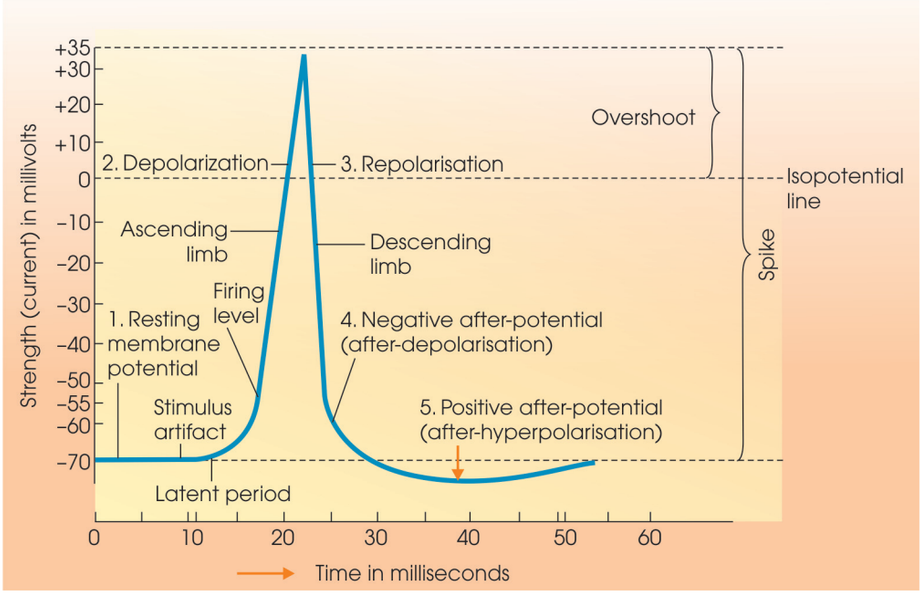

Fig. 21.1: Diagrammatic representation of spike potential (action potential) recorded with the help of microelectrode in the nerve cell

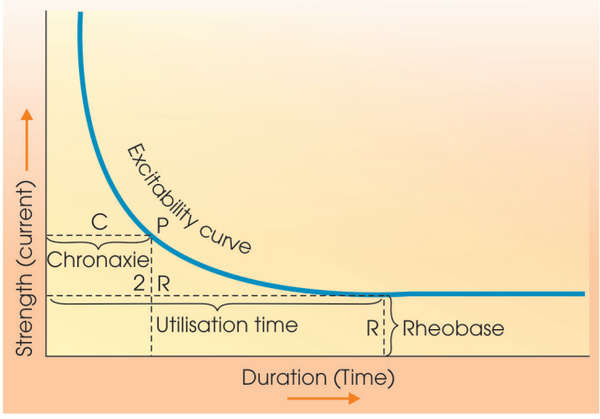

Fig. 21.2: Diagrammatic representation of excitability curve (strength–duration curve) applied to an excitable tissue producing response relating the strength of stimulus

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 is 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.

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.
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 begin to close and gradually are completely closed. Thus, as membrane reaches resting state the activation gates of sodium and potassium channel are closed while inactivation gate of sodium channel opens.
5. **After hyperpolarization:** This increased negativity inside hinder further efflux of K^+. Most of the voltage-gated K^+ channels are closed but as some of the voltage-gated K^+ channels the efflux continues and membrane potential becomes more negative producing the phase of after hyperpolarization. The

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, as Hill suggests, 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 possibly 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 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.

- 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 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

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.

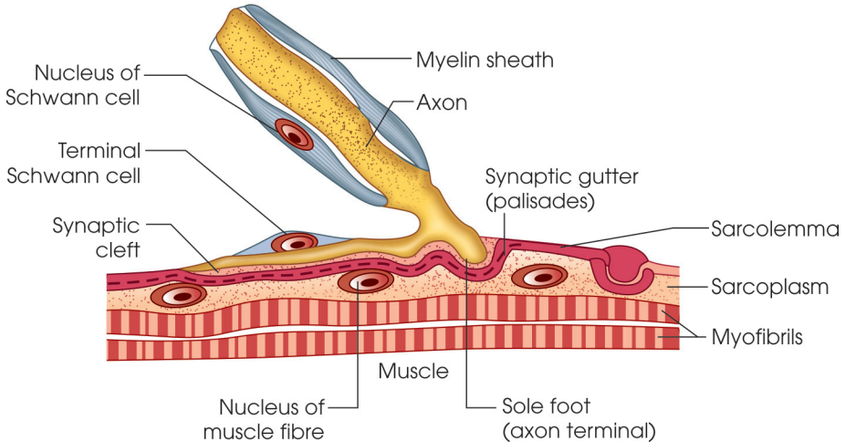

Fig. 22.2: Diagrammatic representation of the relationship of nerve ending to the muscle fibres (*neuromuscular junction*)

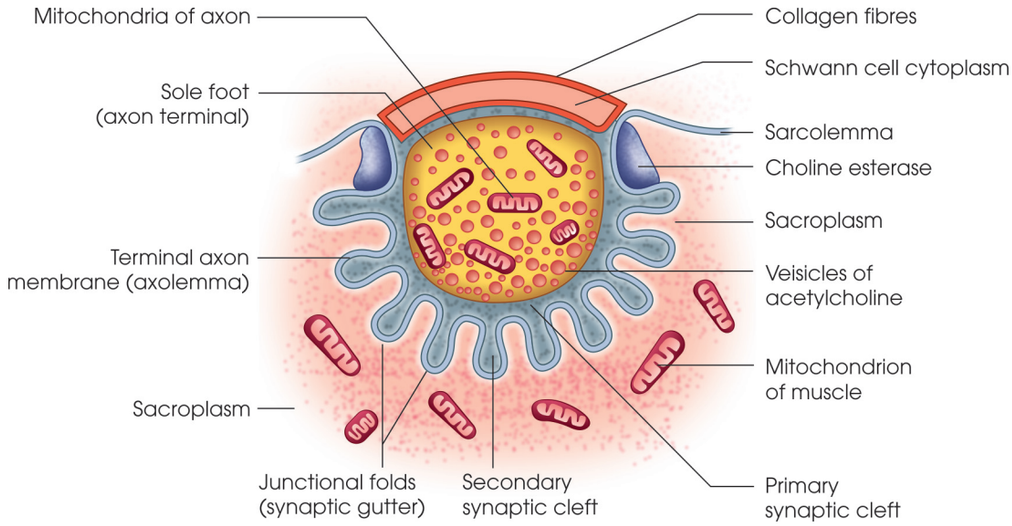

Fig. 22.3: Electron microscopic appearance of myoneural junction at the region of an axon terminal (sole foot) ending in the motor endplate

Synthesis of Acetylcholine in Motor Neuron

The ACh is synthesized locally in the cytoplasm of the nerve terminal, from active acetate (acetyl coenzyme A) and choline. Then it is rapidly absorbed into the synaptic vesicles and stored there. The synaptic vesicles themselves are made by the Golgi apparatus in the nerve soma (cell-body). Then they are carried by axoplasmic transport to the nerve terminal which contains around 300,000 vesicles. Each vesicle is then filled with around 10,000 ACh molecules. As action potential reaches the synaptic knob the Ca channels open increasing calcium permeability. The vesicle fuses with presynaptic membrane to release the neurotransmitter (NT) from synaptic knob to synaptic cleft. The neurotransmitter combines with specific receptors on the other membrane post-synaptic potential to generate end-plate potential. EPP then spread by local current to adjacent muscle fibres which are depolarized to threshold and fire action potential.

Sequence of Events in Neuromuscular Transmission (Fig. 22.4 and Flowchart 22.1)

Pre-synaptic Events

1. The action potential is initiated in the pre-synaptic motor neuron and invades the endplate region.
2. The depolarization of motor neuron up to terminal buttons result in the opening of voltage-dependent calcium channels. There is influx of Ca^{2+}, down its concentration gradient.
3. The increased cytoplasmic concentration of calcium enhances the movement of microfilament and microtubules which moves the vesicle to the pre-synaptic membrane. The fusion of vesicles containing acetylcholine (ACh) to the membrane of the terminal buttons, resulting in exocytosis of ACh. Acetylcholine diffuses across synaptic cleft to the muscle cell.

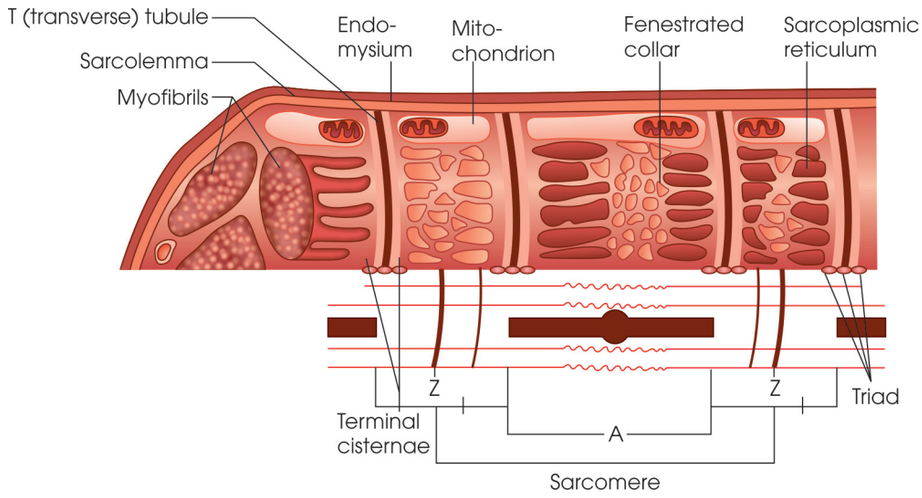

Fig. 24.4: Sarcotubular system of the mammalian skeletal muscles showing the triad to pass at the A-I junction (diagrammatic representation)

with intercommunicating transverse branches. No capillaries penetrate sarcoplasm. The great arteries and veins are seen in the perimysium. The largest and smallest veins possess valves.

- The lymphatic supply communicates with blood vessels of epimysium and perimysium. But lymphatics are not found between these muscle fibres (Flowchart 24.1).
- Myelinated nerve fibres supply striated muscle. The motor nerve endings terminate at end plates. The sensory nerves end in groups of modified muscle fibres known a muscle spindles. Functions of sympathetic nerve here are not known.

Ending of Muscle in Tendon

At the musculotendinous junctions the endomysium, perimysium and epimysium of the muscle become continuous with the fibrous tissue of the tendon.

Red and White (or Pale) Muscles

A muscle fibre, being composed of a number of delicate fibrils surrounded by a more fluid sarcoplasm and having mitochondria and sarcoplasmic reticulum, possess respiratory pigment, myoglobin (muscle haemoglobin) within the sarcoplasm.

1. Red colour of the muscle fibre is due to the presence of myoglobin. This myoglobin acts in the transport

Flowchart 24.1: Micro-anatomical organization of the muscle fibre has been presented schematically