Electrotherapy Explained

Principles and practice

Second edition

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PRINCIPLES

All stimulators of nerve tissue (except implanted stimulators) are in fact transcutaneous electrical nerve stimulators (TENS), but this term is applied only to low-intensity, usually battery-operated, sensory nerve stimulators used for pain control. Such names as faradic, galvanic or diadynamic, historically applied to specify certain therapeutic currents, often overlap and are used inconsistently.

First, it is important to understand the effects of electric charges on the tissues in general terms. These effects depend on the rate of change of the electric pulse:
1. If there is no change, or only a very slow change, and the current is unidirectional there will be a steady flow of ions into and within the tissues causing chemical changes at the electrode—tissue junction, as described in Chapter 2.
2. If the rate of change is somewhat faster and the pulse has a long enough duration, the ionic balance across excitable membranes is disturbed stimulating nerves and muscles (see Chapter 1). If the current is unidirectional it will also lead to chemical changes, as above, but if it is evenly alternating no such changes can occur because any change in one direction is immediately cancelled when the current reverses.

3. If the rate of change is very fast there is insufficient time for transmembrane excitation to occur so that much larger currents can be employed, which can lead to significant heating. This is the basis of diathermy (see Chapter 10).

The extent of the physiological changes will obviously depend on the current intensity; higher currents cause greater effects. The intensity will also determine whether any single electrical pulse has enough energy to provoke a nerve impulse. Thus a given rate of rise, duration and fall of pulse may be too rapid to cause nerve stimulation at a low current intensity, but may do so at a higher intensity.

It is convenient to describe the unit of stimulating current as a ‘pulse’ of current or current phase. This will cause one (or two) nerve impulses. The simple graphs given in Figure 3.1 illustrate the relationship of time and current intensity and hence the rate of change of current. Thus in (a) a slow rise, steady, unidirectional current, and a slow fall illustrate the direct current used in galvanic current or iontophoretic treatments (see Chapter 2). If the rise is made rapid — i.e. there is a high rate of change — then nerve stimulation occurs leading to a nerve impulse, shown in (b) (see Chapter 1). This will happen both when the current rises and when it falls. If the time for which the current flows is made shorter, say 1 ms, as in (c) then there is no time for the nerve membrane to recover and only a single nerve impulse results. The consequence of (b) and (c) would be to stimulate sensory nerves giving a series of single nerve impulses recognized consciously as a series of little shocks. Similarly motor nerves would be stimulated leading to a series of single muscle twitches. If these stimuli are repeated every 10 ms they will cause a steady tingling sensation as they stimulate sensory nerves and a tetanic muscle contraction as they stimulate the motor nerves; this is shown in (d) (see also Chapter 1). Such currents at low intensities produced by small battery-operated electronic stimulators are used for pain control in TENS stimulators. At higher intensities they are used for muscle stimulation, when they are known as faradic stimulators.

The actual strength of muscle contraction or sensory effect will depend on the numbers of nerve fibres stimulated, which depends on the intensity of current. Greater current intensity will spread further in the tissues and hence activate more nerves. If the intensity is increased from zero over a period of a second or so and then decreased to zero again and this sequence is repeated, a series of rhythmic contractions and relaxations will occur like normal physiological muscle action (Fig. 3.1e). This is called a surged current or surging the current; American sources refer to this as ‘ramping’. It is simply current
Fig. 3.1 The relationship of time and current. (a) Direct current; (b) rapid rise and fall in current; (c) 1 ms pulses; (d) 1 ms pulses repeated every 10 ms; (e) surged current.

modulation and can be done automatically, i.e. electronically or manually.
It will be seen that all the currents shown in Figure 3.1 are unidirectional or monophasic so that all would have chemical effects. The individual pulses, except (a), would be described as ‘square wave’ pulses but there are other monophasic pulses which are not in this form, e.g. triangular. These single pulses or phases can be fully described by their:

1. **duration** in seconds, milliseconds (ms) or microseconds (us);
2. **intensity** in milliamps (mA) or voltage (V);
3. **shape** — rate of rise and fall, or how the intensity changes with time.

The term peak current intensity, or peak voltage or peak phase, refers to the highest current/voltage that occurs during the pulse. The mean (average) current/voltage will be less.

The pulse (phase) charge is easily calculated. The charge is the quantity of electric charge in coulombs (C) (see Appendix A). Thus a 1 ms pulse of 1 mA average intensity would have a charge of 1 μC. For further elucidation see Physical Principles Explained (Low and Reed, 1994).

If a series of pulses is considered the pulse rate can be expressed in pulses per second (pps) or the pulse frequency in hertz (Hz). The same information is given by describing the pulse interval, or interpulse interval, expressed in ms or s. Thus a series of 10 ms pulses separated by 90 ms pulse intervals will have a frequency of 10 Hz (Fig. 3.2).

So far consideration has only been given to unidirectional pulses. Many pulses used therapeutically are biphasic. Current passes first in one then in the opposite direction (Fig. 3.3a). Such discrete pulses may be separated by various pulse intervals like monophasic phases or they can be continuous (Fig. 3.3b). When such continuous pulses follow a sine curve the therapeutic current is called sinusoidal current. The mains current is in this sinusoidal form. Due to the constantly changing direction, such currents are called alternating currents. These are evenly alternating but it is common to have uneven alternations, which may be of unequal intensity, unequal duration or asymmetrical shape (Fig. 3.3c).

Clearly if the alternations are equal in charge there will be no total current flow and hence no chemical changes. If the alternations are
such that current in one direction is greater than in the other there will be a net current flow in the former direction. In many clinical sources the difference may be so small as to have little effect.

DEFINITIONS AND DESCRIPTIONS OF TYPES OF CURRENT USED THERAPEUTICALLY

**Direct current**

This refers to any unidirectional current but it is often used to mean constant direct current, that is an unvarying current also known as galvanism or a galvanic current (see Chapter 2).

**Low frequency stimulation**

Each pulse of current depolarizes the nerve fibre. The pulse repetition rate can be up to 1000 pps (1 KHz). The pulses may be all in one direction — uniphasic — or in both directions — biphasic.

Each pulse can also be either constant current or constant voltage. These are both consequences of the way in which resistances in the internal circuit of the machine are arranged; they diminish changes of the electrical pulse due to alterations of external resistance, such as that due to the pads or gel drying out. Where the electrodes are fixed or stationary, constant current is usually used, but if 1 electrode is moved during treatment - a dynamic application - constant voltage
is preferable. This prevents the current density from becoming uncomfortably high if the area of the pad in contact with the tissues is reduced.

**Interrupted direct current**

If the continuous unidirectional current is interrupted it gives a series of pulses or phases of unidirectional current which can be of any duration or shape, repeated at any frequency. Certain durations, shapes and frequencies have acquired particular names so that although any unidirectional pulse is an interrupted direct current (i.d.c.), the term is customarily used to describe only the longer-duration pulses.

**Long duration (of 1 ms or more)**

**Rectangular wave pulses** These are pulses of any duration between 1 and 600 ms separated by pulse intervals of anything from 1 ms to several seconds (Fig. 3.2 and Fig. 3.4). Such pulses can stimulate motor and sensory nerves and can be used to stimulate denervated muscle.

**Accommodation pulses.** Triangular, trapezoidal, sawtooth, serrate, slow-rising, shaped, selective and accommodation pulses are all synonymous terms. Again, these are relatively long-duration pulses, usually 300 to 1000 ms, separated by pulse intervals of one-half to several seconds (Fig. 3.4). These pulses are used to stimulate muscle (as opposed to nerve) tissue selectively and they are able to do so because of differences in muscle and nerve accommodation, hence the names (see Chapter 1).

**Short duration (of 1 ms or less)**

**Faradic-type pulses.** These are pulses of 0.1—1 ms with repetition rates of 30—100 Hz. With pulses repeated at 100 Hz the time period for each cycle is 10 ms, so with a 1 ms pulse the rest period is 9 ms (Fig. 3.4 and Fig. 3.5). These pulses may be unidirectional and are thus short duration i.d.c., or they may be biphasic. Pulses were originally generated by an induction coil and interrupter which, because it was an electromagnetic device, was called a faradic coil, as described in Physical Principles Explained (Low and Reed, 1994). The pulses produced were unevenly alternating biphasic (Fig. 3.5c). The effective nerve stimulus is the spike of voltage, which can be about 1 ins in duration; the rest of the pulse, having much lower voltage, does not cause nerve stimulation.

Although the alternations are uneven in shape they are identical in total charge so that no chemical changes will occur. The repetition rate was dictated by the mass and elastic properties of the mechanical interrupter and was often about 60 Hz. Faradic currents are a succession of these pulses which unmodified would produce a tetanic contraction (Fig. 3.5b). Treatment by faradic current or faradic-type pulses is often
called ‘faradism’. Stimuli used in eutrophic electrotherapy are often 0.08 ms but can be as short as 0.05 ms.

TENS. It has already been pointed out that all nerve-stimulating pulses are TENS but the term is usually restricted to pulses of relatively low intensity used to control pain. Almost all such generators are battery-operated. A variety of pulse forms are available. A few are monophasic, i.e. short pulse i.d.c., but the majority are symmetrically or asymmetrically biphasic (Fig. 3.5d and Fig. 3.4). Pulse durations, often fixed for a given source, can be any length from 0.01 to 0.4 ms. The frequency is usually variable and ranged from 2 to 200 Hz, most devices giving various
Fig. 3.5 (a) Faradic-type current; (b) unmodified faradic-type current; (c) faradic pulses of the form generated by the faradic induction coil; (d) TENS.

frequencies around 100 Hz. Voltage, and thus the applied current, can be varied but is limited to low intensities: the maximum peak current is about 100 mA. Different forms of ThNS are illustrated in Figure 3.6.

H-wave is a form of TENS with a series of exponentially decaying 20 ms pulses of varying low frequencies.
**Electroacupuncture.** Various current forms are in use. It can be a pulse consisting of a few seconds of d.c. or a form of low-frequency, high-intensity TENS.

**High-voltage galvanic stimulation (HVGS) or high-voltage pulsed galvanic stimulation (HVPGS).** This form of current was originally developed in 1945 by Haslip in the USA and called ‘Dyna-wave neuromuscular stimulation’. Later, in the mid 1970s, there was increased interest in this type of current when it became known as ‘high-voltage electrogalvanic stimulation’ or ‘high-voltage pulsed galvanic stimulation’ (HVPGS). The latter is considered the preferred name since it obviates the mistaken idea that this is a constant direct current (Newton, 1987).

The twin pulse waveform has almost instantaneous rises with exponential falls. The pair of pulses lasts for only 0.1 ms and each peak lasts for only a few microseconds; the shape and duration are normally fixed. The frequency of the double pulse can be varied, usually from 2 to 100 Hz. With such short peaks very high voltages are needed (hence the name) to provide high enough currents to stimulate nerve fibres (see Fig. 3.13). Peak currents of 2—2.5 A (Men, 1987) may
be generated during the few microseconds of peak voltage but, of course, the total average current is very low, at around 1.2—1.5 mA.

**Evenly alternating**

**Sinusoidal currents**

Sinusoidal currents are evenly alternating sine wave currents of 50Hz, the form of the UK mains current (see Fig. 3.4). This gives 100 pulses or phases in each second of 10 ms each, 50 in one direction and 50 in the other. It can be produced from the mains by reducing the voltage to 60 or 80 V with a step-down transformer.

**Diadynamic currents**

Diadynamic currents were introduced by Pierre Bernard nearly 60 years ago. They are monophasic sinusoidal currents, being rectified mains-type current Diadynamnic currents have two basic forms:

1. Half-wave rectified sinusoidal current known as MF (monoplese fixe). This consists of a series of 10 ins half sine wave-shaped pulses with 10 ins pulse intervals.
2. Full-wave rectified sinusoidal current known as OF (diphase fixe). This is a continuous series of 10 ms sinusoidal pulses resulting in a frequency of 100 Hz (Fig. 3.7). (Note that the above refers to rectified 50Hz mains current. If 60Hz mains, as in the USA, is used then the pulse lengths and intervals will be 8.333 ms.)

If these two current forms — MF and OF — are applied alternately for I s each the resulting current is called CF module (modulz de en courtes pêriodes). If two MF currents are applied so that one series of pulses occupies the pulse intervals of the other and one is of constant intensity while the other is surged, the result is called LP module (module en longues pêriodes). The length of each surge and surge interval varies with different sources but is usually 5 or 6 s (Fig. 3.7).

The physiological effects of such currents have already been considered and will obviously cause sensory and motor nerve stimulation and thus muscle contraction, as well as chemical changes due to the unidirectional current.

**Medium frequency stimulation**

Medium frequency currents have pulse repetition rates greater than 1 KHz. Because of this, each pulse of current cannot stimulate a nerve’
impulse, as succeeding pulses fall in the refractory period. The nerve membrane potential is thus maintained in a more or less steady state, as occurs with the passage of a steady unidirectional current. In order to stimulate nerve tissue, medium frequency currents must be modulated in some way.

**Medium frequency direct current**

This is referred to in Chapter 2 (p. 19).

**Rebox-type current**

The Rebox is a device that was developed in Czechoslovakia in the 1970s. There is a hand-held dispersive and current is delivered by a point-type electrode. The point electrode is made the negative pole. The current consists of unipolar rectangular pulses of between 0.05 and 0.25 ins at 3000 Hz; thus it is a medium-frequency current. The circuit also contains a microammeter and an earphone and can be linked to a small computer and printer to display a graph of current and other parameters.
‘Russian’ currents

This is an alternating sine wave of 2500 Hz applied at 50 bursts per second — i.e. 10 ms bunches of 25 cycles each — with 10 ms intervals between each burst. Since each biphasic pulse lasts only 0.4 ins it needs a relatively high current to produce adequate nerve stimulation (Alon, 1987).

Interferential currents

The principle of interferential therapy is to cause two medium-frequency currents of slightly differing frequencies to interfere with one another. Where they do so, a new resultant current is set up. The resultant amplitude at any given point is the sum of the two individual current amplitudes, so that where two peaks or two troughs coincide, they will augment each other, but where a peak or trough coincide they cancel each other out (see Fig. 3.8a).

Providing the amplitudes of the two individual currents are the same, the resultant current frequency will be the mean of the two. For example, if current A is 4000Hz and current B is 4100 Hz, the resultant current frequency will be 4050 Hz.

This resultant current varies in amplitude. The frequency with which it varies is called the amplitude modulation frequency, or beat frequency, and is equal to the difference in frequency between the two individual currents (in the above instance, 100 Hz). It is this modulation pulse that triggers the nerve impulse.

Again, if the amplitude of the two individual, medium-frequency currents is equal, the beating will be 100%, as in Figure 3.8a. This is unlikely when two currents are passing in the tissues because they will inevitably have paths of different resistance. In this case there is partial beating (see Fig. 3.8b) and the ‘modulation depth’ will not be 100%.

High-frequency currents

High-frequency currents of millions of hertz are used therapeutically but they cannot stimulate nerve or muscle, because they change too rapidly. They can be safely applied at a high current intensity to produce tissue heating (see Chapter 10).

PRODUCTION OF ELECTRICAL PULSES

Commercially available electrotherapeutic equipment is invariably advertised as being ‘new’ and innovative and considerable efforts are made to connect the trade name of the equipment to the treatment.
Many modern electrotherapeutic stimulators are able to provide a range of pulse widths, frequencies and intensifies which cannot only be separately controlled but can also be programmed (i.e. pre-set) to give a repeatable treatment. The handbooks of many stimulators set out the whole range of possible variables but it must be understood that some combinations are not feasible; pulses of 10 ins duration at 200 Hz are impossible no matter how advanced the technology!

Any report, study or account of electrotherapeutic applications should always include a description of the electrical pulses utilized, in terms of the parameters (duration, intensity, shape, polarity and frequency, as described above on p. 43), as well as the current density employed.

There are several different methods of therapeutic electrical pulse generation which have been used in the past. Faradic currents from an induction coil have already been mentioned, but modern pulse generators are based on integrated circuits of transistors, resistors and capacitors.
The pulse felt at the wrist is simply a wave of fluid, i.e. blood pressure, which is generated by the action of the heart. An electrical pulse — using the word in another context but similar sense — is produced by the same basic mechanism. As the heart contracts (ventricular systole) the pressure in the arteries rises and causes the blood to flow at a higher pressure for a time while the aortic valve is open. When it closes the pressure in the arteries falls (ventricular diastole). This cycle is repeated at a rate determined by the sinoatrial node. Thus a system exerting a pressure (voltage) causes a flow (current) which is turned on and off by a valve (switch). The time for which the pressure is exerted, i.e. heart muscle contraction, is controlled by the rhythmicity of the sinoatrial node acting as a timing device. in a modern pulse generator the mains current is applied via a transformer (see Low and Reed, 1994) to provide a suitably reduced voltage; it is rectified and smoothed. The switch is a transistor which can turn the current on or off very rapidly, and the timing device is a capacitance—resistance circuit.

A capacitance—resistance circuit is analogous to a sandglass eggtimer. If a capacitor is allowed to discharge through a resistance, the time it takes depends on both the size of the capacitor and the resistance. A large capacitor stores more electrons for a given voltage than a small one. The larger the resistance the smaller the current, for a given voltage (Ohm’s law; see Low and Reed, 1994), therefore the capacitor will take longer to discharge. Note that both current and voltage will fall exponentially (Fig. 3.9). In exactly the same way the more grains of sand the eggtimer holds, the longer they will take to pass through the narrow neck, and the smaller the neck the longer the flow of sand will take (Fig. 3.9). The importance of such circuits lies in the fact that by a suitable choice of capacitor and resistance almost any length of time for their
discharge can be arranged. Thus it is possible to time pulses of voltage to last a few microseconds or for several seconds. Not only can the voltage pulse be timed but the period when it is off can be similarly controlled, so that a train of pulses can be generated with identical pulse lengths and pulse intervals. Further, the train of pulses itself can be timed to occur in bursts or surges of pulses separated by timed rest periods.

General structure of electrical pulse generators

Pulse generators may be considered to have four Functional parts:
1 A power source; this may be from the mains supply or a battery.
2 An oscillating circuit to provide a train of pulses, as described above.
3 A modulating circuit to alter the train of pulses, perhaps splitting it up into short bursts or surging it.
4 An amplifying circuit to increase the output voltage appropriately

When power is drawn from the mains it will need to be modified. The voltage will have to be reduced to an appropriate level for the subsequent circuits and output. This is done with a transformer (see Low and Reed, 1994). and rectified by means of a diode (semiconductor diodes are usually used). With a suitable controlling circuit these could be applied as diadynamic current to a patient. If the voltage-reduced rectified mains current is applied to a ‘smoothing’ circuit (a capacitor in parallel and a series inductance) it becomes an unvarying direct current and, if regulated with a potential divider, could be applied to a patient (see Chapter 2).

Modern pulse generators go through the above steps to produce a smooth unidirectional current that can be applied to an oscillator to generate any type of current. For low-intensity TENS currents (used for patient-controlled pain modulation) the smooth d.c. can be supplied by a small battery. There is nearly always a light provided to indicate that the power circuit is on.

The oscillator to generate a train of pulses works, in principle, as a multivibrator, as described in Physical Principles Explained (mw and Reed, 1994). Such circuits are usually manufactured all in one piece as an integrated circuit which can be fitted to appropriate resistors to give the desired pulse lengths and intervals. Where these are to be varied a switch on the panel of the machine connects the necessary resistance to give the pulse length required.

The train of pulses may then be modified, surged for example, by another integrated circuit which may also be controlled by a switch on the machine. A circuit may also make the d.c. pulses biphasic. Finally the output is amplified and applied to a potential divider to regulate the output to the patient. This is illustrated in Figure 3.10.

EFFECTS OF LOW-FREQUENCY ELECTRICAL PULSES ON THE TISSUES

When currents are passed through the tissues two groups of effects can be considered:
There are clear and well documented effects on excitable tissue, that is nerve and muscle, which lead to numerous indirect effects. For example, modifying pain perception in the central nervous system or causing muscle contraction is secondary to the stimulation of the nerve fibre. There is also evidence of direct effects on these tissues affecting their growth and metabolism (eutrophic electrotherapy is considered in this chapter).

Peripheral nerves are composed of many fibres — nerve cell processes — both sensory (afferent) and motor (efferent). The motor fibres are the axons of cells in the anterior (ventral) horn of the spinal cord, hence called anterior horn cells, while the cell bodies of the sensory nerves are found in the posterior (dorsal) root ganglia. The motor nerves to skeletal muscles and the sensory nerves conveying touch and proprioception are all fast-conducting, of large diameter and myelinated (Table 3.1). The majority of fibres making up a typical peripheral nerve are of small diameter, are slow-conducting and non-myelinated; a high proportion are pain-carrying C fibres, while the others are autonomic.

Effects on non-excitable tissue at a cellular level are much less well recognized or understood. There is evidence that direct pulsed currents may accelerate healing in ski, and other tissue, as noted in Chapter 2. It has also been suggested that pulsatile currents may affect cell metabolism leading to arterial, venous and lymphatic exchange at a microcirculatory level, but there is no substantial supporting evidence (Alon, 1987).

To apply electrical pulses to the tissues a complete circuit is needed so two electrodes with suitable conducting material are fixed to the skin. The effects will be evident where the current density is highest, i.e. in the superficial tissues under the smaller (active) electrode. Consequently the cutaneous sensory nerves are affected first and with greater current.
Table 3.1 Classification of peripheral nerve fibres

<table>
<thead>
<tr>
<th>Type of sheath</th>
<th>Fibre diameter (μ)</th>
<th>Conduction speed (m/s)</th>
<th>Classification by letter</th>
<th>Classification by number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myelinated</td>
<td>22</td>
<td>120</td>
<td>Aα: Extrafusal muscle fibres</td>
<td>Ia: Primary sensory fibres of muscle spindles</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Aα: Cutaneous, joint and muscle receptors; large interoceptors</td>
<td>Ib: Sensory fibres from Golgi tendon organs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60</td>
<td>Aβ: Intrafusal muscle fibres</td>
<td>II: Secondary sensory fibres of muscle spindles</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Aβ: Low-threshold mechanoreceptors for light pressure rubbing vibration</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>30</td>
<td>Aγ: Intrafusal muscle fibres</td>
<td>III: Nociceptors pressure-pain</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>4</td>
<td>B: Preganglionic</td>
<td></td>
</tr>
<tr>
<td>Non-Meyelinated</td>
<td>0.1</td>
<td>0.5</td>
<td>C: Postganglionic</td>
<td>IV: Nociceptors</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: Slow pain polymodal nociceptors; thermoreceptors; interoceptors</td>
<td></td>
</tr>
</tbody>
</table>
densities the more deeply placed motor nerves are stimulated. However, the sensory and motor fibres are large-diameter, myelinated, fast-conducting fibres and thus more readily stimulated than the small-diameter pain fibres. If a low current density is applied to the skin the sensory nerves in the skin, which normally transmit touch, temperature and pressure, are the first to be stimulated. This causes a mild tapping sensation which may be due principally to rapidly repeated stimulation of touch receptors. Higher current densities will cause the current to affect more nerves leading to more intense tingling and will eventually spread to motor nerves causing muscle contraction. As still higher currents are applied more motor units will be affected resulting in both stronger and more widespread muscle contractions. Further increases of current will cause pain nerve fibres to be stimulated resulting in perceived pain. These three types of nerve fibres are affected in the same order with any form of stimulating pulse (see below). Clearly the positioning of the electrodes will determine the site of greatest current density and hence which nerves are affected; for example, in order to stimulate a normally innervated muscle effectively but painlessly the active electrode is applied to the motor point. This is a point on the skin surface at which maximum muscle contraction can be achieved because it is close to the point where the motor nerve trunk enters the muscle. Current applied at this point — often at the junction of the proximal third with the distal two-thirds of the muscle belly — will influence a large number of nerve fibres close together. Thus less current density will be needed than if the muscle belly were stimulated at some other place (Fig. 3.11). Notice that the phrase used above — ‘to stimulate a muscle’ — is a convenience. The current is stimulating the motor nerves which convey nerve impulses to stimulate the muscle fibres.

**Nerve stimulation by electrical pulses**

Nerve fibres in a resting state have a potential difference of some 70 mV across the fibre membrane, the inside being negative and the outside positive. The nerve impulse is an electrochemical change that spreads along the fibre, as described in Chapter 1. The impulse can be set off by depolarizing the potential difference across the membrane with an electric pulse. The nerve impulse will travel in both directions but will only cause an effect in one direction (the orthodromic direction) because it is blocked by a synapse in the other direction; but see discussion on eutrophic electrotherapy below.
This occurs because the electrical pulse causes a movement of ions through the tissues and hence across the membrane. However it must be a sufficient disturbance — beyond the threshold value of about 10 mV — to fire the nerve impulse. Once the potential across the nerve membrane is altered beyond its threshold value the full nerve impulse occurs, thus it is an all-or-none response (Fig. 3.12). What the electrical pulse does is to trigger the nerve impulse but it needs a certain minimal amount of
electrical charge to do so. This can be given by a small current for a relatively
long duration or by a larger current for a short period (Fig. 3.13). There is,
however, a certain minimum current needed to fire a nerve impulse at long
durations; it is called the rheobase. This idea is demonstrated in the strength—
duration curve shown in Figure 3.13 — shorter pulse durations need larger
currents to provoke a nerve impulse. It will be seen that as the curves are
exponential an enormous current would be needed at very short pulse lengths so
no practical nerve or muscle stimulation occurs. It must also be understood that
pulses of greater current than is needed to trigger the nerve impulse have no
further effect on that nerve fibre. No matter what the current intensity the same
nerve impulse is triggered. As explained earlier, increased sensory effects or
stronger muscle contractions are due to a larger number of fibres being
stimulated. Similarly if the electrical pulse has a longer duration no further effect
occurs, as illustrated in the strength—duration curve in Figure 3.13, in which the
same current triggers impulses at 1, 10 and 30 ms etc. It is the initial half-ms or so
of the rheobase current which has sufficient charge to disturb the nerve fibre
membrane beyond its threshold. Rather longer pulses of, say, 300 ms will provoke
two nerve impulses, one when the current rises and the other when it falls. The
reason for this is explained later.

**Rate of rise of pulse**

What has been described so far is true provided the rate of rise of the electrical
pulse is very rapid, i.e. it is a square wave pulse. If the rate of rise of the current is
very slow it will not provoke a nerve impulse because the ionic balance across the
nerve fibre membrane is able to adjust itself so that the threshold potential rises in
response to the applied electric charge. This process is called accommodation. The
rate at which accommodation can occur is limited so that the threshold may
eventually be reached by the slow rising pulses of higher currents (Fig. 3.14). This
ability to accommodate is much more marked in nerve than in muscle tissue. This
fact is used to discriminate between innervated and denervated muscle, as
explained in Chapter 4. This also explains why d.c., given for iontophoresis for
example, does not cause nerve stimulation when turned up slowly, as described in
Chapter 2: From what has been noted above it is evident that the electrical pulse
with the least charge that will stimulate a nerve impulse is one that rises rapidly —
square wave — and which is of less than 1 ms duration. Note that in Figure 3.14
the pulses are cut off because their fall would stimulate another nerve impulse.

**Stimulating different nerves**

Strength duration curves have been produced for sensory and motor nerves and
pain responses (Fig. 3.13). It will be seen that greater currents are needed for pain
fibres, less for motor nerves and less still for sensory nerves. The amplitude of
current needed to stimulate a nerve fibre is inversely proportional to its diameter.
Thus the small C fibres carrying pain impulses need the greatest current. It is not a
difference in threshold
but simply that the larger fibres have a lower electrical resistance (due to large cross-section and differing membrane characteristics), allowing a larger current for any given voltage (Ohm’s law; see Appendix A). The difference in sensitivity between motor and sensory fibres is due to their different depths; sensory nerves in the skin receive a higher current density, as explained earlier.

It can also be seen from the strength—duration curves that this separation is greatest at short pulse widths. It is therefore easier selectively to excite motor or sensory nerves without eliciting pain by short-duration pulses, say around 0.05 ms.

**Refractory periods**

The foregoing relates to single electrical pulses causing single nerve impulses. It will be recalled from Chapter 1 that once the nerve impulse has occurred, charging the membrane potential to +34JV, it returns to its resting value in about 0.4—1 ms for A fibres and 2 ms for C fibres. During this time, which is called the absolute refractory period, no stimulus, however large, will cause another nerve impulse. During the next 10—15 ms the nerve impulse can be triggered again but only by a larger stimulus than is normally needed. This is called the relative
Fig. 3.16 Strength of tetanic contraction increasing with rising frequency up to 100 Hz.

refractory period. After this the nerve is in its normal resting state (Fig. 3.15).

These facts have important implications for the frequency of electrical pulses used to stimulate nerves. If a series of electrical pulses is applied to a motor nerve at one pulse per second (1 Hz), a corresponding series of muscle twitches will occur. Similarly, stimulation of a sensory nerve will lead to a series of separately recognized mild shocks. If the frequency is increased to, say, 10 Hz there is a corresponding tremor of the muscle, but if the frequency is increased to, say, 50 Hz the muscle contract continuously — a tetanic contraction. Although the peak current remains the same the strength of the tetanic contraction increases with rising frequency up to about 100 Hz but not beyond. This is illustrated in Figure 3.16. The increase in muscle force occurs because the tension developed during one twitch has no time to relax before the next occurs, so that
successive twitches are cumulative. Beyond 100 Hz the muscle conraction and sensory tingling do not increase with increased pulse frequency; in fact they may diminish unless the current intensity is increased. The reasons for this are obvious from what has been described already. Pulses of 1 ms at 100 Hz will have pulse intervals of 9 ms and at higher frequencies the interval would be shorter still; this means that each pulse is applied during the relative refractory period. In order to excite a nerve impulse during this time a greater current is needed. Thus higher peak currents are necessary to generate impulses at frequencies above 100Hz. If shorter pulse durations are used, say 0.05 ms, to leave a slightly longer pulse interval, then the current must again be increased in order to provide sufficient charge to excite the nerve impulse (Fig. 3.12).

At higher frequencies above 500 Hz for 1 ms pulses, or about 1000 Hz (1 KHz) for 0.05 ms pulses, no increase in the number of nerve impulses can occur no matter how much current is applied, because succeeding electrical pulses would be given during the absolute refractory period. This is sometimes called the maximum depolarization frequency. Above this stimulation frequency the nerve depolarizes at its own frequency. Single pulses of 0.225 ms separated by intervals long enough to allow the nerve to fire and repolarize (i.e. faradic etc.) will cause nerve stimulation. However, if the pulses are applied at 4000 Hz (i.e. medium frequency) they can have no effect on nerve if a steady current is applied. Rapid variation is equivalent to no current, because polarity changes faster than ionic movement can occur. If unidirectional, pulses of 4000 Hz are the same as steady d.c. (e.g. rebox). Therefore they must be modulated in some way to cause nerve stimulation. They can be interrupted, like Russian current, by 50 bursts per second, where the first phase of each burst depolarizes the nerve, or modulated by interference providing amplitude modulation at the beat frequency.

Thus it can be seen that electrical pulses of 0.1—1 ms at 60—100 Hz would stimulate medium and large myelinated nerve fibres with the least possible current. It is notable that these parameters partly encompass both TENS (sensory) and faradic (motor) stimulation. However, the current density in the deeper tissues must also be considered.

Fast twitch muscle fibres, which are phasic and are recruited for added muscle strength and fast movement, respond best to frequencies in the range 50-450 Hz. Slow twitch fibres, which are postural and the first to become active, have a tetanic frequency of 20—30 Hz.

Penetration of electrical pulses through the tissues

The impedance of the skin is very large for both direct current and the longer pulses — much greater than the rest of the tissues — but it is very much less for shorter pulses. Thus the skin may have an impedance of about 1000(1 for a 100ms pulse but only 50(1 for a 0.1xns pulse. This happens because the skin acts as a capacitor which offers much less impedance to short pulse lengths in the same way that it does for high- frequency currents. The distribution of current through the tissues with shorter pulses is therefore more even, so that effectively the current penetrates further.
Consequently the deeper nerves, e.g. motor nerves, are more easily stimulated with the shorter pulses. With longer pulses most of the current is ‘used’ in the skin, stimulating cutaneous nerves.

**Electrical pulses for nerve stimulation**

It can be seen that some of the currents used for stimulation and described earlier are more appropriate to excite a nerve impulse than others. Thus for stimulating motor and sensory nerves, i.e. large-diameter fast-conducting nerves, the pulses should be square wave and of short duration (0.5—0.1 ms). To achieve discrimination from path nerves and greater depth even shorter pulses (0.05—0.02 ms) are appropriate. Evenly alternating pulses have the advantage of avoiding any risk of chemical damage. They are often referred to as depolarized pulses. Frequencies of around 80—100 Hz seem to be the best.

For other purposes, e.g. stimulating superficial pain nerves, longer pulses of around 10 ms would seem appropriate. Similarly if polar effects are needed to promote wound healing obviously direct current pulses are chosen. It must be understood that electrical stimulation has a multiplicity of effects which are not limited to stimulation of the alpha motor neurons and group la fibres only.

**Secondary effects of nerve stimulation**

Therapeutic electrical stimulation of both sensory and motor nerves has been widely used for many years. The principal purposes have been to cause contraction of healthy muscle by stimulating motor nerves and to control pain by stimulating sensory nerves. Various effects have also been claimed as a result of stimulating the autonomic nervous system.

**PHYSIOLOGICAL EFFECTS AND THERAPEUTIC USES**

**The motor unit**

The motor unit consists of an anterior horn cell, the alpha motor neuron emanating from it and all the individual muscle fibres it supplies (see Fig. 4.2). The number of muscle fibres supplied varies from a very few where precise control of movement is required up to one or two thousand for large postural muscles. All the muscle fibres of a particular motor unit are the same type.

Type I are slow twitch, red because they are highly vascular, and predominate in postural muscles. They are slow to contract and relax because the motor neuron supplying them is of small diameter and low conduction velocity and fires at a continuous, low frequency. They have many oxidative enzymes and fatigue slowly.

Type II are fast twitch, white, glycolytic fibres. They are less vascular. The motor neuron supplying them is of larger diameter and has a higher conduction rate. They are divided into two subgroups. Type IIA have fewer oxidative enzymes than type I, but are relatively fatigue resistant. They predominate in
ordinary low force movement. Type IIB have the least oxidative enzymes and fatigue rapidly. They produce a large force for short periods and are only brought into play during strenuous movement.

An individual muscle is made up of many motor units of different types, giving the muscle its particular characteristics.

**Voluntary contraction of muscle**

During voluntary contraction of muscle there is an asynchronous firing of motor neurons resulting in a smooth contraction. The force of a contraction is graded, in general, by an increase in: (a) the number of motor units recruited (spatial summation) and (b) the frequency of nerve impulses (temporal summation); (a) occurs in the early stages and in low force muscle contractions and (b) occurs later and at greater muscle force. In other words, as more motor units become involved, further increase of muscle force is largely achieved by increased rates of nerve impulse firing (Mimer-Brown and Stein, 1975).

Type I muscle fibres are recruited first and later type II. Prolonged muscle contraction leads to fatigue, rapidly in type JIB and most slowly in type I. The order of recruitment is largely fixed but can be influenced by cutaneous stimulation (Gamett and Stephens, 1981).

**Electrical stimulation of innervated muscle**

Electrical stimulation of muscle differs from voluntary contraction in several ways. First, there is synchronous firing of all motor neurons stimulated. Secondly, electrical stimulation will not stimulate motor units in the same recruitment order as voluntary contraction. In fact, it is largely reversed because: (a) larger diameter motor neurons (type II) are more easily stimulated, and (b) sensory nerves are inevitably stimulated. Additionally, the frequency of firing is fixed, unlike voluntary contraction. Therefore, in order to cause stronger muscle contractions the current density has to be increased to stimulate more motor units.

Electrical stimulation of muscle via its motor nerve has both immediate and long-term effects. Muscle contraction and vascular changes are examples of the former, while muscle strengthening and structural changes in muscle fibres may ultimately result from long-term, chronic stimulation. These are considered below.

The structure of living muscle is not fixed. There is, for example, a balance between the synthesis and breakdown of the constituent proteins. The rate of this can be as much as 10% of skeletal muscle protein per day, occurring at a higher rate in type I slow twitch fibres than in type II. More anabolic than catabolic activity will lead to muscle hypertrophy with more muscle and collagenous tissue produced, while the reverse will cause atrophy. Similarly, the form or structure of the constituent muscle fibres can alter in response to changes in long-term stimulation. This plastic adaptation of skeletal muscle tissue is considered to be under both hormonal (e.g. steroidal) and neuronal control. Thus the flow of impulses in the motor nerve serves both to provoke immediate muscle contraction
and, in the long-term, to promote muscle fibre growth and change, as described on p. 69.

Where voluntary active exercise is restricted, electrical stimulation may be substituted. It is usually applied by surging (or ramping) a series of short pulses at frequencies around 50-400 Hz, i.e. a faradic-type current. If the surge of pulses is made to last for, say, 2s and the interval between surges for 4s then a slow physiological muscle contraction and joint motion will be mimicked. Obviously the surge length, rate of rise and fall and interval can be varied (Fig. 33e) Electrical stimulation is used for a variety of therapeutic purposes which may be grouped as follows.

**Muscle strengthening**

The question of whether electrical stimulation of normal muscle can lead to an increase in muscle strength is not entirely resolved in spite of much research. The gist of this seems to be that electrical stimulation does increase muscle strength although not quite to the same extent as the equivalent voluntary exercise. In an extensive review Lloyd et al. (1986) concluded that in general electrical stimulation was not a satisfactory substitute for voluntary activity. However, a number of the studies showed that electrical stimulation (or electrical stimulation combined with voluntary activity) led to similar or in a few cases even greater strength gains than that due to voluntary exercise alone. Taking a number of such studies together, the average gain in strength due to electrical stimulation would seem to be around 20% over a month or so (see Balogun et al., 1993). Some studies showed marked differences in the response to electrical stimulation of different individuals but there is still much uncertainty, partly due to the variety of techniques and differing protocols adopted by the different studies.

In a well controlled study by Hon Sun Lai et al. (1988) it was shown that electrical stimulation of muscles over a 3-week period produced significant gains in muscle strength, being greater in the group treated with high-intensity electrical stimulation than in the group treated with lower intensities. The force of isometric contraction showed greater gains than that of concentric contraction. Eccentric contraction (isotonic lengthening) showed no significant gains. Although the gain in strength declined when treatment stopped it was still significant for the high-intensity group 3 weeks later. There was also a clear increase in isometric strength demonstrated in the opposite untreated limb, a cross-transfer effect which did not show a marked difference between the high- and low-intensity groups. This cross-transfer effect was also found in a study by Balogun et al. (1993) in which a 24% strength increase occurred in the treated muscles and a 10% increase in the untreated, contralateral muscles after 6 weeks. This study compared groups treated with 3 different stimulation frequencies (20, 45 and 80 pulses/sec), and found no difference.

It is considered that the strength gain can be attributed to neural mechanisms, at least initially. This is suggested by several features: the speed with which the increase occurs - it can be demonstrated in about a week - and the speed with which it can decline, as well as the lack of evidence of any changes in muscle volume. Several neural mechanisms have been proposed. One is the increased activation of the spinal motor neuron pools, which regulate the force of muscle
contraction due to stimulation of afferent neurons. This would account for the
cross-transfer effect. Long-term potentiation has also been suggested. This
involves increased sensitivity of synapses as a result of continuous stimulation of
input fibres; the effect may last for some weeks. Synchronization of motor unit
firing patterns is a further mechanism that has been proposed. The selective
recruitment of large fast-twitch type II fibres over the slow-twitch type I fibres
could also be implicated.

The force of voluntary muscle contraction is greater in most but not all
subjects than the force that can be produced by electrical stimulation of the same
musculature (IDe Domenico and Strauss, 1986; Strauss and IDe Dornenico,
1986). This difference does not seem to be accounted for by the nature of the
stimulating current since different stimulators did not produce significantly
consistent differences in contraction force.

In weakened or weakening muscles the value of electrical stimulation is much
clearer and significant gains have been reported with improvement of muscle
function. Electrical stimulation at 30Hz applied to the quadriceps of immobilized
knees and given in 2s on and 9s off cycles for 1 h each day for 6 weeks has been
shown to reduce muscle atrophy (Gibson et al., 1988). In this study, cross-
sectional area of the quadriceps was found to diminish by 17% in the untreated
group, but there was no significant loss in those patients that were treated. The
effect was considered to be due to the maintenance of protein synthesis in muscles
rather than preventing protein breakdown. Obajulwa (1991) found a significant
increase in mean quadriceps muscle girth after using a surged faradic pulse train
of 3s at a maximum tetanic contraction level, but within the limits of tolerance. The
surge was repeated 10 times, with a rest period of 10s between each. This
regimen was repeated 3 times a week for 10 weeks. Similarly, improvement in
muscle force over a 4-week period of electrical stimulation of chronically
weakened quadriceps was found (Singer, 1986). In this study, however, no
significant increase in muscle cross-sectional area was found but electromyograph
changes suggested increased neuromuscular efficiency to account for the
increased muscle force. Soo et al. (1988) have shown that stimulating the
quadriceps at quite low intensities — 50% of maximum voluntary contraction
applied as eight contractions of 155 duration twice a week for 5 weeks — led to a
statistically significant increase in quadriceps torque. Thus surprisingly little
electrical stimulation, a total of only 2 miii stimulation in each of 10 sessions, has
shov.’n significant effect.

A faradic-type current has been used successfully in the treatment of
chondromalacia patellae Uohnson et al., 1977). Fifty patients given 19 treatments
over a 6-week period showed considerable improvement; at least half became
symptom free. The quadriceps muscle was stimulated to produce 1Os isometric
contractions with a 1Os rest period ten times every treatment. The current
intensity was the maximum the patient could tolerate; in fact the authors
concluded that the efficacy of treatment varies directly with the current intensity -
highest currents give the best results. They also noted that the greater the initial
atrophy the more effective the treatment and consequently felt that nonnal muscle
would gain least from the technique. The body of evidence points to the need for
maximum tolerable levels of contraction for strengthening. However, Snyder-
Mackler and Robinson (1989) warn that, after the motor threshold is exceeded,
very small increases in stimulation amplitude produce relatively large increases in
the force of muscle contraction as recruitment increases rapidly, so care must be
exercised

The strengthening effect has been used to promote greater achievement in
athletics but any advantage this might have over similar amounts of voluntary
effort has not been unequivocally demonstrated. Its use for the prevention of
disuse atrophy appears to be justified; (or a full discussion see Currier (1987).

Interestingly, behavioural styles appear to affect how subjects characterize
discomfort with electrical stimulation (Delitto et al., 1992). They felt that
electrically elicited muscle contractions themselves contributed to the discomfort.

**Facilitation of muscle control**

Stimulation is extensively used therapeutically to initiate and facilitate voluntary contraction of muscle, although it is not possible to distinguish this effect from the strengthening effect already considered. This idea may be applied in several circumstances:

1. Where voluntary muscle contraction is inhibited by pain or injury. Stimulating the quadriceps, especially the vastus medialis, after knee surgery or knee injury, for example, is often utilized (Eriksson and I-laggmark, 1979). Another example is stimulation of the calf and Achilles tendon in chronic and postsurgical cases of Achilles tendon injuries (Grisogono, 1989).

2. In situations where muscle action is not readily under voluntary control without practice: stimulation of the pelvic floor muscle in the control of incontinence (see Cawley and Hendriks, 1992; Blowman et al., 1991; Mills et al., 1990); stimulation of the abductor hallucis for the management of early hallux valgus (see below for further discussion of both of these); and, in circumstances such as postural flatfoot or metatarsalgia, where voluntary lumbrical control is desirable.

3. In circumstances in which a new muscle action has to be learned, for example where a muscle or a motor nerve has been transplanted.

4. In the later stages of a recovering peripheral nerve lesion to encourage voluntary muscle contraction where reinnervation has only recently occurred.

5. In situations in which it is necessary to demonstrate to the patient that a particular muscle action or movement can occur normally, where hysterical paralysis is present, for example.

6. For children with cerebral palsy, where electrical stimulation may enhance muscle contraction and provide sensation so that a child can add a weak response with effective results (Carmick, 1993).
Maintenance or increase of range of joint motion

The motion may be limited by different tissues and from different causes. Electrical stimulation of muscle to stretch the shortened tissues has been used in:

1 Contractures of fibrous tissue and scarring. Limitation of joint motion due to shortening of soft tissues on one side of the joint has been treated by cyclical electrical stimulation of the muscles that stretch the contracture. This has been successful in increasing the range of movement in hemiplegic patients (Baker and Parker, 1986), especially for the patient with shoulder pain and for the reduction of shoulder subluxation.

2 Loss of motion due to spasticity of muscles in hemiplegia or other neurological conditions. To maintain the normal range of motion in such patients regular passive movements are recommended, often carded out at home by the patient’s own family. Electrical stimulation has been applied as an alternative to manual passive movement to help prevent the loss of motion due to spasticity of the opposing muscles. This has been used successfully on the wrist and in finger movements of hemiplegic patients (Baker et al., 1979).

3 Scoliosis. In the treatment of scoliosis the lateral trunk muscles on the convexity of the curve are stimulated electrically. Surface electrodes (carbon rubber) are attached to the patient’s back and muscle contraction is provoked in short cycles at a level that allows the patient to sleep during the treatment (Eckerson and Axelgaard, 1984). In moderate scoliosis (20—45°) it has been shown that a progression of the curve can be halted in over 80% of patients (Axelgaard and Brown, 1983). A later overview of scoliosis treatment (Aebi, 1991) concluded that electrical stimulation is unsuccessful in altering the natural course of idiopathic scoliosis. Similarly, Cassella and Hall (1991) found over half the subjects treated with nightly electrical stimulation were not benefited.

Effects on muscle metabolism and blood flow

Electrical stimulation will have the same effect as normal voluntary muscle contraction in causing a temporary increase in muscle metabolism. There will be the associated consequences of increased oxygen uptake and carbon dioxide, lactic acid and other metabolite production, as well as raised local temperature and greater local blood flow. Many studies have demonstrated an increased blood flow, for example Currier et al. (1986). Using 10 and 30% of maximum voluntary contraction these authors quantified a 20% blood flow increase which occurred about 1 mm after electrical stimulation had started and continued for some 5 mm after it had finished.

Not only is the intramuscular blood flow increased but as a consequence of regular muscle contraction and relaxation the flow in adjacent soft- walled veins will be increased — the muscle pumping action. This effect is used therapeutically to help control limb oedema by raising the rate of flow in venous and lymphatic vessels. It has been found that stimulating parts of the quadriceps with 0.4 ms pulses at 50 Hz in 45 on/4 s off cycles leads to art 18.5% increase in blood flow in the femoral artery (Tracy et al., 1988). This study used sufficient current to
stimulate the muscle to 15% of its maximum voluntary contraction and measured the blood flow in the femoral artery with an ultrasonic Doppler device. The increased blood flow was noted within 5 mm of the start of electrical stimulation and fell to normal levels within 1 mm of cessation of stimulation.

Fatigue of muscle

Musch fatigue as a consequence of voluntary contraction is well-known but it is a complex and not fully understood phenomenon. Initially it is due to depletion of muscle glycogen and available blood glucose with other biochemical limitations. Ultimately the rate of oxygen utilization is important. Fatigue at submaximal contractions is controlled by varying the particular motor units involved. Prolonged contraction shows increased recruitment of motor units to maintain the same muscle force as fatigue occurs (Berger, 1982). It would therefore be expected that electrical stimulation of muscle via the motor nerve would lead to relatively rapid muscle fatigue, since a fixed set of motor units are being stimulated with the fast twitch type II fibres preferentially selected. This has been shown to occur by Currier and Mann (1983) and Rankin and Stokes (1992). The former authors showed that muscle fatigue due to electrical stimulation was greater than that due to isometric, voluntary contraction of equal force. The degree and duration of fatigue appears to be directly related to the extent of the electrical stimulation. Rankin and Stokes found some evidence of fatigue persisting for surprisingly long periods after a therapeutic protocol of electrical stimulation applied to healthy subjects. Full recovery was delayed for up to 24 h and, in some cases, 48 h.

Fatigue after exercise, including electrically induced exercise, may be a necessary stimulus for muscle strengthening, but whether stimulation of already fatigued muscle could be harmful is not known. The possible risks due to functional electrical stimulation (see p. 71) have also been considered by Stokes and Cooper (1989) but there seems to be no evidence of any structural or functional damage due to electrical stimulation.

Changes in the structure and properties of muscle: long-term electrical stimulation

The ability to modify the properties of mammalian skeletal muscle by means of long-term electrical stimulation was first demonstrated by Buller and colleagues in 1960. By reversing the nerve supply between a predominantly fast contracting muscle with that of a slow contracting one in the cat, it was shown that not only the contractile properties were exchanged but extensive sequential changes also occurred in the metabolic and histological properties. Thus fast twitch type II fibres were converted to slow twitch type I. Similar changes occurred when animal muscle was subjected to long-term, low-frequency stimulation (Salmons and Vbrova, 1969). Other animal studies have shown that the transformation process takes some 6—8 weeks and is sequential, starting with changes in the muscle membrane and capillary circulation and completed with an exchange of fast to slow type myosin isoenzymes (Pette and Vbrova, 1985). Although there is
overwhelming evidence that variation from the usual neuronal activity is a key factor in provoking these changes, and that the nature of these variations differs between muscles, it is not known how best to effect and exploit this ability to alter muscle properties.

These effects have also been demonstrated in humans (Scott et al., 1985). In this study, low-frequency (10Hz) stimulation of the lateral popliteal nerve was given for 3 periods of an hour daily for 6 weeks, at sufficient intensity to give a visible contraction of the tibialis anterior and movement of the foot. This led to a significant increase in resistance to fatigue in the stimulated muscles when compared with the unstimulated controls, suggesting a change in the properties of the type II fibres.

Later work by Rutherford et al. (1988) on normal subjects compared the effect of long-term, tow-frequency stimulation with a non-uniform pattern of stimulation incorporating a range from 5Hz through to 40Hz. This study showed similar changes in the fatigue characteristics in response to both patterns of stimulation. However, muscles stimulated with a low-frequency pattern lost muscle strength, whereas muscles stimulated using a mixed pattern of stimulation became stronger.

More recent studies have investigated both maximum voluntary strength and changes in contractile characteristics of various groups of normal subjects ranging from the very young to active elderly subjects. Comparative studies have also been undertaken to monitor these changes in patient groups with neuromuscular disease (for a review see St Pierre and Gardiner, 1987). Overall, it would appear that changes in contractile properties may often accompany loss of strength and alterations in neuronal activity.

The frequencies of the motor unit action potential have been recorded electromyographically and used to determine the frequency of electrical stimulation of the muscle (Kidd and Oldham, 1988a,b). This form of stimulation has been called eutrophic electrotherapy to indicate its growth promoting or nourishing effect. The application of eutrophic electrotherapy to the intrinsic hand muscles of patients with rheumatoid arthritis has been reported (Kidd et al., 1989) in which the patterned stimulation was derived from a fatigued motor unit of the first dorsal interosseous muscle of a normal hand. Significant improvements in functional ability and endurance were reported. Similar techniques have been applied to the quadriceps muscles of elderly subjects with disuse atrophy (T. E. Howe, personal communication) with less obvious benefit, although the patterned stimulation appeared to be more effective than conventional uniform frequency stimulation in restoring function in some patients. Eutrophic electrotherapy has been applied in cases of non-recovering Bell’s palsy (Farragher et al., 1987). The mean frequency of motor unit firing in particular facial muscles was used to dictate the frequency (5—8 Hz) of 0.08 ms rectangular pulses applied to the facial nerve, with 2s on and 2s off, for up to 8 h over several weeks. Very considerable objectively assessed improvement was noted. Stress incontinence has also been treated with eutrophic electrotherapy (see p. 93).

In summary, the motor nerve apparently sends two types of encoded information to muscle fibres. One type causes immediate muscle contraction, the other is trophic, causing adaptation over a long period of time and is non-uniform and patterned. By stimulating muscle appropriately with chronic, low-frequency electrical pulses, adaptation can occur with a resultant increase in
strength and endurance. There is considerable uncertainty concerning the optimum patterns of stimulation and this may well be the key factor. There are, however, other features of this therapy, such as the effect of loading and normal use of the muscle during stimulation, as well as patient compliance and acceptability (Baker, 1987) which need to be resolved (see Ciba Foundation, 1988).

**Functional electrical stimulation**

Functional electrical stimulation is the electrical stimulation of the intact lower motor neuron to initiate contraction of paralysed muscle to produce functional movement.

**Electrical stimulation to replace splinting.** This involves the use of faradic-type or similar electrical pulses applied to the skin to cause muscle contraction. These systems encompass both control and rehabilitation of the neuromuscular complex in that strengthening occurs when muscle is regularly and repeatedly stimulated as considered above. There may also be a beneficial effect on muscle spasm (see below). Electrical stimulation of the dorsiflexors in hemiplegic patients triggered by a switch in the shoe has been used. Similarly stimulation of the deltoid has been used to prevent glenohumeral subluxation in hemiplegic patients (Baker, 1987). Rather more complex systems are being devised to enable paraplegic patients to gain control of standing and walking (see Ferguson and Granat, 1992; Isakov and Mizrahi, 1993; Barr et al., 1989).

**Electrical stimulation for the control of spasticity.** The effects of electrical muscle stimulation on spasticity are not clearly established and reported results are variable. This is partly due to the difficulty of measuring and defining spasticity. In general there have been three approaches: first, stimulation of antagonists to utilize the effect of reciprocal inhibition, secondly, stimulation of the spastic muscles themselves, and thirdly, alternately stimulating agonist and antagonist muscles. This latter approach has been tried using low-frequency (3—35 Hz) 0.2 ms pulses, for some minutes daily over several weeks. The pulse frequencies are based on eutrophic stimulation as considered above and on the principle that afferent stimulation reinforces the presynaptic inhibition of motor neurons, thus reducing spasticity. Although some success has been claimed (Shindo and Jones, 1987) a later similar single-blind trial found only short-lived subjective improvement in spasticity (Livesley, 1992). Studies on the other approaches are also inconsistent. However, none appear to support the opinion, sometimes expressed, that spasticity may be exacerbated by electrical stimulation (Baker, 1987). Lagassé and Roy (1989) studied the effects of a functional electrical stimulation training programme on the co-contraction level of spastic hemiparetic patients during a maximal speed forearm extension movement. The pattern of electrical stimulation was adjusted individually from electromyograph parameters of the non-affected limb. This treatment reduced the antagonist co-contraction level associated with spasticity.
Stimulation of denervated muscle

Denervated muscle is different in many respects from innervated muscle, including its response to electrical stimuli. Without a functional nerve supply muscle can only be caused to contract by direct stimulation of the muscle fibre. There are therefore differences between stimulating muscle via its nerve and direct denervated muscle stimulation.

1. Muscle tissue is less excitable than nerve so that a greater electric charge is needed. This is evident from the strength—duration curve for denervated muscle (Fig. 4.3). Thus a square wave pulse of suitable current intensity will stimulate denervated muscle to contract provided the pulse is of sufficient duration, i.e. more than 30 ms. Greater currents will be needed for shorter pulses to provoke a muscle contraction.

2. When a bundle of motor nerves is stimulated at the motor point it causes the simultaneous stimulation of many motor units, each of which activates many muscle fibres thus causing the synchronous contraction of a large part of the muscle. This is evident as a brisk twitch, or a series of twitches, or a tetanic contraction if the frequency is high enough. If there is no nerve the individual muscle fibres are stimulated when the current density across them reaches sufficient intensity so that the contraction spreads slowly through the muscle. Furthermore, the rate of contraction and relaxation of denervated muscle fibres is slower than that of normal muscle. Both these effects contribute to the different quality of contraction which is sometimes called a ‘worm-like’ contraction.

3. It has already been noted that nerve is able to accommodate to ionic changes across its membrane provided those changes are not too rapid. Muscle tissues have less ability to accommodate than nerve so that quite slow changes can stimulate the muscle fibre, as described below. This property provides a means of selectively stimulating muscle, as opposed to nerve tissue, by means of slow-rising triangular pulses.

If a rectangular wave pulse is applied to a nerve fibre an impulse is evoked at a certain current intensity, the rheobase. The term ‘rheobase’ is defined as the minimum current needed to stimulate a nerve or muscle using a pulse of infinite duration. The rate of rise of an electrical pulse can be described in terms of the time that it takes to reach the rheobase current for a square wave pulse. (Square wave pulses rise very rapidly and hence reach the rheobase almost instantly.) If a triangular pulse is applied, taking 10 ms to reach the same current intensity, a very slight increase in current is needed to cause the nerve impulse because the nerve impulse threshold has had little time to change. A pulse taking 20 ms to reach the rheobase for a rectangular wave would need a greater intensity to evoke the impulse and one taking 30 ms would need a current nearly twice the rheobase before it ‘caught up’ with the threshold to trigger an impulse. Clearly, if the rate of rise is made just a little longer (33 ms to rectangular wave rheobase) the current can rise to a very high intensity without catching the rising threshold and hence without stimulating the nerve (Stephens, 1965). This rate of rise has been called the ‘liminal current gradient’. Slower rates of rise will be unable
Fig. 3.17 The liminal current gradient.

Fig. 3.18 The effect of a 600 ms triangular pulse on excitable tissue thresholds.

to stimulate nerve. The idea is shown in Figure 3.17 (see also Fig. 3.14).

The liminal current gradient typical of a nerve can thus be described as taking about 33 ms to the square wave rheobase. If the rheobase is 10 mA, this means the current takes 33 nis to reach that intensity or 300 mA in 1 s (30 rheobases). The gradient for muscle fibre which is normally innervated is less steep, taking about 100 ms to reach the rheobase, which is 100 mA/s. The gradient for denervated muscle fibre is very much less steep, being 300 ms to rheobase or 30 mA/s. Note that this refers to the rate of rise to the square wave rheobase not the pulse length, which is longer (see Fig. 3.17). Note also that, for a given pulse length, the gradient, of course, depends on the intensity of current. As this increases it successively cuts through the different liminal current gradients (see Fig. 3.18).

This provides a useful way of selectively stimulating different tissues. To stimulate nerve, as previously discussed, a square wave pulse is best.
To stimulate muscle fibre but not nerve, a triangular pulse with a rise time of 50 ins (100 ins pulse length) is used. To stimulate denervated muscle fibre only, a rise time of longer than 100 ms is best; 300 or 500 ms triangular pulses are often selected. In practice, the optimum pulse length is found by selecting the one which requires the least current amplitude to produce a contraction.

From the above it is evident that denervated muscle can be made to contract with square wave pulses of sufficient duration (30 ins or more) or triangular wave pulses of long duration (rise times of 100—500 ms). In both cases the current needs to be applied through the muscle tissue itself, because there is no motor point. The current is usually most successfully applied in the long axis of the muscle fibres, that is with the stimulating electrodes at each end of the muscle belly.

Since such slowrising pulses will selectively stimulate muscle tissue, but not its motor nerve supply, it may be asked what effect these pulses have on sensory nerves. Accommodation is most evident in large diameter, rapidly conducting nerve fibres, such as A alpha motor and sensory nerves and less so in the small diameter A delta and C pain fibres. Very high-intensity pulses may stimulate these fibres. Furthermore such long (300—500ms) unidirectional pulses provoke chemical changes in the tissues, as noted in Chapter 2, detected by sensory nerves if of sufficient intensity. This accounts for the curious stinging sensation experienced when such pulses are applied at high intensity to normally innervated skin.

There has been confusion and controversy over the therapeutic use of electrical stimulation of denervated muscles for many years. The rationale for such treatment is to maintain the muscle in as healthy a state as possible by electrically induced artificial exercise while awaiting reinnervation. It seems reasonable that making muscles contract with electrical stimuli would substitute for the beneficial effects of normal muscle contraction. Newham (1991) points out that skeletal muscle has a great capacity for regeneration. For this to occur with the restoration of normal function it is essential that the blood and nerve supply are maintained and that tissue loss is not too extensive. The evidence, however, is somewhat contradictory and in particular clinical benefit has not been unequivocally demonstrated in humans (see Bdlanger, 1991).

When muscle is denervated many structural and functional changes occur. Firstly there is an immediate loss of all voluntary and reflex activity. Secondly there is atrophy, degeneration and fibrosis progressing over weeks and months. There is a rapid loss of muscle weight during the first few months which slows to become almost steady later. The actual loss of muscle fibre is even greater because there may be an increase in connective tissue during atrophy (Sunderland, 1978). The question of when and why denervated muscles fibrose is of utmost importance because ultimate reinnervation is of no use if there is no muscle tissue left. There seems to be no definitive answer but in humans degeneration of muscle does not seem to be complete for some 3 years after the nerve lesion. It has been suggested that prolonged intramuscular stasis and perhaps thrombosis due to vasoconstriction paralysis and loss of muscle pumping action lead to deficient muscle nutrition and hence
degeneration (Sunderland, 1978). In addition, trauma — excessive stretching, heat and cold — has been implicated in accelerating the degenerative processes of denervated muscle. (Innervated muscle seems better able to repair itself.) This suggests that measures to diminish oedema and prevent injury will benefit denervated muscles.

In addition to these changes fibrillation occurs. This is the spontaneous contraction of individual muscle fibres in an irregular pattern. It occurs intermittently during denervation and can usually be detected by electromyography.

There is considerable literature concerning the effects of electrical stimulation on denervated muscle (Spietholz, 1987). In humans there would be no point treating with electrical stimulation the muscles paralysed due to a neurapraxia which will recover before significant muscle atrophy can occur or in any circumstances in which recovery will occur in a few months for the same reason.

There is evidence that electrical stimulation will retard muscle atrophy and degeneration but not completely prevent it. The type and amount of stimulation used to achieve this is very variable. The best results (Hnik, 1962) seem to have been achieved with vigorous isometric muscle contractions — to the point of fatigue — for two or three sessions each day separated by at least 10 mm intervals. Davis (1983) concluded from a review of the literature that all the denervated muscle fibres must be activated, that isometric contractions are more effective than isotonic and that regular stimulation should commence as soon as possible after denervation. From a therapeutic point of view such regimens are difficult to apply except to a few superficial muscles at a time. This, then, tends to be limited to a small number of muscles and to those that patients can be taught to stimulate for themselves. Further, if this treatment is to be useful it will require considerable compliance and tolerance on the part of the patient to continue treatment over a long period of, perhaps, 1 or 2 years. Some clinical studies have been unable to demonstrate any benefits due to the application of electrical stimulation over long periods provided other appropriate care is given to the paralysed muscles. In summary, the value of electrical stimulation for denervated muscle is not proven and its application to gain what may be only a small benefit is often not justified.

The contradictory experimental findings may be due to variations in the type of electrical stimulation — the parameters of frequency, rise time, intensity and so forth — or the varying amounts of stimulation given or, perhaps, it is the trophic effects that are important. In view of this and the availability of conveniently small muscle stimulators that can be used by the patient, the benefit of denervated muscle stimulation may well be demonstrated in the future; see also eutrophic electrotherapy, which is probably the treatment of choice if some motor units are still available for stimulation.

**Stimulation of afferent nerves**

Electrical stimulation is extensively used for the control of pain. Although the idea had been proposed for many years the rationale was provided
by the gate control theory of pain proposed in the mid 1960s by Meizack and Wall (1965). To account for the effects of electrical stimulation it is necessary to consider pain mechanisms first.

**Pain**

All sensations recognized at a conscious level can be altered or modulated by the central nervous system. This is true of path. Two distinct kinds of pain may be described. Fast pain is usually equated with acute path. Receptors, present in the skin all over the body surface, respond to the noxious stimulus and the impulse is carried by small myelinated A delta fibres (Table 3.1). Fast pain seems to be functionally concerned with helping the bodS’ avoid tissue damage since it provokes an immediate flexor withdrawal reflex and evokes rapid well localized conscious awareness. Slow pain, or second pain, on the other hand is equated with chronic pain and results from tissue damage. It is carried by unmyelinated C fibres (Table 3.1), whose free nerve endings are found in all innervated body tissue except the central nervous system. Functionally it appears to enforce inactivity of damaged tissue in order to allow healing to occur. It is thus associated with muscle spasm. Both these types of path are due to stimulation of peripheral receptors and seem to have clear physiological functions. A third type, neurogenic pain, is entirely different in quality — often a burning sensation — and may be associated with autonomic disturbance. It is due to some form of neuronal damage; causalgia and postherpetic neuralgia are examples.

What has been said is somewhat simplified. The nature of chronic pain is not completely understood; some low-level prolonged path seems to occur unrelated to tissue damage and evident damage occurs with little or no pain. Pain is also influenced by hormones and, markedly, by psychological factors. So much so that types of pain are often classified as neurogenic, somatogenic (acute or chronic) or psychogenic.

**Pain receptors**

The A delta receptors respond to strong mechanical stimulation and some to damaging heat, i.e. above 45°C. When these fibres are stimulated experimentally they cause a pricking or stinging s’mssation. The C fibre receptors appear to be sensitive to many kinds of stimuli — mechanical, chemical and heat (hence they are called polyiriodal receptors) — but probably they are sensitive to a chemical released from tissue damaged by any stimulus.

**Pain pathways**

The cell bodies of these neurons are found in the dorsal root ganglia and their central connections enter the spinal cord via the dorsal root, except for some 30% of the C fibres which return to the peripheral nerve and
enter the cord via the ventral root (Fig. 3.19). C fibres synapse in laminae I and II and A delta fibres in laminae I and V of the substantia gelatinosa. Some of the axons end in the spinal cord and brain stem. The rest ascend in the arterolateral funiculus, including the lateral spinothalamic tract, and a few in the posterolateral section of the cord. Some of the ascending fibres go to the thalamus and then to the post-central gyrus and other parts of the cortex. Many go to the reticular formation, while others go to the hypothalamus and the periaqueductal grey matter. The synaptic transmitter of the primary afferent pain fibres is substance P (see Fig. 3.19).
Control of pain

The gate control theory of pain was suggested in 1965 by Melzack and Wall and has been expanded and modified since. The essence of it is that pain perception is regulated by a ‘gate’ which may be opened or closed, thus increasing or decreasing the pain perceived by means of other inputs from peripheral nerves or from the central nervous system. Some low-threshold mechanoreceptors from skin and elsewhere pass, without synapsing, up the posterior columns of the spinal cord. These A beta fibres give off collaterals, which impinge on nociceptor cells of the A delta and C pain fibres in laminae I and II of the posterior horn. It is believed that the input of these mechanoreceptors effectively reduces the excitability of the nociceptor cells to pain-generated stimuli; it is referred to as presynaptic inhibition.

Thus electrical pulses which stimulate these A beta mechanoreceptor fibres are effective in reducing pain perception. As noted already, these relatively large-diameter nerves are capable of being stimulated at low current intensities (Fig. 3.13) and will convey impulses at quite high frequencies. Therefore low-intensity (i.e. just perceptible), high-frequency (100—200 Hz) TENS is appropriate and effective.

Morphine acts on the C fibre system and hence controls tissue-damage pain but not other types of pain. This occurs because morphine imitates naturally occurring neurotransmitters, encephalin and endorphin. In the substantia gelatinosa (lamina II) there are interneurons which can produce encephalin to inhibit the C system cells in this region. Collateral branches of A delta fibres in the posterior horn connect with these interneurons and stimulate them. Thus stimulation of the A delta fibre by electrical pulses will damp down C fibre system-type pain. This is reputed to be the mechanism by which acupuncture works, since these A delta nerve fibres are considered to be stimulated by pinprick. (Acupuncture points seem to be where bundles of these nerves pierce the deep fascia (Bowsher, 1988). The stimulation must be done in the same neural segment.) However, Levin and Hui-Chan (1993) were unable to demonstrate stimulation of A delta fibres in normal subjects and concluded that pain relief was due to stimulation of A alpha and beta fibres by both conventional and acupuncture-like TENS. They concede that patients afflicted with chronic pain may tolerate higher TENS intensities, possibly sufficient to stimulate A delta fibres. Stimulation of these fibres by high-intensity, low-frequency TENS, however, remains as a rational explanation of the inhibition of C fibre-type pain.

It is also recognized that activation of these A delta pain fibres may provoke impulses in the midbrain that then travel back down the spinal cord to inhibit nociceptor neurons at the original level: a descending pain suppression system (De Domenico, 1982; Bowsher, 1988). This system also generates encephalin in the substantia gelatinosa. The A delta nociceptors in the spinothalamic tract give of f collateral branches to the periaqueductal grey matter in the midbrain. Descending neurons from this region use serotonin (5-hydroxytryptamine) as the neurotransmitter. Other descending pain-inhibiting pathways are also known that involve noradrenaline as the neurotransmitter (Bowsher, 1988).
Fig. 120 Control of pain.

The mechanisms so far described are summarized in Fig 3.20 and below:

1. The pain gate effect on both A delta (fast) and C (slow) path fibres in the posterior horn due to stimulation of mechanoreceptors (A beta) fibres by high-frequency, low-intensity electric pulses, sometimes called hi-TENS or traditional TENS.

2. A morphine-type effect on the C fibre system occurs. This is due to encephalin produced by interneurons in the posterior horn, which have been stimulated by A delta pain receptor fibres. These A delta fibres are themselves stimulated by low-frequency, high-intensity electrical pulses, which are called jo-TENS or acupuncture TENS.

3. Morphine-type (encephalin) effect on C fibre system as in point 2 above but via centres in the midbrain and involving serotonin as a neurotransmitter; also activated by A delta stimulation by low-frequency, high-intensity stimuli.
Stimulation at frequencies above 50Hz may be able to produce a physiological block in both types of peripheral path fibres. This effect is rather unclear and its clinical value is uncertain (De Domenico, 1982). However, a recent, well-conducted study (Walsh et al., 1993) has shown that different combinations of TENS frequencies and pulse lengths have differing influences on peripheral nerve conduction latency. It was shown that negative peak latency was markedly increased (i.e. conduction was slowed) in the superficial radial nerve of those who had received 0.2 ms pulses at 110 Hz for 15 min, and to a lesser extent with other stimulation parameters. Thus it would seem that TENS may act directly on peripheral nerves, as well as at their connections in the spinal cord, as indicated above. Furthermore, the effect appears to depend on the stimulation parameters in an, as yet, unelucidated way. These facts may go some way to account for the inconsistency of results from clinical trials, as discussed below.

Electrical stimulation also reaches the cerebral cortex in the sense that the patient is aware of a prickling or tingling sensation. This may contribute to the placebo effect that occurs with all treatments.

As noted earlier, most of the treatments for pain relief are given by means of small battery-operated TENS stimulators which provide fairly short — around 0.05 ms — pulses of low intensity and variable frequency — often 50 or 100 Hz but may be up to 200 Hz. Similar effects are, of course, produced by similar electrical pulses. Many different sources of electrical stimulation, e.g. diadynamic, interferential and faradic-type currents, can lead to pain relief, which may be due to one or more of these mechanisms.

**Therapeutic effectiveness**

The effectiveness of TENS for pain relief has been well supported in quite a large number of clinical studies and trials (see Thorsteinsson, 1983; Fahrer, 1991). However, there are also a substantial number of studies that have shown no benefit from the application of TENS. Fahrer (1991) considered 25 trials reported between 1975 and 1990. He suggests that, as the number of trials interpreted as ‘efficacious’ (14 of 25) dominate slightly over the ‘unproven’ and ‘not efficacious’ studies, TENS should be regarded as an adjunctive analgesic aid. He notes that these studies evaluating TENS are not easy to compare because of large differences in trial design and other parameters. In fact, the average number of patients or subjects for each trial is somewhat lower in the ‘efficacious’ trials quoted, which rather diminishes their effect. In general, when treating acute and some neurogenic pain, the results appear to be better than those achieved in the treatment of chronic pain. Several studies, including
one particularly large and thorough piece of work (Deyo et al., 1972), have found no benefit beyond placebo for the use of TENS in chronic low back pain. Successful treatment of neurogenic pain has been reported (Meyer and Fields, 1972). Perhaps not surprisingly, postoperative and obstetric pain have been quite successfully treated (Santiesteban, 1981), but some studies were unable to show benefit for the pain of labour. Sim (1991) also concluded that, following cholecystectomy through a right upper paramedian incisional approach, the limited benefits of TENS do not justify the additional cost or time. However TENS has been shown to be as effective for postoperative analgesia as nitrous oxide and oxygen mixture (Entonox) inhalation (Jones and Hutchinson, 1991). Both provided short-term pain relief during postoperative physiotherapy, but TENS was preferred due to the lack of side-effects. TENS stimulation of an acupuncture point on the thumb has been shown to be effective in improving antiemetic control following opioid analgesia in orthopaedic surgery in females (McMillan, 1994). In a double-blind trial the efficacy of TENS has been found to be much greater than would be accounted for by the placebo effect. Pain was relieved in 48% of instances due to TENS and in 32% of instances due to placebo treatment (Thorsteinsson et al., 1977).

A careful recent study (Marchand et al., 1993) concluded that high-frequency, low-intensity TENS reduced both the intensity and unpleasantness of pain (which were separately measured) due to chronic backache. Much of the reduction of pain unpleasantness was considered a placebo effect, which, had it been combined with the effect on pain intensity, would have disguised the real effect. The authors conclude that TENS is a useful, short-term analgesic for low back pain. Failure to distinguish between the affective and other components of pain could well account for the confusion and uncertainty over the effects of TENS and similar treatments.

There is a definite difference in the mode of action of high-frequency, low-intensity TENS compared to that of the low-frequency, high-intensity type which acts by the release of morphine-like neurotransmitters, shown by the fact that it can be blocked by a morphine inhibitor (Sjolund and Eriksson, 1979). This difference does not seem to be reflected in any differences in clinical use; both types were reported to be successful in similar conditions. It has been suggested (Thorsteinsson, 1983) that high-intensity, low-frequency TENS gives somewhat better results but is less well tolerated. The pain relieving effects of TENS have been demonstrated with experimentally induced ischaemic pain in normal subjects (Roche et al., 1984; Woolf, 1979). Both studies showed that high intensity, continuous TENS increased tolerance to ischaemic pain (i.e. the time when the subjects reported intolerable pain). Roche et al. also found that low-intensity TENS, in trains or bursts, acted to suppress the pain threshold (i.e. when pain was first reported). This led them to suggest that low-level pain might be effectively modified by low-intensity trains of TENS, while the application of continuous, high-intensity TENS might better increase tolerance and endurance of moderate pain. These results illustrate the need to match the intensity of pain with the appropriate therapy. It has also been found that, while conventional TENS had no effect on sharp pinching pain, it increased the path threshold to dull pressure-type pain in healthy volunteers (Sirmonds et al., 1992).
The duration of pain relief due to TENS is very variable. One study (Thorsteinsson et al., 1977) found a mean of 4—7 hours, but it is not clear why pain should be relieved for such long periods due to any of the described mechanisms.

The effects of other types of current on pain have not been nearly so extensively researched. Although it is widely agreed that interferential currents have a pain-relieving effect, there seems to be a dearth of objective studies. It has been shown that a rise in the pain threshold may occur after interferential treatment (Partan et al., 1953), but a more recent study on jaw pain (Taylor et al., 1987) could find no significant difference between interferential and placebo treatments. A small plot study investigating the effect of interferential therapy on induced ischaemic pain in healthy volunteers (Scott and Purves, 1991) found a pain relieving trend in the treatment group which was not statistically significant. Quirk et al. (1985) found that the symptoms of osteoarthritis of the knee were significantly relieved by treatment using interferential therapy and exercises, shortwave diathermy and exercises, or exercises alone. Overall analysis revealed no significant difference between the three regimens except that the only patients to deteriorate during treatment were those in the exercise-only group. For a full discussion of the way interferential currents may relieve pain see De Domenico (1982).

There is, of course, good reason to suppose that many types of electrical currents which are not customarily regarded as TENS, such as interrupted direct currents, faradic-type currents, sinusoidal currents, HVPGS and diadynamic currents, could all relieve pain by the mechanisms described above. Indeed other modalities such as mechanical vibration and heat will also act in the same way.

TENS has also been utilized in the successful treatment of patients suffering from persistent, abnormal skin sensations, such as formication or feelings of worms wriggling under the skin (dysaesthesia) (Bending, 1993). The mode of action would presumably be similar to that leading to pain relief, so that similar therapeutic parameters should be used.

**Other effects**

**Effects on blood flow**

Cutaneous vasodilation occurs in the area of application of some electrical stimulation, often observed with faradic-type currents and sinusoidal or diadynamic currents of sufficient intensity. This is considered to be due to stimulation of sensory nerves causing arteriolar vasodilation by means of the axon reflex at first and subsequently due to the release of histamine-like substances causing capillary dilation (Wadsworth and Chanmugan, 1980). The effect can be quantified by recording the change in skin temperature. With monophasic (unidirectional) currents the pH and other chemical changes seem to cause the vasodilation, as described in Chapter 2.
Reduction of oedema

There are claims that various therapeutic current applications will help to reduce tissue oedema. These are based on at least three distinct mechanisms. First, the muscle pumping action, noted above (p. 2), in which intermittent muscle contraction mechanically compresses adjacent soft-walled venous and lymphatic vessels to increase the centripetal flow of their contents. The consequent reduction of interstitial pressure is considered to be effective for all oedema, whatever the stage or cause. Secondly, there is a hypothetical mechanism which suggests that the application of current displaces the negatively charged plasma proteins of the interstitial fluid of a traumatized region. The increased mobility of albumin in particular should accelerate the normal lymphatic capillary uptake, so increasing the fluid return in the lymphatic system and reducing oedema. While there is some evidence supporting this hypothesis and it is often asserted (e.g. Newton, 1987), careful experiments on animals have been unable to demonstrate this particular effect, although a symmetrically biphasic current appeared to hamper absorption (Cosgrove et al., 1992; Mohr et al., 1987). These studies applied cathodal high-voltage pulsed currents at 24-hour intervals after the initial trauma and were thus treating already resolving oedema. Other animal experiments, however, applied similar currents immediately after trauma (i.e. while the oedema was developing), and found the oedema formation clearly retarded (Taylor et al., 1991; Bettany et al., 1990). A third suggested mechanism (Mendel et al., 1992; Reed, 1988) is that the current acts to decrease the permeability of capillaries in some way, thus diminishing fluid and plasma protein loss to the interstitial space. This suggests that the time at which the current is applied is important. It must be understood that effects on animals, both supporting and refuting a mechanism, are not necessarily transferable to humans. However, these studies provide some important evidence, not previously demonstrated, which may eventually lead to more enlightened clinical applications.

Effects on the autonomic nervous system

It is to be expected that some autonomic nerves would be stimulated by electrical pulses of suitable intensities since somatic nerves of similar size are stimulated. Such effects are frequently postulated as an explanation for therapeutic benefit, particularly in connection with interferential currents. The evidence for these effects, such as it is, seems to be inconsistent.

Altering the ionic distribution around the cell

Altering the ionic balance around the cell — as electrical stimulation inevitably does — would be expected to lead to some effects, but clear evidence and clinical correlation are lacking at present. It has been indicated in Chapter 2 that monophasic currents can accelerate the healing of cutaneous wounds and bone. Remodelling of bone and fibrous tissue have also been proposed. Many other effects have been considered to occur, such as increases in cell metabolism and exchange across the cell membrane, both being associated with increased microcirculation (Mon 1987); see also Chapter 1.
Summary

The effects of electrical stimulation have sometimes been described in a confusing and illogical manner with no distinction being made between the direct and indirect effects. A more rational approach has been proposed (Mon, 1987) in which the physiological responses to electrical stimulation are organized into cellular, tissue, segmental and systemic levels. Thus nerve excitation occurs at a cellular level and the muscle contraction it induces is an effect at the tissue level. Muscle group contraction and its effect on venous and lymphatic flow occur at a segmental level whilst the analgesic effects due to the release of endorphins and encephalins is an effect at the systemic level.

A rather simplified summary of some of the major effects is given in Table 3.2, but it must be understood that neither the magnitude nor the therapeutic importance is taken into account in this table.

PRINCIPLES OF APPLICATION

Electrical energy for therapy must be applied to the body tissues with at least two electrodes to form a complete circuit. The transition of an electric current of conduction in the wires (electron movement) to a convection current in the tissues (ionic movement) is complex and very important in determining the resulting effects.

Electrode—tissue interface

The changes that occur between the conducting metal and conducting fluid on and within the tissues consist of complex dynamic electrochemical interactions. The simple consequences of these have been described in Chapter 2 and Appendix B. If the applied current is evenly alternating (biphasic) there are no significant chemical changes; also if the total current, although unidirectional, is very small (low-intensity and/or very short pulses) the chemical effects will be negligible.

A layer of ion-containing fluid is needed to pass current from the electrode to the tissues, normally skin. This is usually water or conducting gel. This serves to ensure a uniform conducting pathway between the electrode and the epidermis and secondly to make the electrochemical changes occur outside the epidermis. Since the epidermal surface is very irregular a flat electrode pressed onto it would be in contact at only a few points, leading to a high current density at these points. Further, the epidermal surface has a high electrical resistance because it is largely dry keratin, and because of the presence of oily sebum. This resistance is lowered by wetting the skin surface.
Table 32 Outline of effects of pulsed electric currents on excitable cells

- Prickling sensation
- Vasodilation—due to axon reflex
- Pain relief—due to pain gate mechanism
- Re-education of movement
- Increase in strength and endurance
- Increase in intracutaneous blood flow
- Pumping action on veins, lymphatics leading to increased blood flow in tissues
- Trophic changes (eutrophic effects)
- Altered vessel diameter resulting in possible altered blood flow
- Pain sensation
- Pain relief via encephalins, endorphins and other central nervous system mechanisms
- Pain perception
- Muscle contraction
Types of electrode

There are three basic electrode systems:

1. A malleable metal electrode such as tinplate or aluminium coupled to the skin with water retained in a pad of lint, cotton gauze or some form of sponge material, e.g. Spontex (Fig. 3.21). The water provides the uniform ion-containing low-resistance pathway for the current while the absorbant material simply serves to keep the water in place. Ordinary tapwater is suitable in most instances but in some soft-water areas a little salt or bicarbonate of soda may need to be added. The whole assembly is fixed in place by a strap, bandage or by suction. The thickness of the pad needed, and hence the quantity of water, depends on the irregularity of the skin surface and on whether significant chemical changes will occur. If the latter is the case then about 1.25cm (16 thicknesses of lint) is considered an appropriate thickness. Otherwise rather thinner (0.5—1 cm) wet thickness seems to be sufficient for most treatments.

   In a system in which current passes through the body the total current at each of the two electrodes must be equal but the important factor is the current density, i.e. current per unit area. Thus if two pads are of unequal size, most effect will occur close to the smaller one, which is called the active electrode. The other electrode is called the indifferent or dispersive electrode. In order to limit the effects to an area such as the motor point of a muscle, the active electrode can be a small metal disc covered with lint or other suitable material and attached to a handle. This is often called a button electrode.

2. The second system involves electrodes that will conform to the body surface more easily than the metal electrodes described above. These are made of carbon-impregnated silicone rubber. They may be used with sponge pads or coupled to the skin by a thin layer of conducting gel and fixed in place either with a strap or adhesive tape. A somewhat similar system for more lengthy application of TENS involves karaya gum (obtained from a particular kind of tree in India) which when wetted is both conductive and adhesive. Some synthetically produced polymers act in the same way (Paterson, 1983).

   In general, the carbon rubber and similar electrodes used with conducting gel are convenient for long-term use and repeated self-application by the patient, whereas the water pad conduction methods, whether with metal or carbon rubber electrodes, are more appropriate for treating larger areas with higher currents and are usually used in the physiotherapy department. Metal electrodes are somewhat more efficient in passing current to the tissues than carbon rubber and other similar types in that they have a lower impedance (Nelson et al., 1980). However, carbon rubber appear, on the whole, to have
lower impedance than many other commercially available polymer electrodes, some of which exhibit remarkably high impedance (Nolan, 1991). It should be noted that, where the electrode is coupled to the skin by a wet pad, current density is determined by the area of the pad, but, where the electrode is in direct contact with the skin, it is determined by the area of the electrode. Paterson (1983) noted that carbon rubber electrodes have significant resistance compared with the electrode—tissue junction, so that most current will take the shortest pathway. Thus, where it is directly coupled to the skin by gel, the current density is likely to be higher near where the metal wire enters the electrode.

3 The third system is by means of a water bath (or baths) in which the body part is immersed with an electrode. Current is passed from electrode to tissues through the water. This system is considered later.

**Current flow in the tissues**

The quantity of current that flows in the tissues and the path it follows will depend on the impedance of that pathway. The impedance includes the ohmic resistance, capacitive resistance (or reactance) and the inductive resistance. The latter is negligible in the tissues but the two former have an important influence on the effects of the electrical stimulation. Generally, watery tissue such as blood, muscle and nerve has low ohmic resistance; bone and fat has rather higher and epidermis has the highest of all. The ohmic resistance is determined therefore chiefly by the thickness and nature of the skin under the electrodes and, to a much lesser extent, by the inter-electrode distance. Where two low-resistance regions are separated by a high-resistance region, i.e. a near insulator, a capacitor is formed and capacitive effects occur. Thus where an electrode is separated from nerve and muscle by skin and fat and there is a capacitor. The concept of these electrical pathways is illustrated in Figure 3.22,

For direct current (unidirectional current) and slowly changing pulses of current the skin resistance is high and thus most of the electrical energy is released in the skin and subcutaneous tissues, hence cutaneous nerves are affected. As the current spreads through the low-resistance pathway of the deeper tissues it can have less effect. However, capacitive resistance diminishes for short pulses of current or alternating (biphasic) currents of higher frequencies,
thus the current can pass through the skin more easily and relatively more energy is released in the deeper tissues. This explains why short pulse (phase) lengths are able to penetrate the skin more easily. The effect occurs with both single pulses and alternating pulses of appropriate frequency (i.e. a 4000 Hz medium-frequency current is a series of 0.125 ms phases (half cycles) and behaves in the same way as separate single pulses of this duration). These points are further explained in Physical Principles Explained (Low and Reed, 1994).

Taken together the effects described above suggest that some deeply placed low-threshold nerves, such as motor nerves, would be more efficiently stimulated by shorter pulses, say about 1/20th of a millisecond (0.05 ms) because of the skin capacitance. On the other hand to stimulate high-threshold unmyelinated pain fibres (C fibres) in the skin it would seem sensible to use longer pulses of a few milliseconds.

**Arrangements of electrodes**

It has been noted already that what matters for producing an effect in the tissues is the current density. Adding to the size of the electrodes will decrease the current density. This occurs whether the greater size is due to a single larger electrode or to one or more additional parallel connected electrodes. Since the water in the pad has a very low ohmic resistance the effective area of application is that of the pad. With carbon-rubber electrodes there may be a slightly higher current through the region where the wire enters the electrode but since these electrodes are usually small the effect is not great.

The position of the electrodes will obviously determine the path that the current will follow in the tissues. In many situations a small electrode is used to give a high localized current density, such as to stimulate the motor point of a muscle or an acupuncture point. In these circumstances the dispersive (or indifferent) electrode can be placed on any convenient area of skin that is reasonably close. The further away it is placed the more current will be needed and less effective localization will occur. If the two electrodes are of similar size the current density under each will be similar and therefore effects such as sensory stimulation will occur under both. If the electrodes are placed close together the effects will be localized to the region between them, e.g. placing electrodes at either end of the long axis of the belly of a muscle will cause local stimulation of that muscle, or stimulating sensory nerves in a local area of skin will give pain relief. If the electrodes are placed too close together current will be localized to the adjacent edges and to the intervening small area of skin rather than passing through the whole area of electrode and epidermis in contact.

**Water baths**

The hand, forearm, foot and leg can conveniently be put into baths or bowls of water with electrodes to provide a means of passing current to
the tissues. Such an arrangement can be used to provide a large area for an indifferent electrode, as described in Chapter 2. They can also be used as a method of applying muscle-stimulating currents. If two electrodes are placed in the same water bath with the part to be treated, current will pass both through the water and through the tissues — two pathways in parallel (Fig. 3.23). The current density in the tissues is critically dependent on the position of the two electrodes in the bath and on the relative resistance of the two paths. Such a system, called a faradic foot bath, is often used to stimulate the intrinsic muscle of the foot for reeducation.

Unipolar and bipolar

The system described above, with both electrodes in the same bath, is referred to as ‘bipolar’ whereas if one electrode is in the bath and the circuit is completed by a pad electrode or an electrode in another bath it is called ‘unipolar’. It may be noted that adding salt to a unipolar bath results in a greater current passing through the tissues because it lowers the resistance in series thus reducing the total resistance. Adding salt to a bipolar bath will decrease the current through the tissues since greater current will now pass in the parallel water pathway due to its lowered resistance. In some publications (Alon, 1987) bipolar treatments are described as those in which both electrodes or pads are applied to the area being treated, whereas unipolar are those involving a (usually) larger indifferent electrode applied at some distance from the active electrode which is sited over the target tissue. However, it must be emphasized that all these treatments involving a flow of current in the tissues are essentially bipolar in the sense that there must be two connections to the tissues. The use of the terms ‘unipolar’ and ‘bipolar’ sometimes creates confusion.

Lowering the electrical resistance at the skin surface

As has been noted already, the electrical resistance of the epidermis is high. It can be reduced by washing the surface to remove some of the
keratin and sebum and leaving the skin wet. Warming the skin also helps to lower its resistance by increasing the rate of particle and ionic movement, also perhaps increasing the activity of the sweat glands and blood flow. Thus warming, washing and wetting the skin will allow larger currents to flow for the same applied voltage.

To record small currents generated by the tissues, such as the electrocardiogram or surface electromyogram, it is sometimes necessary to reduce the skin surface resistance still further by gently scraping or sandpapering some of the surface epidermis off — removing some of the dead cells that form the outer epidermis — before fixing the electrodes.

In all cases it must be realized that maintaining the same skin—electrode junction throughout treatment is essential. If the adhesion of the electrode to the skin surface alters, or the pressure of sponge or pad decreases, this can lead to a higher resistance; consequently the fixation of the electrodes to the body surface is very important in keeping a constant uniform low resistance at this junction.

**Checking for areas of abnormal resistance**

The skin should be inspected before treatment to check for any low resistance areas, such as cuts or abrasions, or any other circumstances which might lead to uneven distribution of the current. Sometimes the pad/electrode can be conveniently moved to avoid this area but, if not and the area of low resistance is small enough, it can be protected with a layer of petroleum jelly covered with cotton wool. Abnormal epidermal tissue, such as warts or scars, may present areas of higher resistance, which could alter current distribution if they are extensive. Similarly, grease from emollients may need to be removed.

**Safety recommendations**

Electrical units should be energized by pressing the power switch to the ‘on’ position before connecting the patient to the circuit. This is because, on some equipment, there is a spike of output before the machine stabilizes. Current density for any electrode/skin contact area should not exceed 2 mA rms/cm’

**APPLICATION AND USES OF SPECIFIC CURRENTS**

**Faradic-type currents**

Electrical muscle stimulation is usually achieved by faradic-type currents (0.1—1 ms duration at any frequency between 30 and 100Hz). In order to localize the current to individual muscles a small active electrode, i.e. a small pad or button electrode, is applied to the motor point of the muscle, the circuit being completed with a larger dispersive electrode sited in some convenient, usually proximal, area. The motor points of some superficial muscles are often indicated on charts (Fig. 3.24). Such charts act as a guide but a knowledge of the relevant anatomy coupled with a
Fig. 3.24 Guide to motor points.
little trial and error will locate the precise point at which the muscle is most effectively stimulated. The usual site is in the lower part of the proximal third of the muscle belly but there are many exceptions. It is obvious that deeply placed muscles can only be successfully stimulated where their fleshy belly emerges, for example, the extensor hallucis longus emerging in the lower part of the leg between tibialis anterior and extensor digitorum longus (Fig. 3.24).

As mentioned previously, if a choice is available, constant current pulses are preferred for techniques in which the pads are fixed, because they are reputed to be more comfortable. In this case, as the skin resistance fluctuates, the voltage alters to maintain a constant current. However, for labile techniques, where one pad is moved, constant voltage is preferable. In these applications, the effective area (i.e. the area in contact with the skin) of the pad/electrode changes, which alters the current density. If the area of the pad in contact with the tissues becomes smaller, the resistance increases. However, if the voltage remains constant, the current intensity will fall so that the current density will remain approximately the same.

**Technique of application**

The patient is positioned so that the part to be treated is comfortably supported with the muscles to be stimulated in a shortened position, although this may be modified when movement is to be produced, e.g. slight knee flexion allowing quadriceps stimulation to cause extension.

The skin surfaces to which the current will be applied must be examined and any cuts, abrasions or other lesions that might cause uneven current distribution insulated (with a dab of petroleum jelly) or avoided. These areas should be washed to remove sebum and epithelial cells and left damp; using hot water warms the skin and helps to lower the resistance further.

The size of the active electrode, which may be a small plate and pad or a button electrode, is chosen by considering the size of the area to be treated; the motor point of a small muscle close to others is clearly best stimulated with a small button electrode. In all cases the dispersive or indifferent pad should be two or three times larger. Leads are connected to the machine and attached to the electrodes. The metal electrode should be smaller than the pad or sponge material to prevent the edge of the electrode being bent down on to the skin which could lead to high current density at that point and would be very uncomfortable.

Holding the two electrodes, separated, in one hand allows the machine and connections to be tested by the therapist. Observing this may help to allay the anxiety of a patient experiencing this treatment for the first time.

The pads or sponges should be soaked in warm tap water, saline or sodium bicarbonate solution, which are somewhat better conductors, particularly in soft water areas, and applied to the skin. Fixation is achieved with a rubber strap, a crêpe or similar bandage, or simply by body weight. A piece of polythene or other waterproof material is placed on the pad to prevent the bandage becoming wet. If the whole bandage gets wet, it becomes effectively a circular pad.
The nature of the treatment and the sensations to be expected — a tingling sensation and muscle contraction — should be explained to the patient with reassurance that there is no way that any damage can be caused by this treatment. When a single muscle is to be stimulated the active electrode is placed firmly over the approximate motor point, indicated in Figure 3.24, and a small current is applied. Small adjustments of the position of the active electrode will allow the best position to be found; the current may need to be increased and then decreased as the exact motor point is found and good contractions can be obtained with less current.

When a muscle group is to be stimulated the active pad can be made to straddle all the motor points or the two pads may be of approximately the same size, placed at either end of the muscle group so that current spreads through the whole group: this happens in the faradic foot bath, shown in Figure 3.23. The stimulation of individual muscles is often done to re-educate the activity. In this case the patient attempts a voluntary contraction at the same time as the current causes the muscle to contract. To enable the patient to co-operate, the length of the contractions and the intervals between them should be suitably long. This allows patients time to match their efforts with the stimulated contractions and an adequate rest between each one.

As an example of re-education, the stimulation of the abductor hallucis will be considered. This muscle in the sole of the foot contributes to the stability of the first metatarsophalangeal joint in walking and running. If the joint is free it will act to flex the toe and abduct it. In those people who have worn shoes throughout childhood the ability to abduct the big toe when the foot is supported on a smooth surface is often absent. However, at least in young adults, a little re-education and practice will restore this ability to some degree. The abductor hallucis may be stimulated in a bipolar bath with two electrodes, one under the heel and the other close to the belly of the muscle; a modification of the arrangement shown in Figure 3.23. Alternatively the indifferent electrode may be a pad fixed behind the medial malleolus while a small button electrode is used over the muscle belly.

The patient is instructed first to feel the movement of the big toe brought about by the faradic current. When he or she is able to appreciate this movement he or she is asked to attempt to join in voluntarily so that the action becomes, in effect, active-assisted. As the patient’s ability increases the intensity of the current is progressively reduced and he or she is asked to hold the contraction at the end of the surge. When he or she can initiate a contraction actively without the assistance of the current, re-education continues by other means (see discussion in Chapter 4).

Re-educating the pelvic floor musculature for the treatment of stress incontinence requires a special technique to stimulate the sphincter muscles of the urethra. This can be done with a large dispersive over the lumbosacral region and a rectal electrode as the active with the patient in side-lying or in crook half-lying. In females a vaginal electrode can be used; a button electrode placed over the perineal body can be used as the active electrode in either sex (Wadsworth and Chanmugan, 1980).
Voluntary contraction is attempted with the electrical stimulation. Faradictype currents have been used in the successful treatment of this condition (Montgomery and Shepherd, 1983) as well as interferential currents (see p. 106).

Muscle groups in the limbs can be stimulated rhythmically to effect a muscle-pumping action, enhancing the venous and lymphatic flow to assist the reduction of oedema. This is combined with elevation of the limb and the application of a pressure bandage. The largest volume of muscle that can be stimulated is required, so the quadriceps and plantarflexors of the lower limb and flexors of the elbow and hand in the upper limb are usually chosen. Large pads are applied over these muscle groups, or on the sole of the foot and quadriceps; there are numerous other pad positions to achieve the strong generalized muscle contractions needed. The compression bandage, applied over the pads, should give firm pressure against which the contracting musculature can press but should not be constrictive. Strong slow muscle contractions should be produced with a long period of relaxation (several seconds) to allow vessel filling.

**Interrupted direct current**

The technique of stimulating denervated muscles with interrupted direct current is very similar to faradic-type stimulation. The major difference is in the positioning of the electrodes. Because the maximum number of muscle fibres should be stimulated, the electrodes need to be positioned at either end of the muscle belly. Alternatively, one electrode may be fixed at one end and the other moved slowly down the length of the fibres — a labile technique. If the muscle is very small, one pad can completely cover the fibres and another dispersive electrode can be placed at a convenient place to complete the circuit. Because the muscle is not innervated, and is therefore flaccid, care must be taken not to stretch the muscle during treatment. It is therefore usually positioned in a slightly shortened or neutral position.

**TENS**

It is customary, as already noted, to limit the acronym TENS to low-intensity, short impulses produced by battery-operated sources specifically for pain relief. As local areas are usually treated and self-treatment is common, small carbon-rubber electrodes are usually employed. Since both the intention and effect of the treatment are to relieve pain it is important to be certain that this is appropriate and does not lead to the neglect of the underlying causes of the pain. It is also very important that the pain should be evaluated both initially and during the course of the treatment. This serves both to monitor the effectiveness of the particular treatment parameters used, such as the position of the electrodes, and to measure the progress of the treatment. Any objective methods that are appropriate, such as
measuring the range of movement, should be used but often subjective pain assessment is the principal means used. A 10cm horizontal visual analogue scale (on which the patient marks the intensity of pain between one end marked ‘no pain’ and the other end ‘worst pain ever’) and pain behaviour analysis have been recommended (Frampton, 1988).

The application of TENS requires decisions about where to place the electrodes and what current parameters to use.

**Electrode placement**

There are four approaches to be considered. The most usual is to site electrodes close to where the pain is perceived to be; often one electrode is sited over the place where the most intense pain is felt or the greatest tenderness elicited. Secondly, the electrodes may be placed within the same dermatome, myotome or sclerotome. They may be placed to pass current through the long axis of the dermatome. In many, but not all, circumstances the dermatome, myotome and sclerotome overlap. Thirdly, trigger or acupuncture points may be the preferred sites of current application. It is considered (Klein and Pariser, 1987) that acupuncture points can be located by their lower resistance compared with the surrounding skin (due to active sweat glands and/or local vasodilation); they can be found by using an electronic probe. Jones d al. (1990) found no difference in pain relief whether the electrodes were placed over the acupuncture point or para-incisionally for post-cholecystectomy patients. Fourthly, stimulation of peripheral nerves is used. Electrodes are placed in the line of the nerve and where it is particularly superficial. This method is used principally for the treatment of neurogenic path such as postherpetic neuralgia.

It is evident from a clinical point of view that these four approaches are by no means mutually exclusive. Thus trigger points, peripheral nerves and the painful area all lie in a dermatome. The choice of electrode position is often dictated by an effective result, i.e. relief of pain. Several positions may be tried before success is achieved.

**Current parameters**

TENS is most often applied as short pulses of around 0.05 ms at 50-400 Hz; this is called conventional TENS, and is high-frequency, low-intensity stimulation. The intensity is turned up gradually until a prickling or tingling sensation is felt. It should be neither painful nor should it cause a muscle contraction. It is presumed that these low-intensity short pulses will selectively stimulate the large low-threshold A beta fibres to produce pain inhibition by the pain gate mechanism, as described on p. 77. This conventional mode is the most usual method for self-treatment. The recommended duration and timing of such treatments varies from 30 to 60 min sessions once or twice a day (Klein and Pariser, 1987) to continuous TENS for a minimum of 8h/ day or even a full 24 h/day (Frampton, 1988).
High-intensity, low-frequency (acupuncture-like) TENS is another frequently used approach. Pulses of around 0.2 ms at about 2Hz are given at intensities close to the maximum that the patient can tolerate. This stimulates the high-threshold A delta and C fibres which leads to the release of endorphins and encephalins, as described on p. 78. This kind of stimulation is often applied to acupuncture points which, it is considered, are places where small bundles of A delta and sympathetic efferent fibres pierce the deep fascia to become more superficial (Bowsher, 1988). It is also sometimes applied to the motor points of muscle in the segmentally related myotome. In contrast with conventional TENS it is usually applied once per day for 20 or 30mth (Klein and Pariser, 1987). Mannheimer and Lainpe (1984) suggest that acute pain of a superficial nature, including causalgia, responds best to conventional TENS, whereas longstanding, deep, aching path responds best to low-frequency TENS.

‘Burst TENS’ is a series of pulses (i.e. a train), repeated 1—5 times a second, commonly twice. Each train or burst lasts about 70 ms and consists of a number of individual pulses at the usual conventional TENS frequencies of 50-100 HZ. The benefit claimed for this latter method is that it combines both the conventional and acupuncture-like TENS and therefore provides path relief by both routes. These modes of stimulation are illustrated in Figure 3.6.

In ‘modulated TENS’ the pulse width, frequency and intensity are all constantly and automatically varied. As well as the benefits of delivering both the conventional and acupuncture-like modes it is also believed to prevent adaptation of the nerves to the current. It has already been noted (p. 80) that TENS has been widely used for the relief of acute path, including postoperative and obstetric pain, as well as chronic and neurogenic path. Electrodes are fixed to the skin with adhesive tape or bandaged into place. A conducting gel is applied between electrode and skin. The leads from the TENS unit are concealed and fixed in the clothing if continuous home treatment is being given. Convenient ready-gelled adhesive electrodes are available.

**Contraindications to TENS**

TENS should not be used for patients with demand-type cardiac pacemakers. However, it is apparently safe with fixed-rate pacemakers (Eriksson and Schuller, 1978). Electrodes should not be placed over the carotid sinus as this could lead to cardiac arrhythmias. Similarly placement over the pharyngeal region could interfere with normal breathing and swallowing co-ordination. In any circumstances in which the skin is devoid of sensation, uniphasic (unidirectional) TENS should not be used because of the risk of unnoticed chemical change. Electrodes should not be placed over skin lesions or open to wounds because of the different resistance and the risk of infection. Placing electrodes over or close to the pregnant uterus is contraindicated but there is no evidence of any damage; avoidance is usually recommended for medicolegal reasons.
Electroacupuncture consists of two aspects. First, the acupuncture point may be found on the surface by testing the electrical resistance. Second, stimulation of the point may be given with an electric pulse rather than the traditional needle penetrating the skin. Some machines allow both testing and stimulation through the same electrode. Acupuncture points and trigger points (which appear to correspond to each other (Melzack et al., 1977)), apparently have a lower electrical impedance than the surrounding area, as noted above (Klein and Pariser, 1987). These points are located with a point electrode (probe) on the skin and a small applied current is then measured; thus the circuit impedance (or conductance) can be displayed on a meter. The electric pulses used for treatment are usually some form of low-frequency, high-intensity TENS stimulating A delta nerve fibres to achieve encephal'mergic pain relief (see p. 78). TENS applied to traditional acupuncture points for specific therapies is also used. For example, low-frequency 0.1 ms pulses given over the P6 acupuncture point proximal to the s'trist have been successfully used for controlling sickness after chemotherapy (McMillan and Dundee, 1991).

Ryodu-Raku (from Japan) is an example of such a system. The device is called a neurometer and can be used to test the tissue resistance between a point electrode applied to the skin and a dispetsive held in the hand. Where large differences of conductivity are found treatment is applied. This consists of a few seconds of direct current from the same device given either by means of a small surface electrode or a fine needle into the skin.

High-voltage pulsed galvanic stimulation (HVPGS)

Such currents will pass easily through the tissues because they are so brief (see p. 48) and will be relatively comfortable due to their wide discrimination between sensory, motor and pain nerve fibres (see Fig. 3.13).

As well as the frequency the intensity can be varied (0—500 V) and the polarity altered. The pattern of current can be changed by a mode switch. In continuous mode the train of twin pulses is delivered continuously Reciprocate mode refers to the alternate application of trains of pulses to one or other of two active pads and does not mean that the current direction is reversed. Surge mode gives a train of pulses whose intensity

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**Table 33 Summary of TENS**

<table>
<thead>
<tr>
<th>Electrode placement</th>
<th>Stimulus parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Site of pain</td>
<td>1 High-frequency, short pulse length, low-intensity</td>
</tr>
<tr>
<td>2 Dermatome, myotome</td>
<td>conventional TENS</td>
</tr>
<tr>
<td>3 Trigger point,</td>
<td>2 Low-frequency, wider pulse, high-intensity</td>
</tr>
<tr>
<td>4 Peripheral nerve</td>
<td>TENS</td>
</tr>
<tr>
<td>3 Trigger point,</td>
<td>3 1 and 2 mixed; burst TENS</td>
</tr>
<tr>
<td>4 Peripheral nerve</td>
<td>4 1 and 2 varied; modulated TENS</td>
</tr>
</tbody>
</table>
is gradually increased, as indicated with square wave pulses in Figure 3.le. A meter to indicate peak current may be provided. On some machines the interval between the two peaks may be altered; this is called the intrapulse interval. The current is applied by flexible electrodes and sponges. The electrodes are usually small and are sometimes mounted on a handle. Various special electrodes are available.

Uses of HVPGS

**Wound healing.** Since a direct current, albeit of very low total intensity, is being applied, the discussion on this subject in Chapter 2 is relevant. There seems to be evidence that low-intensity currents lead to tissue healing (see discussion in Chapter 2).

**Pain modulation.** How pain may be controlled by electrical stimulation has already been discussed (p. 77). Since both the frequency and intensity of HVPGS can be controlled it is possible to apply both high-frequency, low-intensity stimulation for pain gate control and low-frequency, high-intensity stimulation for encephalin-type pain control. HVPGS has been recommended for controlling all kinds of pain — acute, chronic, neurogenic and path from many sources (Newton, 1987).

**Muscle stimulation.** HVPGS is used for the stimulation of innervated muscle and, due to the short pulses and hence good transmission in the tissues, it is an efficient way of doing so. Consider the strength—duration curve (Fig. 3.13) which shows that short pulses at high intensities will be more selective in stimulating motor rather than pain nerves. HVPGS has therefore been used for muscle strengthening and the reduction of disuse atrophy of innervated muscle. A frequency of around 30Hz has been suggested with long intervals between bouts of tetanic contraction as the optimum schedule.

**Other uses.** Effects on the vascular system are claimed in that rhythmical muscle contraction and relaxation due to HVPGS of motor nerves will have a pumping effect, increasing blood flow in muscle and surrounding tissues, as considered on p. 68. This effect can aid in the reduction of tissue oedema. Fish et al. (1991) found that anodal high voltage pulsed current did not curb oedema formation in frog hind limbs. This contrasts markedly with significant treatment effects found with cathodal HVPGS (Taylor et al., 1991). Direct effects on autonomic nerves leading to local vasodilation, increased fluid exchange in the tissues and other beneficial effects have been claimed (Wadsworth and Chanmugan, 1980; see discussion on oedema reduction, p. 83). Consequent upon its ability to stimulate innervated muscle HVPGS has been recommended in the treatment of muscle spasm and to increase joint mobility (Newton, 1987).

Some uncertainty exists over the advantages of the twin peaks wave form, which is what makes this current unique. It has been suggested that a single pulse could be just as effective (Alon, 1987).
Sinusoidal currents

Effects

If a sinusoidal current is applied continuously it will cause a tetanic muscle contraction and a tingling sensation due to stimulation of motor and sensory nerves. It is usually surged to cause rhythmic muscle contractions. The sensory stimulations can lead to pain relief by some of the mechanisms described in connection with TENS. The rhythmic muscle contractions induced can help reduction of oedema by muscle pumping action and other consequences, discussed on p. 68. Various specific effects have been claimed, such as increased blood flow in the treated region suggested by the marked cutaneous erythema that can develop. Similarly it is claimed that unsurged sinusoidal current will help the absorption of oedema or inflammatory exudate (Wadsworth and Chanmugan, 1980). There seems to be no clear evidence to support these latter claims.

Application

Sinusoidal current can be applied in the same way as other low-frequency currents by means of electrodes and pads. However, because of the marked sensory stimulation this current is often applied to large areas and rarely used for local muscle stimulation. Thus it is applied either through large pads or water baths (see p. 88) or both. For pain control continuous sinusoidal current at intensities close to the limit of tolerance is recommended, increasing the current as the patient accommodates. This is applied for about 5 mm and repeated if there is insufficient immediate effect. For reduction of oedema and to increase the limb circulation surged sinusoidal current is suggested, causing regular rhythmical muscle pumping actions.

Sinusoidal current is rarely used in modern physiotherapy departments. It is interesting, however, to note that the series of 10 ms phases gives marked sensory nerve stimulation in the skin, acting in part like modern TENS stimulators. Recent understanding of pain control (see p. 77) may account for some of the benefits that were claimed for this treatment (Wadsworth and Chanmugan, 1980).

Diadynamic currents

Therapeutic effects

The effects claimed (Rennie, 1988) include:

1. Pain relief due to the mechanisms described already, i.e. pain gate mechanism, pain suppression by neurologically stimulated endorphins and encephalins, removal of irritants from the area by the increased circulation, and the placebo effect.

2. Decreased inflammation and swelling due to the increased muscle pumping action and increased local circulation; changes in cell membrane permeability are also claimed.
Muscle re-education and strengthening are considered to occur due to the stimulation of muscles. Increased local circulation due, it is claimed, both to the altered autonomic activity such as reduced sympathetic tone leading to Vasodilation and the release of histamine-like substances due to the unidirectional effects. Facilitation of tissue-healing due to local circulatory changes noted above and to the polar effects leading to increased cell activity (see Chapter 2).

Application of electrodes

Either metal plate or carbon rubber electrodes may be used with pads. Two equal-sized electrodes on either side of the area to be treated may be used or a small electrode may be placed over a trigger or motor point with a larger electrode placed proximally. Various treatment parameters are suggested. For pain relief and most other effects, an initial minute or so of DF followed by up to 5 mm of CF or LI' (Rennie, 1988). The reasons for preferring these or any other particular regimens are not provided in commentary on this subject.

It is suggested for all treatments that the current intensity should be perceptible but not painful. The major danger with such currents is tissue damage due to the polar effects. These may be avoided by current reversal during treatment.

Rebox

When applied to normal tissue the current rises over a period of about 1 s giving a characteristic displacement of the meter, of sound in the earphone, and of the shape of the graph. When applied to damaged or abnormal tissue a different pattern is said to occur in which the rise of current is slower, taking 3 or 4 s, and may occur in a series of steps. These differences do not seem to have supporting experimental evidence nor is there a clear theoretical basis for them.

Repeated application of this current for a few seconds at a time (up to 20 V leading to a maximum current of 0.3 mA proximal to the injured area), using the device as a treatment is claimed to lead to a normal response when the sante area is subsequently tested, and to therapeutic benefit. It has been used in the treatment of musculoskeletal pains, recent trauma and a number of other conditions. The benefits have been accounted for by postulating that the current causes increased ionic movement in the tissue fluid, and that monitoring rate of change of current in the tissues helps to localize the area of abnormality (Hervik, 1989).

Russian currents

In the 1970s claims were published that the 2500 Hz medium-frequency interrupted current could be used to generate greater muscle force than a maximal voluntary muscle contraction. This current has been described on p. 51. It is called ‘Russian’ because its use was first described by Dr
K. M. Knots in the Russian literature. It provoked much interest because the very successful Russian Olympic team were using it in addition to their usual training methods and it was suggested that its use led to significant (30—40%) gains in muscle strength.

Although it is a medium-frequency current the nerves are stimulated because it is interrupted to give a low-frequency stimulation of 50Hz. Due to the short pulses (of 0.2 ms phase) it will pass fairly easily through the skin and be effective in stimulating motor nerves (p. 64) but the stimulus is due to the initial electrical pulse, thus the purpose of the rest of the 10 ms train is not clear. It is, in fact, like a short-duration faradictype pulse at 50Hz.

The theoretical basis for its use is that maximum electrical stimulation can cause nearly all the motor units in a muscle to contract synchronously: something that cannot be achieved in voluntary contraction, it was claimed. This would allow stronger muscle contractions to occur with electrical stimulation and hence greater muscle hypertrophy. This has not been found to occur in the subsequent research. Many investigations have determined that electrical muscle stimulation leads to muscle hypertrophy but not to any greater degree than voluntary activity (Currier, 1987) — see p. 65.

Not only was it claimed that the electrically generated force was greater than that generated voluntarily but also that this occurred without producing pain. This claim has not been entirely supported either; in one careful study which assessed torque values and pain scores (Gilles and Bélanger, 1987) the assertions were definitely refuted.

This current can be applied in the usual way with electrodes applied over the muscle belly. To achieve muscle hypertrophy, which is the usual purpose, currents of high intensity producing maximum tolerable muscle contraction are given in spells of a few seconds separated by somewhat longer rest periods.

Interferential currents

The principle of interferential therapy, as explained, is to pass two medium-frequency alternating currents which are slightly out of phase through the tissues. Where the currents intersect a new current is set up (Fig. 3.Sa).

Medium-frequency currents will pass much more easily through the skin than low-frequency currents due to the lower impedance offered to very short electrical pulses (p. 87). With a 4000 Hz current a very high current intensity would be required to stimulate nerves. It is the beat effect that acts like any other low-frequency current and stimulates nerves. Thus such currents will pass easily through the tissues because they are medium-frequency but stimulate nerves because of the amplitude modulation.

Although the spread of medium-frequency current in the tissues is more uniform than a low-frequency current it is still at greater intensity
close to the electrodes (Fig. 3.25a, b). The great advantage of true interferential currents is that the low-frequency modulation is made to occur deep in the tissues.

About 40 years ago this idea was developed by Hans Nemec in Vienna and although used quite widely in the intervening years, it has become much better known recently with the development of cheaper electronic circuitry. A later development involves the use of a third current in a path at right angles to the other two; this is called stereodynamic interference current (Kloth, 1987). Thus the tissues are stimulated in a three-dimensional system; however, the usefulness of such a system is not clearly established.

Most interferential machines allow a constant beat frequency to be selected, e.g. 10, 50 or 100 Hz — in fact any frequency from 1 to 250 Hz — called the constant or static mode. They also have an arrangement that allows the beat frequency to change automatically and regularly between

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**Fig. 3.25 Areas of maximum stimulation.**
some pre-set pair of frequencies over a specified time period. This is variously
called a frequency modulation, swing or sweep. Thus the machine could be set to
sweep, for example, between 20 and 80 Hz over a period of 6s and back over the
next 6s. The pattern and timing of this modulation is usually adjustable and is
sometimes called the spectrum. Such an arrangement is useful to prevent nerve
habituation and extend the range of nerve types that can be stimulated. The higher
frequency and hence shorter pulse length will stimulate the low-threshold large
nerve fibres, as already explained. Since the pulse is sinusoidal, not square wave,
it is not easy to define the pulse length.

Figure 3.25c shows the theoretical distribution of current in a homogeneous
medium. The clover-leaf shape of maximum current modulation is due to the fact
that both the current amplitudes and their directions need to be summated, i.e.
vector addition. The distribution shown for four equally spaced electrodes is
unlikely to be realized in real tissue, since variations in tissue resistance and
electrode distances would prevent this neat uniformity. The real pattern is likely to
be much more irregular and diffuse in all tissues between and around the
electrodes. None the less the pattern shown is a valid concept and a useful guide.
This is a static pattern but, by varying the current intensity in the 2 circuits with
respect to each other, it is possible to move the clover-leaf pattern of maximum
modulation to and fro through 45°, thus giving a more uniform total distribution
of the interferential current in the tissues. There are various names for such a
mechanism, including vector sweep’, ‘scanning’, ‘rotating vector system’ or
‘dynamic interference field system’. It serves to increase the area of effective
treatment.

Control of the current intensity is provided to allow more or less stimulation
as needed. There is also usually automatic timing control for the timing of
treatment. Thus, in summary, controls on the interferential machine are:

1  Settings for constant beat frequency, e.g. 80 Hz.
2  Settings for variable beat frequencies, e.g. 20—80 Hz.
3  Control for time of variable beat frequencies cycle, e.g. 6s.
4  Intensity control.
5  Control for using rotating vector mechanisms, e.g. on or off.
6  Control for total treatment time, e.g. 10 mm.

Currents are applied by metal or carbon-rubber electrodes with water-soaked
sponges or lint. Carbon-rubber electrodes may be used with conducting gel, as
already described. Since two circuits are involved, four electrodes, i.e. a
quadrupolar technique, are usually used. These electrodes may be secured by
rubber straps or bandages; alternatively they may be secured by suction. Suction
units can be connected to the interferential machine. Flexible rubber cups are
connected by tubes to a pump that can provide a negative pressure. This suction
may be continuous or variable. Metal electrodes mounted inside the cups are
connected by wires carried within the tubes to the interferential source. Contact is
made by moistened sponges placed inside the cups between the metal electrode
and the skin. The negative pressure is set to vary rhythmically during treatment,
which diminishes the risk of skin damage.
Fig. 3.26 Electrode arrangement for interferential therapy. Plan for coplanar treatment.

It should be adjusted to maintain good electrical contact without causing discomfort. As well as maintaining electrical contact the suction has a mild massaging effect on the skin, stimulating cutaneous sensory nerves and causing slight vasodilation, both of which may contribute to the effectiveness of treatment.

The electrodes must be placed so that the currents cross one another in the target tissue. In Figure 3.26 the current of the first circuit is carried via electrodes I’ and j2 and that of the second circuit by electrodes TI’ and 112, thus generating the interferential field in the deep tissue. The electrodes are positioned in a coplanar arrangement to treat a flat surface such as the back. It is normally recommended to use the largest electrode sizes that can conveniently be applied (Savage, 1984) in order to ensure a comfortable current of sufficient intensity throughout the treated area. The leads (and suction tubes) are colour-coded to ensure correct arrangement of the circuit.

It is also possible to use only two electrodes; this is called the bipolar mode. In this case the two medium-frequency currents are superimposed within the machine, so that the single current produced is amplitude-modulated already. The result is that interference occurs throughout the region between the two electrodes. The modulation is always 100%. For this type of application the two electrodes should be placed opposite one another so that the part to be treated lies between them. Because the current modulation occurs throughout the area, including the superficial tissues and skin, there tends to be more sensory stimulation than with the four-pole technique, although still less than with low-frequency stimulation.

**Precautions**

It would seem wise to wash the skin before treatment to reduce skin impedance and to use large enough (sponge) pads to ensure that neither the electrodes nor their connections touch the patient’s skin. This obviates any risk which could occur with high current densities.

It has been estimated that interferential currents of 50 mA applied to the thorax could induce currents of sufficient intensity to cause ventricular fibrillation.
Comments on the effects

Different frequencies and ranges of frequencies are recommended, often for the treatment of similar problems. The appropriate dosage parameters are not universally agreed. Some researchers (Laycock and Green, 1988) have found that 2 kHz is preferable for muscle strengthening to a 4 or 5kHz carrier frequency. Most commentators suggest a range of amplitude modulation frequencies for different effects (see Savage, 1984; Goats, 1990; De Domenico, 1982). Hogenkamp et al. (1987) however postulate that amplitude modulation frequency has no effect on the selective stimulation of thick nerve fibres, but only determines the frequency with which nerve fibres depolarize. They suggest using a high AMF (70—150 Hz) for acute problems and pain, and frequencies below 50Hz for chronic and subacute conditions and where muscle contraction is required.

Interferential currents are frequently employed for pain relief by means of mechanisms already described and summarized below:

1 Activation of the pain gate mechanism due to stimulation of large- diameter, low-threshold nerve fibres with high-beat frequencies — around 100 Hz.
2 Activation of A delta and C fibres causing encephalin and endorphin release, probably activated by low frequency (10—25 Hz).
3 It is possible that high-frequency (above 50Hz) stimulation may lead to temporary physiological block of finely myelinated and non-myelinated nociceptive fibres.
4 The local increased fluid flow and fluid exchange consequent on mild muscle contraction and possibly stimulation of autonomic nerves may help to remove chemical irritants affecting pain nerve endings and reduce local tissue pressure. When interferential therapy is applied in this way, the varying suction could also contribute to this effect. Savage (1984) states that frequencies of 10—150 Hz stimulate the parasympathetic nerves increasing blood flow through the area, and 0—5 Hz the sympathetic nerves. Nussbaum et al. (1990), however, concluded that interferential therapy does not cause vasodilation.
5 A placebo effect, which occurs in all treatments, is likely, especially since interferential machines are technically impressive and produce a distinct, somewhat unusual but not unpleasant sensation.

While there are many claims that interferential current is effective as a pain-relieving treatment there is little objective evidence in support. A rise in the pain threshold, based on the time taken to elicit ischaemic pain, and changes in the strength—duration curves of muscle were found after interferential therapy in one study (Partan et al., 1953). Other studies have found no change in nerve conduction velocities after interferential and no significant difference between interferential and placebo treatments.
Muscle contraction can also be achieved with the lower range of interferential frequencies. Strong muscle contractions can be achieved without any significantly uncomfortable skin sensation. It is often used in the treatment of stress incontinence McQuire (1975) recommended a frequency swing of 0—100 Hz, with 2 vacuum electrodes positioned on the lower abdomen and 2 on the upper medial aspect of the thighs. In 1988, Laycock and Green suggested a bipolar technique, placing one electrode under both ischial tuberosities and one over the anterior perineum immediately’ inferior to the symphysis pubis for females. Male patients were treated with 2 electrodes placed either side of the gluteal cleft, under the ischiál tuberosities, anterior to the anus. Subsequently, Laycock and Jerwood (1993) reported using a medium electrode over the perineal body and a small electrode immediately inferior to the symphysis pubis. They used 3 specific frequencies for 10 mm each, and found a significant increase in strength in the interferential therapy group; however, there was also improvement in a number of patients receiving placebo treatment, showing a strong placebo effect for interferential procedures.

A study of stress incontinence in the UK (Mantle, 1991) found that, after pelvic floor exercises, interferential was the most widely preferred treatment, but the methods and parameters of application varied widely.

SAFETY WITH ELECTRICAL CURRENTS

Primuni non nocere — firstly do no harm — is a central tenet of every therapy but is especially applicable where damage can easily occur. Damage to the tissues as a result of the passage of electrical currents cart occur in three ways:

1 Direct or uniphasic currents can cause electrochemical damage, often called a chemical burn (see Chapter 2).
2 Currents varying at rates that will stimulate nerve or muscle, that is, all those currents discussed in this chapter, could cause damage by provoking excessively powerful or prolonged muscle contractions or more seriously the heart muscle, thus stopping the circulation.
3 Currents of sufficient intensity can cause heating in the tissues leading to a heat burn.

All these kinds of damage could occur together but serious shocks or fatalities are usually confined to the last two points. if large currents are passed it is called an electric shock. In all cases what matters is the electric charge, that is, the current intensity and the length of time for which it flows.

Damage due to the therapeutic use of electricity is, in fact, extremely rare and most damage that does occur is due to the mains current and is of the kind that may equally well take place in the home with ordinary domestic equipment.
It is usual to distinguish macroshock, in which current passes through the skin, from microshock which refers to the very small currents which are applied directly to, say, the heart as an external pacemaker; in this situation quite small current increases can be fatal. This latter will not be considered further.

It will be apparent from what has been described earlier in this chapter that quite high currents can be passed through the body without ill effects provided they are applied as very short pulses (Fig. 3.13). As a general observation electrotherapeutic equipment is designed so that it cannot deliver pulses of sufficient intensity to be seriously damaging to the tissues, except as considered below.

**Electric shock due to mains-type current**

If an electric current is passed through the whole body it tends to spread throughout the low-resistance subcutaneous tissues. It will be recalled that the skin resistance here is very much greater than that of the internal tissues. Normal skin resistance is many thousands of 5.2, but wet skin can be as little as 10005.1; the internal tissues have resistances of only a few hundred (1; for instance, the resistance between hand and foot excluding the skin resistance is about 50012 (Ward, 1986).

The current through the body — it is the current which is the critical factor — will depend directly on the voltage (240 V root mean square (RMS) in the UK) and inversely on the resistance (1= V/R), where I= intensity, V= voltage and R= resistance. Thus very much larger currents will flow if the skin is wet, because wetting greatly lowers its resistance; this explains why serious domestic accidents invoking electrocution often occur in the bathroom or laundry. Applying Ohm’s law the current through a 1000 5.2 resistance due to 240 V would be 240 mA which is enough to cause ventricular fibrillation in the heart muscle and could well be fatal. With dry skin having a resistance of, say, 100 0002 the current would be 2.4 mA, causing tingling sensations. Most of the therapeutic currents described in this chapter are applied to wetted, hence low-resistance, skin but are driven by voltages much lower than that of the mains; the maximum output from many faradic-type sources is around 40 or 50 V.

At mains frequency a current density 0.5—1 mA/cm² is just detectable and currents around 10 mA cause discomfort or pain. Above this level the current can cause strong painful muscle contractions and at higher levels (50—250 mA) may lead to ventricular fibrillation and hence may be fatal. Currents of still higher intensities tend to provoke complete cardiac arrest and severe heat burns (Ward, 1986). Thus it can be seen why the nature of the electrical contact with the skin is so critical; anything that lowers skin resistance allows larger currents to pass. Voltages higher than that of the mains are proportionally more dangerous because they will cause larger currents; for this reason areas in which high voltages are found, such as transformer substations, are specially protected. It must also be understood that the consequences of electrocution depend on the path the current follows in the body. Thus if the current passes from, say, a hand touching the live wire through the body to earth via the feet.
standing on the ground, the current then passes through the heart, lungs and abdomen and may well cause cardiac arrest and/or the cessation of respiration and thus prove fatal. Touching both contacts of a lighting socket with one finger would be likely to cause a painful shock and burns to the fingertip.

**Immediate treatment of mains current shock**

Firstly, the circuit must be disconnected to stop the flow of current through the victim. This may be simply a matter of switching off and unplugging, but it is important to ensure that disconnection has been made before touching the victim otherwise the rescuer may form another path to earth and also receive a shock.

Secondly, the carotid pulse and respiration should be checked. If absent the airway must be cleared, mouth-to-mouth resuscitation and external cardiac massage immediately started and assistance summoned. In all cases the victim should be medically examined as soon as possible — urgently if there has been any loss of consciousness. Musculoskeletal damage can occur as a result of abrupt muscle contraction due to the electric shock as well as cutaneous burns, which are usually evident.

**Safety features of electrical apparatus supplied from the mains**

The safety of electrical apparatus connected to the mains is ensured in several ways. The metal casing of the apparatus is connected to the large earth terminal of the three-pin plug and socket. Thus if the live wire were to touch the casing of the machine a large current would flow to earth causing the protective fuse to ‘blow’ and interrupting the circuit. If an earth wire is not present, or if it is broken, and the casing becomes live, anyone touching the machine may provide a low-resistance pathway to earth.

Small portable pieces of electrical equipment such as radios and hair-driers are often double-insulated and connected to the mains by only two terminals, live and neutral. In these the casing is made of some non-conducting plastic material and the electrical conducting parts are separately insulated. Any exposed metal, such as the cutters of electric razors, is again separately insulated. All mains equipment is protected in one of these ways.

Two further safety measures are of consequence: the use of isolating transformers and core-balance relays. The way in which these and the 3-wire fused mains system works are considered in Physical Principles Explained (Low and Reed, 1994).

The safety of electromedical equipment is subject to recommendations made by the British Standards Institution (BSI) and the International Electrotechnical Commission (IEC). These bodies specify certain safety conditions such as the maximum permissible leakage current both normally and under fault conditions and designate the equipment accordingly. Most of the electrical stimulators referred to in this chapter (designated type BF) would have more stringent protection against delivering an electric shock than equipment to which the patient is not directly connected, electric heating pads for example, but less than the very
strict precautions taken for equipment directly connected to the heart such as external cardiac pacemakers (designated type CF). The regulations are covered by 85 5724 and its amendments and supplements (section 2.10, 1988).

**Electric shock or damage due to therapeutic nerve and muscle-stimulating currents**

As already noted, these do not usually cause damage because the output of the machine will not permit a sufficiently high current. However, certain situations could lead to pain, alarm and possibly tissue damage.

If current is applied at high intensity very abruptly the sensation and pain caused are likely to frighten the patient. It will be recued that the sensory nerves are stimulated by lower intensities than the pain nerve endings, so that high currents are more painful. Some pain may well be an appropriate part of treatment (see p. 78), but it should never provoke anxiety or fear. It has already been emphasized that the major resistance to current flow is the skin so that any break in the skin provides a low-resistance pathway and therefore a high local current density, which may be painful. Subjects become accustomed or accommodate to electrical stimulation so that high intensities are easily tolerated if the current is increased gradually over a few minutes. The term ‘shock’ is often used confusingly; any sudden sensory stimulation may be described as a ‘shock’ (psychological); this would include electric ‘shock’ which refers specifically to electrical injury.

There is quite widespread apprehension about contact of the body with electricity. This is doubtless partly a fear of the ill understood. However, it is also culturally engendered in many places to suggest fear (and vitalize monsters!) in old films and new videos. There are even allusions to it in literature; for example, in Shelley’s Epipschidion, ‘Her touch was as electric poison’. These anxieties may, at first, cause the sensations due to electric currents to be perceived as painful. As current flows so the feelings become familiar and toleration rises. When applying treatment such fears must be taken into account; the patient must be carefully reassured and treatment applied initially at low intensity which can be gradually increased. It has been noted already that the current density is important in determining the strength of the effects, so the area of skin/electrode contact must be carefully considered. Furthermore, there appear to be very wide differences in sensory responsiveness to electrical stimulation. It has been found that, in general, women appear to have lower thresholds for ‘detection, pain and tolerance to cutaneous electrical stimulation than men’ (Lautenbacher and Rollman, 1993). This is presumably associated with the fact that women are more sensitive to pressure pain than men (Fischer, 1987), although there are no differences between the sexes for thermal thresholds or heat pain. These factors account for the wide discrepancies found among patients and therapists concerning the painfulness or otherwise of particular intensifies of therapeutic current. It may be emphasized that the pain due to electrical stimulation is not associated with, or due to, any tissue damage (except as noted below) but is the result of sensory, including pain, nerve stimulation and is thus harmless.
As these currents can cause strong muscle contraction it is possible that exceptionally vigorous artificially produced contraction might cause mechanical muscle or joint damage. However, such damage seems unlikely in normal tissues because of the protective mechanism of the Golgi tendon organs, and the fact that electrical muscle stimulation rarely produces a greater contraction than can be produced voluntarily. Further, there seems to be no recorded instance of such injuries happening to normal muscles and joints. With tissues that are already diseased or injured, such as partial muscle rupture, overstrong muscle contraction could be damaging. It is also possible that muscle contraction could dislodge the attachment of a deep vein thrombus, causing an embolus. Prolonged and intensive electrical muscle stimulation can lead to muscle soreness like that due to voluntary activity (Hon Sun Lai et al., 1988).

It is recommended (Wadsworth and Chanmugan, 1980) that infected or inflamed areas should not be treated with low-frequency currents because there is a risk of spreading the infection. This is presumably due to the muscle-pumping effect; there seems no other reason why infection should spread. Certainly, as far as acute inflammation is concerned, any increased activity would be undesirable. Since these currents provoke nerve impulses it is often recommended that areas in which autonomic nerves might be inappropriately stimulated should be avoided; for example, the region of the carotid sinus (Frampton, 1988). Similarly, treatment close to the pregnant uterus might provide undesirable uterine movements. While neither circumstance appears to have been reported it would seem proper to be cautious.

If haemorrhage, either on the surface or in the tissues, is occurring or likely to occur then electrical stimulation by causing muscle movement and vasodilation could prevent or disrupt clotting; it should therefore be avoided. Although there appears to be no evidence for this effect it is usual and reasonable to avoid direct treatment of neoplastic tissue in case metastasis is provoked, or growth encouraged.

Currents applied in the region of an implanted cardiac pacemaker could alter the stimulus leading to cardiac arrhythmia. A separate slight risk is of the electromagnetic field generated by the therapeutic stimulator interfering with demand-type pacemakers, this is a remote possibility with almost any piece of electrical equipment.

A significant danger arises from failure to recognize when the current applied is a direct current, or has a direct current component with sufficient charge an electrochemical bum can result. Even extremely low current densities, such as that from some TENS machines, can have this effect given sufficient time. In most circumstances the patient’s sensation warns of this danger and nothing more than skin irritation occurs. Electrochemical burns seem to develop because the sensations of burning and pain experienced by the patient due to the current are not particularly sharp. There may also be a gradual increase of current, due to falling skin resistance, to which the patient accommodates. It is therefore important that adequate explanation and warning should be given to the
CONTRAINDICATIONS
Contraindications to electrical stimulation may be summarized as circumstances in which:

1. Strong muscle contraction might cause joint or muscle damage; detachment of a thrombus; spread of infection, and haemorrhage.
2. Stimulation of autonomic nerves might cause altered cardiac rhythm or other autonomic effects.
3. Currents might be unduly localized due to open wounds or skin lesions, e.g. eczema.
4. Currents might provoke undesirable metabolic activity in neoplasms or in healed tuberculous infections.
5. Current is not evenly biphasic, leading to possible skin damage or irritation, especially if there is loss of sensation.

REFERENCES
Bending J. (1993). TENS relief of discomfort ‘like worms wriggling under the skin’.


Lautenbacher S., Rollman C. B. (1993). Sex differences in responsiveness to painful and non-painful stimuli are dependent upon the stimulation method. Pain, 53,


