CHAPTER 21

Properties, Classification of Nerve Fibres and Nerve Action Potential

Competency achievement: The student should be able to:
PY3.2: Describe the types, functions and properties of nerve fibres.
PY3.3: Describe the degeneration and regeneration in peripheral nerve.
PY3.8: Action potential in skeletal muscle.
PY3.17: Describe the strength duration curve.

INTRODUCTION: PROPERTIES OF NERVE FIBRES

Nerve fibres show the following properties:
1. Excitability
2. Conductivity
3. All-or-none law
4. Refractory period
5. Summation
6. Adaptation
7. Accommodation
8. Indefatigability

1. EXCITABILITY

The nerve fibre can be stimulated by a suitable stimulus, which may be mechanical, thermal, chemical or electrical. In experiments, electrical stimulation is usually employed, because its strength and frequency can be accurately controlled.

The following changes will show that a nerve has been excited:

On stimulation of a nerve by a threshold stimulus will generate an action potential and this wave of negative potential passes along the nerve and can be detected by galvanometer or by CRO.

Generation of Action Potential and Excitability of the Nerve

Key Points

Phases of Action Potential

1. **Resting potential:** An electrical disturbance always accompanies the travelling nerve impulse. In resting cell, the surface is positively charged and the interior is negatively charged. When the surface is stimulated and the permeability is increased, as a result, there is reversal of polarization. The surface at the stimulated point becomes negative (cathode) causing catelectrotonic change.
2. **Depolarization:** When this change rises to threshold level, impulse will pass like self-propagated disturbance by drawing positively charged particles from the neighbouring points which in turn become cathode. The depolarization of the membrane is the first step of the manifestation of an impulse. After an initial slow rise, depolarization wave overshoots rapidly and reaches up to the potential line (zero line) to approximately +35 mV.
3. **Repolarization:** After that it reverses and begins to fall very rapidly towards the resting level (–70 mV). At approximately two-thirds of repolarization, the rate of fall is being abruptly slowed.

   This slower fall is known as negative after potential (after-depolarization). The rapid rise of depolarization wave and the rapid fall of repolarization wave are known as spike potential.
4. **Hyperpolarization:** After reaching the basal level the wave overshoots slightly but slowly in the hyperpolarizing direction. This is known as positive after-potential (after-hyperpolarization). The whole sequence of potential changes in the nerve following excitation is known as action potential or membrane potential (Fig. 21.1).

Ionic Basis of Excitability of Nerve

Key Points

1. **Resting state:** In resting state, the nerve fibre remains in polarized state and the membrane potential lies within –70 mV. The inside of the nerve is negative and the outside of the nerve is positive (Fig. 21.2). Na^+ concentration outside the membrane is higher than that of inside the membrane. K^+ concentration inside the membrane is also higher than that of outside the membrane. K^+ can permeate through the membrane at resting state but the Na^+ cannot permeate.
2. **Depolarization (excitability):** Permeability of Na^+ to membrane is increased only after excitation and

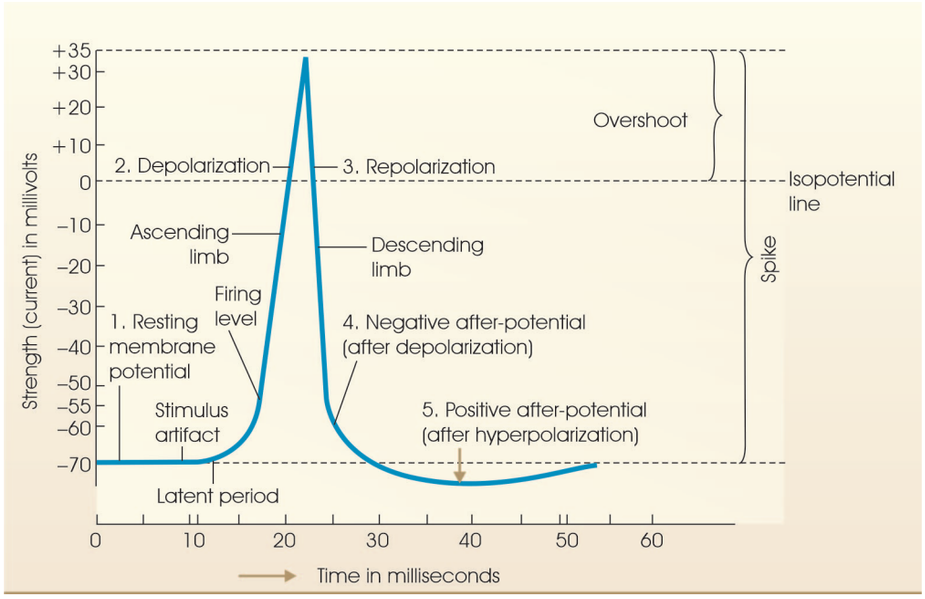


Fig. 21.1: Diagrammatic representation of spike potential (action potential) recorded with the help of microelectrode in the nerve cell

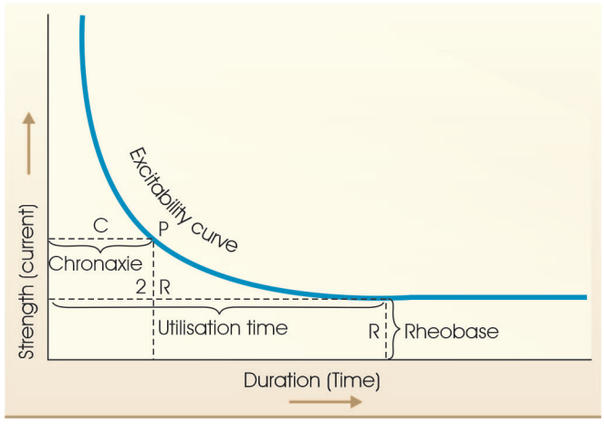


Fig. 21.2: Diagrammatic representation of excitability curve (strength–duration curve) applied to an excitable tissue producing response relating the strength of stimulus

it is the first event of the action potential. The threshold stimulus leads to influx of sodium through leaky channels and via the opening of the voltage-gated sodium channel. The membrane potential decreases from –70 to –55 mV.

As the depolarization proceeds further, a large number of voltage-gated channel opens. So the depolarization starts with the onset of Na^+ entry and thus an increase in Na^+ conductance is taken place. The tremendous increase in Na^+ conductance during this period is known as activation of membrane produce large and sweep depolarization and the membrane potential reaches to +35 mV. Thus, the *reversal* of potential is caused with the development of positivity inside the membrane and negativity outside. The Na^+ sodium influx stops due to inactivation of gates of sodium channel. The sodium channel remains open for very brief period of time. Thus, this speedy closure produces auto-deactivation of the sodium channel. The voltage-gated K^+ channels fully open at +35 mV causing efflux of K^+ ions. Repolarization starts as voltage-gated K^+ channels open and potassium ions efflux starts over. The action potential termination as a result of activation of voltage-gated potassium channel is a negative feedback process mechanism.

3. **Repolarization:** But as soon as the action potential attains the voltage approximately +35 mV, K^+ efflux out from inside the membrane. The inside of the membrane becomes negative and outside becomes positive again. This stage is the repolarization phase and K^+ conductance is increased to the maximum. But at the later period of this phase (at the termination of spike potential) K^+ conductance is slowed down as potential returns to resting level.
4. **After depolarization:** But at the later period of this phase (at the termination of spike potential) K^+ conductance is slowed down. As the membrane potential reaches to iso-potential level and as it is reaching towards the resting membrane potential, the inside of the membrane achieves negativity; this limits efflux of potassium ions. Thus, a few milliseconds are delayed in restoring the membrane potential. This state is known after depolarization phase-potential and is attributed to slow efflux of potassium ions. In the later phase of repolarization the sodium channel is closed and then its inactivation gate opens slowly while the K^+ channel begins to close and gradually is completely closed. Thus, as membrane reaches resting state the activation gates of sodium channel are closed while inactivation gate of sodium channel opens.

5. **After hyperpolarization:** This increased negativity inside hinders further efflux of K^+. Most of the voltage-gated K^+ channels are closed, but as some of the voltage-gated K^+ channels are about to close the efflux continues and membrane potential becomes more negative producing the phase of after hyperpolarization. The resting membrane potential is yet to be achieved. **Achieving resting membrane potential:** It is achieved by the complete closure of voltage-gated K^+ channel. The resting ionic composition is restored by the active Na^+-K^+ pump mechanism (increased activity of Na^+-K^+-ATPase). **In this way resting normal ionic status is established.**

Competency achievement: The student should be able to:
PY3.17: Describe strength duration curve.

Excitability Depends upon Following Factors

1. **Strength of stimulus** (Fig. 21.2): A minimum strength is essential to excite a tissue.
   Strength-intensity of stimuli: Current intensity of stimulus which is just adequate to cause an impulse is called the threshold. Intensity below the threshold is known as subliminal. Magnitude of current just sufficient to excite a nerve or muscle is called rheobase.
2. **Duration of stimulus** (Fig. 21.2)**:** The stimulus must continue for a certain minimum period, which varies inversely as the strength. The minimum time required to have a response is known as utilization time. The shortest duration of current flow which will excite the nerve or muscle under current strength equal to twice the rheobase is called the chronaxie. Chronaxie value is a useful index of the relative excitability of the tissues.
   Excitability of a nerve fibre can be determined by studying its strength–duration relationship (threshold stimulus intensity and duration) of the stimulus. To obtain an excitability curve (strength–duration curve), a minimum current strength for exciting a nerve or muscle is first determined and chronaxie is then obtained by determining the shortest duration of stimulus with double the rheobase voltage (Fig. 21.2).
3. **Direction of the current:** If the current is passed transversely across the nerve, no effect will be produced. When it passes along the length of the nerve, there is the maximum chance of stimulation.
4. **Frequency of stimulus:** A single stimulus will generate a nerve impulse, but if the stimulus be strong, more than one impulse may follow. Ca^{++} lack increases this tendency of multiple responses.

Compound Action Potential

Action potential recorded from a group of nerve fibres (e.g. sciatic nerves) or a nerve trunk is called compound action potential as it is the summated action potentials of different types of nerve fibres having different conduction velocities (Fig. 21.3). The multi-peaked shape of the compound action potential is due to the activity of the different nerve fibres of varying conduction velocity. Most nerves are composed of myelinated nerve fibres of various diameters and also unmyelinated fibres of quite large number. The results obtained by stimulating one end of a frog nerve and recording from a point as far away as possible were described by Erlanger and Gasser.

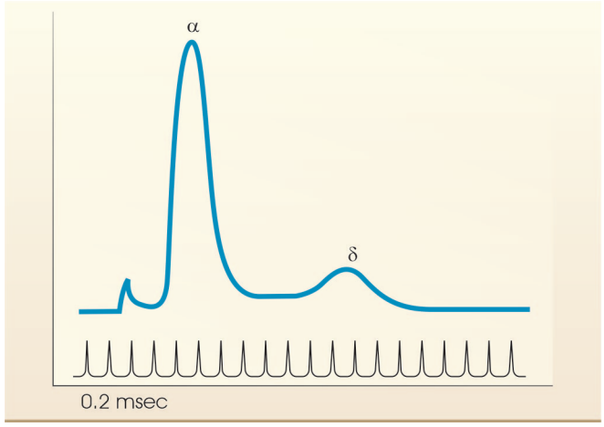


Fig. 21.3: Compound action potential

With a large shock, the action potential appears as in Fig. 21.4. It is conventionally split into three waves called A, B, C. The A wave itself is divided into α, β, γ and δ sections (Fig. 21.5).

By adjusting both the stimulus strength and the pressure it is thus possible to record any component of the action potential. For example, if it is desired to fire γ fibres along the stimulus, strength will be increased until α, β and γ waves appeared and then knock out the β components by application of a pressure block.

2. CONDUCTIVITY

The nerve impulse is conducted along the nerve fibre. Conductivity shows the following characteristics:

1. Impulse is propagated normally from synaptic junction to axon terminal and is referred to as orthodromic conduction. While conduction in reverse direction is referred to as anti-dromic conduction as observed in sensory nerve supplying the blood vessels.
2. ***Velocity of nerve impulse:*** The nerve impulse is propagated with a definite speed (other conditions remaining same). The conduction velocity depends upon the diameter of the nerve fibres, the thicker fibres showing higher velocity. The conduction velocity also depends upon the presence or absence of myelination and also on temperature.
   - *Myelination:* The conduction velocity of myelinated fibres is proportional to the diameter of nerve fibres. The conduction rate in msec is approximately 6 times the fibre diameter in microns in fibres larger than 3 μm. The unmyelinated fibres have a conduction velocity proportional to the square root of the diameter of

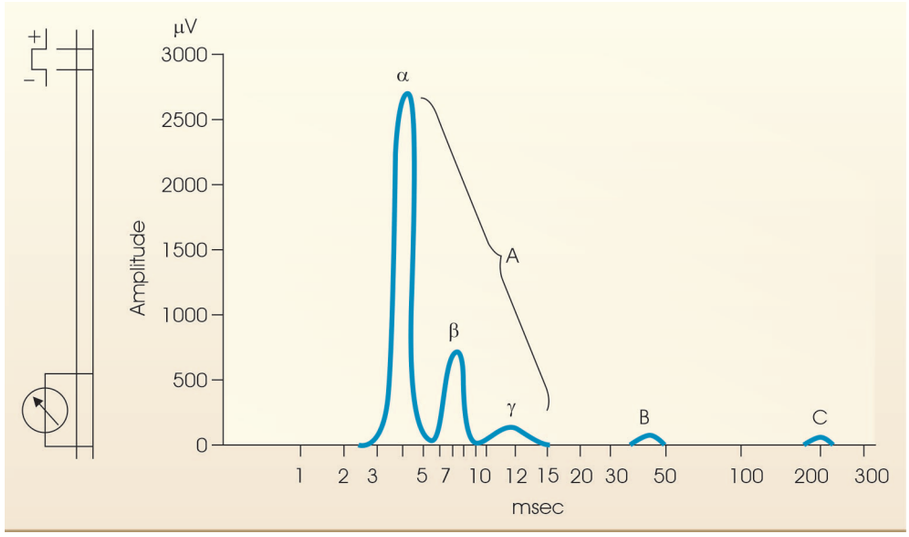


Fig. 21.4: A compound action potential as might be recorded from any nerve containing both myelinated and unmyelinated fibres. The A wave represents myelinated fibres and the C wave unmyelinated ones. The B wave may be mixed

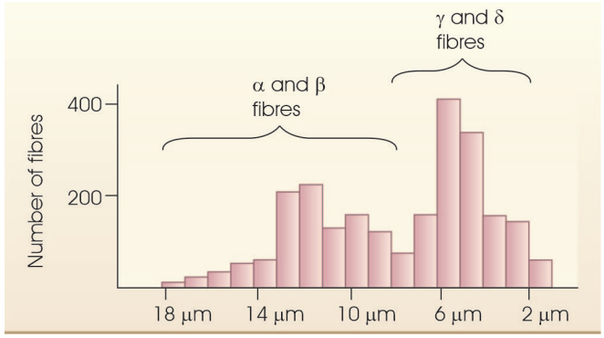


Fig. 21.5: The graph showing the numbers of myelinated nerve fibres of different diameters in nerve

nerve fibres. With a diameter of 1 μm the conduction velocities are approximately the same. Below 1 μm unmyelinated fibres have a faster conduction rate than myelinated fibres. Thus to conclude conduction rate is faster in myelinated nerve.

3. ***Temperature*** has got an immense role in conduction of nerve impulse. In the cold-blooded animal conduction velocity is lower than those of the warm-blooded animal.

Conduction velocity calculation: The conduction velocity of a bundle of nerve fibres can be studied by stimulating the nerve bundle at one end and recording compound action potential at other end. If the length of nerve in mm from the placements of the stimulating electrodes to the recording electrodes is noted, then the conduction velocity V (msec) can be calculated easily by diving this length of nerve fibres (L) with the latency (in msec) of action potential: V (velocity in msec) = L (length of the fibre in mm)/latency in msec (Fig. 21.6). In general, greater the diameter of the nerve fibre, higher is the velocity of conduction.

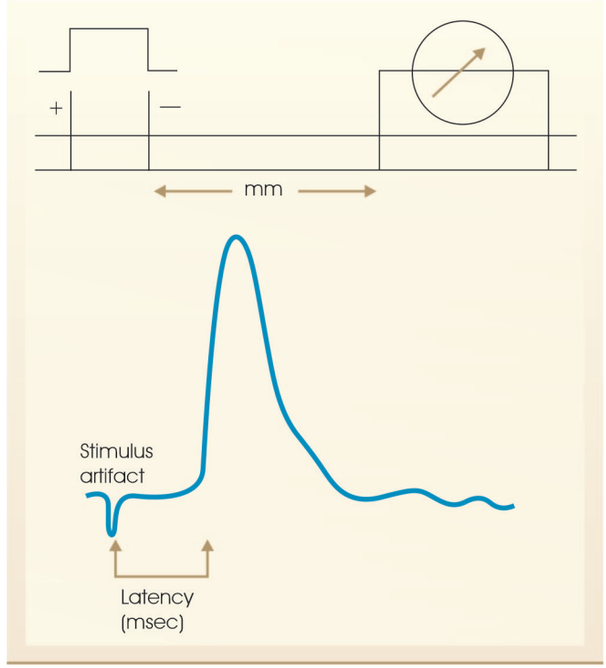


Fig. 21.6: Calculation of conduction velocity

The conduction velocity of peripheral nerve fibres in man has been studied intensively and it lies in between 60 and 120 msec.

Factors Affecting Conductivity and Excitability

1. Temperature—cooling diminishes and warming increases these properties.
2. Mechanical pressure—depresses conductivity and excitability.
3. Blood supply—if blood supply be cut off both these properties of conductivity and excitability are lost.

4. Chemicals—CO_2 and narcotics, viz. ether, chloroform, alcohol, Novocain, etc. diminish and finally abolish excitability and conductivity.
5. H^+-ion concentration—increased pH (alkali) increases and decreased pH (acid) diminishes conductivity and excitability.
6. Increased Ca^{++} diminishes and decreased Ca^{++} increases conductivity and excitability.
7. O_2 lack—depresses conductivity and excitability and if continued abolishes these properties. If O_2 is readmitted, they return.

3. ALL-OR-NONE LAW

If the stimulus be adequate, a single nerve will always give a maximum response. If the strength or duration of the stimulus be further increased, no alteration in the response will take place. This property is present in single fibre preparation. In the whole nerve this property is different.

4. REFRACTORY PERIOD

When the nerve fibre is once excited, it will not respond to a second stimulus for a brief period. This period is called absolute refractory period. The absolute refractory period means that the nerve is completely refractory to stimulation—in other words, it is incapable of eliciting an action potential at any intensity of stimulation. During the absolute refractory period there is total inactivation of the sodium carrier mechanism and as the Na^+ ions cannot enter the fibre, there is no development of the action potential. Immediately following this, there is a brief relative refractory period, during which the excitability is subnormal but gradually rising. This is succeeded by a third brief period of increased excitability, known as supernormal phase. Lastly, there is a period of subnormal excitability—subnormal phase. Figure 21.7 demonstrates the different refractory periods.

When two stimuli are applied to one end of a nerve at different time intervals, compound action potentials are recorded and two different action potentials are recorded (I, II). But as the time intervals are gradually decreased, a time will come when the second shock will fail to produce compound action potential of equal length and the height will be decreased (III, IV). This is happened in absolute refractory period. But if the time interval is further decreased, then the second stimulation will fail to elicit any response. This period is the absolute refractory period (V).

Note

In the large mammalian nerve fibres the durations are as follows: Absolute refractory period: 2 to 3 milliseconds in frog but in mammal it is 0.5 millisecond. Relative refractory period is 10 to 30 milliseconds in frog but in mammal it is 3 milliseconds. Supernormal phase is 12 milliseconds and subnormal phase may be up to 70 milliseconds.

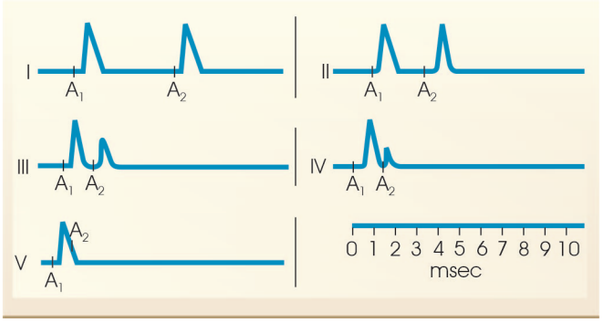


Fig. 21.7: Diagram shows the nature of action potential recorded by successive stimuli at different levels

5. SUMMATION

Applying a subthreshold stimuli will produce no response or generate action potential. When subthreshold stimuli are applied in rapid successive manner, the effect will get summated producing an action potential.

6. ADAPTATION

The nerve fibre quickly adapts itself. Due to this adaptation there is no excitation during the passage of a constant current. Only when the strength of the current is suddenly altered or the current is made or broken, excitation takes place. A gradual change will fail to excite.

7. ACCOMMODATION

When a continuous stimulus is applied, it decreases the excitability of the nerve fiber. This phenomenon is called accommodation. The accommodation is at level of nerve fiber and more specifically at nerve ending. The transmission of impulse at the nerve ending is decreased. Moreover, when a nerve is stimulated with constant current strength, the site becomes less excitable as there is rise of threshold for its stimulation.

8. UNFATIGABILITY

Even if the nerve is stimulated repeatedly, they are not fatigued as these nerve fibers primarily conduct impulses which do not expend energy in propagation of action potential.

HEAT PRODUCTION IN NERVE FIBRE

As already mentioned the metabolism in the nerve fibre is very low. During rest a minute quantity of heat is produced which increases during activity. Heat is evolved in three phases: The first phase is called initial heat and the other two phases are seen during recovery and therefore known as the recovery heat or the delayed heat.

Initial heat: It is about 10% of the total heat (5–10 microcalories per second per gram of nerve fibre) but the

rate of evolution is very brisk being 5000 times greater than that of delayed heat. It is anaerobic and coincides with the spike potential. Its cause may be the breakdown of ATP, creatine phosphate or, due to the discharge of an electric double layer located at the surface of the nerve fibre.

Delayed heat: It is aerobic and is 8.5 times more than the initial heat. This energy is used for the resynthesis of ATP and creatine phosphate and as such, for restoring the normal excitability of the nerve fibre.

It comes in **two phases:**
The *first phase* lasts for a few seconds and the quantity of heat is small and is about the same as the initial heat.
The *second phase* may last for 10–30 minutes and contributes the greatest proportion of both total and delayed heat.

Key Note

- Increase in the strength of the stimulus does not raise heat production. But increased frequency increases about 25%.
- It is to be noted that heat production in the grey matter (nerve cell) is enormously greater than that in the nerve fibre.

Classification of Nerve Fibres

Nerve fibres have been classified in different ways:
1. **Histologically:** Medullated and non-medullated
2. **Functionally:** Motor (efferent) and sensory (afferent).
3. **Chemically:** Adrenergic (producing norepinephrine) and cholinergic (producing acetylcholine).

According to diameter and conduction velocity (Erlanger and Gasser): The physiological properties of nerve fibres vary with their diameter and conduction velocity. Thicker the fibre, higher will be the impulse velocity and spike potential but lower will be the refractory period and stimulus threshold (chronaxie). Erlanger and Gasser have divided the nerve fibres into A, B and C.

On systematic examination of the compound action potential of various nerves, it reveals that:
1. A fibres are myelinated, somatic, afferent and efferent axons.
2. B fibres are pre-ganglionic, myelinated, efferent, and sympathetic axons.
3. C fibres are sympathetic and somatic, unmyelinated axons. The C fibres are differentiated into two groups—the sC and drC on the basis of differences in their after-potential. The drC group has got no negative after-potential. C groups of fibres are efferent, post-ganglionic sympathetic axons and the drC groups of fibres are the small afferent axons found in peripheral nerves and dorsal roots.

In peripheral somatic fibres, both A and C fibres are present. If such fibres are stimulated at one end and recorded through oscilloscope at other end, then the compound action potential formed in A fibres is of four different deflections—α, β, γ and δ. These different deflections are due to corresponding stimulation of different fibres of different conduction velocities.
- α deflection is due to stimulation of nerve fibres having comparative larger diameter with higher conduction velocity.
- δ deflection is due to stimulation of fibres having lowest diameter and slowest conduction velocity.

Tables 21.1 and 21.2 and Flowchart 21.1 depict classification according to conduction velocity and diameter of the nerves.

MECHANISM OF CONDUCTION OF THE NERVE IMPULSE

According to the membrane theory, the nerve impulse is a propagated wave of depolarization. As soon as the fibre is excited at a point the polarity is changed and for a brief period, it is actually reversed. This reversed polarity is due to increased permeability of Na^+ to the membrane and this depolarization of wave is developed. A local circuit current flows between the depolarized membrane and the resting membrane areas. Positive current flows inward through the depolarized membrane and outward through the resting membrane and in this way circuit is completed. This local depolarization current then excites the adjacent portions of the membrane producing progressively more and more depolarization. The depolarization wave travels in all directions along the

Table 21.1: Numerical classification of nerve fibres

Types of fibre	*Diameter of fibre in μm*	*Velocity of conduction in msec*	*Duration of spike potential in msec*	*Absolute refractory period in msec*	*Function*
A-α	12–20	70–120	0.4–0.5	0.4–1.0	Proprioception; somatic motor
A-β	5–12	30–70	—	—	Touch, pressure
A-γ	3–6	15–30	—	—	Motor to muscle spindle
A-δ	2–5	12–30	—	—	Pain, temperature
B	Less than 3	3–15	1.2	1.2	Preganglionic sympathetics
C dorsal root (drC)	0.4–1.2	0.5–2.0	2.0	2.0	Pain reflex response
C sympathetic (sC)	0.3–1.3	0.7–2.3	2.0	2.0	Postganglionic sympathetics

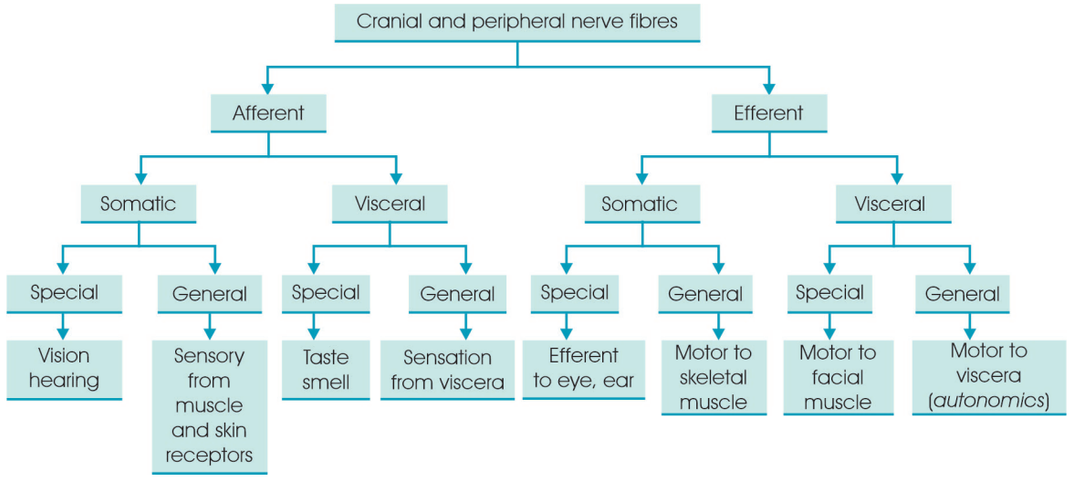
%Numerical classification of sensory nerve fibres: Sometimes sensory nerve fibres are numerically classified and have been presented along with Erlanger and Gasser latter system in the following Table.

Table 21.2: Classification of nerve fibres on physio-chemical basis

Group	*Letter system*	*Origin*	*Reflex response*	*Central reflex connection*	*Destination*
Ia	A-α	Muscle spindle: Annulo-spiral spindle ending	Myotatic reflex of muscle (tendon jerk)	Monosynaptic with motor neuron of muscle of origin	Spindle of extensor of flex or muscle
—	—	—	Relaxation of antagonist muscle during myotatic contraction of agonist muscle	Disynaptic with antagonist motor neuron	Antagonist muscle
Ib	—	Golgi tendon organ	Reaction lengthened	Disynaptic with motor neuron of muscle of origin	Muscle of origin
II	A-β and A-γ	Muscle spindle Flower-spray ending, skin, touch-pressure receptors	Relaxation of extensors and excitation of flexors (flexor withdrawal)	Polysynaptic	Extensor and flexor motor neurons
III	A-δ	Muscle and skin, pain-temperature receptors	Flexion withdrawal of same limb, crossed extensor of opposite limb	—	—
IV	Dorsal root C fibres	Muscle and skin, pain receptors	—	—	—

% On physio-anatomic basis—the peripheral nerves can be classified into afferent and efferent and each of which is again subdivided as presented in Table 21.2 and Flowchart 21.1.

Flowchart 21.1: Classification of nerve fibres on anatomical basis



entire length of the nerve fibre. This type of conduction is observed in the non-medullated nerve fibre (Fig. 21.8).

Repolarization wave occurs from the point of stimulus, a few ten thousandths of a second later than the depolarization wave and spreads progressively along the membrane following same directions as the depolarization has spread previously.

Saltatory Conduction in the Myelinated Nerve Fibre

In the myelinated nerve fibre, condition depends upon a similar pattern of circular current flow. Myelin sheath is an effective insulator. Ions cannot pass through the myelin sheath, and nodes of Ranvier permeate ions to pass through it more easily. That is why the membrane

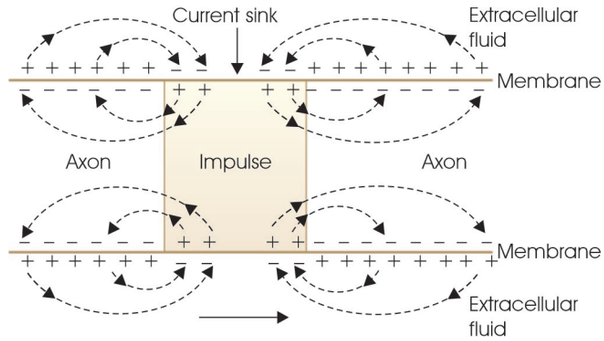


Fig. 21.8: Diagrammatic representation of current flow around an impulse of axon in unmyelinated nerve fibre representing movements of positive charges. A straight arrow indicates direction of propagation

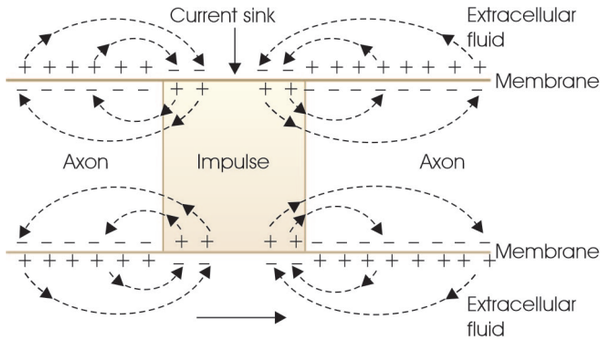


Fig. 21.9: Diagrammatic representation of local current flow around an impulse, an axon in the myelinated nerve fibre (saltatory conduction) representing movements of positive charges. A straight arrow reveals direction of propagation

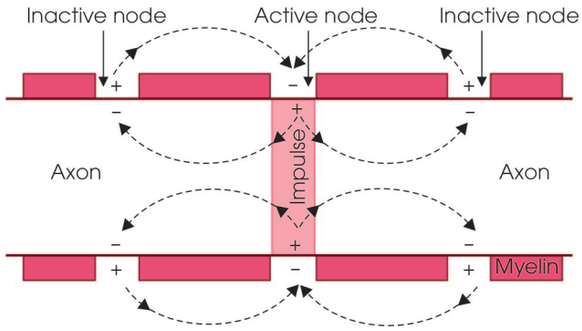


Fig. 21.10: Diagrammatic representation of the saltatory conduction along a myelinated axon. The straight arrow represents direction of propagation

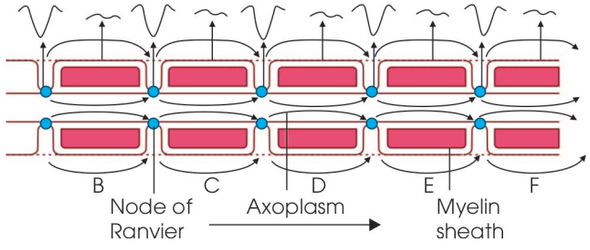


Fig. 21.11: Huxley and Stampfli's experiment to demonstrate the conduction of nerve impulse depending upon action current flowing outside the myelin sheath

at nodes of Ranvier is 500 times as permeable as it is in unmyelinated fibres. For this reason the impulse is transmitted from node to node rather than continuously along the entire length of the fibre (Fig. 21.9). The depolarization in myelinated axon jumps from one node of Ranvier to the next. This jumping or leaping of depolarization from node to node is known as saltatory (saltare = to dance) conduction (Figs 21.10 and 21.11).

The current which remains confined to the nodes depolarizes the internodal part by local circuit action. The myelin sheath increases the velocity of conduction.

PHYSIOLOGICAL PROPERTIES OF THE NERVE FIBRES

Physiological properties of the nerve fibre can be studied through the cathode ray oscilloscope (CRO). Action potential or membrane potential or a single nerve fibre can be studied by microelectrode placed within the nerve fibre and a differential electrode placed outside the nerve fibre. Each microelectrode is a minute capillary glass tube with a tip of 0.25 to 2 microns filled with a very concentrated potassium chloride solution acting as an electrical conductor. The other ends of both the electrode are connected to the cathode ray oscilloscope (CRO). The development of action potential in the nerve fibre is viewed on the oscilloscopic screen of the CRO.

Competency achievement: The student should be able to:
PY3.3: Describe the degeneration and regeneration in peripheral nerve.

Degeneration and Regeneration of Nerve

Nerves injury may occur due to compression, ischaemia, laceration, traction or burning. The damage to nerve may vary in severity. The injury might be transient and quick recovery of lost functions or it may lead to degenerative changes.

Sunderland Classification of Nerve Injury

First degree injury: It constitutes transient ischaemia and neurapraxia, the effects of which are reversible.

Second degree injury: Axonal degeneration takes place, but as the endoneurium is preserved, regeneration can lead to complete, or near complete recovery.

Third degree injury: The endoneurium is disrupted but the perineurial sheaths remain intact and there is limited internal damage. The chances axonal regeneration exist but fibrosis and crossed connections will limit recovery.

Wallerian degeneration: It occurs when a nerve axon is cut, crushed, or frozen.

It is called anterograde degeneration. It is named after Augustus Waller, a neurophysiologist (1816–1870), who first described the process degeneration of injured nerve fibres. Post injury as the axon is disrupted from the neuron's cell body, it degenerates distal leading to Wallerian degeneration. Degeneration usually occurs within a day or two after a nerve injury. The axon's neurolemma is the outermost layer of the neuron made of Schwann cells. It does not degenerate and remains as a hollow tube.

The changes due to nerve injury may progress as follows:

Early changes

1. Synaptic transmission disruption.
2. The cut ends pull apart and seal up, and swell, due to axonal transport in both directions.

After a few hours later

3. Synaptic terminal degenerates and there is accumulation of neurofibrils, vesicles, etc.
4. Astroglia surrounds terminal normally; after axotomy. It interposes between terminal and target due to which terminal get pulled away from post-synaptic cell.

After days–weeks

5. Myelin breaks up and leaves debris (myelin hard to break down).
6. Axon undergoes Wallerian degeneration.
7. Chromatolysis: Cell body swells; nucleus of the nerve cell becomes eccentric and Nissl bodies are sparse.

Regeneration

Regeneration takes place only outside the central nervous system where neurolemma is present. Presence of neurolemma is, therefore, essential for the process. Hence, in the central nervous system, neurolemma being absent, nerve fibres do not regenerate at all.

The following steps are seen during regeneration:

1. The axis cylinder grows out from the central cut end as a rounded sprout and proceeds towards the solid neurolemmal cord.
2. The proliferated Schwann tissue in the peripheral cut end and its prolongation towards the central cut end provide an influence (neurotropism) which guides the approaching axis cylinder.
3. Each growing fibre splits up into numerous neurofibrils (even up to 100), the Schwann cells disappear and the fibrils enter the newly-made neurolemmal tubes (2–3 weeks after the section, the inner walls of the tube may contain a number of fibrils. All the fibrils degenerate, excepting a single one, which gradually enlarges and occupies the central part of the whole length of the tube proceeding peripherally.
4. The daily rate of growth is about 0.25 mm in the scar tissue between the two cut ends and 3–4 mm in the peripheral neurolemmal tubes.
5. Myelin sheath begins to appear in about 15 days and proceeds peripherally along the fibre at a slower rate than the growing axis cylinder. Increase in the diameter of the fibre takes place very slowly. The diameter of the fibre is limited by the size of the neurolemmal tube and that of the parent nerve cell.
6. With a clean sharp wound and the cut ends being in apposition, some degree of recovery usually takes place in 6–24 months. For a motor nerve, recovery may be complete. But for a mixed nerve, it is rarely so.
7. In the regenerated fibres the axis cylinder and myelin sheath are reduced in thickness, the internodal distance is also diminished. But the rate of conduction of nerve impulses in the regenerated fibres remains the same.

DEGENERATION AND REGENERATION OF NERVE

The framework of both sensory and motor endings can resist degeneration for months. If the nerve fibres fail to regenerate, the endings also atrophy. But if the fibres regenerate, the living frameworks of the nerve endings quickly establish connection with the growing fibres and start functioning. Some of the newly growing fibres may establish connection with new types of endings in new situations. It is also possible that some growing nerve fibres may reach a place where there was no nerve ending at all and absolutely fresh nerve endings may develop around them. Complete functional regeneration occurs after histological regeneration—3 weeks in case of motor nerve fibres and 5 weeks in case of sensory nerve fibres.

Transneuronal Degeneration

When a neuron or its motor fibre degenerates the neuron next in the chain is often found to degenerate also. This takes place in spite of the fact that there is no anatomical conditions continuity through the synapses. It is probably an example of disuse atrophy. In many conditions, this type of degeneration occurs, e.g. after section of the optic nerve, the cells in the lateral geniculate body degenerate. After section of the posterior spinal root, the posterior horn cells degenerate. In lesions of the motor cortex or pyramidal tracts, the anterior horn cells may degenerate. This type of degeneration may be the underlying cause of the so-called system diseases, viz. amyotrophic lateral sclerosis, etc. where degeneration of anterior horn cells follows that of the pyramidal tracts.

APPLIED PHYSIOLOGY

Cathode Ray Oscilloscope (CRO)

Karl Ferdinand Braun
1850–1918

The cathode ray oscilloscope is used to estimate or measure the electrical changes of the living tissues. A cathode emits electrons when a high voltage is applied to it with a suitable anode in a vacuum. These emitted electrons are directed into a focused beam which strikes the face (screen) of the glass tube of the CRO. The screen of the CRO is coated with a number of fluorescent substances (phosphors) which emits light when struck by electrons.

Karl Ferdinand Braun invented the Cathode Ray Tube Oscilloscope in 1897 when he was experimenting on physics principles of applying an oscillating signal to electrically charged deflector plates in a phosphor-coated CRT.

In a CRO to record the action potentials, two electrical circuits must be employed which are as follows:

1. An electronic sweep circuit
2. An electronic amplifier.

The electronic sweep circuit is connected with two vertical metal plates (X-plates) on either side of the beam in the cathode ray (CR) tube. By altering the electrical potentials at a very high speed it moves the beam of electrons horizontally across the screen of the tube from left to right and when the beam reached the right extremity, it jumps back to the left side and in

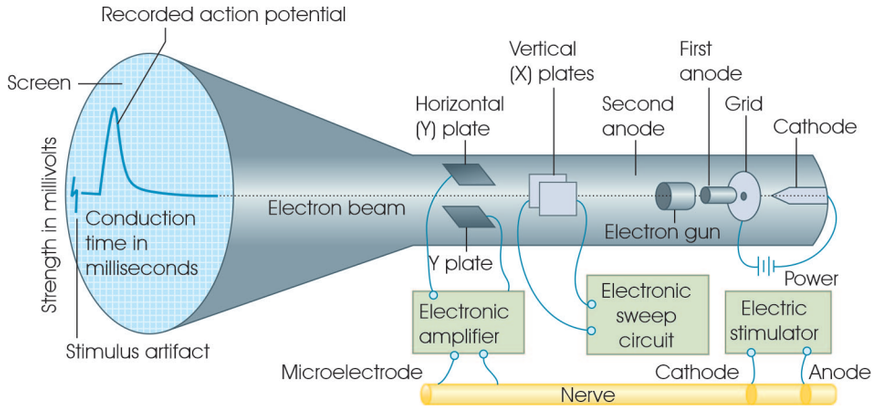


Fig. 21.12: Simplified, diagrammatic representation of the main connections of the CRO to action potential changes

this way the beam is always moving from right. There are two metal plates, in the cathode ray tube, which arranged horizontally, one above and the other below the electron beam. The Y plates (horizontal) are connected, with the electronic amplifier. This set of deflecting plates move the electron beam up and down with the change of action potential in them. For experiments, the electrodes are place on the nerve fibre or the tissue of which the electrical potential change is to be measured. These electrodes are connected with the amplifier circuit, thereby to the horizontal plate (γ-plates). The potential change in the tissues after proper amplification is transmitted to the Y plates and potential changes are recorded as vertical deflections of this electronic.

Beam on the screen which may be photographed for permanent record. Within the CR tube itself, cathode serves as a source of electrons; grid controls the intensity of electron beam and brightness of the spot. The first anode compresses the flow of electrons into narrow beam, whereas the second anode (electron gun) is highly positive and accelerates the beam of electrons. The electric stimulator, of which one is connected with cathode and the other with anode, applies a short (or as chosen) voltage (PIP) to the nerve stimulating microelectrodes (Fig. 21.12).

EXAM-ORIENTED QUESTIONS

Essay

1. Discuss the properties of nerve fibre.
2. Classify nerve fibres. How is the impulse conducted in various nerves?
3. Describe the mechanism of Wallerian degeneration and regeneration of nerves.

Short Notes

1. Excitability of nerve
2. All-or-none law in nerve
3. Conductivity in nerve
4. Metabolism in nerve fibres
5. Electrotonic current
6. Electrotonus
7. Cathode ray oscilloscope