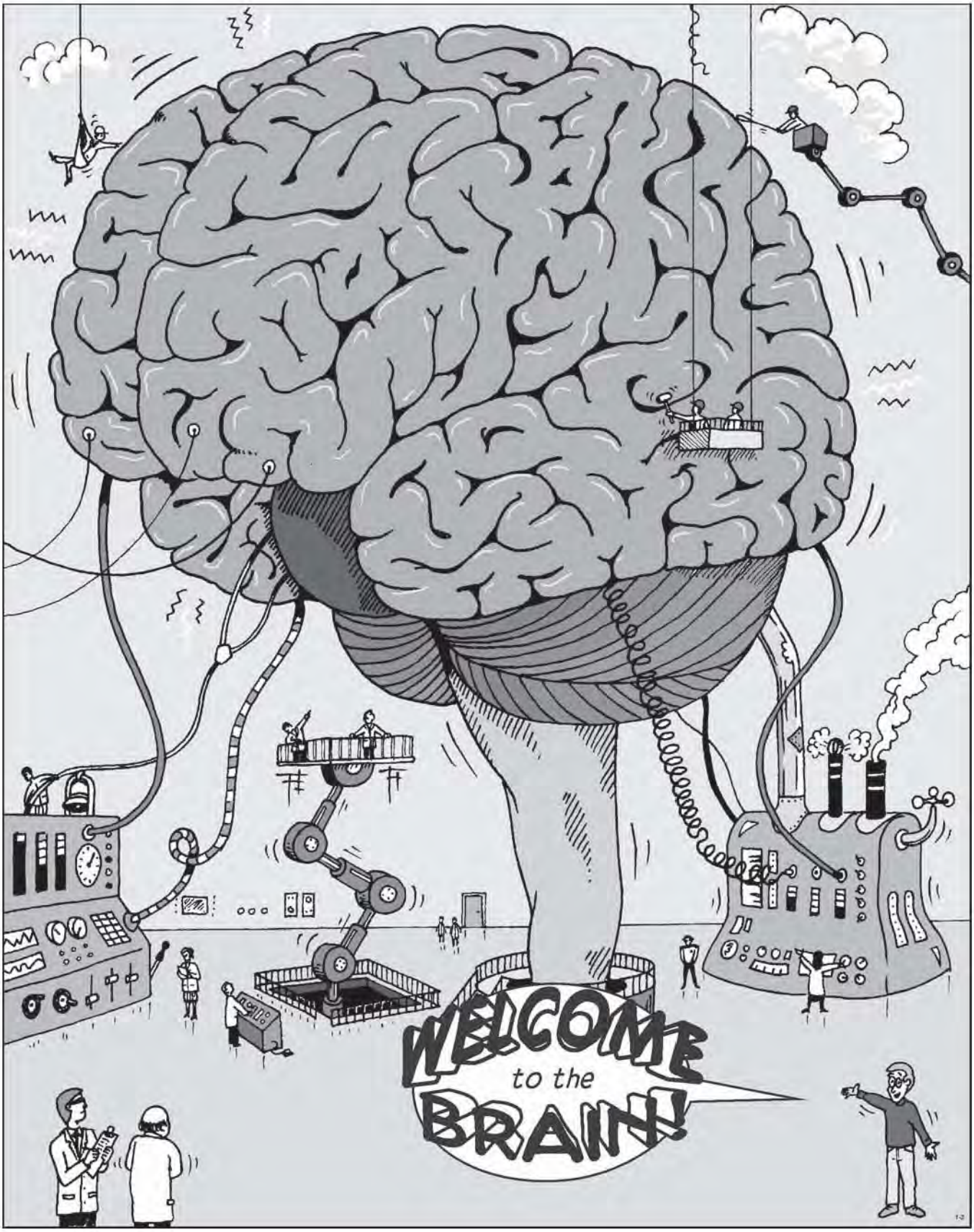
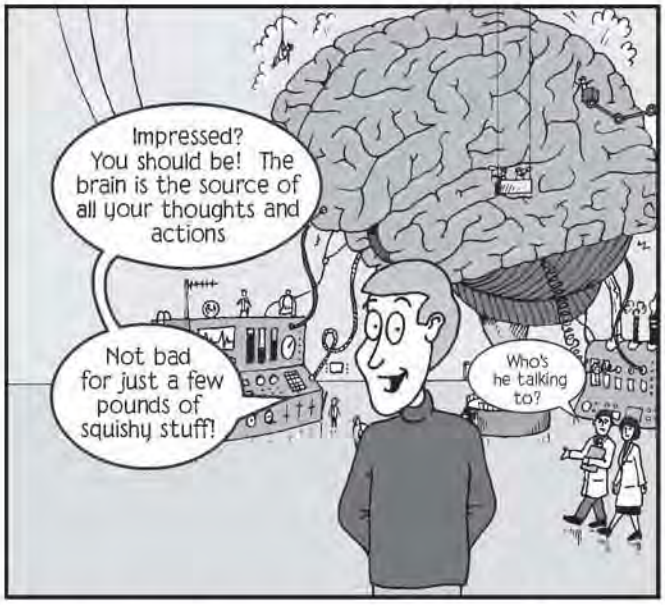
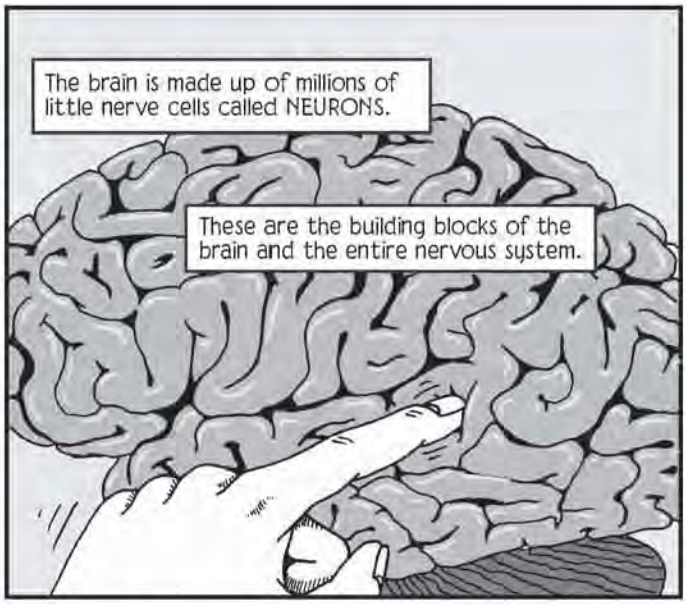
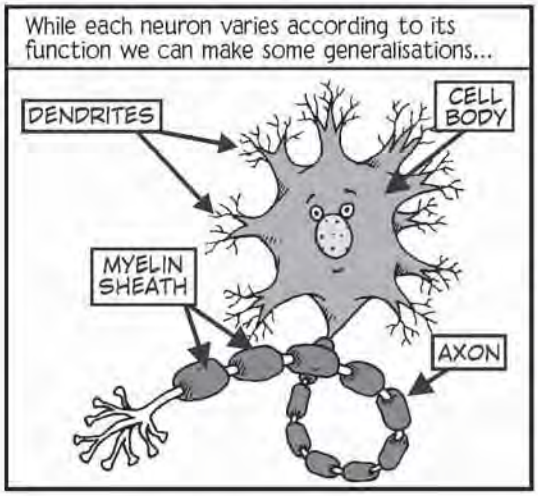
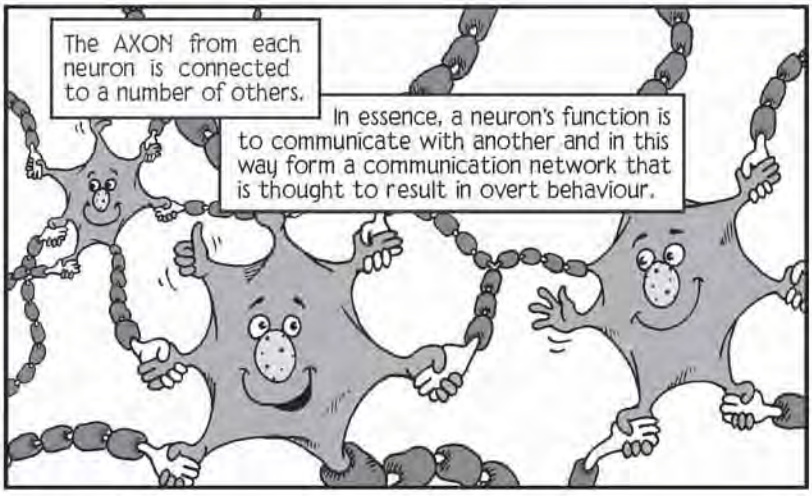
CHAPTER 1
THE BRAIN AND THE NERVOUS SYSTEM

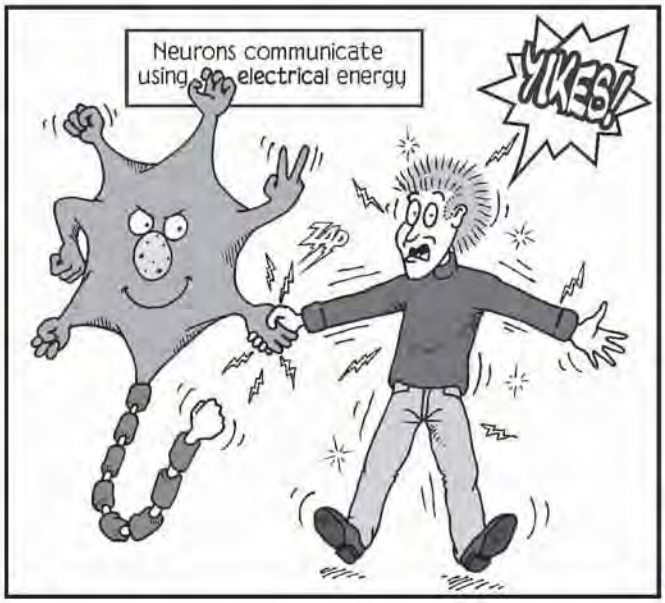

WELCOME
to the
BRAIN!

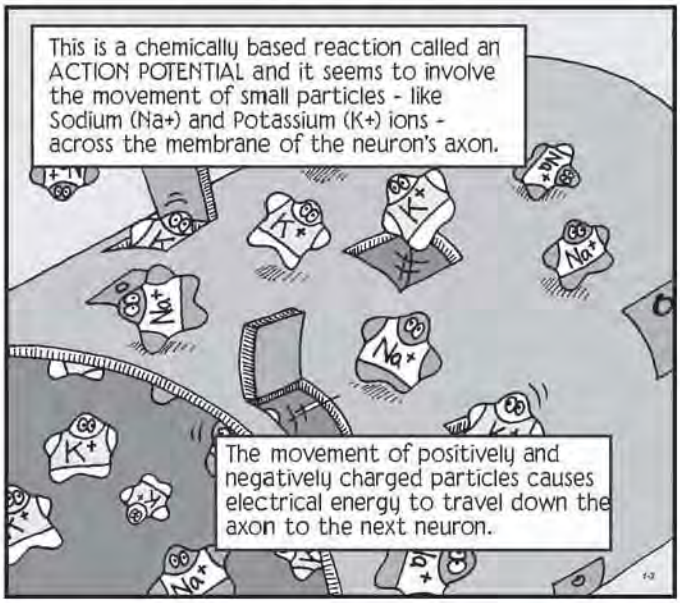

Impressed? You should be! The brain is the source of all your thoughts and actions
Not bad for just a few pounds of squishy stuff!
Who's he talking to?

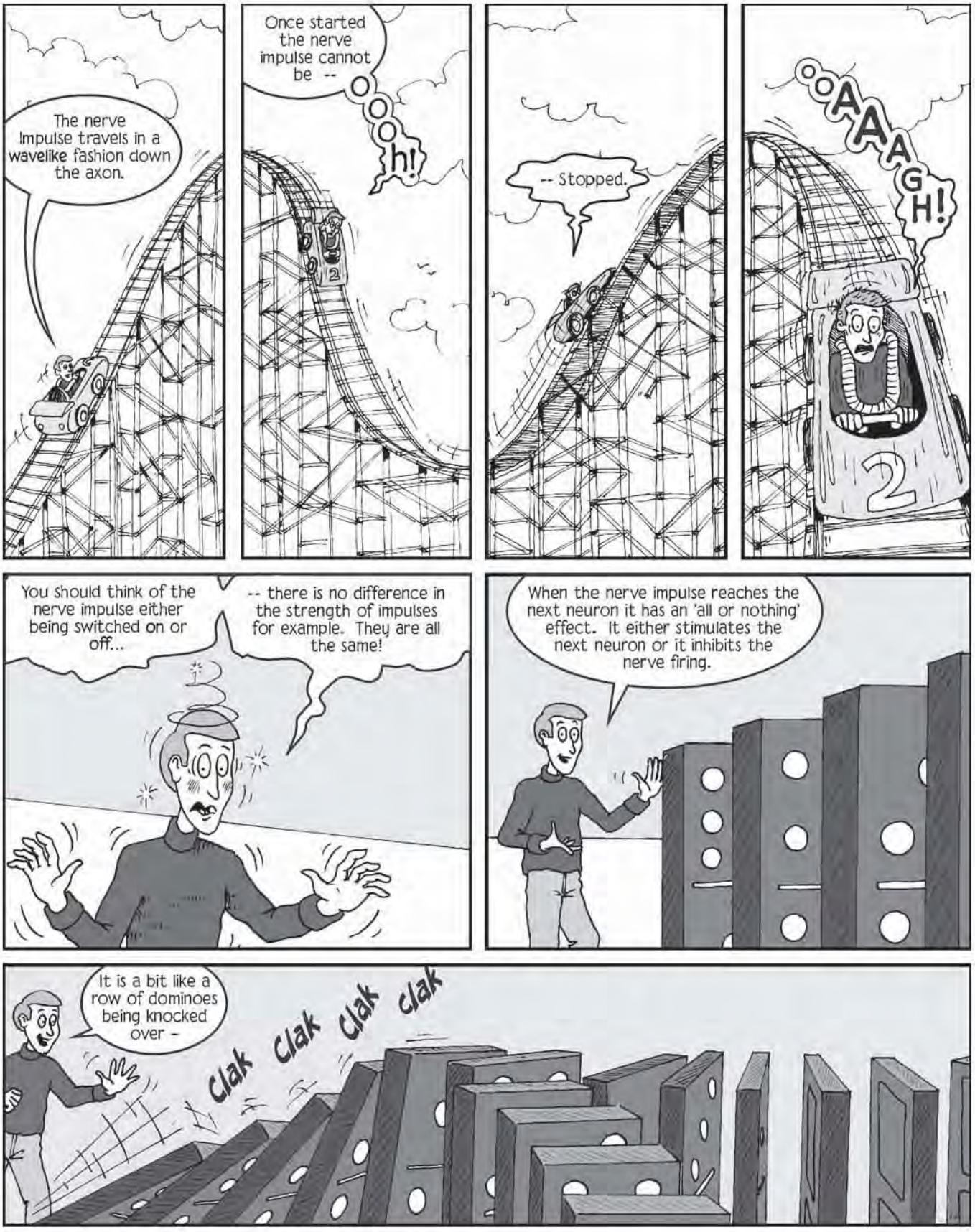

The brain is made up of millions of little nerve cells called NEURONS.
These are the building blocks of the brain and the entire nervous system.

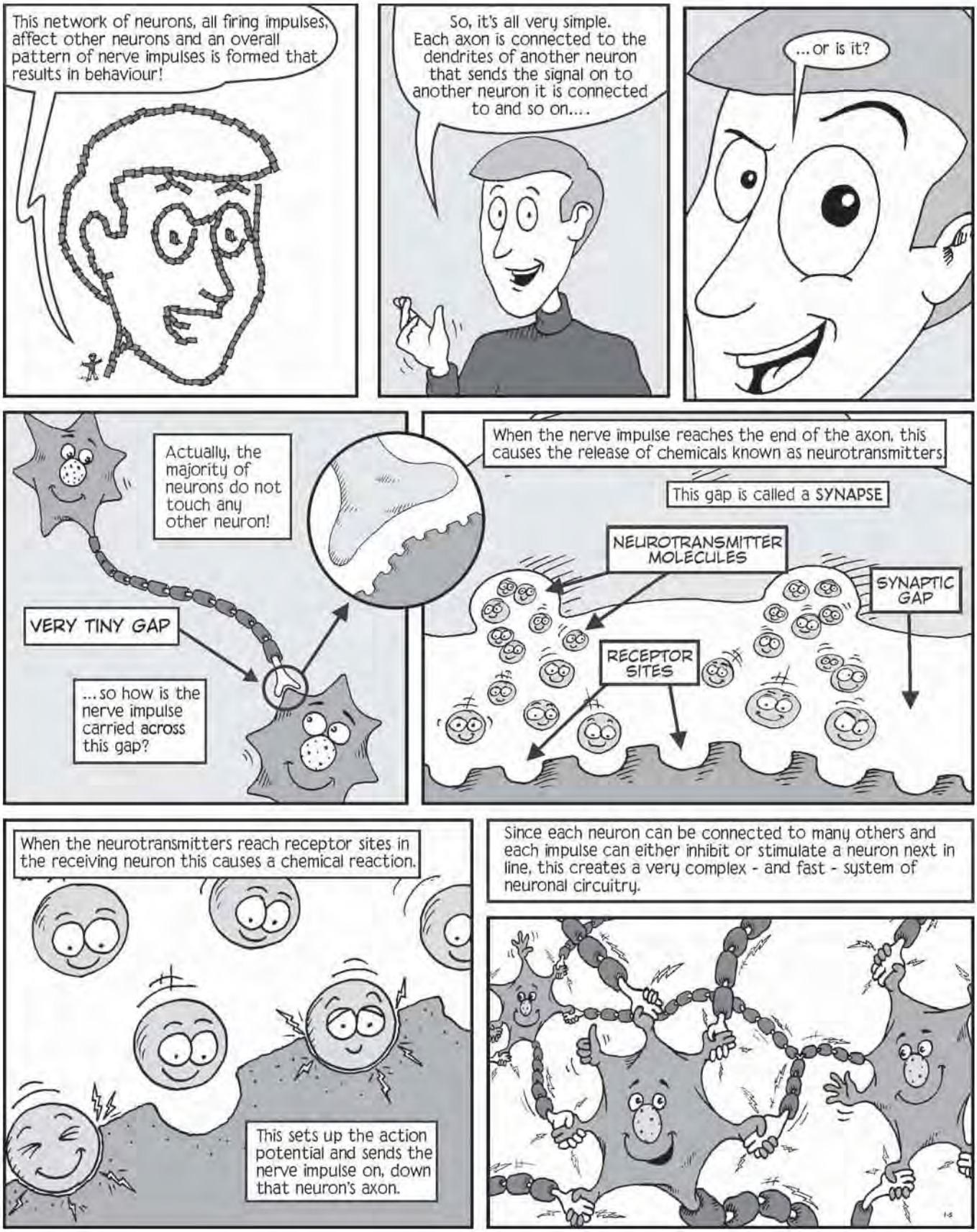

While each neuron varies according to its function we can make some generalisations...
DENDRITES
CELL BODY
MYELIN SHEATH
AXON

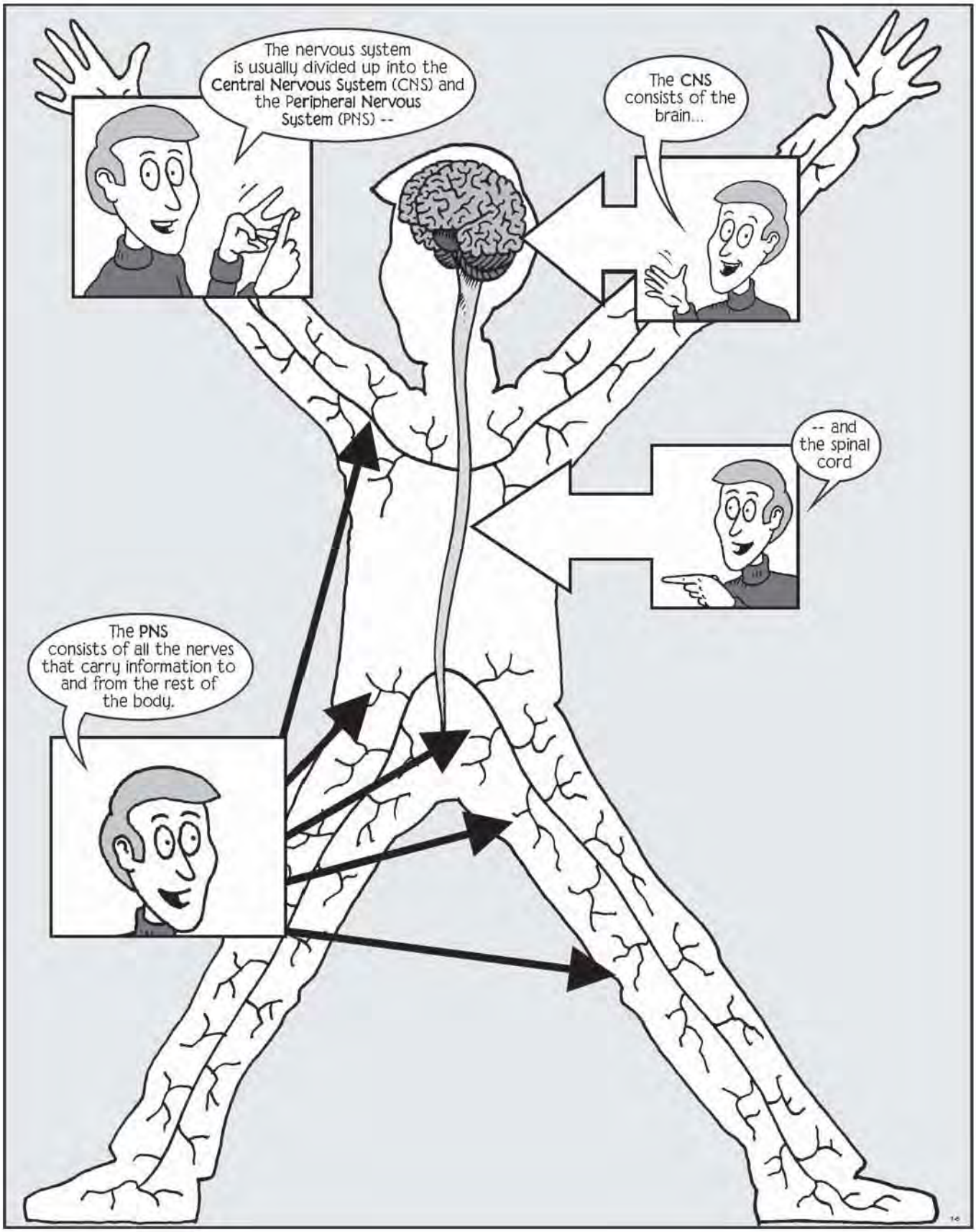

The AXON from each neuron is connected to a number of others.
In essence, a neuron's function is to communicate with another and in this way form a communication network that is thought to result in overt behaviour.


Neurons communicate using electrical energy
YIKES!
ZAP


This is a chemically based reaction called an ACTION POTENTIAL and it seems to involve the movement of small particles - like Sodium (Na+) and Potassium (K+) ions - across the membrane of the neuron's axon.
The movement of positively and negatively charged particles causes electrical energy to travel down the axon to the next neuron.


The nerve impulse travels in a wavelike fashion down the axon.
Once started the nerve impulse cannot be --
OOOh!
-- Stopped.
OOAAAGH!
2
You should think of the nerve impulse either being switched on or off...
-- there is no difference in the strength of impulses for example. They are all the same!
When the nerve impulse reaches the next neuron it has an 'all or nothing' effect. It either stimulates the next neuron or it inhibits the nerve firing.
It is a bit like a row of dominoes being knocked over -
Clak Clak Clak Clak


This network of neurons, all firing impulses, affect other neurons and an overall pattern of nerve impulses is formed that results in behaviour!
So, it's all very simple. Each axon is connected to the dendrites of another neuron that sends the signal on to another neuron it is connected to and so on....
...or is it?
Actually, the majority of neurons do not touch any other neuron!
VERY TINY GAP
...so how is the nerve impulse carried across this gap?
When the nerve impulse reaches the end of the axon, this causes the release of chemicals known as neurotransmitters
This gap is called a SYNAPSE
NEUROTRANSMITTER MOLECULES
SYNAPTIC GAP
RECEPTOR SITES
When the neurotransmitters reach receptor sites in the receiving neuron this causes a chemical reaction.
This sets up the action potential and sends the nerve impulse on, down that neuron's axon.
Since each neuron can be connected to many others and each impulse can either inhibit or stimulate a neuron next in line, this creates a very complex - and fast - system of neuronal circuitry.


The nervous system is usually divided up into the Central Nervous System (CNS) and the Peripheral Nervous System (PNS) --
The CNS consists of the brain...
-- and the spinal cord
The PNS consists of all the nerves that carry information to and from the rest of the body.

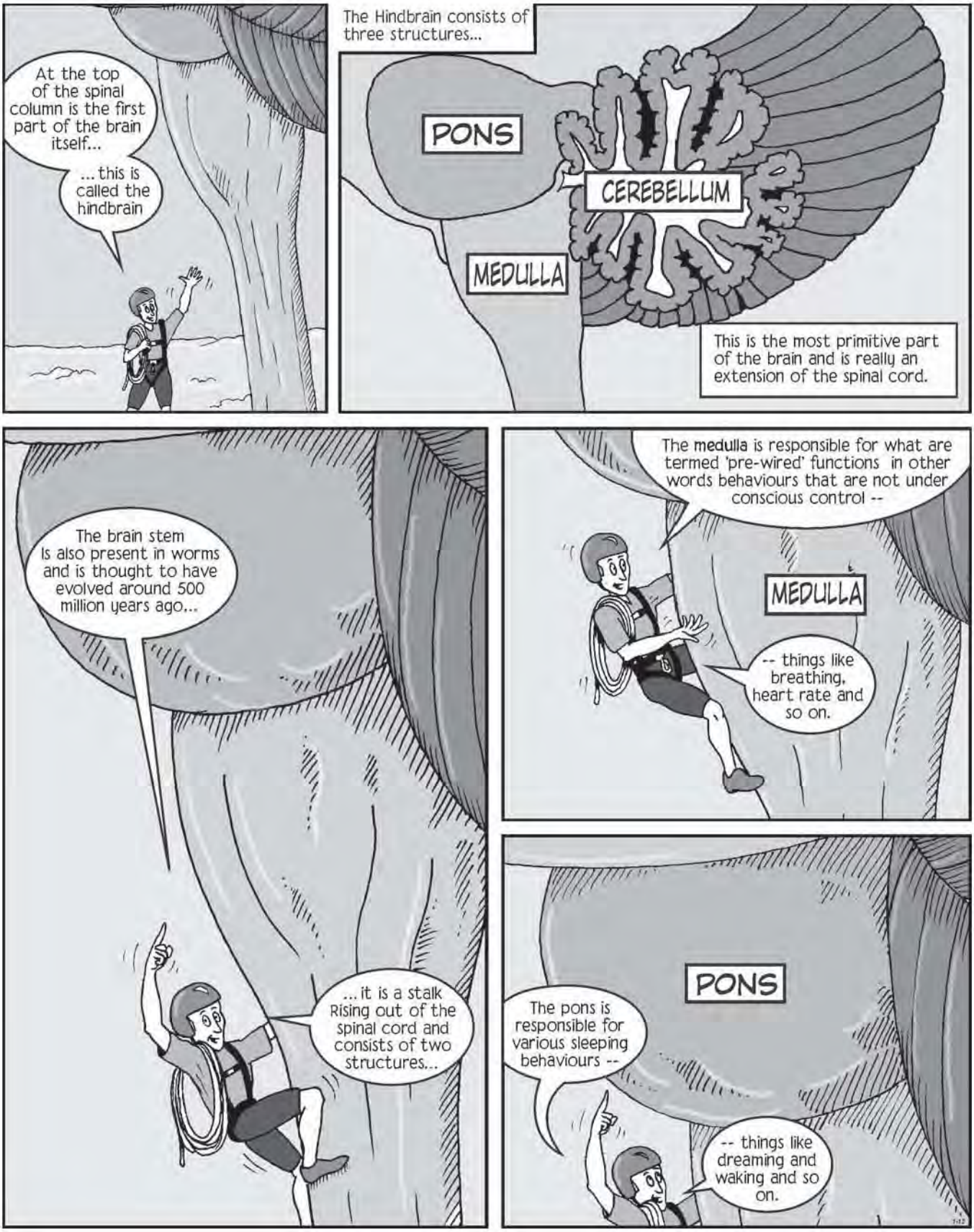

MOTOR NERVES
TO MUSCLES 3200 nm
NEXT SERVICES 20 nm
SENSORY NERVES
TO CNS 2500 nm
TAKE A BREAK!
Peripheral nerves are made up of motor nerves, which carry information to the body from the CNS --
-- and sensory nerves that convey information to the CNS
The PNS is further divided into the somatic nervous system and the autonomic nervous system...
The somatic nervous system is involved in the control of voluntary actions such as running...
The autonomic nervous system, on the other hand, controls non-voluntary functions like the beating of your heart, your blood vessels, glands and internal organs.
BBDMM
BBDMM


The ANS has two functions commonly called the sympathetic and the parasympathetic nervous system.
Imagine that you are running for a bus...
VROOOOM!
Eat FOOD!!!
EWAN MEE
YOR BUS
Hey.... Stop....That's my bus!!
When you're... pant... running... the sympathetic nervous system is responsible... hufff....pant....for getting your... hfff... body...ready for action.....
huff... pant... puff... it raises... auhgh... your heart rate, breathing rate... hufff... and so... on.
When you stop the activity... ahuu... it is the parasympathetic nervous system that slows the physiological processes of the body down again... hufff...
So the sympathetic and parasympathetic nervous systems work in opposing ways depending on what the body needs.
Don't worry love, there's another no. 42 along in 10 minutes!

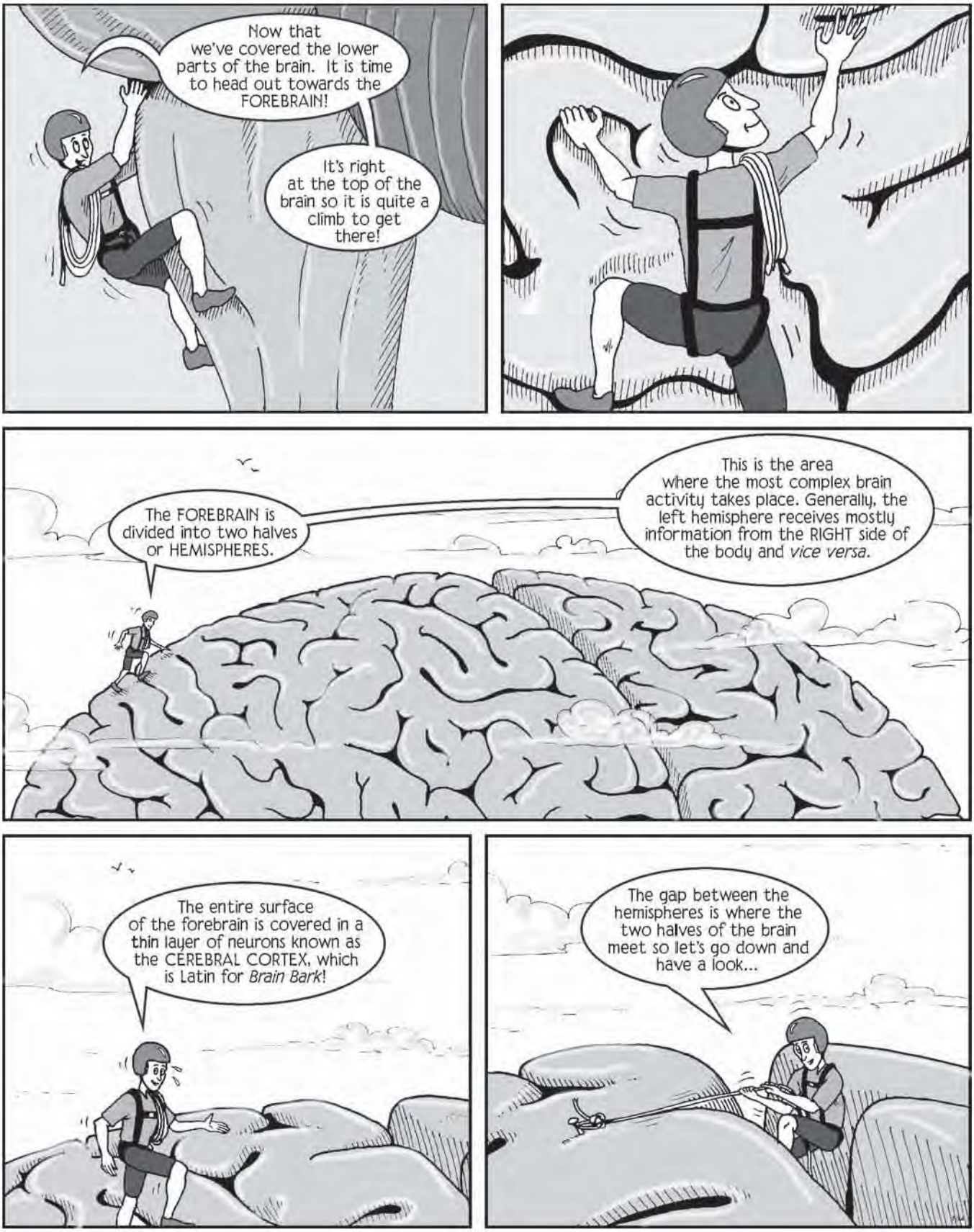

But that is enough about the peripheral nervous system... lets go on to explore the central nervous system.
So the next thing we need to look at is the basic anatomy of the brain...
"Hang on" I hear you saying...
Isn't that a bit too *Biological*?
Hey what's he doing here?!
Beep Beep
Well... yes it is fairly 'Biological' but it is included here for a number of good and proper reasons...
Firstly, many of these specific structures are associated with behaviours and so it is useful if you are familiar with the terms when they are mentioned later on...
Secondly, other books and texts will mention these structures a lot! So it is good if you have at least a working knowledge of what they are...
and thirdly .... It gives me an excuse to go on safari!

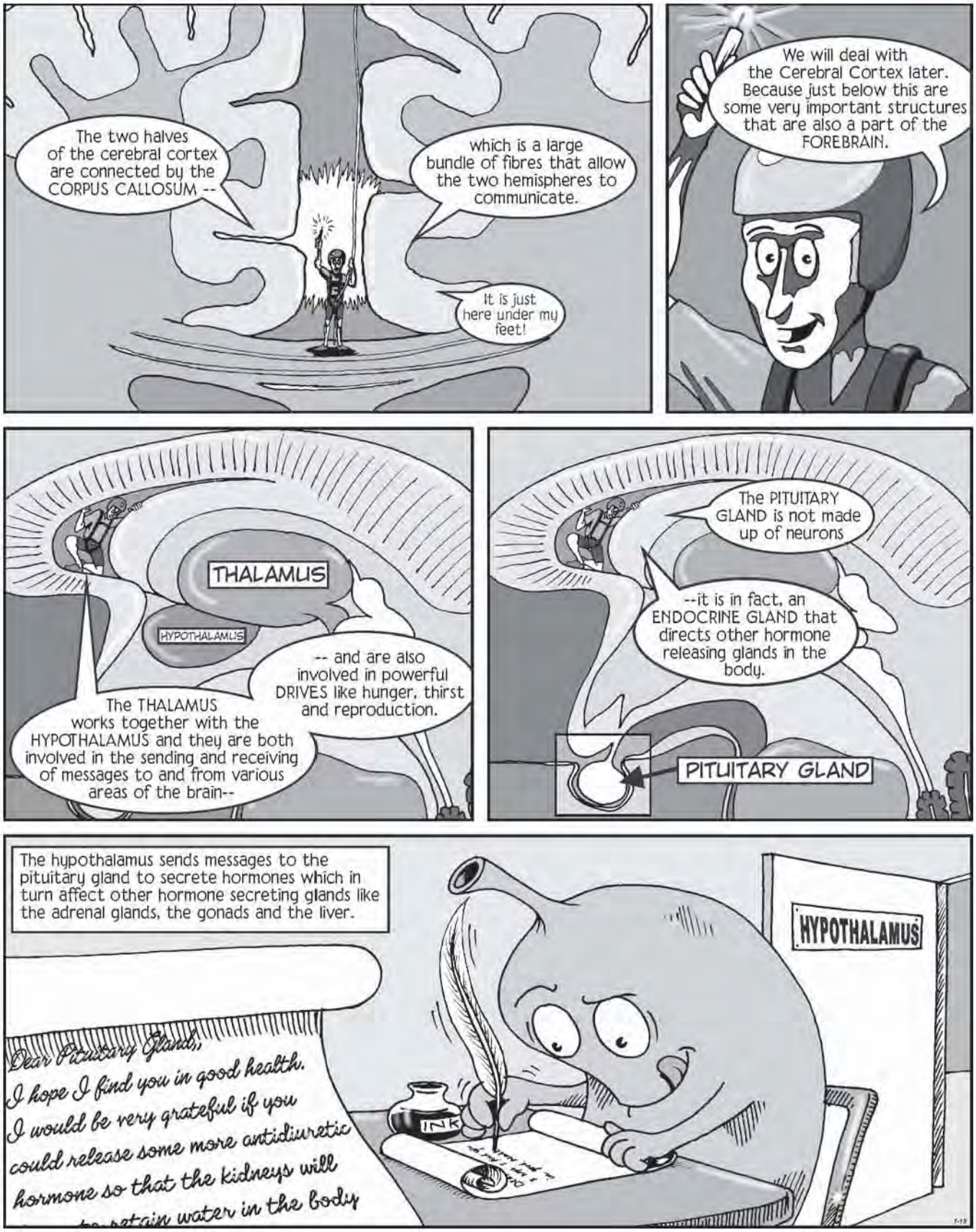

Our 'safari' through the central nervous system begins with the spinal cord!
CNS TOURS
Most of the peripheral nervous system is connected directly to the spinal cord....
The spinal cord acts as a kind of 'bridge' between the body (via the PNS) and the brain.
So you might now simply expect me to head straight for the brain...
But no!
There is *more* to the spinal cord than just a glorified link to the brain.
In fact the spinal cord is responsible for some very important behaviours completely independent of the brain.
SCREECH!

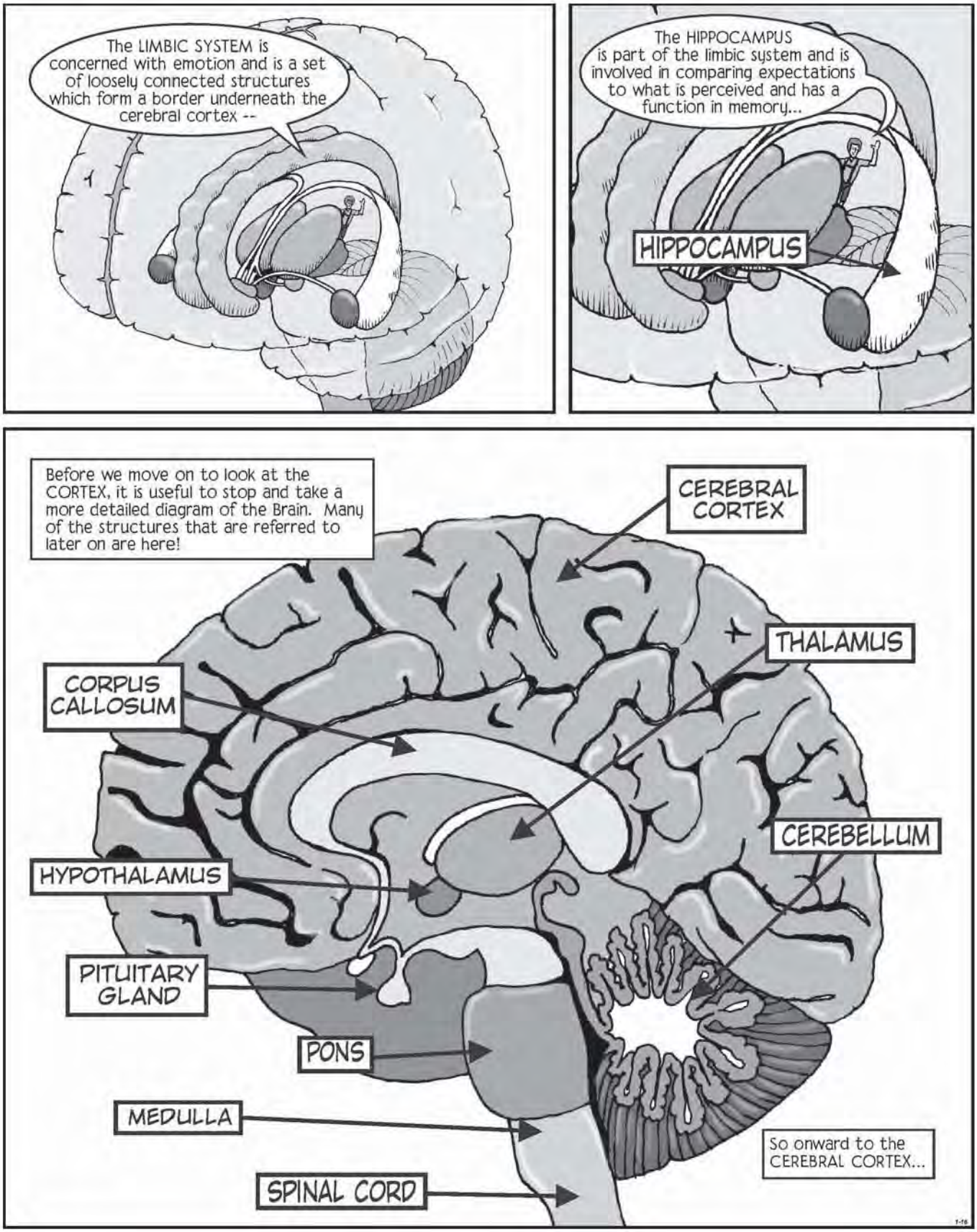

The spinal cord is largely responsible for controlling reflex actions.
A reflex is a "*a simple, automatic movement in response to a sensory stimulus*".
Reflexes need to be quick so the commands go through the spinal column bypassing the brain
MEDICAL HELP
THE DOCTOR IS IN
One of the best known is the knee jerk reflex.
Go ahead doctor...
TAP!
YOW!
SMACK!
THUMP!
So the spinal cord **controls** the knee jerk and other reflexes completely independently from the brain.
Of **course**, the brain is aware of the reflex and can even stop it from happening.
But in this case it was funnier if my brain didn't!
MEDICAL HELP
Speak for yourself mate!
THE DOCTOR IS OUT

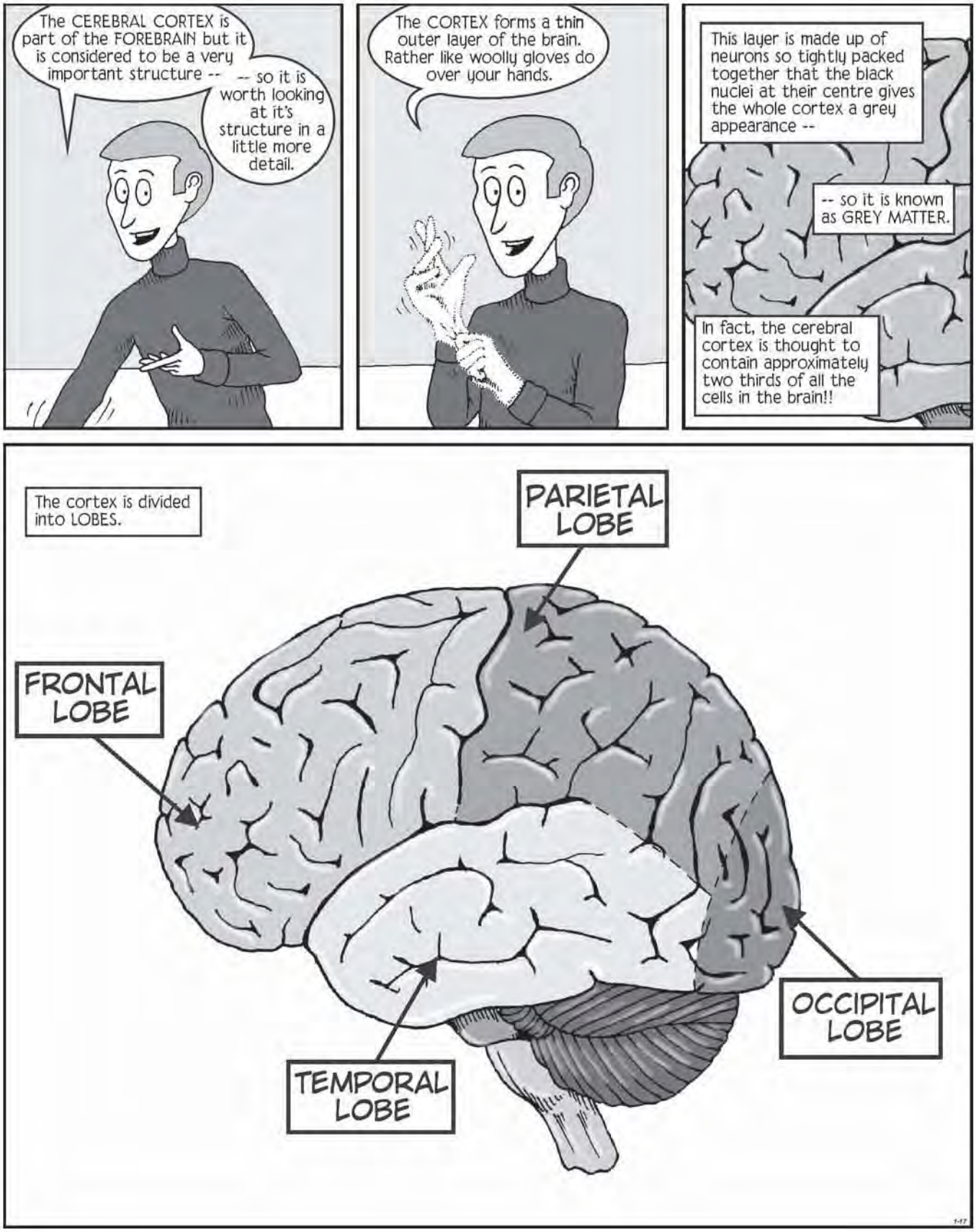

At the top of the spinal column is the first part of the brain itself...
... this is called the hindbrain
The Hindbrain consists of three structures...
PONS
CEREBELLUM
MEDULLA
This is the most primitive part of the brain and is really an extension of the spinal cord.
The brain stem is also present in worms and is thought to have evolved around 500 million years ago...
... it is a stalk Rising out of the spinal cord and consists of two structures...
The medulla is responsible for what are termed 'pre-wired' functions in other words behaviours that are not under conscious control --
MEDULLA
-- things like breathing, heart rate and so on.
PONS
The pons is responsible for various sleeping behaviours --
-- things like dreaming and waking and so on.

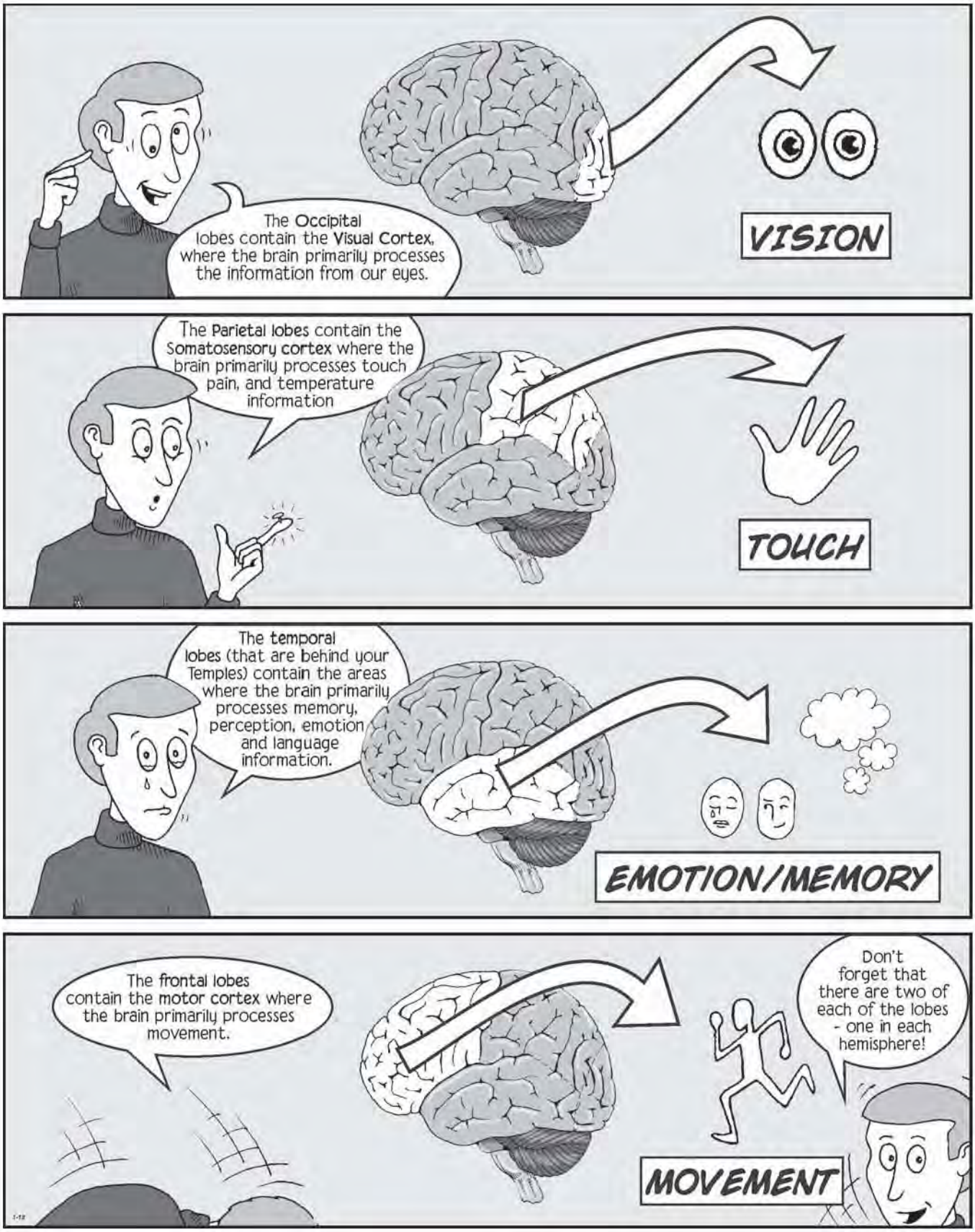

The medulla and the pons also contain the reticular formation that extends from the brain stem into the higher centres of the brain (the midbrain and the forebrain).
The functions of the reticular formation are to act as a sort of arousal or attention centre. It screens out irrelevant information and passes the relevant stuff to the rest of the brain and highlights important material for these higher centres.
The last major part of the hindbrain is the small squiggly part at the back of the brain.
this is known as the cerebellum
which means *lesser or little brain*!
Traditionally the CEREBELLUM has been thought of as having many functions related to movement, especially balance and coordination --
-- for example people with damage to the cerebellum are clumsy and tend to lose their balance.
However--
-- more recent work has suggested that the role of the Cerebellum is much more complex and involves areas like timing and visual attention.
Just above the hindbrain structures is the MIDBRAIN
...in essence, the MIDBRAIN is a collection of neural fibres that connect the lower parts of the brain with the higher centres of the brain.
er... excuse me
Actually, some researchers consider the midbrain areas to be simply a part of the brain stem...and not a separate section of the brain at all!!
Yes!
Yes!
Yes that's right!
Yes... you are absolutely right!
Well I'm not sure really...


Now that we've covered the lower parts of the brain. It is time to head out towards the FOREBRAIN!
It's right at the top of the brain so it is quite a climb to get there!
The FOREBRAIN is divided into two halves or HEMISPHERES.
This is the area where the most complex brain activity takes place. Generally, the left hemisphere receives mostly information from the RIGHT side of the body and *vice versa*.
The entire surface of the forebrain is covered in a thin layer of neurons known as the CEREBRAL CORTEX, which is Latin for *Brain Bark*!
The gap between the hemispheres is where the two halves of the brain meet so let's go down and have a look...


The two halves of the cerebral cortex are connected by the CORPUS CALLOSUM --
which is a large bundle of fibres that allow the two hemispheres to communicate.
It is just here under my feet!
We will deal with the Cerebral Cortex later. Because just below this are some very important structures that are also a part of the FOREBRAIN.
THALAMUS
HYPOTHALAMUS
The THALAMUS works together with the HYPOTHALAMUS and they are both involved in the sending and receiving of messages to and from various areas of the brain--
-- and are also involved in powerful DRIVES like hunger, thirst and reproduction.
The PITUITARY GLAND is not made up of neurons
--it is in fact, an ENDOCRINE GLAND that directs other hormone releasing glands in the body.
PITUITARY GLAND
The hypothalamus sends messages to the pituitary gland to secrete hormones which in turn affect other hormone secreting glands like the adrenal glands, the gonads and the liver.
HYPOTHALAMUS
INK
Dear Pituitary Gland,,
I hope I find you in good health.
I would be very grateful if you could release some more antidiuretic hormone so that the kidneys will
to retain water in the body


The LIMBIC SYSTEM is concerned with emotion and is a set of loosely connected structures which form a border underneath the cerebral cortex --
The HIPPOCAMPUS is part of the limbic system and is involved in comparing expectations to what is perceived and has a function in memory...
HIPPOCAMPUS
Before we move on to look at the CORTEX, it is useful to stop and take a more detailed diagram of the Brain. Many of the structures that are referred to later on are here!
CEREBRAL CORTEX
THALAMUS
CORPUS CALLOSUM
CEREBELLUM
HYPOTHALAMUS
PITUITARY GLAND
PONS
MEDULLA
SPINAL CORD
So onward to the CEREBRAL CORTEX...


The CEREBRAL CORTEX is part of the FOREBRAIN but it is considered to be a very important structure --
-- so it is worth looking at it's structure in a little more detail.
The CORTEX forms a thin outer layer of the brain. Rather like woolly gloves do over your hands.
This layer is made up of neurons so tightly packed together that the black nuclei at their centre gives the whole cortex a grey appearance --
-- so it is known as GREY MATTER.
In fact, the cerebral cortex is thought to contain approximately two thirds of all the cells in the brain!!
The cortex is divided into LOBES.
PARIETAL LOBE
FRONTAL LOBE
OCCIPITAL LOBE
TEMPORAL LOBE


The Occipital lobes contain the Visual Cortex, where the brain primarily processes the information from our eyes.
VISION
The Parietal lobes contain the Somatosensory cortex where the brain primarily processes touch pain, and temperature information
TOUCH
The temporal lobes (that are behind your Temples) contain the areas where the brain primarily processes memory, perception, emotion and language information.
EMOTION/MEMORY
The frontal lobes contain the motor cortex where the brain primarily processes movement.
Don't forget that there are two of each of the lobes - one in each hemisphere!
MOVEMENT


The frontal lobes are particularly special in human beings...
... the area right behind your forehead is known as the PRE-FRONTAL CORTEX and its size varies in different animal Species...
In cats the Pre-Frontal Cortex accounts for 3.5% of the total cortex.
In dogs the Pre-Frontal Cortex accounts for 7% of the total cortex.
In humans the Pre-Frontal Cortex accounts for 29% of the total cortex!
GRRR
This suggests that something uniquely Human is happening in this area of the brain --
-- and this seems to be HIGHER MENTAL PROCESSES.
FSSST
MAOWWW
WOOF
One of the most famous case studies in Biological Psychology highlights the function of the Pre-Frontal Cortex...
NEW ENGLAND USA
SEPTEMBER 1848
Phineas Gage was a 25 year-old railroad worker whose duties included demolition.
Part of his job was to pack explosives into a drilled hole using an iron bar.
Unfortunately, on this occasion, the explosives went off prematurely.
KABOOM
This propelled the iron bar into Gage's cheek, out through the top of his head and 30 feet into the air!


Remarkably, Gage survived and reportedly walked to the cart that took him to hospital.
Even more remarkably, most of Gage's mental faculties remained unchanged and he attempted to return to work a while later.
However, his friends and family said that 'he was no longer Gage'
YOU CAN TAKE YOUR JOB AND SHOVE IT UP YOUR $%* !!!
He'd gone from being mild mannered and kind to being obnoxious and rude.
Although not widely accepted at the time, Dr. John Harlow suggested that Gage's personality change had been a result of damage to the frontal lobes of the brain.
Frontal Lobes
The Brain
$E=mc^2$
Phineas Gage died in May 1860 but his skull was kept at Harvard University.
MODERN COMPUTER IMAGING WORK BY HANNA DAMASIO AND HER COLLEAGUES CONFIRMED THAT HARLOW WAS INDEED CORRECT: THE DAMAGE TO GAGE'S BRAIN WAS CONFINED TO THE PART OF BOTH FRONTAL LOBES THAT DEAL WITH EMOTIONS AND MAKING DECISIONS IN SOCIAL SETTINGS.


The case study of Phineas Gage was the first clue that suggested that the pre-frontal lobes of the brain are responsible for higher mental processes like personality, the ability to plan behaviours, set goals and intention behaviour.
The case of Phineas Gage also highlights the early thinking on what is known as the *Localisation of Function* in the brain.
In other words it shows that some behaviours can be *localised* to specific parts of the brain.
PAUL BROCA was an early pioneer of localisation of function. He discovered that damage to a small area on the left side of the brain had caused his patient to lose the ability to speak.
This area is now known as BROCA's AREA and is an area of the brain important for language production.
By the 1880s most researchers in the area were convinced of the concept of localisation of function in the brain...
...although, it was later discovered that the extent of the localisation is not as great as was once thought!
These and other case studies led early researchers to see the brain as *explainable*.
This meant that studying the brain could provide answers for what had, up to that point, been seen as the domain of a separate concept - 'the *mind*.'
The rest of this book will discuss the various functions of the brain in more detail and at times we will refer to specific parts of the brain to understand how things 'work'.
THIS BOOK!
For now, I just want you to take a few moments to think about the brain in all its glory and the amazing things you are capable of simply because you have one of your very own!
Er... you do own one, don't you?

The brain and the nervous system

▶ PAGE 1

Biological Psychology is the application of biological principles to the study of behaviour. The term *behaviour* refers to a wide variety of phenomena including both internal events like thinking and emotion as well as overt behaviour that can be seen by others.

A great deal of biological psychology is concerned with the physiology of the nervous system and especially the brain. Other terms are used to describe the same area of research: Physiological Psychology, Psychophysiology, Biopsychology, Biological Bases of Behaviour and so on.

▶ PAGE 2

The average adult human brain is actually around the size of a grapefruit or a small melon and is pinkish-grey in colour. It has many folds and creases and looks a little like a large walnut.

▶ PAGE 3

Panel 1

These days we are quite accustomed to seeing the brain as the source of our thoughts and actions. However, this was not always the dominant idea. Ancient cultures, including the Egyptian, Indian and Chinese, considered the *heart* to be the seat of thoughts and emotions. The ancient Greek philosophers Hippocrates (460–370 BC) and Galen (AD 130–200) both suggested the brain as the source of these phenomena whilst Aristotle (384–322 BC) believed the brain was there to cool the passions of the heart!

French philosopher Renée Descartes (1596–1650) was one of the first people to see the human body as a machine and he suggested that a separate entity called the *mind* controlled the brain and nervous system and it worked as a sort of hydraulic pump.

The next major innovation was in the late 1700s by Italian philosopher Luigi Galvani who discovered that he could make a frog's leg twitch by stimulating a nerve with electricity. Later on, Fritsch and Hitzig (1870) succeeded in producing movement in dogs by stimulating their brains with electricity. German physicist Herman von Helmholtz (1821–94) later discovered that the nerves were not simply 'wires' since he calculated that the speed of nerve conduction of around 30 to 40 metres per second was far slower than the flow of electricity or around 3×10^8 metres per second (the speed of light).

All of these pioneering ideas led to the concept that the brain behaved like a biological machine and that this could be investigated using scientific principles.

The adult brain weighs around 1400 grams and has a gelatinous consistency. A living brain is so soft and squidgy that it can be cut with a blunt knife.

Panel 2

There are approximately 100 billion neurons in the human brain (Williams & Herrup, 1988). However, neurons only make up approximately 10 per cent of the cells in the brain. The rest are known as *glial* cells, and these provide a supporting role for the neurons themselves. Neurons are larger than glial cells however and make up about 50 per cent of the volume of the brain.

The idea that the neuron is the unit of brain was suggested by the Spanish Nobel prize winner Santiago Ramón y Cajal from work carried out between 1887 and 1903.

Panel 3

It should be noted that there are a number of different types of neuron. The type depicted here is based on a 'typical' motor neuron. Oh and just for clarity's sake, neurons do not have faces!

The cell body of a neuron contains (amongst other things) the cell nucleus that contains the genetic material and the other structures that keeps the neuron alive.

The dendrites are points on a neuron where information from other neurons are received.

The axon is the long part of a neuron that sends the nerve impulse. Axons can be quite long.

The myelin sheath that surrounds the axon is the insulating material (rather like the plastic around an electric cable). It is made up of a fatty material and has a white appearance.

Panel 5

Technically, it needs to be pointed out that the nerve impulse only happens down the neuron's axon so you could not get a shock in this way. However, it should also be pointed out that this scene is impossible since neurons are microscopic cells and they do not have 'hands'!

Panel 6

Note: The explanation of the nerve impulse involves a complex number of disciplines including concepts from chemistry, physics and biology and many find these difficult to understand. The following explanation is unashamedly simplified although we are aware that some of the terms may appear like a foreign language that need some interpretation.

The concepts described in this explanation baffle even the most qualified individuals and this is not as a result of their intellect, but rather an issue with their background. The following description has been 'run by' an experienced psychologist with a Ph.D. who finds this area taxing and is assured that it is basic enough! Obviously, some may find the material overly simplistic and to these readers we recommend further reading.

Please note that this explanation concerns **only** the electrical nerve impulse *within* a single neuron. It does **not** deal with the conduction of impulses across different neurons.

The Nerve Impulse

The fluid inside body cells has certain chemicals that are electrically charged (this means that these chemicals are moving between positive and negative charges in currents, similar to that in the electricity in your home). These are called ions and the important ones for nerve conduction are sodium and potassium (that both have one positive charge), calcium (that has two positive charges), certain proteins called organic anions (that have a negative charge) and chloride (that has one negative charge). The positive ions are attracted to the negative ions and *vice versa*. Hence these chemicals tend to move towards each other. The fluid inside the cell is separated from fluid outside the cell by a cell membrane that allows some chemicals through and not others (it is known as a *semi-permeable* membrane). Therefore, not all the ions can move freely to where they are attracted.

At rest, a nerve cell (neuron) has a negative charge inside the cell and a positive charge outside the cell. This is because the membrane allows positive potassium ions (K^+) easily through to the inside while negative chloride ions (Cl^-) and positive sodium ions (Na^+) have more difficulty. Additionally, there are the organic anions (A^-) inside the cells. Furthermore, there is a pump mechanism that moves sodium ions out of the cell relative to the number of potassium ions within the cell (for those who want to know, the cell 'allows' one sodium ion for each potassium ion, the pump moves sodium out of the cell until this ratio is achieved). When the correct ratio of sodium and potassium ions is achieved, and thus the inside of the cell has a correct negative charge, the neuron is in a sate of balance that is called a *Resting Potential*. If you were to anthropomorphosise the neuron at this point it would be a 'very happy bunny'!

During this state of balance, this resting potential, it is possible to measure the amount of electricity being generated using a gadget called a voltmeter. When this is carried out, it is found that the resting potential of an average neuron is around −70 millivolts (mv) which means that the inside of the cell is 70mv less than the outside. Bear in mind that this is a very small amount of electricity. A portable CD player usually requires two 1.5 volt batteries to operate (a total of three volts, approximately forty times that in a neuron in a state of resting potential!).

Keeping the neuron in this 'happy' state requires a lot of work. In fact, the resting potential uses up approximately 40 per cent of the neuron's energy. However, despite using up such a great deal of the neuron's energy, this is worthwhile because the resting potential is absolutely essential in powering the actual nerve impulse.

When a neuron at rest is stimulated (by another neuron for example), this causes its voltage to move towards 0 mv (in other words from −70mv to 0mv). Before it reaches this, however, at around −55mv, this causes the nerve cell to *fire* or *spike*. This is called an *Action Potential.* When the neuron fires (i.e. it has an action potential) it sends an electrical impulse down its axon (which results in behaviour, for example the movement of a muscle). This value of −55mv is called a firing *threshold*. If it is not achieved, the neuron will not send a message.

The Action Potential

The action potential occurs because of an exchange of ions across the neuron's semi-permeable membrane. When the firing threshold level is reached (i.e. −55mv), this causes the cell to open sodium channels ('holes' in the semi-permeable membrane) that allow sodium ions to rush into the cell. This causes the rapid change from a negative charge of −55mv to a positive one of around +30mv. As soon as this happens, the cell tries to recover its resting potential state by closing sodium channels and opening potassium channels. The now negative charge on the outside of the cell then causes the movement of potassium ions to the outside of the cell, bringing the resting potential back. The final stage is for the sodium-potassium pump to help in the removal of sodium ions back to the outside of the cell.

What does this achieve?

All of this happens on one tiny spot on the cell membrane. However, the nerve impulse (which remember is carrying the message) has to travel the whole length of the neuron's axon. The 'struggle' that causes the action potential and then a return to the resting potential on one part of the membrane creates an imbalance in the spot on the membrane next to it. So, this next spot on the membrane opens the sodium channels and begins the action potential at that point. This creates a chain reaction of action potentials down the length of the neuron's axon. In the chapter, dominoes were used to highlight this wave of electricity that moves down the neuron's axon, but you could also see it as a worm's movement along the ground. One segment of its body pushes another segment which pushes on another and so on.

▶ **PAGE 4**

Panel 6

The 'all or nothing' effect is concerned with the *threshold* of −55mv. If this is not reached then the action potential does not occur. It often takes many neurons stimulating a single neuron to achieve this threshold.

▶ **PAGE 5**

Panels 2 to 6

Note: The following explanation deals with the transmission of the nerve impulse from one neuron to another.

Chemical transmission at the synapse

It was Ramón y Cajal who showed that neurons were not physically touching each other. These physical gaps are the reason von Helmholtz (see page 3 panel 1 above) did not find the nervous system transmitting electrical messages at the speed of light. The synaptic gaps slow down the message considerably.

Up until the 1920s, it was thought that the synapse was bridged by an electrical impulse. It took German physiologist Otto Loewi (1953) to show that synapses are bridged by sending chemicals across the gap. Chemical transmission is how most synapses are bridged.

We now know that there are a few neurons that **do** bridge the gap electrically by sending ions across the synapse. In other words, the action potential is physically carried from one neuron to the next. These are called *Electric Synapses* and are present in situations where very fast nerve transmission is very important. Electric synapses are rare and tend to occur in invertebrate animals. The crayfish, for example, has electric synapses that control the movement of its tail allowing it to escape from predators very quickly.

How is the chemical transmission achieved?

The end of a neuron's axon ends in what is described as the synaptic knob or the *pre-synaptic terminal* (it is called **pre**-synaptic because it is found *before* the synaptic gap). The pre-synaptic terminal is a sort of swelling at the end of the axon. Inside this swelling, there are small pockets that contain certain chemicals. These are called synaptic *vesicles* ('vesicle' means 'little bladder' – which is a very good description of what they are actually like!). The chemicals inside these 'little bladders' are called *neurotransmitters* and these are the chemicals that cross the synaptic gap and pass on the message from one neuron to another.

When the action potential arrives at the pre-synaptic terminal this causes calcium channels to open in the terminal's membrane (remember, these are 'holes' in the semi-permeable membrane that temporarily open to allow certain ions – in this case calcium – into the inside of the neuron). The calcium causes the vesicles near the terminal's membrane to fuse with it and thus open into the synapse. This allows the neurotransmitters inside them to spill out of the neuron into the synaptic gap.

What happens next?

The neurotransmitter chemicals flow or *diffuse* across the gap and reach the receiving part of the next neuron. This is called the **post**-synaptic neuron since it is *after* the synaptic gap. The neurotransmitter molecules fit into small 'holes' called *receptor sites* on the surface of the receiving neuron. This is rather like a key fitting into a lock so that the neurotransmitter molecules are the 'keys' shaped in such a way that they fit into the corresponding 'locks' of the receptor sites. This chemical 'jump' across a synapse takes only about 2 milliseconds (a millisecond is a millionth of a second!).

The locking of a neurotransmitter at a receptor site can have one of three effects:

1) *Ionotropic* effects
   Some neurotransmitters cause the postsynaptic neuron to open ion channels to allow a particular ion into the neuron.
   The neurotransmitter *glutamate,* for example, causes the sodium channels to open and therefore initiate an action potential. This is called an *excitatory* effect. The neurotransmitter *Gama-amniobutyric acid (*GABA) opens chloride gates that make the inside of the neuron more negative and hence stops an action potential taking place. This is called an *inhibitory* effect. However, both glutamate and GABA have ionotropic effects. Ionotropic effects are fast and are used when a quick response is needed such as in moving muscles.
2) *Metabotropic* effects
   Metabotropic effects are much slower and longer lasting than ionotropic effects. Metabotropic effects take place by creating a sequence of chemical (metabolic) reactions. When a neurotransmitter locks onto a metabotropic receptor this causes chemical changes inside the neuron that can have a variety of effects from opening an ion channel to switching on the effect of a chromosome. *Dopamine* is an example of a neurotransmitter that can have metabotropic effects.
3) *Modulatory* effects
   Some neurotransmitters act as what are known as *Neuromodulators*. Neuromodulators diffuse to more than one neuron. They then lock to all the correct receptor sites of the neurons close by. This is rather like a radio signal reaching all the radios that are tuned in to it.
   The effects of neuromodulators on neurons are quite small. They alter (modulate) the effect of the neurotransmitters (Millhorn *et al*., 1989). Some neuromodulators, for example, can prolong or limit the effect of a neurotransmitter whilst others can limit the release of neurotransmitters. Endorphins (see Chapter 3) are examples of neuromodulators that have the effect of reducing pain responses.

Stopping the effect of neurotransmitters

Once the neurotransmitter has locked onto the receptor site and the desired message has been transmitted, it makes sense for the synapse to return back to its normal state in readiness for the next message. In order for this to be achieved, the neurotransmitter must be removed from the receptor sites and any that may be left in the synaptic gap. There are four ways in which the effect of the neurotransmitter is removed. Firstly, some of the neurotransmitter left in the gap simply flows away from the synaptic gap and is unable to bind

to the receptors. Secondly, there are other chemicals called *enzymes* that are released into the gap that break up the neurotransmitters. Thirdly, there are chemicals that bind to the neurotransmitter molecules and absorb them back into the presynaptic neuron to be used again later (this is called the *reuptake* mechanism). And fourthly, there are supporting cells (glial cells) that absorb the neurotransmitter into themselves for re-use by the neuron.

Why have chemical transmission at synapses?

The chemical transmission at a synapse significantly slows down the nerve impulse. This must therefore have an important purpose. Given that neurons are either activated or not (in other words there is only a 'yes' or a 'no' response from each neuron) the chemicals at the synapse provide a great deal of complexity to the communication system. By using many different types of neurotransmitter molecules and different types of receptor sites on many different synapses, the nervous system can create a complicated code rather like having many letters in an alphabet to create a language.

▶ PAGE 10

Panel 3

The spinal cord is a segmented structure. Each segment has on each side a sensory nerve that receives information from the body and a motor nerve that sends information to the body. A cross-section through the spinal cord shows a darker 'H' in the centre that represents the tightly packed neurons. The white matter around this 'H' is made up mainly of axons with white myelin sheaths around them. The core of the spinal cord is a fluid-filled channel called the *central canal*.

Each segment of the spinal cord receives information from the brain and sends information to the brain. If the spinal cord is cut at a specific segment, then the brain loses all sensation from that segment and any segments below that one. Similarly, all motor control is also lost to the part of the body connected to this segment and the ones beneath it.

Protection for the central nervous system

The spinal cord and the brain are protected by fluid filled membranes called the *meninges.* The space between the meninges and the brain and spinal cord is filled with a liquid called *cerebrospinal fluid.* The brain and spinal cord therefore float in a bag of fluid. This protects the delicate structures from damage by impact. Sometimes the meninges become infected and this results in the condition known as *meningitis.*

In addition, the brain is protected from harmful chemicals by the *blood-brain barrier*. This is a set of tightly packed tiny blood vessels that prevent chemicals with large molecules from entering the brain. Anything that dissolves in fat can pass freely into the brain but there are many chemicals that only dissolve in water that need to be actively transported across the blood-brain barrier in order to reach the brain.

▶ PAGE 11

Panel 1

This definition of a reflex is from Garrett (2003, p.74).

▶ PAGE 12

Panel 1

The hindbrain is also sometimes referred to as the *rhombencephalon*.

▶ PAGE 13

Panel 5

The midbrain is also known as the *mesencephalon* and consists of two structures the *tectum* and the *tegmentum*. The important structures in the tectum are the *superior colliculi* that are involved in vision and the *inferior colliculi* that are concerned with audition. One important structure in the tegmentum is the *substantia nigra* that contains neurons that produce dopamine. It is the death of neurons in this area that is believed to be largely responsible for the movement disorder called Parkinson's disease (see Chapter 5).

The midbrain is relatively small in humans. In birds, reptiles, amphibians and fish the midbrain structures are much more noticeable.

▶ PAGE 14

Panel 1

The forebrain is sometimes referred to as the *prosencephalon*.

▶ PAGE 15

Panel 1

The *Corpus Callosum* is the largest of the *cerebral commisures*. These are dense fibres that carry information between the two cerebral hemispheres. The two hemispheres carry out slightly different tasks and therefore need to communicate with each other. Additionally, information from the senses is often directed to one specific hemisphere. For example, visual information on the left is sent to the right hemisphere while visual information on the right is sent to the left hemisphere. This information needs to be shared with the other hemisphere through the corpus callosum and the other comissures. Sometimes, surgeons cut the corpus callosum so that those suffering from epilepsy can confine their fits to just one cerebral hemisphere (this is only done in the most serious of cases). These individuals therefore have no internal communication between the cerebral hemispheres and have been studied to determine the different functions of the two hemispheres. These studies have shown that, for example, the left hemisphere is generally more involved in language than the right hemisphere and that the right hemisphere is more involved in spatial tasks and face recognition (e.g. Gazzaniga, 1967; McKeever, Seitz, Krutsch, & Van Eys, 1995; Nebes, 1974).

▶ PAGE 16

Panel 3

What this diagram omits are the brain *ventricles*. The ventricles are hollow spaces inside the brain filled with cerebrospinal fluid. There are four ventricles in the brain connected to the central canal of the spinal cord (see notes for page 10 panel 3 above) and to the meninges.

▶ PAGE 19 (Panels 4 to 7) and PAGE 20 (Panels 1 to 5)

The case of Phineas Gage

At the time of his accident, Phineas Gage was 25 and working as a railroad construction foreman near the town of Cavendish in Vermont, USA. One of his duties was to drill holes in rock in order to place explosive charges in them so that the rocks could be removed. The drilled hole had to be filled with the explosive powder and then sand placed on top. This mixture was then packed together using a three and a half foot (approximately 1.07 m) long iron rod. On the fatal day (13 September 1848 at about 4.30pm) Phineas Gage either forgot to add the sand or was packing the explosives before adding the sand when he was distracted and this caused a spark that ignited the explosive powder. The force of the explosion caused the iron rod to fly up through Gage's skull and into the air. The 3 cm diameter metal rod passed through his skull and caused damage to his brain (Damasio, 1994; Macmillan, 1986).

After his recovery from the accident, Gage failed to be re-employed by the railroad company and in 1850 he spent about a year as a side-show attraction at P.T. Barnum's New York museum displaying his injury (and the tamping iron!) to paying customers. He later spent some time in Chile as a coach driver before returning to his home in San Francisco in 1859 to become a farm worker before his death in 1860.

The type of deficit suffered by Phineas Gage is actually open to some debate. It is often stated that Gage suffered personality changes. This is usually referenced back to John Harlow (1868), the doctor who attended to Gage's injuries. He stated that:

> 'His contractors, who regarded him as the most efficient and capable foreman in their employ previous to his injury, considered the change in his mind so marked that they could not give him his place again. He is fitful, irreverent, indulging at times in the grossest profanity (which was not previously his custom), manifesting but little deference for his fellows, impatient of restraint or advice when it conflicts with his desires, at times pertinaciously obstinate, yet capricious and vacillating, devising many plans of future operation, which are no sooner arranged than they are abandoned in turn for others appearing more feasible. In this regard his mind was radically changed, so decidedly that his friends and acquaintances said he was 'no longer Gage' ". (Harlow, 1868 in Neylan, 1999, p. 280).

However, MacMillan (2000) has cast some doubt over the scientific accuracy of the personality changes ostensibly suffered by Gage. He points to the fact that at the time of the injury Harlow (writing in 1848, see Neylan, 1999) did not mention much regarding psychological changes. Similarly, Bigelow (1850), who was professor of medicine at Harvard University and examined Gage after his recovery, stated that he had recovered both in body and in mind and made no note of psychological changes. It was Harlow's quote above in 1868 (eight years after Gage's death) that seems to have been embellished by later writers and coupled with Gage's sensationalistic appearances at Barnum's museum. We therefore cannot be certain about the details of the case of Phineas Gage since, as MacMillan has pointed out; very few of the deficits attributed to Gage have been based on original sources written at the time. Many deficits have been suggested based on modern ideas of frontal lobe damage instead.

Panel 6

Damasio *et al.* (1994) conducted neural imaging studies on the surviving skull of Phineas Gage and reconstructed the path of the iron bar through his skull. They suggested that the accident damaged the part of both frontal lobes involved in making decisions in personal and social matters. More recent work by Ratiu *et al.* (2004) using computed tomography scanning (CAT scan) has cast some doubt over Damasio *et al.*'s

conclusion, however. Ratui *et al.*'s work suggests that the damage to Gage's brain was much less extensive than was hitherto believed as they suggest that only Gage's left frontal lobe was actually damaged.

Despite these controversies, the case of Phineas Gage really does represent a milestone in terms of 'tipping the balance' in favour of the idea of localisation of function in the brain.

Phineas Gage – another perspective

Most introductory psychology textbooks mention the case of Phineas Gage (approximately 60 per cent according to MacMillan, 2000). However, in reading some of these, the case is generally described in a sanitised way. In these sources' excitement to place the case of Phineas Gage in its deserved important historical context, Phineas Gage the human being is lost. Little mention is made of Gage's horrific injuries except to say that the hot iron rod 'cauterised' the wound on its way through Gage's skull. Much is made of Gage walking to the cart that took him to the town, implying that despite the injury, Gage came away relatively unscathed, at least physically.

However, the reality seems very different if you read the original account from Harlow (1848; see Neylan, 1999). Phineas Gage suffered a great deal. Harlow attended to Gage about one and a half hours after the accident at approximately 6pm. This is his description of how he found his patient:

> 'He seemed perfectly conscious, but was getting exhausted from the haemorrhage, which was very profuse both externally and internally, the blood finding its way into the stomach, which rejected it as often as every 15 or 20 minutes. Pulse 60, and regular. His person, and the bed on which he was laid, were literally one gore of blood' (Harlow, 1848 in Neylan, 1999, p. 281).

He also mentions that Gage's hands and forearms were deeply burned up to the elbow; something which is rarely mentioned in other sources.

Harlow also describes the subsequent recovery of his patient from the 13 September until the 18 November 1848. During this time he mentions further haemorrhaging, vomiting, severe swelling of the face, 'foetid' discharge from the scalp intermingled with particles of brain, the formation of an abscess on one of the facial muscles and fungal growth from within the wound. Ever the clinician, Harlow does not attempt to describe the level of pain that Gage must have suffered.

This level of 'gory detail' is included here to remind us that despite the controversies over Gage's deficits there is a man at the heart of this case. Whether you believe that Gage suffered personality changes or if the phrase 'he was no longer Gage' was indeed spoken by his friends or not, should not detract from the horrendous physical suffering that Phineas Gage endured as a result of what happened on 13 September 1848.

▶ PAGE 21

Panel 3

Broca's Area

Pierre Paul Broca (1824–80) was a French surgeon who in 1861 treated a patient for gangrene. This patient had earlier lost his ability to say anything except the word 'tan' and to utter an oath. Five days later the patient died and Broca was able to examine his brain. He found damage to a specific part of the

left frontal lobe. On the basis of this and other cases, Broca concluded that this part of the brain must be intact for speech production despite intact vocal apparatus and normal language comprehension.

Broca is often cited as one of the first to show localisation of function in the brain although others also wrote about this area at around the same time (Finger & Roe, 1996). The area of the frontal lobe is now known as *Broca's Area* and is generally seen as a language production area of the brain. Any serious impairment of language production is known as *Broca's Aphasia* regardless of whether the damage is to Broca's Area or not. Aphasia is the term used for any language impairment.

We now know that speaking involves a large part of the cortex, especially in the left hemisphere and is certainly not confined to Broca's Area (Wallesch, Henriksen, Korhuber & Paulson, 1985).

Wernicke's Area

In 1874 Carl Wernicke discovered that damage to an area in the left temporal lobe of the cortex produced an aphasia that was very different to that discovered by Broca. This is known as *Wernicke's Aphasia* or *Fluent Aphasia*. In this case the language impairment is characterised by an inability to remember names of objects and general impairment of language comprehension. The person is often seen as speaking very fluently despite difficulties in finding certain words (known as *Anomia*). Additionally, these individuals find it very difficult to understand both spoken and written speech. Like with Broca's Aphasia, these types of deficit are called Wernicke's Aphasia regardless of whether the damage is in the same part of the brain.