemispheres had become almost identical, ‘representing a return toward a normal condition’ (Liepert et al; 2000).

**FIGURE 4.19** Constraint-induced movement therapy involves immobilising a non-affected limb to force usage of the affected limb.
Most people who have had a limb amputated continue to experience sensations from where their missing limb was originally located. This is called **phantom limb syndrome**. The missing limb feels as if it still exists and some even report feeling a persistent itch, extreme discomfort or chronic pain where it was located. Phantom limbs may also be experienced as moving normally, or missing arms may ‘gesture’ during conversations as if they really existed.

Such experiences have long intrigued psychologists and a number of explanations have been proposed to explain them. Indian-born psychologist Vilayanur Ramachandran, a leading researcher on phantom limb syndrome, has proposed that it can be attributed to the brain’s plasticity.

Ramachandran stimulated the skin surface in various points on the face, arms and upper body while using functional neuroimaging to monitor brain activity in volunteer participants with an amputated limb. The same was done with a control group of participants who did not have an amputated body part. The scans showed areas of somatosensory cortex that were activated when different parts of the body were stimulated by touch.

In one experiment, Ramachandran and his colleagues (1992) found that participants with an amputated hand reported that stimulation of their cheek through touch (with a cotton swab) was perceived as if the stimulation was on their now-missing hand. In some cases, this sensation was quite precise. When specific areas of the face were stimulated, participants reported sensations in a particular area, such as just one finger, of the phantom hand.

The scans also revealed that stimulating the cheek activated an area in the somatosensory cortex that previously would have been activated by their hand. Both the cheek and hand are represented next to each other in the somatosensory cortex.

Following loss of the hand, the adjacent cortical area had both taken over the unused cortex previously representing the hand and also assumed its function. The new face and arm representations were now connected with each other, filling in the space occupied by the hand representation. Through its plasticity, the brain had reorganised itself to compensate for the loss of sensation from the missing hand.

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**LEARNING ACTIVITY 4.7**

**Review questions**

1. Explain the meaning of plasticity in relation to the brain.
2. Explain why the brain is considered to have plasticity with reference to two key points.
3. In what way does plasticity account for the fact that no two human brains are identical?
4. (a) Explain the meaning of adaptive plasticity with reference to brain injury.
   (b) Describe two neural mechanisms or processes that indicate and enable adaptive plasticity.
5. Give an example of research findings that support the occurrence of adaptive plasticity in response to:
   (a) everyday experience
   (b) recovery from brain injury.
6. Explain how adaptive plasticity enables compensation for lost brain function and/or maximises remaining functions in the event of brain injury.
7. Will the brain recover to some degree from all types of injuries? Explain your answer.
8. What role does rehabilitation play in plasticity and recovery from brain injury?
PARKINSON’S DISEASE

Parkinson’s disease is a progressive neurological disorder which is characterised by both motor and non-motor symptoms (Parkinson’s Australia, 2017a). Motor symptoms such as tremors, muscle rigidity, slow movements and unstable posture, primarily result from the progressive degeneration of neurons in the substantia nigra, which is located in the basal ganglia in the midbrain (see figure 4.18).

Neurons in the substantia nigra produce the neurotransmitter called dopamine so when the substantia nigra is diseased or damaged, the amount of dopamine available is markedly reduced. Dopamine carries messages between neurons to help ensure effective planning, initiation and maintenance of movements, both at rest and during periods of activity. If there are fewer neurons in the substantia nigra, less dopamine will be produced. This means that the motor cortex higher up in the brain receives fewer or irregular messages on how to control movements. Movement commands are disrupted because essential information about how and when to move has gaps or has not been received. This is what primarily contributes to the motor symptoms that are characteristic of Parkinson’s disease and often lead many people to define it as a ‘movement disorder’. However, Parkinson’s disease does not only affect movement, nor does, a decrease in dopamine necessarily account for all symptoms experienced with the disorder (Brain Foundation, 2017; Parkinson’s Australia, 2017a).

Although Parkinson’s disease is linked to the degeneration of dopamine-producing neurons, it is not known what actually causes this specific type of neurological disorder. Therefore, it is described as idiopathic, which means ‘having no known cause’.

Parkinson’s disease is not considered to be genetic though there is a family history of the disorder in a small percentage of cases. Neurotransmitters other than dopamine have also been suggested as contributory factors in the disease. For example, dopamine producing neurons in the midbrain also release the neurotransmitter called GABA (gamma amino butyric). This is the main inhibitory neurotransmitter in the central nervous system (but also found in the peripheral nervous system). Generally, GABA dampens (‘lowers’) the activity of other neurotransmitters to help stabilise neuronal activity and maintain balanced overall functioning of the nervous system. Without GABA, neurons fire too often and too easily.

Since people with Parkinson’s disease also have a reduced level of GABA, this can make it difficult to isolate the effects of dopamine and GABA deficiencies.

**FIGURE 4.21** Parkinson’s disease is a disorder of the central nervous system, involving primarily a degeneration of neurons deep within the brain in an area called the basal ganglia, and in particular a loss of dopamine-producing neurons in the substantia nigra. This cross-section shows the basal ganglia and substantia nigra, which interact with other structures in controlling and coordinating movement. The basal ganglia comprises three smaller structures (‘nuclei’) made up by clusters of neurons. The neurotransmitter GABA is also released by the dopamine producing neurons which has led researchers to investigate the role of GABA in Parkinson’s disease.
However, some researchers have proposed a range of possible effects of GABA or GABA depletion; for example, that GABA blocks the effect of dopamine and thereby worsens symptoms; that it contributes to the death of dopamine-producing neurons; that it makes the nervous system vulnerable to disturbed neuronal activity, and, that it contributes to the degeneration of the nervous system (Blaszczyk, 2016; Di Michele et al, 2013; Tritsch, Ding & Sabatini, 2012).

Environmental factors such as head injury and exposure to pesticides and other chemical toxins have been suggested as contributory factors or possible causes. The most significant risk factor seems to be age. Generally, however, the disease is currently thought to be the result of a complex interaction between genetic and environmental factors. (Parkinson’s Australia, 2017a).

**Symptoms**

The symptoms of Parkinson’s disease develop slowly and gradually progress over years. They tend to vary greatly between individuals diagnosed with the disorder and no two people will experience the condition the same way. According to Parkinson’s Australia (2017a), there are four key symptoms used for diagnostic purposes. All of these are motor symptoms.

**Motor symptoms**

1. **Tremor** involving continuous, involuntary shaking (trembling) of the body is the best-known symptom (but not present in all cases of Parkinson’s).

   Most often, tremors are ‘resting tremors’ and occur when the limb is not in use. Sometimes ‘action tremors’ are experienced. These occur when commencing some form of motor activity; for example, when the person walks, their hands may begin to shake. Usually tremor is worst at rest, improves during voluntary movement and disappears during sleep.

   ‘Restless legs’ is also common. This is when the person’s legs appear to move or feel as if they are moving constantly, especially at night.

2. **Muscle rigidity**, or ‘stiff muscles’, whereby the muscles seem unable to relax and are tight, even at rest, is another key symptom.

   Individuals report feeling that their muscles will not do what they want them to do. They may have difficulty performing automatic movements, such as swinging their arms when walking or rolling over in bed. They may feel their muscles are so tight that they have frozen and won’t actually move.

   Rigidity can also lead to lack of facial expression through loss of facial muscle tone. This sometimes gives the face a mask-like appearance.

3. **Slowness of movement** (called bradykinesia), particularly when initiating and executing movement and in performing repetitive movements, presents in a variety of ways, including difficulty starting new movements or stopping an ongoing movement.

   There is a decrease in fine motor coordination required for ‘delicate’ work with the hands such as when doing up buttons, putting on make-up, shaving or slicing vegetables. Difficulty with turning over in bed is common, as are problems with handwriting becoming slow and small.

   Because of its impact on everyday activities, bradykinesia can be one of the most disabling symptoms.

4. **Postural instability**, balance problems and gait (walking) disturbances occur later in the course of the disease. Inability to maintain a steady, upright posture or to take a corrective action to prevent a fall often results in just that — falling. Individuals tend to go backwards as well, and a light shove may cause them to continue taking many steps backwards or to fall.

   Gait disturbance is apparent in the short, shuffling steps taken by individuals and reduced arm swing.

**FIGURE 4.22** The use of a balance aid often becomes necessary late in the course of Parkinson’s disease to assist with mobility and help prevent falls.
Non-motor symptoms
Lack of dopamine is believed to also contribute to many of the symptoms classified as non-motor.

Speech problems, especially change in verbal fluency, are a common non-motor symptom. The muscles involved in speech may be affected which can reduce the volume, clarity and speed of speech. For example, speech can become rapid, with the words crowded together, similar to the short, shuffling, ‘propelling’ steps when walking. The muscles involved in swallowing can also be affected, making it difficult to chew or swallow.

Other symptoms may include a decrease or loss of sense of smell, pain and discomfort in an arm or leg, tiredness and disturbed sleep, constipation, problems urinating, and mental health problems such as confusion, panic attacks, anxiety disorder and depression.

Problems with cognitive function such as slowness of thinking, impaired planning and decision making and memory loss may occur in up to 40–50% of people with Parkinson’s disease, especially late in the disease and in older people. However, cognitive impairments are also associated with other age-related disorders (such as dementia) so it can be difficult to isolate the actual cause (Golbe, Mark & Sage, 2014; Parkinson’s Australia, 2017a).

Due to the very slow onset of Parkinson’s disease, it can take a while for people to notice then realise their reduced ability to control movement and other motor or non-motor symptoms. For some, a slight tremor of the hand when it is relaxed and not in use will be the first sign that something is wrong. For others, deterioration in the sense of smell, difficulty with walking, or falling due to disturbed balance control, may be the first sign of the disease. The symptoms of Parkinson’s disease also tend to vary in severity from day to day and at different times throughout the day.

Diagnosis and treatment
The average age of diagnosis of Parkinson’s disease is between 55 and 65 years, though it can affect anyone at any time, including much younger people. It is estimated that about 1 in 350 people in Australia have the condition, with the incidence increasing to about 1 in 100 people over the age of 60. It is slightly more common in men than in women (Brain Foundation, 2017; Parkinson’s Australia, 2017a).

Diagnosis is based on the individual’s presenting symptoms, a neurological examination, a review of their past medical history and their response to Parkinson’s medications if the disease is suspected. However, there are no really adequate or specific biological or neuroimaging tests available for a diagnosis that would confirm the presence of the disorder.

At present, there is no known cure for Parkinson’s disease. It is not contagious, lifespan is not necessarily shortened and medications can help treat symptoms and improve quality of life for a very long time. Because of the complex nature of Parkinson’s disease, for each individual its management requires a holistic, biopsychosocial approach which takes account of all aspects of the affected person’s life, not just their motor problems. Given that no two people are affected in the same way, so management will vary (Brain Foundation, 2017).

Motor symptoms such as tremor, muscle rigidity and slowness of movement may be relieved by medications that restore the deficiency of dopamine. Two types of dopamine-affecting medications can be used — those that can be converted into dopamine by the brain and those that are able to effectively stimulate dopamine by the neurons. Other medications that influence the activity of other neurotransmitters that can directly or indirectly affect motor symptoms may also be used.

In some cases, deep brain stimulation of the substantia nigra within the basal ganglia may be a treatment option, depending on the symptoms. As a result, the individual may be able to treat the amount of medication previously required. However, not all Parkinson’s symptoms will necessarily respond to the stimulation.

Other ‘neurosurgical’ options involve cutting (lesioning) specific parts of the brain to alleviate targeted motor symptoms. However, none of the drugs or other interventions can prevent the progression of the disease (Parkinson’s Australia 2017a)

FIGURE 4.23 According to Parkinson’s Australia (2017b), exercise has been proven to be essential in maintaining mobility and quality of life.
One of the most commonly used and effective medications is L-dopa, made from levodopa, a chemical that is converted to dopamine by neurons and thereby replaces dopamine lost in Parkinson’s disease.

It is relatively common for people to require high doses of medication and therefore experience side effects as the disease progresses and natural dopamine production is reduced. In many cases, medication has a maximum benefit for a period of 5–10 years. Many patients report that some of the side effects are as disabling as the disease itself (Parkinson’s Victoria, 2015).

**Use of animal studies and neuroimaging techniques to develop understanding of Parkinson’s disease**

Researchers often use animal studies and neuroimaging techniques to understand human neurological disorders such as Parkinson's disease. The main goals of this research are to understand the physiological changes associated with the disorder, to prevent the disease, to slow its progression after its onset and to develop treatments (e.g. treat the symptoms with as few side effects as possible). In this section we examine examples of research studies that have used animals and/or neuroimaging techniques.

**Animal studies**

There have been numerous animal studies conducted throughout the past four decades to investigate various aspects of Parkinson’s disease. Most have used mice, rats, rabbits and monkeys. These mammals are substitutes for people because of obvious ethical constraints for research with humans.

The use of animals allows researchers to investigate Parkinson’s disease in ways which would not be possible with humans. This includes performing procedures on animals to induce Parkinson’s symptoms, such as damaging the relevant dopamine-producing brain area or dopamine pathways to the motor cortex. Alternatively, animals may be injected with substances for use in drugs being developed to prevent or treat symptoms. For example, researchers can administer chemical agents that target neurons in the substantia nigra and induce their degeneration so that dopamine production is restricted. They can then use a drug under development to test whether it can counter the effects of their experimental manipulation (Blandini & Armentero, 2012).

It is assumed that causes and treatments of Parkinson’s disease will be similar in both the animal species selected for study and in humans because of similarities in brain structures and functions. This is important so that the animal reacts to Parkinson’s or its treatments like humans do. For example, small mammals have been used in studies on Parkinson’s disease because they have dopamine in their brain and nervous system and it functions in much the same manner as it does in people. In particular, dopamine has been found to have a role in their control of voluntary movements.

So, researchers find it reasonable to claim that we can learn about Parkinson’s disease (and other neurological disorders) in humans from the study of the brain and behaviour in animals. The expectation is that discoveries made in the animal studies will provide valuable insights into the disease in humans that may otherwise have not been possible. Of course, care is taken when drawing conclusions and making generalisations from one species to another.

**Discovery of levodopa**

Some animal studies have been particularly valuable when considered in terms of their immediate or long-term outcomes. For example, Swedish doctor Arvid Carlsson was awarded the Nobel Prize in Physiology or Medicine in 2000 for his animal studies on Parkinson’s disease. His experiments revealed that dopamine played a role in the control of voluntary movements, and was linked to Parkinson’s disease. His experiments also led to the development of levodopa for treating the disorder.

![Swedish doctor Arvid Carlsson used animal studies to develop levodopa, the most commonly used medication for treating symptoms of Parkinson’s disease.](Image)
Carlsson (1957) studied rabbits to which he administered a drug (reserpine) to decrease the level of dopamine in their brain. He hypothesised that this would cause loss of movement control, which is what occurred in a very dramatic way. The rabbits became very lethargic and their movements were significantly impaired. The effects were very similar to the symptoms of Parkinson’s. However, he also found that their movement could be restored by injecting them with levodopa, a chemical that is converted to dopamine by neurons.

When follow-up studies by Carlsson led him to conclude that a lack of dopamine caused Parkinson’s disease, it became apparent that levodopa could be used as a drug to alleviate its symptoms. This led other doctors to try using levodopa on patients with Parkinson’s disease, and found it alleviated some of the motor symptoms in the early stages of the disease. Levodopa, also called L-dopa, subsequently became the first treatment for Parkinson’s disease.

More recently, researchers have used mouse models to help understand why the standard levodopa treatment for Parkinson’s disease is often effective for only a limited period of time. They found that midbrain dopamine producing neurons also released GABA, thereby raising such questions as to what role GABA has in Parkinson’s disease and which Parkinson disease effects are due to loss of GABA and which are due to loss of dopamine. This has led to further research to investigate more effective long-term management of symptoms (Tritsch, Ding & Sabatini, 2012).

**Deep brain stimulation treatment**

Studies with rats and monkeys led to the development of a surgical treatment for Parkinson’s disease when levodopa and other drugs are not effective. The treatment is called **deep brain stimulation**, an invasive procedure whereby a surgeon implants electrodes (tiny wires) within the basal ganglia. The electrodes, which are connected to a pulse generator implanted under the skin of the chest, stimulate the target area with tiny amounts of electric current. The electrical activity blocks the faulty neuronal activity that causes tremor, rigidity, and other motor symptoms. The pulse generator is similar to a heart pacemaker and about the size of a stopwatch. Because the left hemisphere controls the right side of the brain and vice versa, deep brain stimulation is commonly performed on both sides of the brain.

Deep brain stimulation has been found to improve motor symptoms and reduce the need for levodopa in over 80,000 human patients throughout the world. However, as with any neurosurgery, there are risks.

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**FIGURE 4.25** Animal studies led to the development of deep brain stimulation as a surgical treatment for some people with Parkinson’s disease. The apparatus shown allows precise positioning of electrodes for stimulating the substantia nigra or any other targeted brain area. Neuroimaging is used to assist with mapping, measuring and monitoring of electrode insertion.

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`<Permission clearance pending>`
involved. In the case of deep brain stimulation, these include death, stroke, speech changes, difficulties with decision making or infection. In addition, some people may experience increased depression or anxiety which may not be reversible (Frank, et al., 2007; Parkinson’s Australia, 2017c).

**Neuroimaging studies**

Structural and functional neuroimaging techniques have long been used in the study of Parkinson’s disease. These have enhanced understanding of brain areas and processes underlying the disease, enabled earlier and more precise diagnosis, and supported evaluation of existing and potential new treatments. Neuroimaging studies are not conducted with only human participants. Many animal studies have also been conducted, often with monkeys.

CAT and MRI mainly provide detailed images of degeneration in brain areas and pathways. These are especially useful for diagnostic purposes to assess the nature and extent of damage and to monitor degeneration over time. Images can be taken at the neuronal level within dopamine-producing areas and neural pathways along which dopamine is used.

However, CAT and MRI images are static (still). They provide useful clues about brain function on the basis of structural abnormalities but do not actually display its activity. When used for diagnostic purposes, the image can also help rule out other conditions which may resemble Parkinson’s disease or structural abnormalities which may result in Parkinson’s-like symptoms (Parkinson’s Australia, 2017c).

Functional neuroimaging techniques such as PET and fMRI are preferred for research purposes as they provide detailed images of both brain structure and activity. Researchers can use these techniques in very precise ways; for example, to measure changes in the release of dopamine at synapses within the basal ganglia and observe the impact in dopamine pathways for movement. Importantly, it is possible to observe activity within various dopamine-producing and motor areas throughout the brain at the same time.

Functional neuroimaging techniques have also increased diagnostic accuracy and enabled earlier diagnosis through their very detailed images at the neuronal level. They allow more detailed monitoring of the rate of progression of the disease, as well as the impact of different treatments. In particular, functional techniques have been increasingly used to study symptoms other than the motor ones of tremor, muscle rigidity and bradykinesia. For example, researchers have used PET and fMRI to study changes in mental processes among people with the disorder, such as cognitive functioning and impairments in personality or social behaviour (Niethammer, Feigin & Eidelberg, 2012).

There has been a rich variety of neuroimaging studies on Parkinson’s disease. Some of the more recent investigations have investigated and enhanced understanding of:

- brain functionality and processes underlying different motor symptoms e.g. areas of high and low activity when compared with non-Parkinson’s patients
- consequences of reduced dopamine levels on non-motor mental processes and behaviour
- consequences of reduced levels of neurotransmitters on symptoms, neural activity or nervous system degeneration
- non-motor symptoms of the disease that may precede the motor symptoms and provide evidence of onset
- activity in specific areas of the motor cortex associated with different symptoms such as hand tremors and rigidity

![FIGURE 4.26 PET scans comparing dopamine activity in the substantia nigra of a healthy individual and a patient with Parkinson’s disease. The radioactive tracer is seen as yellow, green and orange. Note the greater uptake of the tracer and activity in the scan from a healthy person.](image-url)
• the impact of different treatments, particularly medications e.g. effects of various dosages and frequency of use, how quickly levodopa impacts on dopamine levels and usage, responsiveness to the medication at the individual symptom level, age-related differences in responsiveness, differences in performance on motor tasks by patients taking and not taking levodopa, differences between patients who have levodopa wear off and those who don't

• the effects of gene therapies that may slow the disease's progression and avoid side effects of existing treatments e.g. insert copies of a gene that may minimise dopamine depletion

• new ways of compensating for the loss of neurons e.g. implant fetal stem cells in the brains of individuals with Parkinson's disease to establish new, healthy connections where dopamine may be released and thereby minimise the symptoms or their effects.

**BOX 4.6 Motor neurone disease**

Motor neurone disease (MND) is a general term used to describe a group of diseases in which motor neurons controlling the muscles degenerate and die. This gradually impairs the abilities to move around, speak, breathe and swallow. With no neurons to activate them, muscles gradually weaken and waste (MND Australia (MNDA), 2017a).

Like Parkinson's disease, MND is degenerative — the person's ability to voluntarily control their muscles gradually deteriorates after its onset. However, MND is terminal, whereas Parkinson's is not.

MND may be diagnosed in anyone at any age but it tends to be most common among adults between the ages of 50 and 60. It is a much rarer disorder than Parkinson's disease. There are over 400,000 people worldwide with MND, and about 2000 people in Australia. There are slightly more men than women. About 10% of people with MND have a family history of the disorder.

Motor neurone disease can be difficult to diagnose because the initial symptoms can be similar to many other conditions. The early symptoms are mild, and may include feeling unbalanced and wobbly when standing or stumbling due to weakness of the leg muscles, difficulty with holding objects or turning on a tap due to weakness of the hand muscles, slurring of speech, or swallowing difficulties due to weakness of the throat and tongue muscles.

About 50% of people with MND may experience some change in cognitive abilities, language, behaviour and personality, but usually change is mild. These changes are due to impaired functioning of the frontal and temporal lobes and are associated with MND. The functions of the bowel and bladder are usually not affected, nor the senses of sight, hearing, smell and touch.

Once the symptoms appear, average life expectancy is 2.5 years, although it can occasionally be longer than 6 years. The effects of MND on each individual vary enormously in relation to the initial symptoms, rate and pattern of progression, and survival time after diagnosis.

There is currently no cure for MND but some treatments can help people living with the disease to live better for longer. Only one medication is approved for treatment in Australia. This is called riluzole and is sold as Rilutek™ or APO-Riluzole. Riluzole prolongs survival by about two to three months. However, research indicates that people who start taking riluzole early in the onset of MND are more likely to remain in the milder stages of the disease for longer than those not taking riluzole (MNDA, 2017b).

The famous physicist Stephen Hawking, who has helped to bring his ideas about black holes and quantum gravity to the general public, was first diagnosed with a form of MND (called amyotrophic lateral sclerosis) when he was aged 21 and was not expected to live much longer. Now aged over 70, he spends most of his waking life in a wheelchair and speaks through a computer system which he operates with his cheek. However, he works as the director of research at the Centre for Theoretical Cosmology at Cambridge University in England and continues to generate new theories.

**FIGURE 4.27** Stephen Hawking has lived with a form of MND since he was aged 21, which is most unusual.
LEARNING ACTIVITY 4.9

Review questions

1. (a) Explain what Parkinson’s disease (PD) is with reference to key motor and non-motor symptoms. (b) How may speech fluency be influenced by motor impairment?
2. Give two examples of how PD may impact on psychological and social functioning.
3. Why can PD be described as (a) a neurological disorder, (b) a neurodegenerative disorder and (c) an idiopathic disorder?
4. Explain how a low level of dopamine is believed to impair motor activity.
5. Briefly describe two possible treatments for PD, outlining how they work and several potentially significant side effects of each treatment.
6. List three outcomes from animal and/or neuroimaging studies that have led to benefits for people with PD.

LEARNING ACTIVITY 4.10

Reflection

Consider some of the findings of animal studies on Parkinson’s disease. Comment on whether animal studies on the disorder are justifiable.

LEARNING ACTIVITY 4.11

Media response

View this short video on 5 daily exercises to boost your brain power.
1. Comment on whether these types of exercise can actually ‘boost brain power’.
2. Which one or more of the exercises would change the brain through plasticity? Explain your answers.

eGuideplus

Weblink

Video on five daily exercises to boost your brainpower 1 min 54 s
KEY TERMS

- adaptive plasticity p. 000
- brain injury p. 000
- myelination p. 000
- Parkinson’s disease p. 000
- plasticity p. 000
- rerouting p. 000
- spatial neglect p. 000
- sprouting p. 000
- synaptic pruning p. 000
- synaptogenesis p. 000
- traumatic brain injury p. 000

LEARNING CHECKLIST

Complete the self-assessment checklist below, using ticks and crosses to indicate your understanding of this chapter’s key knowledge (a) before and (b) after you attempt the chapter test on pages 215–17. Use the ‘Comments’ column to add notes about your understanding.

<table>
<thead>
<tr>
<th>Key knowledge I need to know about the neural basis of learning and memory</th>
<th>Self-assessment of key knowledge I understand before chapter test</th>
<th>Self-assessment of key knowledge I need to do more work on after chapter test</th>
<th>Comments</th>
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<td>Development of myelin</td>
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<td>Synaptogenesis and synaptic pruning</td>
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<td>Frontal lobe development</td>
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<td>Diagnosis and treatment</td>
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<td>Use of animal studies and neuroimaging techniques to develop understanding of Parkinson’s disease</td>
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<td>• Discovery of levodopa</td>
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<td>• Neuroimaging studies</td>
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study on

- Unit 2
- Area of study 1
- Topic 1

Concept screens and practice questions
SECTION A — Multiple-choice questions

Choose the response that is correct or that best answers the question. A correct answer scores 1, an incorrect answer scores 0. Marks will not be deducted for incorrect answers. No marks will be given if more than one answer is completed for any question.

Question 1
Which of the following statements about brain growth and development is correct?
A. All human brains are identical.
B. Experience cannot change the brain’s neuronal organisation.
C. Infancy and adolescence are periods of rapid development and changes in brain structure and function.
D. Sensory and motor areas in the cerebral cortex develop and mature before hindbrain areas.

Question 2
Brain size is about 90–95% adult size by the age of about
A. 1 month.
B. 6 months.
C. 6 years.
D. 25 years.

Question 3
Synaptogenesis
A. is an acquired brain injury.
B. starts before birth.
C. starts after birth.
D. ‘fine tunes’ neural connections.

Question 4
When neural connections are repeatedly used, then it is likely that they will
A. strengthen.
B. weaken.
C. be pruned.
D. disappear.

Question 5
Two bursts of brain myelination occur
A. shortly before and shortly after birth.
B. shortly after birth and during adolescence.
C. shortly before and shortly after adolescence.
D. during adolescence and shortly before adulthood.

Question 6
The last brain area to reach maturity tends to be the
A. motor cortex.
B. sensory cortex.
C. cerebral cortex.
D. prefrontal cortex.

Question 7
The case study of Phineas Gage demonstrates the effects of brain injury to the cortex in the _____ lobe.
A. frontal
B. parietal
C. temporal
D. occipital

Question 8
Spatial neglect is most commonly associated with damage in the _____ lobe.
A. frontal
B. occipital
C. temporal
D. parietal

Question 9
Motor symptoms of Parkinson’s disease are believed to primarily result from
A. depleted amounts of dopamine produced in the motor cortex.
B. loss of dopamine as it travels along motor pathways.
C. over-production of dopamine in the basal ganglia.
D. depleted amounts of dopamine produced in the substantia nigra.

Question 10
Parkinson’s disease is best described as a _____ disorder.
A. central nervous system
B. somatic nervous system
C. cognitive
D. terminal
Question 11
A synapse is
A. a neural connection.
B. a type of neurotransmitter.
C. the place where neurons communicate.
D. the part of the neuron where small extensions (branches) grow.

Question 12
The process of eliminating synaptic connections is called
A. synaptic pruning.
B. dendritic sprouting.
C. adaptive plasticity.
D. dendritic growth.

Question 13
When a brain area assumes or ‘takes over’ the function of an adjacent damaged brain area, this is best described as
A. synaptic pruning.
B. dendritic sprouting.
C. adaptive plasticity.
D. dendritic growth.

Question 14
A brain injury due to neural degeneration
A. has gradual onset.
B. is a traumatic brain injury.
C. has sudden onset.
D. is not classified as acquired.

Question 15
Brain maturation is generally complete by the end of
A. infancy.
B. childhood.
C. adolescence.
D. early adulthood.
SECTION B — Short-answer questions

Answer all questions in the spaces provided. Write using black or blue pen.

**Question 1** (1 mark)
Explain the meaning of neurodegenerative in relation to brain injury.

**Question 2** (2 marks)
Explain the difference between a brain injury and a head injury.

**Question 3** (1 mark)
Describe a potential biological, psychological or social change that may be caused by a severe brain injury to the prefrontal cortex.

**Question 4** (2 marks)
One characteristic of the general pattern of brain growth and development is bottom to top growth; that is, the hindbrain develops first, then the midbrain then the forebrain. What are two other characteristics?

**Question 5** (4 marks)
Name and describe two processes other than genes and maturation that contribute to growth in brain size soon after birth.

**Question 6** (1 mark)
Describe a potential benefit of studying changes in patients with cerebral cortex damage.
Question 7 (2 marks)
Explain why an immature or under-developed prefrontal cortex in adolescence is believed to contribute to a higher incidence of poor decision making and impulsive behaviour during that period compared with other periods in development.

Question 8 (4 marks)
(a) Explain the meaning of adaptive plasticity in relationship to the brain.

(b) When during the lifespan is adaptive plasticity more likely to assist recovery from brain injury?

(c) Explain your answer to (b) above.

(d) Give an example of a naturally occurring neural mechanism that assists recovery from brain injury through adaptive plasticity.

Question 9 (1 mark)
Give an example of a type of study on Parkinson’s disease that may be conducted using a neuroimaging technique.

Question 10 (2 marks)
(a) What is an assumption underlying animal studies on Parkinson’s disease?

(b) Give an example of a research finding from animal studies that improved understanding or treatment of Parkinson’s disease.