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# The anatomy and physiology of pain

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# 1

The human body is able to experience a range of sensations, from the pleasant, soothing texture of velvet to the extremely unpleasant sensation of pain. For many years it has been acknowledged that the process of pain does not consist solely of a physiological set of sensations: it is a combination of physiological sensations that requires complex physiological, psychological and behavioural interactions to enable the human to interpret and subsequently respond (Wall and Melzack, 1999).

The aims of this chapter are:

- To discuss the concepts underpinning the physiology of pain.
- To explore the gate control theory of pain.
- To highlight the changes that occur within the nervous system as a result of ageing that may impact upon the pain experience as the person ages.
- To demonstrate how an understanding of these factors may influence practice.

Generally, everyone perceives the pain experience to be unpleasant and to be avoided at all costs. Only a few reported individuals are known to have never experienced pain, and this is now a recognized syndrome (hereditary sensory and autonomic neuropathy type 4). Pain is wholly subjective, and the perceived intensity and discomfort for any one known controlled stimulus varies from person to person. The actual perception of pain requires a complicated integration of sensory nerves, motor nerve pathways and chemicals that serve to enhance the

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pain. All of these can be influenced by the genetic make-up of the individual, their past experiences and emotional contributors. This means that the sensation of pain is greater than the sum of its parts.

Although pain pathways, physiology and local hormone production play only a small part in the overall sensation of pain, the efficacy of analgesics and other pharmacological therapies is based on the modulation of the nervous system and its role in the sensation of pain. It is essential for any health-care professional to have good understanding of the anatomy and physiology of pain in order to make informed decisions regarding the most appropriate therapy.

### Learning point

Revise some of the following terminology:

- peripheral and central nervous system
- spinal cord
- sensory cortex
- simple spinal reflex
- synapse, neurotransmitters and receptors
- sensory afferents
- motor and autonomic efferent
- autonomic nervous system.

You may wish to read the paper by Davis (1993) and the book by Melzack and Wall (1996) to support your learning.

## Pain and sensation

The definition of pain as

an unpleasant sensory or emotional experience associated with actual or potential tissue damage or described in terms of such damage (Merskey and Bogduk 1992, p. 210)

suggests that pain may be the result of actual or potential tissue damage and that it prevents the individual from bodily harm, or from the injury, disease or harm becoming worse. It is a dramatic mixture of emotional and physiological reactions (Mountcastle, 1980; Merskey, 1986; Wall and Melzack, 1999).

There are certain things that we now know to occur within the nervous system when a disease or injury arises, but there are still some things we don't know about. Research into these mechanisms is ongoing. In this section we discuss the basic physiological concepts and then we consider the issues that are particularly relevant for the care of older people.

Imagine putting your hand on to a hot stove. This will initiate a series of responses within the nervous system that will eventually be perceived as pain. The whole process begins at the site of the injury, or 'where your hand is touching the hot stove'.

Physiological pain arises from chemical, thermal or mechanical stimulus of the small-diameter sensory afferent fibres found in the tissue. These actually detect injury and are known as **nociceptors**, which derives from the Latin word meaning injury. It is important to be aware of this as it helps us to understand the concepts of **neuropathic** and **nociceptive** pain.

### Learning point

Can you identify the differences between neuropathic and nociceptive pain?

Think about the types of neuropathic pain that you see in your area.

There are two types of nociceptors: **A $\delta$**  (A delta) and **C fibres** (Cesare and McNaughton, 1997). These are different from other sensory afferent nerve fibres in that the noxious stimulus has to be of a sufficient intensity and duration to bring about tissue damage. In other words, these fibres have a high stimulation threshold. Tactile fibres such as **A $\beta$**  (A beta) fibres have a low threshold and follow slightly different spinal tracts to the brain. They also transfer information related to pressure and texture, but not pain. To illustrate this, imagine what it would be like if pain was initiated by a soft touch – such inappropriate misfiring would make life impossible. Equally, if the nociceptors' threshold is set too high then tissue damage would result before avoidance action could be taken. Hence the stimulation intensity is set to prevent unnecessary tissue damage or discomfort.

Modulation and regulation of all of the incoming information is carried out by nerves that descend from the brain to the spinal cord and contribute to the analysis of the sensations at this level. These **descending tracts** are responsible

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for the regulation of sensations that actually reach the brain and allow the individual to divert their attention elsewhere. This is the rudimentary basis of the **gate control theory** which we will return to later.

We can consider two categories of pain:

- **Physiological pain:** The pain response to high-intensity stimuli is transient if the tissue damage is prevented by a simple spinal flexion reflex arc (Willer, 1979). Consider striking a match and touching the flame with your fingers – you would drop the match instantly before damage could occur. The speed with which this reflex occurs prevents deep tissue damage and allows only a brief moment of discomfort. This is caused by a simple spinal reflex mediated by the high-intensity thermal stimulation of small sensory nerve endings in your fingers.
- **Pathological pain:** This results from sensitization of the nerves in the periphery and the spinal cord. Peripheral nerve endings are made more sensitive to noxious stimuli through tissue damage, action of local hormones such as prostaglandins, histamine, serotonin and bradykinin, and also by direct nerve damage – this is called **peripheral sensitization**.

When the **neurons** involved with the transmission of pain along the spinal cord to the sensory cortex in the parietal lobe of the brain are sensitized by a barrage of impulses from the site of tissue damage, this is referred to as **central sensitization**. As a result the nerve fibres of the central nervous system begin to respond to non-noxious stimuli such as gentle touch as if they were pain impulses. Peripheral and central sensitization of the neural pathway can produce pain without a clear external stimulus. So, for example, gentle stroking can become pain – this is termed **allodynia**. Furthermore, an exaggerated response to low-threshold noxious stimuli can occur (**hyperalgesia**) (Woolf, 1989, 1991; Rang, Dale and Ritter, 1999). In acute pain, this is quite an important concept as potentiation of pain will encourage rest and thus prevent further tissue damage (Woolf, 1991). However, should this continue after the acute phase (i.e. in **chronic pain**) it will serve no useful purpose and become a clinical problem in its own right. This will be discussed later, in Chapter 6.

### Summary

- Pain is an unpleasant sensation which warns of impending tissue damage.
- Pain develops as a result of chemical, thermal or mechanical stimuli.
- Activation of A- $\delta$  and C fibres occurs; these are known as nociceptors and they detect injury, not pain.

- A- $\beta$  fibres transmit pressure, not pain.
- The physiological response to high-intensity is transient if the tissue damage is prevented by a simple spinal flexion reflex arc.
- Sensitization of nerves in the periphery and spinal cord is known as pathological pain.
- Tissue damage or local hormone action can make peripheral nerve endings more sensitive this is known as peripheral sensitization.
- When the central nervous system responds to A $\beta$  fibres as if they were conducting pain impulses, central sensitization occurs.

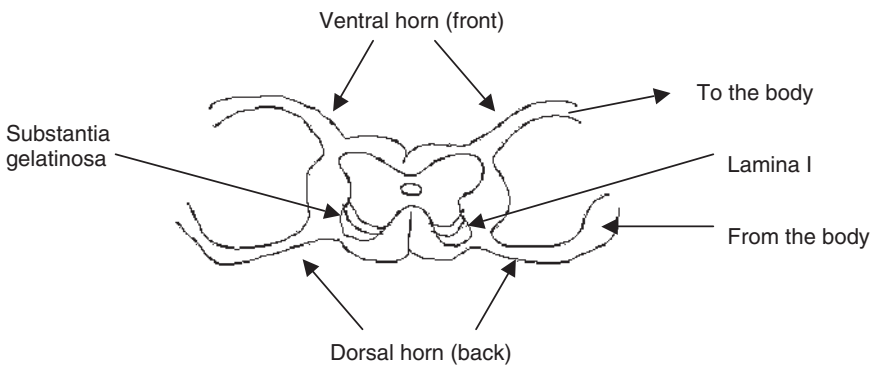
## Neural pain pathways

When the sensory neurons synapse with the motor neurons and transmission neurons in the dorsal horn of the spinal cord, pain is detected. As seen in Figure 1.1, the nerve fibres within the dorsal horn (rear) carry information back to the spinal cord and brain. The ventral horn (front) carries autonomic efferents and motor nerves away from the spinal cord and brain back to the body.

The terminal nerve endings of the sensory nociceptors release the neurotransmitters **substance P** and **glutamate**. These chemicals in turn bind to the surface of the dendrites of the transmission neurons, propagating the signal forward either to a motor nerve or up to the brain via the spinal cord.

### C fibres

These are fine **unmyelinated fibres**, 0.23–1.5  $\mu\text{m}$  in diameter, which respond to chemical, thermal or mechanical stimuli. Because they have more than one mode



**Figure 1.1** Cross-section of the spinal cord

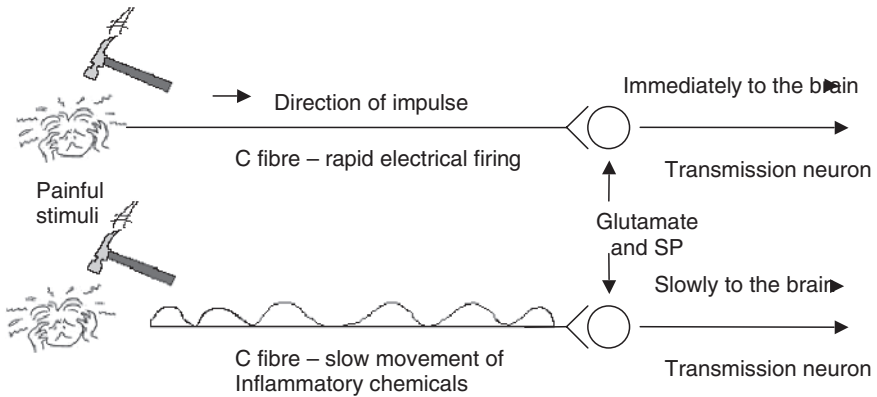


Figure 1.2 The rapid and slow effects of the C fibre in the dorsal horn

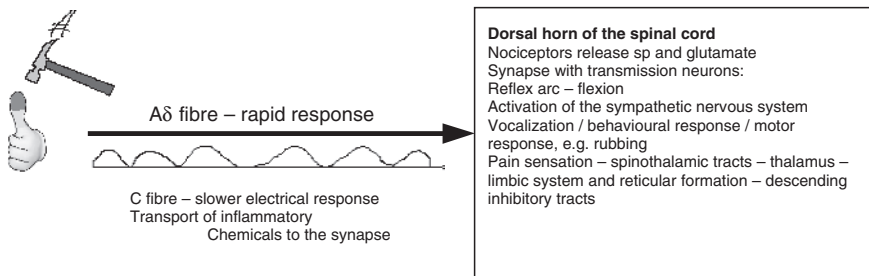


Figure 1.3 Physiological pain responses

of stimulation they are also known as **polymodal fibres**. It is believed that C-fibre activity is associated with dull, diffuse pain and once initiated can continue for up to 80 hours. The conduction velocity (speed with which the pain message travels) is  $<2.5$  m/s (Figure 1.2).

Along with sending electrical messages to the spinal cord by the movement of potassium and sodium ions into and out of the axon, C fibres are also responsible for the absorption of inflammatory chemicals such as **bradykinin** along the length of the axon to be released within the spinal cord at the synapse with the transmission neuron (Wall and Melzack, 1999). This process provides a dull, diffuse and profound ache that often follows relatively minor injuries such as a sprained ankle, resulting in the whole leg aching for days after the injury.

### Aδ fibres

These are medium-sized (1–5  $\mu$ m diameter), myelinated, fast-acting neurons with a rapid conduction velocity ( $>2$  m/s) (Figure 1.3). It is believed that these neurons

are responsible for the sensation of well-localized, sharp and intense pain (Rang, Dale and Ritter, 1999). The function of  $A\delta$  fibres is similar to that of C fibres, but they react more rapidly and are sensitive to thermal and mechanical stimuli only.

Think of how it feels when you prick yourself with a needle. Initially you feel the exact point of the pain. But a few minutes later, the pain became more widespread and it becomes difficult to locate the exact site of the injury. What we are feeling is the initial  $A\delta$  pain followed by the C fibre pain. Both  $A\delta$  and C fibres are found in large numbers in the skin, but C fibres predominate in the internal organs, muscles and viscera. However, both types of fibres set up a reaction that moves along the axons to the synapse with a number of transmission neurons within the dorsal horn of the spinal cord and along tracts to the brain.

At the site of injury we have a group of nerve fibres that will begin the process.

### Learning point

Can you complete the table below?

Nerve fibre	Myelin sheath	Type of sensation
$A\delta$	?	?
C	?	Dull aching
$A\beta$	Yes	?

## Dorsal roots

The next stage of the pain processing pathway is the spinal cord. Both  $A\delta$  and C fibres enter the spinal cord at the **dorsal horn**. The dorsal roots are made of layers, known as **laminae**, into which the sensory nerve fibres enter. Synapses are made with the transmission neurons that direct the impulse across the spinal cord to motor neurons and can elicit a reflex flexion arc from the offending source of the noxious stimuli. Alternatively, the impulse may ascend the spinal cord to the brain.

Within the spinal cord is a neuron-rich area which is known as the **substantia gelatinosa**. All sensory fibres have to cross this area before forming a synapse with spinal neurons in the various laminae. It is believed that the  $A\delta$  and C fibres connect within the layers I–III.

The substantia gelatinosa contains short nerve fibres (**interneurons**) which regulate the transmission of impulses from nociceptors and other sensory nerve

fibres. Interneurons are rich in neurotransmitters which resemble opiates and are therefore very important in the modulation of nociception through an opiate receptor mechanism. These chemicals, known as endorphins, are very similar to morphine.

### Learning point

Endorphins are opiates produced naturally by the body.

It may be useful at this point to consider the marathon runner. After running for about 10–15 miles – around half way in the 26-mile race – the runner experiences excruciating pain, but if they continue to run, they pass through what is known as the **pain barrier**. At this point the pain begins to subside, because the person has started to produce the endogenous opioids which act like morphine and control the pain. Although we are all capable of producing these chemicals, most of us tend to ask for drugs instead of relying on our internal mechanisms. It has been suggested that it can take a few days to get these opioids out of the system.

Furthermore, the interneurons inhibit the response of the transmission neurons to stimulation from an A $\delta$  fibre when impulses generated by an A $\beta$  fibre are also arriving at the synapse with the transmission neurons. At a time of high input from nociceptors the large A $\beta$  fibres which respond to pressure and mechanical stimuli are filtered through the substantia gelatinosa in the spinal cord. These A $\beta$  fibres are much larger than the other two types and therefore can transmit their sensations much quicker, thus blocking the pain messages being carried by the other two. The A $\beta$  fibres are activated by rubbing the area.

### Learning point

Massage is a pain-relieving technique that works on this principle.

## Spinal cord to brain

As the impulse travels through the various centres within the brain, the sensation of pain is perceived. Furthermore, the sympathetic nervous system is aroused and consequently the individual experiences an increased blood pressure, heart rate



and increased blood flow. The organism experiences a heightened sense of arousal or wakefulness. The sympathetic nervous system, which responds to **E situations** (emergency, excitement and embarrassment), dominates the regulation of the body and prepares it for 'fight or flight'. Adrenalin is secreted by the adrenal medulla and noradrenaline is secreted into the injured tissue.

Simultaneously, the individual experiences higher brain centre responses which include vocalization and behavioural responses – oh \*\*\*\*! Arousal and emotional effects occur as a result of increased involvement in other areas of the brain.

The problem is that there is no one centre in the brain that is responsible for pain processing. Therefore almost all of the brain becomes involved, which is why pain is often difficult to treat.

### Learning point

Read around the role of the brain and identify the functions of some of the major structures listed below:

Structure	Function
Cortices	
Thalamus	
Limbic system	
Reticular activating system	

The A $\delta$  and C fibres exist throughout the body, on the periphery, viscera and internal organs – with one exception. There is one place where we do not feel pain. Think of Hannibal Lecter! That's right; the internal substance of the brain has no pain receptors. But before you test this theory by putting a hatchet in someone's head, remember that the scalp and the outer coverings of the brain do contain the receptors that enable us to feel pain.

## Descending tracts and substantia gelatinosa

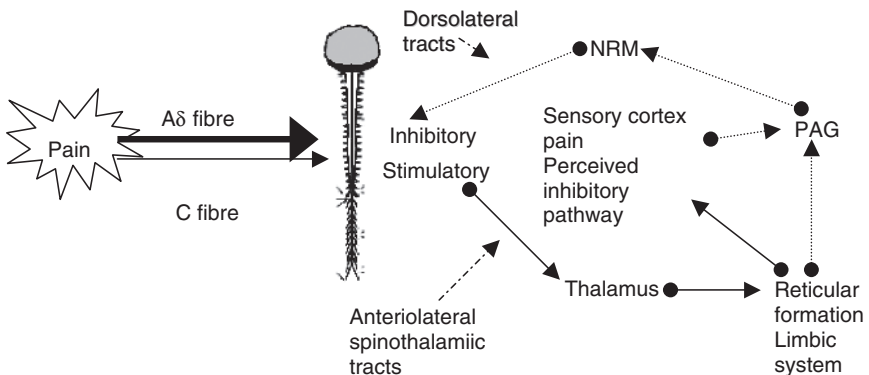
Descending tracts are efferent fibres which leave the reticular formation within the brain, travel along the spinal cord and synapse with the transmission and interneurons within the substantia gelatinosa. The descending tracts function in order to modulate incoming messages from peripheral nerves. Thus, they act as

a filter and partial inhibitor of the messages ascending the spinal cord from the nociceptors. This limits transmission of the impulses from the sensory A $\delta$  and C fibres along the transmission fibres.

The descending nerve fibres arise at the **periaqueductal grey** within the reticular formation and flow into the **medulla** (brainstem). From the specific area called the **nucleus raphe magnus** in the medulla, impulses then pass down the dorsolateral tracts in the spinal cord to connect with the transmission neurons and interneurons in the substantia gelatinosa of the spinal cord (Fields and Basbaum, 1994). The major neurotransmitter from the descending tracts is **serotonin**, which stimulates interneurons within the substantia gelatinosa to release peptides, noradrenaline and endogenous opioids such as **endorphins**, **enkephalins** and **dynorphines**. All of the areas associated with pain processing are rich in opiate receptors, which could explain the actions of our analgesic preparations. The pain pathway is a cycle of events within the central nervous system, interpreting and modulating the impulses that are generated in the peripheral nerves of the body.

### Learning point

Revise the autonomic, peripheral and central nervous systems.



**Figure 1.4** The ascending pathways and descending inhibitory pathways. SG, substantia gelatinosa; PAG, periaqueductal grey; NRM, nucleus raphe magnus. Dashed lines show pain modulation pathways (descending); solid lines show pain sensation pathways (ascending)

## Summary

- Nociceptors synapse with motor and transmission neurons in the dorsal horn of the spinal cord.
- Transmission fibres within dorsal horn carry information to the brain and spinal cord.
- The dorsal horn is made up of layers (laminae) which contain many transmission neurons.
- A $\delta$  and C fibres synapse with transmission neurons in the first three layers of the spinal cord (laminae I–III).
- Nociceptors release the neurotransmitter (substance P or glutamate).
- The substantia gelatinosa is a neuron-rich area through which all nociceptors pass before forming a synapse with spinal neurons in the various laminae.
- The interneurons within the substantia gelatinosa regulate the transmission of impulses from the nociceptors and other sensory nerve fibres to the various laminae. These are rich in neurotransmitters that resemble opiates and are important in the modulation of nociception through an opiate receptor mechanism.
- C fibres are unmyelinated and respond to chemical, thermal and mechanical stimuli; they precipitate dull, diffuse pain.
- A $\delta$  fibres are myelinated and therefore produce sharp, shooting stabbing pain that is well localized.

## The pain gate

The pain gate was proposed by Melzack and Wall back in 1965. It is the most widely accepted theory of pain, and explains a great many of the pain phenomena. But the theory is by no means complete, and work continues to refine it. The **plasticity** of the nervous system – its ability to become desensitized and sensitized – also adds an extra dimension (Figure 1.5).

A $\delta$  and C fibres synapse within the dorsal horn of the spinal cord with both transmission fibres and interneurons. This is true for the A $\beta$  fibres also. Tissue damage produces high-intensity messages which move along the nociceptors to the transmission neuron. The nociceptors also form synapses with small excitatory interneurons. Concurrent stimulation of the excitatory interneuron as well as the transmission neuron will augment the nociceptors' output and hence potentiate the pain experience. Rubbing the affected area will also stimulate the

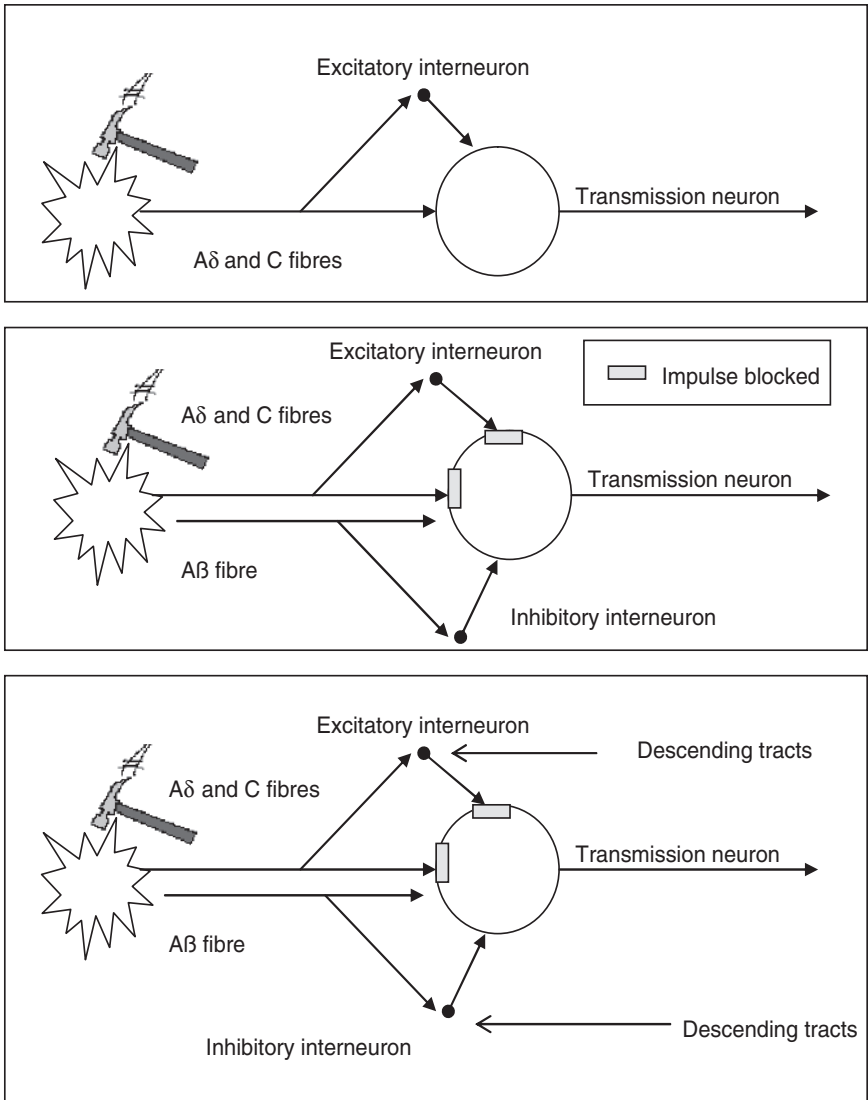


Figure 1.5 A simple pain pathway

low-threshold  $A\beta$  fibres which in turn synapse with the **inhibitory interneuron** which decreases sensitivity of the transmission neuron to the nociceptors' outputs. The descending pathways of the periaqueductal grey will attempt to modulate the activity of the interneuron by stimulating the inhibitory interneurons to release endogenous opioids, thus blocking the nociceptive pathway.

## Learning point

How does this fit with some of the strategies that we may use for pain control, such as heat or TENS?

## Summary

- The gate control theory is the most widely accepted theory of pain sensation and inhibition. It has yet to be disproved, but other theories do exist.
- Plasticity is the ability of the nervous system to become sensitized or desensitized.
- $A\delta$  and C nociceptors and  $A\beta$  sensory fibres synapse with the same interneurons.
- Painful stimuli excite the nociceptors, which in turn excite the transmission neuron and excitatory interneurons
- Rubbing the affected area excites  $A\beta$  sensory fibres. These synapse with an inhibitory interneuron, which decrease sensitivity of the transmission neuron to the nociceptors outputs.
- The descending tracts modulate the pain sensation by stimulating the inhibitory interneurons to release endogenous opiates, blocking the nociceptive pathway.

For a recent survey of the gate control theory, see the article by Dickenson (2002).

## Chronic pain

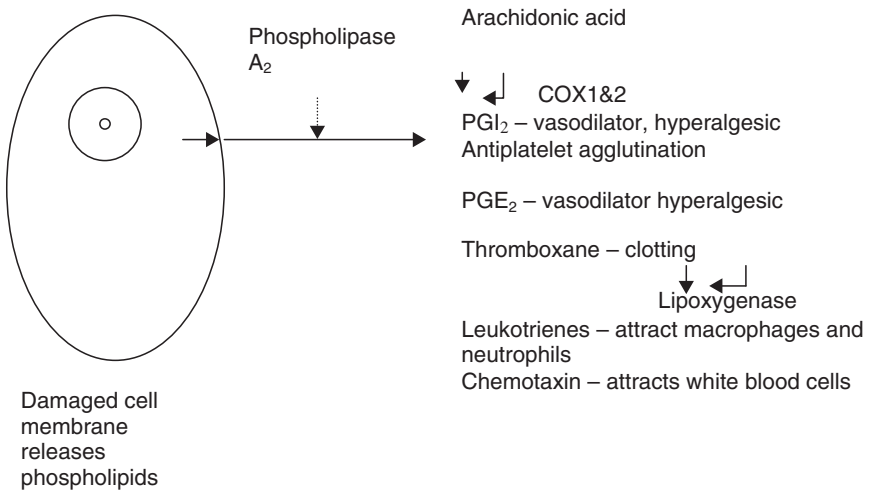
All of this physiology appears fairly straightforward. But as we can see in Chapter 6, the whole system becomes much more complicated when we discuss chronic pain or pain that appears to have no identifiable physical cause. We are aware that pain sensations travel through many parts of the brain in their journey towards interpretation. This journey could in some way explain the ramifications and issues that arise from chronic pain as the individual becomes increasingly depressed and disillusioned, along with the involvement of the sympathetic nervous system and the inability to find sleep or comfort that is often described by chronic pain sufferers. However, this is not the complete picture. The plasticity of the central nervous system and the change in sensitivity of peripheral nerves

and central pathways also adds support to the signs and symptoms reported by patients with chronic pain. Therefore when dealing with chronic pain, we need to look beyond the usual approaches with analgesics and adopt more creative approaches that would not be considered for the management of acute pain.

## Peripheral sensitization

When tissues are damaged they cause the release of phospholipids from the plasma cell membrane. Enzymes such as phospholipase  $A_2$  exist in the cell membrane and catalyse the breakdown of phospholipids into arachidonic acid which can be further modified by other enzymes (cyclooxygenases 1 and 2 and lipogenase) to produce a family of chemicals known as **eicosanoids**. These are often referred to as **local hormones** and include substances such as prostaglandins, leukotrienes, lipoxins, chemotaxins and thromboxanes. These eicosanoids act on the C fibres and thus increase their sensitivity, which increases the unpleasant experience of pain. The C fibres can also absorb many of these chemicals and pass them along to the dorsal horn via the axons (Figure 1.6).

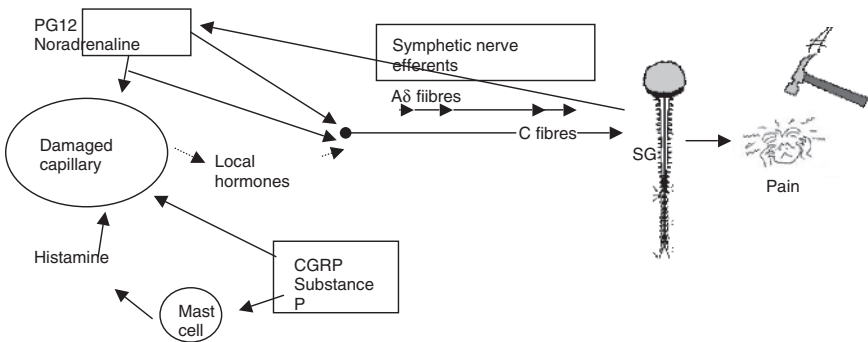
The nociceptors also release inflammatory mediators into the surrounding tissue, which adds impact to the action of local hormones. Hence when stimulated by high-intensity heat, chemical or mechanical activity, the nociceptors also release **calcitonin gene related peptide (CGRP)** and substance P (Brain and Williams, 1985). These chemicals act directly upon mast cells (connective tissue



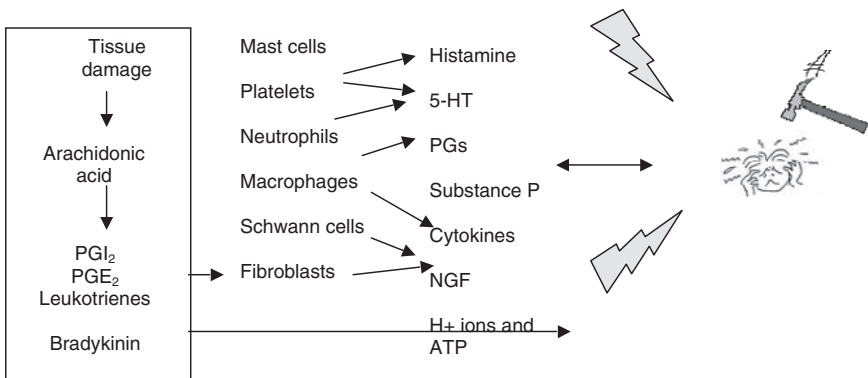
**Figure 1.6** Cell damage and arachidonic acid

cells containing granules of histamine), causing the release of histamine and serotonin. Substance P and CGRP also act upon the local blood vessels, causing vasodilatation and increased capillary permeability (Figure 1.7).

The sympathetic nerves in the damaged area add to the cocktail of inflammatory chemicals by releasing prostaglandin I<sub>2</sub> and monoamines such as noradrenaline. The inflammatory cocktail alters the threshold of peripheral nociceptors to increase sensitivity of the neuron. This is done in several ways; by coupling to the receptors on the neuron and opening up ion channels, thus lowering the action potential threshold, or indirectly by increasing the number of ion channels within the nociceptive membrane (Figure 1.8).



**Figure 1.7** Peripheral sensitization by the sympathetic efferents, local hormones and local release of nociceptors derived from substance P



**Figure 1.8** The inflammatory chemical cocktail and increased peripheral pain perception

The inflammatory mediation of peripheral sensitization mechanisms explains the anti-inflammatory and analgesic effects of non-steroidal anti-inflammatory drugs (NSAIDs) and glucocorticoids (steroids) which inhibit the lipoxygenase and cyclooxygenase enzymes and so reduce the quantity of prostaglandins and leukotrienes at the site of tissue damage.

## Summary

- Cell-membrane phospholipids are converted to arachidonic acid by phospholipase A<sub>2</sub>.
- Arachidonic acid is the chemical precursor of many inflammatory mediators (eicosanoids) of which leukotrienes and prostaglandins are examples.
- Arachidonic acid is broken down into prostaglandins by the cyclooxygenase enzymes.
- Prostaglandins are a family of chemicals that are also involved in pain potentiation, platelet aggregation and vascular resistance. They are absorbed by C fibres and transmitted along the axon lengths to be released within the dorsal horn.
- Substance P and CGRP are released by nociceptors into the surrounding damaged tissues which potentiates the action of the inflammatory mediators by increasing mast cell degranulation, histamine release and vasodilatation.
- The inflammatory cocktail is augmented by the release of prostaglandin I<sub>2</sub> and noradrenaline which is caused by the sympathetic nerve endings in the damaged tissue.
- NSAIDs and glucocorticoids (steroid drugs) inhibit the lipoxygenase and cyclooxygenase enzymes and so reduce the quantity of prostaglandins and leukotrienes at the site of tissue damage (Table 1.1).

## Central sensitization and A $\beta$ fibre mediated pain

The transmission neurons within the spinal cord are subject to a barrage of impulses from peripheral nerves and from centrally descending tracts. Substance P and glutamate are released by A $\delta$  and C fibres which act upon specific receptors on the dendrites of the transmission neurons. The receptor-neurotransmitter complex brings about depolarization of the transmission fibre which generates an



**Table 1.1** Sources and actions of local hormones

<b>Substance</b>	<b>Released from</b>	<b>Action</b>
Hydrogen ions	Intracellular fluid	Excites nociceptors
ATP	Intracellular fluid	Acts on ATP receptors on macrophages inducing macrophage degranulation
Histamine	Macrophages, mast cells, basophils, histaminocytes (in the stomach) and histaminergic neurons	Vasodilatation, increases plasma permeability. Increases gastric acid secretion. Smooth muscle contraction, with the exception of smooth muscle
Bradykinin	Cleaved from kininogens found in the tissue fluid	Activates sensory neurones, fibroblasts, endothelial cell secretion, liberates arachidonic acid
Prostaglandins	Derived from arachidonic acid by cyclooxygenase	Sensitizes nociceptors to the actions of bradykinin, 5-HT and mechanical and thermal stimuli
5-HT (serotonin)	Platelets and mast cells	Both sensitizes and activates nociceptors
Substance P	Sensory nerve endings	Induces mast cell degeneration
Leukotrienes	Derived from arachidonic acid by lipoxygenase	Sensitize nociceptors possibly by stimulating macrophages and basophils to release eicosanoids. Encourages release of substance P
Cytokines/ interleukins	Neutrophils, phagocytes	Possibly by increasing the amount of prostaglandin formation

action potential along the length of the axon. The activity of the transmission neuron is prolonged for some time after the sensory fibre has ceased firing as a result of substance P and glutamate. Also, the transmission neuron has a special excitatory amino acid receptor which is connected to an ion channel. When the transmission neuron is resting or stimulated by low-threshold sensory nerve fibres ( $A\beta$  fibres) this receptor ion channel remains inoperative, blocked by magnesium ions. The receptor ion channel can only be activated when all the ions are displaced (Mayer, Westbrook and Guthrie, 1984). This displacement of magnesium ions occurs when the transmission neuron is stimulated by an excitatory neurotransmitter such as substance P, glutamate or CGRP. The ion channel now opens and allows the influx of sodium and calcium into the neuron, producing prolonged depolarization. If another impulse arrives at the synapse before the transmission neuron slowly returns to a resting stage, another more rapid action potential is generated by the transmission neuron. Hence the transmission neuron becomes increasingly sensitive to any excitatory impulse arriving at the spinal cord. Thus the usually non-nociceptive impulses generated by  $A\beta$  fibres now produce altered sensory perception in terms of nociception.

## Summary

- Modulation of the transmission neuron causes central sensitization.
- The transmission neuron has a number of receptors that are sensitive to substance P and glutamate. There is also a novel amino acid receptor which is connected to an ion channel.
- Magnesium ions block the receptor-ion channel and remains inoperative during tactile stimulation or when nociceptors are inactive.
- When magnesium is displaced, the receptor-ion channel is activated.
- When substance P, glutamate or CGRP (neurotransmitters) stimulate the transmission neuron, the displacement of magnesium can occur.
- Stimulation of the transmission neuron by neurotransmitters released from nociceptors opens the ion channel to allow influx of sodium and calcium into the neuron, producing a prolonged depolarization.
- The transmission neuron now becomes increasingly sensitive to any excitatory impulses arriving at the spinal cord, which are therefore interpreted as painful.
- The usually tactile stimulus generated by  $A\beta$  fibres now produces an altered sensory experience, felt as pain.

Now, of course we also know that pain is **individual**. Two patients who have the same condition may respond very differently: patient A may be up and about while patient B is lying in bed, not even moving.

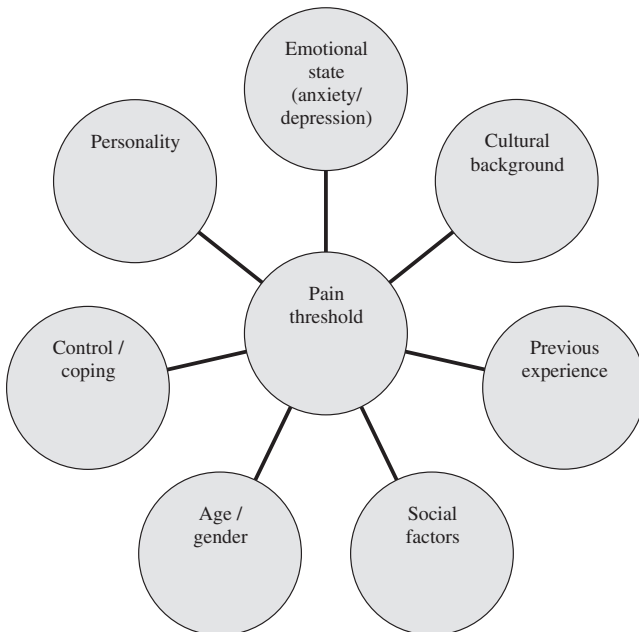
### Learning point

Can you think of the factors that make the pain an individual experience?

- Age: do older or younger people cope better?
- Gender: do men or women cope better?

All of these factors are known to contribute to the individuality of the pain or the **pain threshold**, which is the amount of pain an individual is prepared to tolerate. It can vary from hour to hour or from day to day, and is influenced by a combination of the factors identified above. We also know that as health professionals we can influence this threshold in how we react to patients when they are in pain.

Here are some of the things that you probably identified (Figure 1.9):



**Figure 1.9** Factors influencing the pain threshold

## Learning point

Consider the following statements – are they true or false?

**True / False**

The attitudes of health-care staff have no effect on the patient's pain

Giving information makes the pain worse

Listening to patients' fears, previous experience and coping strategies can improve pain

Staff are in the best position to know what the patient is experiencing

All pain procedures have the same amount/level of pain

Self-medication/patient controlled analgesia make the pain worse

Apart from the factors that are internal to the individual, we are also influenced by those around us. For example, as carers it is important to consider how we respond to those in pain. What is the **attitude** of staff towards those in pain? Consider the place where you work and your colleagues. How do you respond to someone in pain – are you empathetic? Or do you sometimes avoid the person, maybe because you do not know what to do or say?

Many years ago Jack Hayward published *Information: A Prescription Against Pain* (1975). This work was continued by Jennifer Boore in 1978 – *Information, A Prescription for Recovery*. Both studies demonstrated that giving people information gave **control** and subsequently they were able to **cope** better. It has taken us many years to adopt these principles, but we are getting better at giving information. Think about your own clinical area – how is information given to patients/clients? Can you think of any ways that this could be improved? Is the information appropriate for older people with visual or hearing difficulties?

It is often difficult to find time to **listen** to patients. Sometimes it is easier to reach for the pharmacological approaches, and yet allowing the person to express their fears and worries is sometimes all that is needed to make them feel better. Furthermore, people tend to employ their own ways of **coping** and this may be something totally different from what you would expect, for example heat/cold or even acupuncture. Giving the person time to express their preferences may be very enlightening.

It is often said that you should be a patient to know what it is like, and this is true of pain. We cannot take a photograph of someone's pain and so we have to believe what they are telling us. As Wittgenstein (1967, p. 102) stated.

I can only know that I am in pain – I have to accept what someone is telling me as I cannot see their pain.

How many times have you heard someone say ‘Oh – they have only had their appendix removed, they shouldn’t be in that much pain’ or ‘They should be on oral analgesics by now’. These are examples of **preconceived expectations** that are often held by nursing and medical staff, which ultimately lead to poor pain control.

Finally, there is an issue about **control** that is associated with type of analgesia. Whenever patients come into hospital, we take their medications from them and expect them to ask when they need it – of course this just makes the pain worse. Self medication systems and patient-controlled analgesia can help prevent this.

So, to summarize, all of the factors highlighted above can influence the whole pain experience. Consider a patient in your care – how many of these factors are involved in their pain experience? Both the physiological/biochemical mechanisms and the factors influencing pain form what is known as the **puzzle of pain**.

Now we have looked at the mechanisms of pain, it is useful to be able to define pain.

Pain is what the experiencing person says it is and occurs when he/she says it does. (McCaffery, 1979 p. 95)

Do you see any problems with this definition? It has been suggested that it is a little simplistic. In addition, it requires that the individual has a command of language that we can understand. But what about those who are learning disabled, or cognitively impaired – or babies. They cannot tell us they have pain, and so traditionally they have received little attention within the literature. A more appropriate definition is that proposed by the International Association for the Study of Pain (IASP) on page 2.

## Older people

Now we have discussed the physiological concepts underpinning pain processing. How does this relate to the older adult? Consider the following scenario of an elderly patient visiting her GP:

Patient	Doctor, I have pain in my leg.
GP	Oh it’s to be expected at your age
Patient	But my other leg doesn’t hurt and it’s the same age!

This takes us back to the familiar misconceptions that are held by many people who assume that pain is to be expected as part of ageing. However, in terms of

physiological changes, there is little evidence to support the fact that anything happens to the pain pathways as we get older. Although occasionally older patients may be admitted with silent myocardial infarctions or abdominal catastrophes, there is no evidence to suggest that pain pathways deteriorate with age at all. However, a number of key factors have been noted:

- Older adults tend to have reduced sensitivity to noxious stimuli, but this does not mean that when pain is present they experience it less intensely. When older adults report pain, they are likely to be afflicted with greater levels of underlying pathology than their younger counterparts who report the same level of pain (Gagliese and Melzack, 1997; Weiner and Herr, 2002).
- Older adults tend to under-report pain, because of misinterpretation of physical sensations (e.g., 'hurt' rather than 'pain'), difficulty using standard pain assessment scales, particularly in those with cognitive impairment, and false beliefs about pain and its management.
- Examples of chronic pain conditions common in adults of advanced age include osteoarthritis, postherpetic neuralgia, spinal canal stenosis, cancer, fibromyalgia, post-stroke pain and diabetic peripheral neuropathy. But is also important to remember that more than one clinical diagnosis typically contributes to chronic pain in older adults (Jones and Macfarlane, 2005) and there is also an increased likelihood of atypical pain presentations in this group, due to diminished physiological reserves and interacting comorbidities (Helme and Gibson, 2001).
- It has been suggested that the pain in older adults tends to be constant, of moderate to severe intensity, lasting for several years, multifocal and multifactorial (Brattberg *et al.*, 1996).
- The main problem with pain in older adults relates to impaired quality of life secondary to pain which may be expressed by depression (including increased suicide risk), anxiety, sleep disruption, appetite disturbance and weight loss, cognitive impairment, and limitations in the performance of daily activities. These added burdens are expected to improve with effective pain management (AGS Panel, 2002). Older adults with persistent pain tends to consider their health to be poorer (Reyes-Gibby, Aday and Cleeland, 2002) and use more health-care services than those without pain (Lavsky-Shulan, Wallace and Kohout, 1985).
- The prevalence rate of dementia doubles every five years from age 60 to 24% at age 70 and 30% at 85 years (Helme *et al.*, 2003) and some suggest that the prevalence of pain appears to decrease with increased cognitive impairment. However, in patients with hip fracture, severe pain or inadequate analgesia after surgery can lead to increased confusion, slower recovery, and poorer

ambulation and function (Morrison *et al.*, 2003a,b). Again there is no evidence to support the theory that pain processing changes with dementia and we should therefore treat all people the same regardless of their age or cognitive ability.

### Learning Point

Can you write down a situation from your practice whereby you perceived an older person with/without cognitive impairment to be in pain?

- How was this situation handled by the staff?
- How did you know this person was in pain?
- What have you learned from this?
- How will you deal with the situation differently in the future based upon your new knowledge?

Many of us know when someone is experiencing pain, and we need to be confident in our perception and deal with it. Of course we talk about being the patient's advocate, and recognizing pain and doing something about it is fundamental to the principals of advocacy:

- Recognizing pain-provoking situations.
- Pre-empting pain.
- Fostering a multidisciplinary approach to pain management.

## Conclusion

Our understanding of the physiology of pain remains rudimentary, particularly in scientific terms. The relationship between the pain pathways and the higher centres within the brain remains uncertain. The concept of individuality or threshold also complicates the picture. Think of the disproportionate pain caused by something minor such as a paper cut – and yet we often see major injuries that do not appear to produce much pain. The physiology of pain is important, but only goes some way to explain the phenomenon. Scientists, clinicians and researchers are in agreement that further work needs to be done.

Understanding the physiology of pain is essential if practitioners are going to be in a position to prescribe appropriate analgesics or other forms of treatment.

Pain is a common symptom of disease, and the one problem that drives individuals to seek help.

The population is getting older, and we are likely to see increasing numbers of older adults who are experiencing pain. It is essential therefore, that health-care professionals are aware of the basic principals of physiology and the pain pathways to enable them to understand and deal with the problems. The gate control theory remains our primary theory of pain and stands the test of time, so familiarity with this theory will help us to understand pain and apply appropriate management strategies.

\*All figures taken from Drago (2005) and adapted by Matthew Schofield.

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