

Local Analgesia.

Learning Objectives:

1. To understand the applications of local analgesia in podiatry
2. To understand how these drugs bring about their analgesic affect
3. To be able to correctly calculate a maximum safe dose for an individual patient
4. To know which patients are unsuitable for local analgesia
5. To be able to recognise the signs and symptoms of anaphylaxis and toxicity, and to be able to act appropriately should they occur
6. To understand the legal, ethical and moral implications surrounding consent issues
7. To know the correct procedure for common administration techniques in podiatry.

Different techniques to reduce the sensation of pain have been used since ancient times, but modern application of drugs for this is attributed to Karl Koller, an Austrian ophthalmologist. Working in the nineteenth century it was very difficult to perform surgery, and Koller recognised the potential pain-killing properties of cocaine. Prior to this he had tested solutions such as chloral hydrate and morphine as anaesthetics in the eyes of laboratory animals without success, but with the use of cocaine Koller was able to demonstrate its potential as a local anaesthetic to the medical community in 1884, and was seen as a breakthrough in patient management. Interestingly, the adverse effects of cocaine were not reported until 1885 by Halsted who produced one of the first papers warning of the addictive properties of cocaine. An addict himself, he was successfully treated using morphine, which at the time was used to cure cocaine addiction.

The early success of this approach produced the well-recognised problems associated with addiction and toxicity, and from this, the need arose to find alternatives which produced the desired effect without the associated complications. In 1904 Einhorn synthesised the drug 'procaine', the first ester type local anaesthetic drug. Not until nearly forty years later was the first amide type local anaesthetic synthesised by Lofgren (1943). Since this time there has been a continued introduction of newer local anaesthetic drugs in an attempt to produce the 'ideal' drug. Lignocaine, prilocaine, bupivacaine and mepivacaine remain the most widely used local anaesthetic agents, and in Podiatry have been available for use since the 1970's. Recently, two new preparations were added to the list - levobupivacaine, and ropivacaine.

Previous to the use of drugs a number of other techniques had been employed-

Ischaemia - Esmarch, (a prominent military surgeon) introduced the 'Esmarch Bandage' which was used to maintain haemostasis and analgesia during operations. This type of tourniquet is still used today in a range of surgical procedures to provide haemostasis although more effective methods are employed to provide anaesthesia. Application of a tourniquet proximal to an injection site will prolong the duration of local anaesthetic action by reducing systemic absorption. This combination is used commonly in podiatric surgery although the prime purpose of the tourniquet is to maintain a bloodless operative field. Whilst ischaemia does alter nerve conduction, it is neither a practical nor effective means of achieving clinical anaesthesia.

Compression - Ambroise Pare (1510 - 1590) identified that direct compression on a nerve induced a state of local analgesia which could be used to perform amputations. Over time, several pieces of equipment to maintain compression over nerves in different regions of the body were designed, and much discussion occurred with respect to the mechanism by which compression brought about this analgesia, leading to two schools of

thought at this time. Some believed that it was the compression alone that provided the analgesia, whilst others considered the compression merely produced ischaemia which was known at that time to produce analgesia in the affected limb. Despite much interest, these early techniques proved to be unsatisfactory. The technique was unreliable, and could cause nerve damage.

Use of Cold - Hunter (1793) examined this 'scientifically' on himself by immersing his hand in a wasps nest. He noted the pain from the stings was reduced whilst the hand was immersed in cold spring water. Cold remains a valuable component of physical therapy, although analgesic rather than anaesthetic affects are produced.

Applications of Local Analgesia

It is important to remember that when using the term 'analgesia' it implies that the patient remains conscious throughout the procedure, whereas 'anaesthesia' implies a loss of consciousness, therefore care should be taken as the terms are not inter-changeable. Analgesia need not be confined to use in surgical procedures only, but can also be used in basic pain control, diagnostic procedures, and in musculo-skeletal management.

Diagnostic Peripheral Nerve Blocks

Under normal circumstances, painful sensations are conveyed centrally along the afferent nerve fibres to the spinal cord. They may then pass to the brain in the ascending columns of the spinal cord and thereby we become aware of the presence of a painful stimulus and its location. Pain thought to arise peripherally can originate centrally (in the spinal cord or brain). A good example of this is seen in spinal nerve entrapment - patients may experience symptoms of paraesthesia in the foot as a result of irritation of the L5 nerve root. Local anaesthetic blocks the transmission of both afferent and efferent impulses along the nerve by preventing the generation of an action potential. For example, a patient experiences paraesthesia in

the right second and third digits. If a local analgesic agent is introduced, and this relieves the symptoms, the underlying problem must arise distal to the site of injection. If symptoms still persist despite blockade, the problem must arise proximal to the site of the injection (assuming correct injection technique). In this example, the first injection can be placed in the metatarsal inter-space. If a neuroma is the cause of symptoms, the pain will disappear as the analgesic takes effect. If the patient has a tarsal tunnel syndrome, this approach will fail to resolve the discomfort. If a proximal tibial block at the ankle alleviates the symptoms, then this would confirm the diagnosis of tarsal tunnel syndrome.

Peripheral Vasodilatation for Ischaemic Complications

The prolonged duration of action of some local analgesic agents has been employed in the management of ischaemic ulcerations in the foot via sympathetic blockade using posterior tibial nerve analgesia. The theory behind this technique is that blockade of the sympathetic flow would bring about vasodilatation distal to the site of injection. There is no doubt that administration of local anaesthetic produces vasodilatation to the area of blockade - observation confirms that the area becomes noticeably warm. The advantage of using a local block to improve blood flow to an area lies in the ability to localise the block to a particular site. This is not possible with systemic medications which may preferentially increase flow through more competent vessels. The major disadvantage of using local analgesics in this role arises from the necessity to perform repeat injections. Additionally, tissue viability must be maintained after wound healing. It is difficult to see what role local analgesia would have for long term treatment.

Manipulation of Joints

Occasionally aggressive manipulation may be required to increase joint motion. This technique requires a local analgesic block for three reasons:

- Reduce pain of the manipulation
- Diminish muscle tension around joint
- Avoid reflex contraction during manipulation

Manipulation of the metatarsophalangeal joints may be facilitated by a posterior tibial block supplemented by local infiltration just proximal to the joint. Manipulation of more proximal and larger joints would require more proximal nerve blocks, namely common peroneal and posterior tibial. In view of the loss of protective pain responses, care should be taken when performing joint manipulation under anaesthesia to avoid tearing periarticular soft tissue structures or fracturing osteophytes. Patients should be warned that a moderate degree of discomfort is to be expected once the blockade has worn off. In addition, use of analgesia in this manner should be restricted to practitioners who have had specific training in joint manipulation.

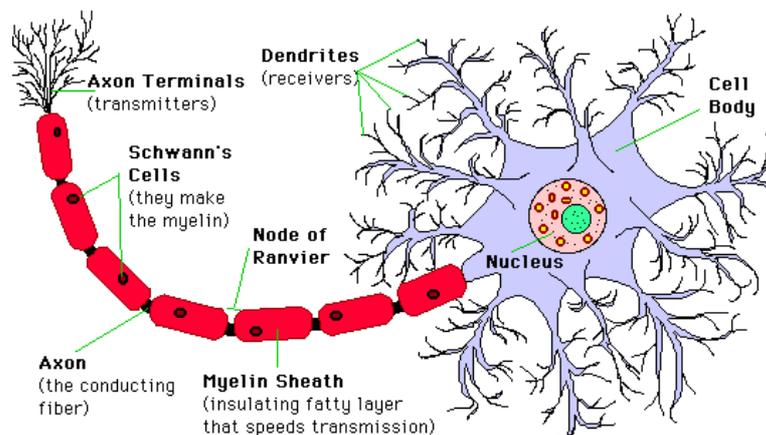
Arrangement of the Nervous System

The nervous system can be divided into two parts: the central nervous system and the peripheral nervous system. The central nervous system includes the brain and spinal cord, enclosed in the cranium and the vertebral canal. The peripheral nervous system includes 12 pairs of cranial nerves and their branches and 31 pairs of spinal nerves and their branches. The peripheral nervous system provides input to the central nervous system from sensory receptors and output from it to effectors (muscles and glands). Communicating networks within the central nervous system and various brain centres which process incoming sensory information make possible the appropriate unconscious or conscious response to sensory input. For convenience, peripheral efferent nerve fibers distributed to smooth muscle, cardiac muscle, and glands are usually referred to as the autonomic nervous system.

The nervous system is composed of a special tissue containing two major types of cells: neurons, the active conducting elements, and neuroglia (glia, meaning 'glue'), the supporting elements.

Neurones

The basic unit of the nervous system is the neurone, or nerve cell, which conducts an electrical impulse from one part of the body to another. The neurone itself consists of a cell body containing a single nucleus, and processes which transmit impulses to and from the cell body.



Neurons have two types of processes: axons and dendrites. An axon is a single, elongated, cytoplasmic extension carrying nerve impulses away from the cell body. The axon substance, or axoplasm, is jelly-like. The axon itself has a smooth outline, is of constant diameter, is sheathed, and terminates in more minute branches, which form junctions with effectors and other neurons. There is only one axon per neuron, but side branches, called collaterals, may arise along the course of an axon.

The dendrites are processes that carry impulses toward the cell body. The word dendrite describes the manner in which the processes appear in true dendrites - numerous, short, branching, and thickened at their point of origin. True dendrites are unmyelinated and their surfaces have spine-like projections (dendrite spines) that are the principal sites of junctions between dendrites and axon terminals, where nerve impulses are transmitted from one to the other. Sensory neurons (those conducting sensory information to the central nervous system) have a single process that bifurcates a short distance from the cell body. One branch (the peripheral process) runs from a receptor to the cell body located just outside the central nervous system. The other (the central process, or axon) runs from the cell body to the central nervous system. The peripheral process has the smooth surface, is myelinated, and is in other respects similar to axons. Sensory neurons, therefore, do not have true dendrites, although the peripheral process is sometimes called a dendrite because it conducts impulses to the cell body.

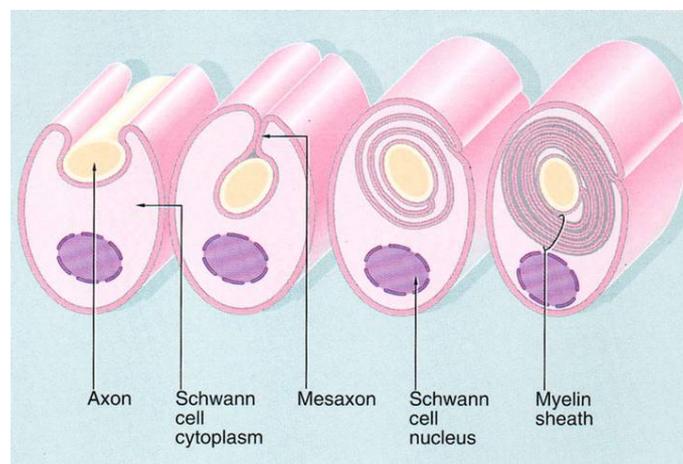
Cytoplasmic Organelles.

Located in the cell body are the endoplasmic reticulum and associated ribosomes (collectively called Nissl bodies), the Golgi apparatus, mitochondria, and lysosomes. True dendrites have a similar organelle composition but lack the Golgi apparatus and have lesser amounts of endoplasmic reticulum. Ribosomes and the Golgi apparatus are not present in axons. Nissl bodies characteristically respond to injury of a nerve fiber by breaking up into a powder-like mass and dispersing with a loss of affinity to

stains, a change called 'chromatolysis'. Also present throughout the neurone are microtubules, neurofilaments, and microfilaments. These organelles play a role in transporting neuronal substances, especially neurotransmitters or enzymes needed for their synthesis from the cell body to axon terminals. They also appear to be involved in nerve fiber growth.

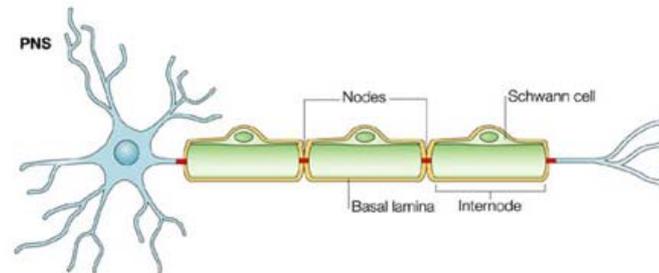
The Nerve Fiber.

The term 'nerve fiber' refers to any long neurone process, such as an axon or peripheral process of a sensory neurone. All fibers of the peripheral nervous system have a wrapping outside the cell membrane formed by accessory cells of the peripheral nervous system called Schwann cells. Fibers less than about one micrometer in diameter have a thin wrapping. In the case of the larger-diameter fibers, repeated wrappings form a thick sheath called myelin. When this sheath is formed, the bulk of the cytoplasm of the Schwann cell is expelled as it wraps around a segment of the nerve fiber, so that what remains is a tightly wound spiral of Schwann cell membrane with occasional clefts of cytoplasm.



The outermost wrapping, containing the flattened nuclei of the Schwann cells and the greater part of their cytoplasm, is referred to as the neurilemma or sheath of Schwann. Myelin covers the entire fiber except at its termination, which is enveloped by the neurilemma, and at periodic gaps called nodes of Ranvier, where the neurilemma dips inward with finger-like processes to cover the fiber. Segments between nodes are called

internodes, each formed by a single Schwann cell. Fibers wrapped in a myelin sheath are called myelinated fibers. The small-diameter fibers, which lack the multilayered myelin sheath, are called non-myelinated fibers.



In the central nervous system, where Schwann cells are absent, the myelin sheath is formed by accessory cells called oligodendroglia. However, there is an important difference between myelin formed by oligodendroglia and Schwann cells. A whole oligodendroglial cell does not wrap itself around a segment of a neurone fiber; rather, it sends out processes (the average number may be as high as 40), each of which wraps around a segment of an adjacent fiber. Hence, these fibers lack an outer, nucleus-containing wrapping (neurilemma).

Myelin is about 80 per cent lipid and is an effective insulator. It increases the rate at which impulses are conducted along nerve fibers, a property described as saltatory conduction.

There are three classes of neurones entering into the formation of nerve pathways. Sensory, or afferent (afferre, to carry to), neurones convey impulses from the skin or other sense organs to the central nervous system (spinal cord and brain). Motor, or efferent (efferre, to carry away), neurones carry impulses away from the central nervous system to muscles and glands. The third class consists of neurones which lie entirely within the central nervous system. These neurones receive input from sensory neurones and communicate with one another or with motor neurones.

KEY LEARNING POINTS.



- 1. The basic cell of the nervous system is the neurone, consisting of a cell body, axons, and dendrites.**
- 2. Dendrites carry impulses towards the cell body, whilst axons carry impulses away.**
- 3. Schwann cells act to insulate the nerve, speeding up the rate of impulse transmission.**
- 4. Sensory neurons carry information from the peripheral nervous system to the central nervous system, motor neurons transmit away from the central nervous system to effector organs.**

The Nerve Impulse - Action potential generation

Neurones function to conduct signals from one part of the body to another. The capacity for selective permeability to ions is a function of the cell membrane, and it is this property of the nerve cell which is involved in the transmission of the nerve impulse. In the resting state the interior of the nerve fiber is negative to the exterior by approximately 70 to 90 millivolts. This difference in potential across the membrane is called the resting membrane potential of the nerve fiber. In general terms, the origin of the resting membrane potential is accounted for as follows: the active transport of positively charged sodium ions to the outside of the cell (the so-called sodium pump) with the reciprocal transfer of positively charged potassium ions to the inside maintains a high concentration of sodium outside and a high concentration of potassium inside the cell.

Diffusion of sodium and potassium across the cell membrane in response to the concentration gradients created by the active transport mechanism results in the 'leaking' of sodium back into and potassium out of the cell. However, the cell membrane is much more permeable to potassium than to sodium. Very little inward diffusion of sodium occurs, and the greater outward diffusion of potassium creates a deficit of positive charges on the inner surface of the membrane that is responsible for the resting membrane potential. The relatively high permeability of the membrane to potassium is accounted for by the presence in the membrane of a class of permanently open channels selectively permeable to potassium.

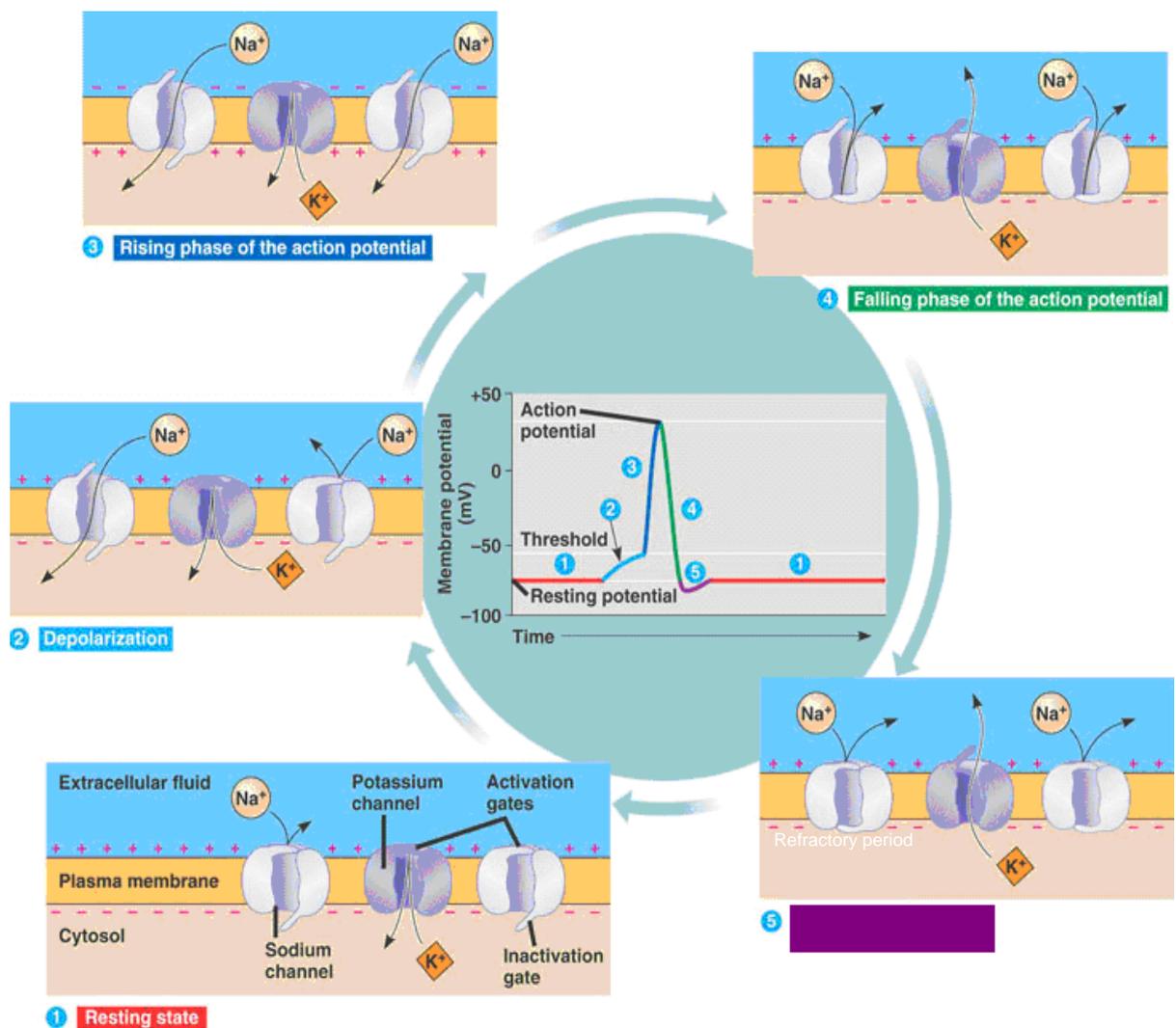
When a stimulus is applied to a nerve cell, an impulse, a transient reversal of the membrane potential, is transmitted along the nerve fiber. This comes about as follows:

the stimulus causes changes in the conformation of membrane proteins functioning as sodium and potassium ion channels that are closed in the resting state. This results first in opening of the sodium channels and a rapid inflow of sodium, which changes the membrane potential locally from negative to positive. This is followed by closing of the sodium channels and opening of the potassium channels. A rapid outflow of potassium returns the membrane potential to negative. These changes are known as the action potential. The local disturbance stimulates the adjacent regions of the nerve fiber, and the action potential sweeps along the fiber. In the body the stimulus is normally received at one end of the neuron and is propagated in one direction, but if a nerve fiber is artificially stimulated in the middle the action potential will be transmitted in both directions.

For a brief period following stimulation of a nerve fiber it will not respond to a new stimulus. The interval of complete unresponsiveness is called the absolute refractory period. The absolute refractory period is followed by a relative refractory period, during which time a stronger than minimum effective stimulus will lead to the transmission of a nerve impulse. For a large mammalian myelinated nerve fiber the absolute refractory period

ranges from 0.4 to 1 millisecond. Excitability gradually returns to about 95 per cent of the resting level in 10 to 30 milliseconds.

The absolute refractory period corresponds to a period when the inflow of sodium ions is completely inactivated. The sodium channel protein apparently has three conformations. In the resting state the conformation is closed. A stimulus changes it to an open conformation. This change from a closed to an open conformation is terminated after a brief interval by a process called sodium inactivation. The closed conformation in the inactivated state is different from the resting state conformation, and until the channel returns to the resting state it will not respond to a new stimulus.



All-or-None Principle.

The transmission of a nerve impulse by a nerve fiber is said to work on an all-or-none principle. This means that nerve fibers will not transmit an impulse unless the stimulus has a certain strength (the threshold of the nerve fiber). If the threshold is reached, the impulse is maximal. Each type of nerve fiber sends an impulse of only one strength - its characteristic impulse. A stronger stimulus does not lead to a larger impulse.

A stimulus just strong enough to lead to a propagated impulse is called a threshold stimulus. A subthreshold stimulus will cause the internal potential to become briefly less negative, deflecting the voltage upward. Only when the threshold voltage of the nerve fiber is reached will the sodium influx be of sufficient magnitude to cause the sharp spike potential and propagated impulse.

Different nerve fibers have different thresholds - some will fire only with very strong stimulation, others with very weak stimulation, but all fibers work characteristically and on the all-or-none firing principle.

Saltatory Conduction.

Myelin is resistant to the flow of ions, and the thick myelin sheath of myelinated fibers prevents continuous passage of impulses along the length of the fiber. Ion flow at nodes of Ranvier sets up potential differences between nodes, and the impulse jumps from one node to another. This process, called saltatory conduction, greatly increases the transmission velocity of nerve impulses.

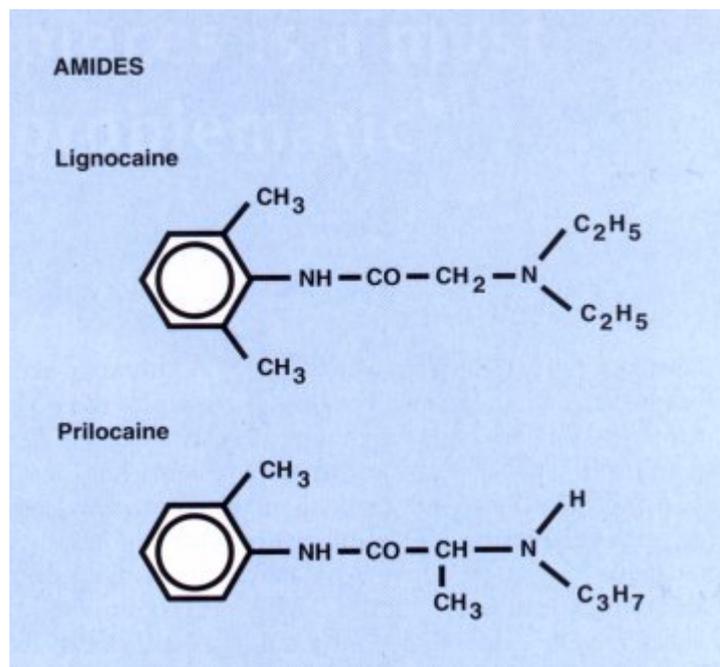
KEY LEARNING POINTS.



- 1. The diffusion of sodium and potassium ions across the neurone membrane bring about the production of an action potential.**
- 2. The relative opening and closing of sodium channels, followed by potassium channels, controls the generation process.**
- 3. the all-or-nothing principle means that a threshold change in charge across the membrane is required, but once this is reached, the maximum response is produced.**

Pharmacology

Modern local analgesic drugs consist of molecules that have both a benzene ring and an amide group, linked by an intermediate chain. The older agents have an ester group in place of the amide, but this makes the molecule less stable, and predisposes to increased toxicity, therefore the amide-based molecules are used preferentially.



The benzene ring allows the molecule to be lipid-soluble (lipophilic), aiding the molecule to move across cell membranes. The amide group makes the molecule water-soluble (hydrophilic), allowing it to dissolve in water, and aiding dispersal in the tissues. Amide agents include lignocaine, mepivacaine, prilocaine, bupivacaine, ropivacaine, and levobupivacaine. To improve the stability of amide agents, they are usually combined with a hydrochloride eg, lignocaine hydrochloride, which is both stable, and soluble in water. Generally these drugs are all metabolised in the liver, although prilocaine is also metabolised in the lungs.

How do analgesic agents work ?

Local analgesic agents act as membrane stabilisers, meaning that they prevent the passage of sodium ions across the cell membrane, and therefore prevent depolarisation. They do this by blocking the sodium ion channels. Once the agent has been injected into the tissues, it diffuses across the cell membrane, and once inside the axon, the molecules of agent combine with hydrogen ions, and this complex binds to a receptor on the ion channel, causing the channel to close, thereby preventing the influx of sodium. It is important that hydrogen ions combine with the agent molecule, as the analgesic molecule on its own cannot bind to the receptor and block the ion channel.

However, if the agent molecules combine with hydrogen ions before they have diffused across the cell membrane, then the agent-hydrogen complex cannot get into the axon, and therefore the analgesic is ineffective. This situation can occur when infection is present in the tissues, which creates an acidic environment. In acidic conditions there are more free hydrogen ions available, and therefore these will bind with the analgesic before it has a chance to diffuse into the cell, hence the problem with analgesic effectiveness in the presence of infection.

Local Analgesic Drugs.

Currently a number of agents are licensed for use in Podiatry for the purposes of inducing local analgesia. These are ;

1. lignocaine
2. mepivacaine
3. prilocaine
4. bupivacaine
5. levobupivacaine
6. ropivacaine

The choice of which agent is the most suitable will depend on the characteristics of each of these agents, such as onset, duration, distribution, regression, toxicity, and also the type of procedure to be performed.

The onset of the drug refers to the time it takes from administration to the onset of analgesia. Factors that affect this length of time include the amount of myelination of the nerve, and also the diameter of the nerve. The larger the nerve and the more myelin present, the longer it will take for the onset of analgesia. Additionally, sensory loss will occur before motor loss.

The duration, as the term suggests, relates to the length of time the analgesic effect is in place. This can be affected by the amount of drug which has been administered. Once the sensation begins to return, this is known as regression, and refers to the time when sensation begins to return, to the point of complete return of function.

Distribution refers to the ability of the drug to move through the tissues, and reach the site where its effect is required, i.e. the nerve axon. The more water-soluble the drug, the easier it can be distributed within the tissues.

The toxicity of the drug relates to the potential side-effects it can produce. Clearly, the fewer side-effects the better, but it is accepted that all drugs have some undesirable effects. In the case of local analgesia, toxicity is more likely related to intra-vascular injection, or by injecting excessive amounts of drug.

Lignocaine.

Lignocaine was first introduced as 'xylocaine' in 1949, and is also currently known as lidocaine. As well as its role in analgesia it is also used in the management of cardiac arrhythmias as one of its effects to slow the heart

rate (bradycardia). Lignocaine can also produce a local vasodilatory effect. Around 90% of the administered lignocaine is metabolised by the liver, therefore in patients with liver disease this drug should be used with caution. Lignocaine may also interact with cimetidine. It has a fairly rapid onset time of approximately ten minutes, with a duration of between 90 and 120 minutes. The recommended maximum safe dosage is 3mg per kilo of body weight.

Mepivacaine.

Mepivacaine is similar to lignocaine, but without the potential cardiac side effects, and produces less vasodilation. It has a similar onset time to lignocaine, but has a slightly longer duration time. However, it is less toxic than lignocaine, and has a maximum safe dose per kilo of 6mg.

Prilocaine.

Prilocaine also has a similar onset time to lignocaine, and its effects last for up to two hours. It also has a maximum safe dose per kilo of 6mg, but there is a possibility of the development of methaemoglobinaemia due to one of its metabolic by-products, and therefore should be used with caution as the uptake of oxygen can be compromised. Prilocaine is also metabolised in the liver, as well as the lungs and the kidney. This drug also has a useful topical effect in combination with lignocaine in the form of EMLA cream, although there are limitations to effectiveness which depend upon the thickness of the skin at the application site - the thicker the skin, the less effective the preparation, therefore EMLA has little if any use in digital or ankle analgesia.

Bupivacaine and Levobupivacaine.

Bupivacaine is the most toxic agent of the analgesic agents available to Podiatrists, with a maximum safe dose per kilo of 2mg. It can have cardiotoxic effects, and is associated with cardiac arrest in 0.5%-0.75% of cases. The most potent of the group, it is usually used in concentrations of 0.5% or 0.25%. Its duration of action is significantly longer than the others,

reported as up to eight hours, but additionally it's onset time is much slower, being approximately 30 minutes. Levobupivacaine is an isomer of bupivacaine, and is less toxic, but still has the same MSD of 2mg/kg. The extended action time of these drugs is related to protein-binding - these two agents are 90% protein bound, whereas lignocaine is only 64% protein bound. The protein bound agent acts as a reservoir which replaces non-bound drug as it is absorbed and metabolised, therefore the higher the percentage of protein binding, the larger the reservoir from which to provide free drug, and therefore the longer the analgesic effect.

Ropivacaine.

This is similar to bupivacaine, but is considered to be less cardiotoxic. It differs in that it has less of a motor blockade effect whilst achieving a good sensory blockade.

Contra-indications for the use of Local Analgesia.

It is generally accepted that these drugs are very safe when used appropriately, therefore careful patient selection and assessment are important. In certain situations extra caution is required :

Liver disease - the liver is the prime site of metabolism for many drugs, therefore patients with liver pathology may well have difficulty in breaking down drugs. This means that the active drug will stay in the circulation longer, with the greater chance of toxic side-effects occurring.

Kidney disease - as well as metabolising some drugs, the kidneys are also necessary for excretion of the drug metabolites, therefore good kidney function is required.

Diabetes mellitus - this is not usually a contra-indication unless there has been poor blood glucose control. However, as there may be some immune-compromise, extra vigilance is required to reduce the risk of infection. It should also be remembered that there may be peripheral neuropathy, which may affect the action of LA.

Peripheral vascular disease - moderate to severe PVD would preclude both the use of LA and surgical procedures on these patients.

Local sepsis - injection at the site of infection may contribute to infection spread, simply because introduction of the needle crosses different tissue planes. Additionally, infection in the tissues creates an acidic environment, increasing the levels of available hydrogen ions in the area, thereby reducing the effectiveness of the analgesic agent.

Epilepsy - most local analgesic agents can act as cerebral stimulants if given in high enough doses, or accidentally injected intra-vascularly, therefore unless the epilepsy is stable, they should be avoided.

Pregnancy - whilst there is little evidence suggesting that these drugs are potentially harmful to the fetus, it is sensible to avoid their use in the first trimester of pregnancy, but once past this stage they are said to be safe to use. However, it may be wise to exercise caution, and use judgement as to whether conservative treatment is a better option until after delivery.

Haemophilia - due to the increased bleeding times in this situation, it may be wise to avoid injections on these patients.

Leukaemia - the potential for the risk of bleeding and the reduced resistance to infection may cause the practitioner to think carefully before using LA.

Working out drug dosages.

In order to use these drugs safely it is essential that the practitioner be aware of how much drug can be used for each patient. There is guidance on the maximum safe dose for each of the drugs, and this should be used in order to work out the maximum safe dose (MSD) for each patient, related to their body weight.

Generally, when using these drugs, it should be remembered that ;

- if 10mg of drug is present in 1ml of solution, this is 1% solution.
- if 20mg of drug is present in 1ml of solution, this is 2% solution.
- if 30mg of drug is present in 1ml of solution, this is 3% solution.
- if 0.5mg of drug is present in 1ml of solution, this is 0.5 % solution.

Consider the following patient:

Mr M is in good general health, and requires a total nail avulsion to the left first digit. He weighs 80 kg. You wish to use 1% lignocaine.

To work out the MSD, the following calculation is used:

Body weight x MSD per kg

Mg of drug per %

Therefore the calculation should be : $\frac{80 \times 3}{10} = \frac{240}{10} = 24$

So for Mr M, weighing 80kg, using 1% lignocaine, the maximum volume of drug given is 24mls (although all of this amount does not need to be administered!). In the calculation, the '3' is the 3mg per kilo of body weight allowed for lignocaine, and the '10' is the 10mg per ml of 1% solution.

In the same patient, but using a 2% solution, the calculation would be:

80 x 3 = 12ml

20 the '20' represents the 20mg of drug per ml of 2% solution.

Again with the same patient, but using a standard 3% mepivacaine solution :

80 x 6 = 16ml

30 where '6' is the MSD per kg for mepivacaine, '30' because it's a 3% solution.

(There are some calculations in the course workbook for you to practice on)

KEY LEARNING POINTS.



- 3. Analgesic agents have both fat-soluble and water-soluble properties, making it easier for them to cross the cell membrane, and also disperse through the tissues.**
- 4. If the drug combines with hydrogen ions before it crosses the cell membrane, it will not be able to cross the membrane, and the analgesic effect will not occur.**
- 5. Drugs licensed for use by Podiatrists are amide analgesic drugs, which are generally stable, and safe to use, but must always be used in plain solution, without the addition of a vasoconstrictor.**
- 6. The practitioner must ensure that they have obtained sufficient information from the patient regarding potential contra-indications to the use of local analgesia.**
- 7. The practitioner should always calculate the correct MDS for each patient, based upon weight, and this should always be recorded in the patient's notes.**

Recognising anaphylaxis and toxicity.

In addition to this information, it is strongly recommended that you read the document found at the following link :

<http://www.resus.org.uk/pages/reaction.pdf>

Anaphylaxis is a potentially life-threatening, severe, whole-body allergic reaction. After being exposed to a substance (eg, food, insect venom, drugs), the immune system becomes sensitised to that allergen. On a later exposure, an allergic reaction may occur. This reaction is sudden, severe, and involves the whole body.

Like the majority of other allergic reactions, anaphylaxis is caused by the release of histamine and other chemicals from mast cells. On their surfaces, mast cells display antibodies (IgE) which detect environmental substances to which the immune system is sensitive. Substances from a genuinely threatening source, such as bacteria or viruses, are called antigens. A substance that most people tolerate well, but to which others have an allergic response, is called an allergen. When IgE antibodies bind with allergens, they cause the mast cell to release histamine and other chemicals, which also affect neighbouring cells.

The interaction of these chemicals with receptors on the surface of blood vessels causes the vessels to leak fluid into surrounding tissues, causing fluid accumulation, redness, and swelling (oedema). On the smooth muscle cells of the airways and digestive system, they cause constriction. On nerve endings, they increase sensitivity and cause itching.

In anaphylaxis, the dramatic response is due both to extreme hypersensitivity to the allergen and its usually systemic distribution. Allergens are more likely to cause anaphylaxis if they are introduced directly into the circulatory system by injection. However, exposure by ingestion, inhalation, or skin contact can also cause anaphylaxis. In some cases, anaphylaxis may develop over time from less severe allergies.

Symptoms

Symptoms develop rapidly, often within seconds or minutes. They may include any of the following:

- Abdominal pain or cramping
- Abnormal (high-pitched) breathing sounds
- Anxiety
- Confusion
- Cough
- Diarrhea
- Difficulty breathing
- Fainting, light-headedness, dizziness
- Hives, itchiness
- Nasal congestion
- Nausea, vomiting
- Sensation of feeling the heart beat (palpitations)
- Skin redness
- Slurred speech
- Wheezing

Signs include:

- Abnormal heart rhythm (arrhythmia)
- Fluid in the lungs (pulmonary oedema)
- Hives
- Low blood pressure
- Mental confusion
- Rapid pulse
- Skin that is cyanotic (blue) from lack of oxygen, or pale from shock
- Swelling (angioedema) in the throat that may be severe enough to block the airway
- Swelling of the eyes or face
- Weakness
- Wheezing

Treatment

Anaphylaxis is an emergency condition requiring immediate medical attention - it is wise to call '999' and request Paramedic help. In addition, immediate treatment should begin :

Check the ABCs (airway, breathing, and circulation) in all suspected anaphylactic reactions. CPR should be started, if needed. People with known severe allergic reactions may carry an Epi-Pen or other allergy kit, and should be helped to administer it if they are conscious.

The dosage should be as follows :

Intra-muscular (IM) doses of 1:1000 adrenaline

Adult :	500 micrograms IM (0.5 mL)
Child more than 12 years:	500 micrograms IM (0.5 mL)
Child 6 -12 years:	300 micrograms IM (0.3 mL)
Child less than 6 years:	150 micrograms IM (0.15 mL)

The dose should be repeated after 5 min if there is no improvement.

The use of an EpiPen (preloaded injection device) may make administration simpler, although suitable training in their use is required.

In addition to adrenaline, patients may also be subsequently given antihistamines, such as diphenhydramine, and corticosteroids, such as prednisone, to further reduce symptoms (after lifesaving measures and adrenaline are administered). However, these are administered by either Paramedics or Medics, not by Podiatrists.

Anaphylaxis is a severe disorder that can be life-threatening without prompt treatment. However, symptoms usually get better with the correct therapy, so it is important to act immediately.

Toxicity.

Toxic reactions can occur but are more usually associated with exceeding the maximum safe dose, or intra-vascular injection. These agents are lipid-soluble therefore they cross the blood-brain barrier - many of the side-effects seen with toxicity are in the central nervous system. Initially this may lead to CNS stimulation, but severe toxicity will lead to CNS depression, and possibly death.

Some of the signs include:

- Restlessness
- Excitement
- Dizziness
- Tinnitus
- Blurred vision
- Numb lips
- Muscle tremors or twitching
- Convulsions

Management of toxicity will always require medical intervention, therefore medical help should also be called. The practitioner should be capable of performing cardio-pulmonary resuscitation (CPR) if necessary until help arrives.

KEY LEARNING POINTS.



- 1. The practitioner MUST ensure that they can recognise the signs of anaphylaxis and toxicity, and know the appropriate management in each situation.**
- 2. The practitioner must engage in annual updates in CPR and basic Life Support.**

Consent

(Much of this information reflects current Department of Health guidelines of seeking and gaining consent for treatment)

Patients have a fundamental legal and ethical right to determine what happens to their own body. Valid consent to treatment is therefore absolutely central in all forms of healthcare, from providing personal care to undertaking major surgery. Seeking consent is also a matter of common courtesy between health professionals and patients.

Consent is a patient's agreement for a health professional to provide care. Patients may indicate consent non-verbally (for example by presenting their arm for their pulse to be taken), orally, or in writing. For the consent to be valid, the patient must:

- be competent to take the particular decision
- have received sufficient information to be able to understand
- not be acting under duress.

The context of consent can take many different forms, ranging from the active request by a patient for a particular treatment (which may or may not be appropriate or available) to the passive acceptance of a health professional's advice. In some cases, the health professional will suggest a particular form of treatment or investigation and after discussion the patient may agree to accept it. In others, there may be a number of ways of treating a condition, and the health professional will help the patient to decide between them. Some patients, especially those with chronic conditions, become very well informed about their illness and may actively request particular treatments. In many cases, 'seeking consent' is better described as 'joint decision-making': the patient and health professional need to come to an agreement on the best way forward, based on the patient's values and preferences and the health professional's clinical knowledge.

Where an adult patient lacks the mental capacity (either temporarily or permanently) to give or withhold consent for themselves, **no-one else can give consent on their behalf**. However, treatment may be given if it is in their best interests, as long as it has not been refused in advance in a valid and applicable advance directive.

For some procedures, it is sensible to document clearly both a patient's agreement to the intervention and the discussions which led up to that agreement. This may be done either through the use of a consent form (with further detail in the patient's notes if necessary), or through documenting in the patient's notes that they have given oral consent.

Consent is often wrongly equated with a patient's signature on a consent form. A signature on a form is **evidence** that the patient has given consent, but is not **proof** of valid consent. If a patient is rushed into signing a form, on the basis of too little information, the consent may not be valid, despite the signature. Similarly, if a patient has given valid verbal consent, the fact that they are physically unable to sign the form is no bar to treatment. Patients may, if they wish, withdraw consent after they have signed a form: the signature is evidence of the process of consent-giving, not a binding contract. It is rarely a legal requirement to seek written consent, except in certain circumstances, but it is good practice to do so if any of the following circumstances apply:

1. the treatment or procedure is complex, or involves significant risks (the term 'risk' is used throughout to refer to any adverse outcome, including those which some health professionals would describe as 'side-effects' or 'complications')
2. the procedure involves general/regional anaesthesia or sedation
3. providing clinical care is not the primary purpose of the procedure
4. there may be significant consequences for the patient's employment, social or personal life

Completed forms should be kept with the patient's notes. Any changes to a form made after the form has been signed by the patient, should be initialled and dated by both patient and health professional.

It will not usually be necessary to document a patient's consent to routine and low-risk procedures, but if there is any reason to believe that the consent may be disputed later or if the procedure is of particular concern to the patient (for example if they have declined, or become very distressed about, similar care in the past), it would be helpful to do so.

Where an adult patient does not have the capacity to give or withhold consent to a significant intervention, this fact should be documented, along with the assessment of the patient's capacity, why the health professional believes the treatment to be in the patient's best interests, and the involvement of people close to the patient.

An apparent lack of capacity to give or withhold consent may in fact be the result of communication difficulties rather than genuine incapacity. Appropriate colleagues should be in making such assessments of incapacity, such as specialist learning disability teams and speech and language therapists, unless the urgency of the patient's situation prevents this. If at all possible, the patient should be assisted to make and communicate their own decision, for example by providing information in non-verbal ways where appropriate.

Where analgesia will be administered, it is the responsibility of the person giving the analgesia (not that of the surgeon) to seek consent, having discussed the benefits and risks. The practitioner should ensure that the discussion with the patient and their consent is documented in the patient's notes or on the consent form. Where the clinician providing the care is personally responsible for analgesia (eg where local anaesthesia or sedation is being used), then he or she will also be responsible for ensuring that the patient has given consent to that form of treatment.

Treatment of young children

When babies or young children are being treated the practitioner should discuss with the parent(s) the proposed treatment, and ensure that they give their consent for these interventions in advance. If parents specify that they wish to be asked before particular procedures are initiated, you must do so.

Only people with 'parental responsibility' are entitled to give consent on behalf of their children. You must be aware that not all parents have parental responsibility for their children, eg, unmarried fathers do not automatically have such responsibility although they can acquire it. If there is any doubt about whether the person with the child has parental responsibility for that child, this must be checked.

The provision of information is central to the consent process. Before patients can come to a decision about treatment, they need comprehensible information about their condition and about possible treatments or investigations, and their risks and benefits (including the risks/benefits of doing nothing). They also need to know whether additional procedures are likely to be necessary as part of the procedure. Once a decision to have a particular treatment/investigation has been made, patients need information about what will happen.

Patients and those close to them will vary in how much information they want: from those who want as much detail as possible, including details of rare risks, to those who ask health professionals to make decisions for them. There will always be an element of clinical judgement in determining what information should be given. However, the presumption must be that the patient wishes to be well informed about the risks and benefits of the various options. Where the patient makes clear (verbally or non-verbally) that they do not wish to be given this level of information, this should be documented.

Refusal of consent

A competent adult patient is entitled to refuse any treatment, except in circumstances governed by the *Mental Health Act 1983*. If, after discussion of possible treatment options, a patient refuses all treatment, this fact should be clearly documented in their notes. If the patient has already signed a consent form, but then changes their mind, this should be noted.

Where a patient has refused a particular intervention, the practitioner must ensure that they continue to provide any other appropriate care to which the patient has consented. It should also be ensured that the patient realises they are free to change their mind and accept treatment if they later wish to do so. Where delay may affect their treatment choices, they should be advised accordingly.

If a patient consents to a particular procedure but refuses certain aspects of the intervention, the practitioner must explain to the patient the possible consequences of their partial refusal. If it is genuinely believed that the procedure cannot be safely carried out under the patient's stipulated conditions, the practitioner is not obliged to perform it, but there must be provision of any other appropriate care. Where another health professional believes that the treatment can be safely carried out under the conditions specified by the patient, the practitioner must, on request, be prepared to transfer the patient's care to that health professional.

KEY LEARNING POINTS.

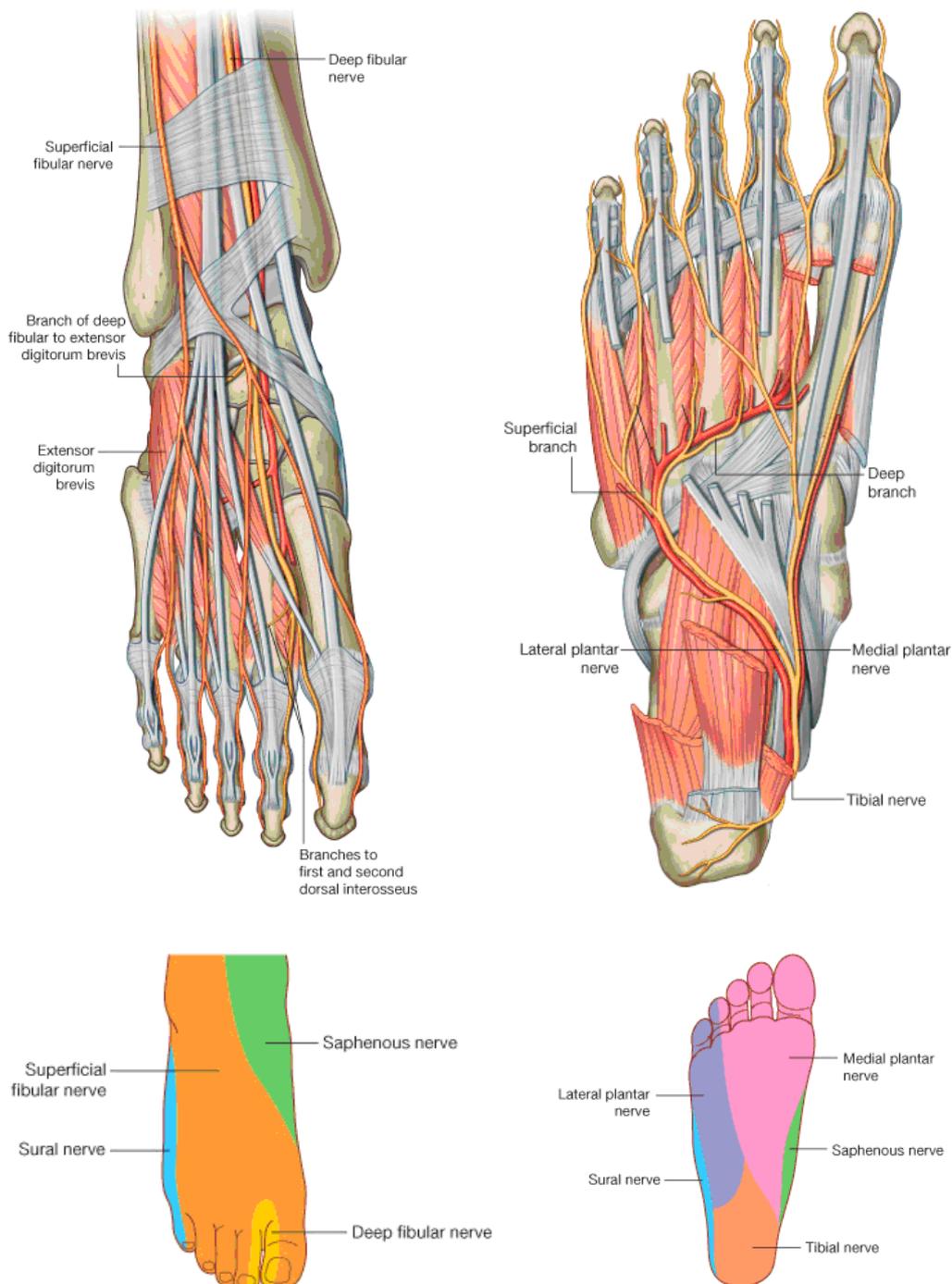


- 1. Every practitioner should ensure that they know the legal requirements relating to patient consent for treatment for adults, children, and also for those not capable of giving consent for themselves.**
- 2. Clear and unambiguous records should be made relating to discussion with patients relating to consent.**
- 3. Practitioners must respect a patient's right to refuse consent, without this being prejudicial to the patient's treatment.**

Common administration techniques in podiatry

Digital Block.

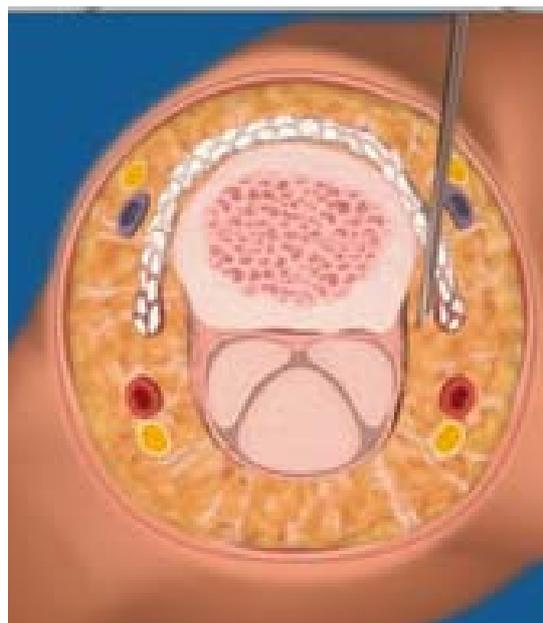
This is probably the most commonly used technique in Podiatry, and provides analgesia to all surfaces of the digit involved. The aim is to deposit the agent around the dorsal and plantar digital nerves, of which there are four in total.



The dorsal digital nerves are extensions of the superficial peroneal (fibular) nerve, and supply the dorso- medial and dorso-lateral aspects of all the digits, with the exception of the lateral aspect of the first and medial aspect of the second digits, which are supplied by the deep personal nerve.

On the plantar aspect, sensation is provided via the medial plantar nerve to the first, second, third, and medial aspect of the fourth digits, whilst the later aspect, and all of the fifth digit is served by the lateral plantar nerve. In addition, these two nerves also supply the apices of all five digits. The medial and lateral plantar nerves are branches of the tibial nerve.

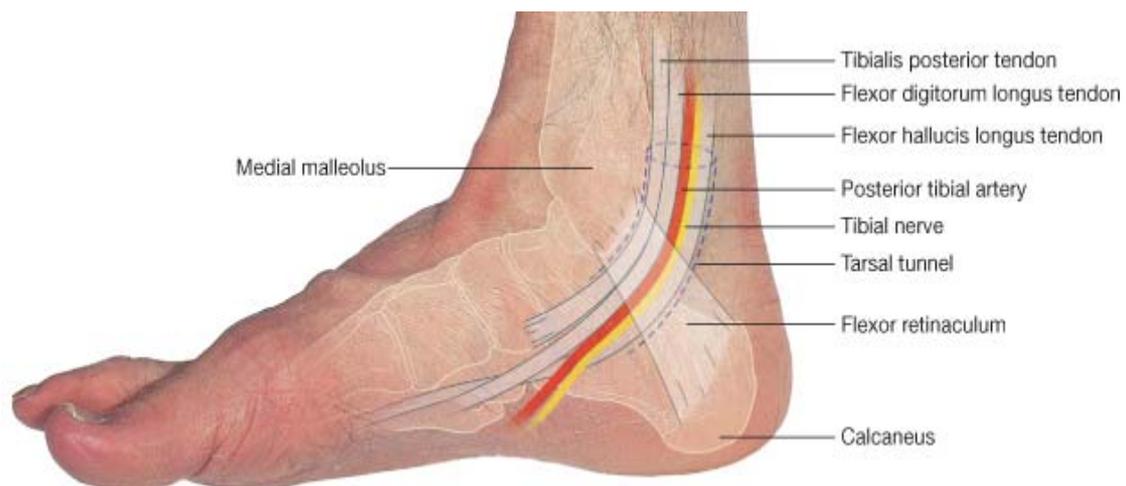
The technique consists of introducing the needle through the dorsum of the digit on the medial side first, advancing the tip through the tissues towards the plantar aspect of the toe to be adjacent to the plantar digital nerve, where a small amount of fluid can be deposited. It is important to aspirate before depositing fluid to ensure that intra-vascular injection has not occurred. The needle is then withdrawn to a level where the dorsal digital nerve is located, aspiration is performed again, and a further small amount of fluid is deposited. The needle is then completely withdrawn from the digit, and the procedure repeated on the lateral aspect of the digit.



It is possible to check the position of the needle in relation to the plantar digital nerve as there will be a bulging and blanching of the tissues as the needle tip progresses towards the base of the toe. Additionally, as the fluid is introduced into the area, there will be expansion of the tissues in order to accommodate the fluid, and this is another indication that intra-vascular injection has not occurred. Usually, analgesia should have been achieved within 10 minutes of a completed injection.

Tibial nerve block

If analgesia to the plantar aspect of the foot is required then blockade of the tibial nerve can be performed. The technique involves a deep injection, and can use up to 5 mls of fluid to produce a good level of analgesia.



The tibial nerve runs behind the medial malleolus, together with the posterior tibial artery, within the tarsal tunnel, and underneath the flexor retinaculum. It gives rise to the medial and lateral plantar nerves, as well as supplying sensation to the plantar aspect of the heel.

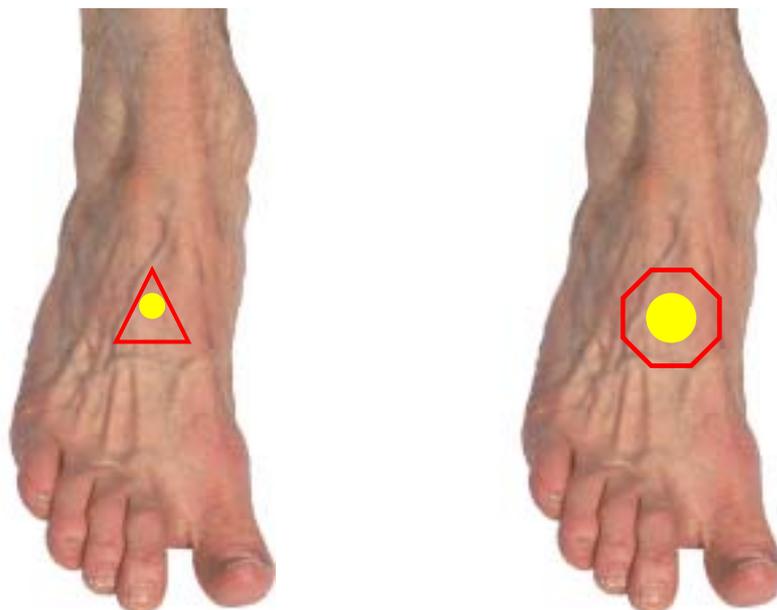
For this technique the patient should be supine, and it helps in stabilising the foot if the ankles are crossed, the foot to be anaesthetised on top. The posterior tibial pulse is located with the fingers, as the nerve runs directly posterior to it, and vessel palpation helps prevent intra-vascular injection.

The needle should be introduced directly behind the pulse, and the tip advanced in the direction of the tip of the fourth digit, until the tibia is reached. The tip should then be withdrawn slightly, and aspiration should be performed. Once the operator is sure that the tip is not within the blood vessel, fluid can be deposited in this area, before withdrawing the needle. In the case of a tibial block at this level, analgesia can take up to 30 minutes to become fully effective.

Infiltration

This aim of this technique is to encircle a lesion with agent, ideally to allow more extensive treatment to be pain-free. It is really only suitable for relatively superficial lesions, but is not recommended for the plantar aspect of the foot, as this would be an extremely painful!

Infiltrations can differ depending on the size of the lesion involved. For smaller lesions, a simple triangular approach is suitable, but for larger lesions, an encircling technique is preferred. For either situation, the method of introducing the needle through the skin remains the same. It is best to decide beforehand whether triangular or encirclement infiltration is suitable. The operator should then visualise this on the foot.



The needle is introduced so that it will lie subcutaneously, at one of the 'angle' points of the visualised shape, and the tip advanced to the next 'angle'. The fluid is injected as the tip of the needle is withdrawn back toward the point at which it was inserted. The needle is then removed, and the procedure repeated at the next angle, until the lesion has been surrounded. Once this is complete, there should be a wall of fluid around the lesion that also extends underneath. The analgesic effect should be apparent in around 10 minutes.