Revision of the Resting membrane potential

1. Intracellular concentration of K+ ions in neurones is high and Na+ ions is low relative to the extracellular space.
2. The reversal potential is the potential towards which the membrane would move if it was freely permeable to this particular ion, and no other ions. At this potential chemical driving force is exactly equal electrical driving force and the net ion flux is nil.
3. The reversal potential for K+ ions in neurones is very negative (~ -95 mV) while for Na+ ions positive (~ +60 mV).
4. Resting membrane potential is generated mainly by a steady flux of K+ ions through ion channels embedded into the membrane of the neurone.

What is the action potential?

AP is a brief "all-or-none" depolarisation of the neuronal membrane which, once initiated propagates without decrement.

Features of the AP - 1:
Propagation along the cell's membrane and along the axon

Features of the AP - 2:
"All-on-none" nature

In contrast to the "small, sub-threshold" potentials, action potentials have very similar amplitude and shape. Once the threshold has been reached, their amplitude will not increase even if a stronger stimulus is applied. In terms of information transfer they are like logic "ones", as compared to "zeros".
Glossary of the terms

Overshoot
Repolarisation
Hyperpolarisation
Depolarisation
Resting membrane potential

Ionic basis of AP: Na ions are responsible for depolarisation, K ions are responsible for re-polarisation. Fluxes of both ions are coordinated by voltage-gated ion channels.

Na+ channels are controlled by the potential of the membrane.

"Positive feedback" is responsible for the very fast dynamics of AP

1. Depolarisation
2. Opening of voltage-gated Na+ channels
3. Sodium currents depolarise membrane further

Something has to happen for this process to terminate!

At positive potentials voltage-gated Na+ channels become “inactivated”
Ion channel activation during an action potential

REMEMBER:
Na+ fluxes depolarise the membrane (EP ~+60),
K+ fluxes hyperpolarise the membrane (EP ~-90)

Na+ channels opening
K+ channels opening
Na+ channels inactivate

Two factors responsible for AP termination:
1. Inactivation of Na+ channels
2. Delayed activation of K+ channels (delayed rectifiers)

Molecular nature of the voltage-gated Na+ and K+ channels

A voltage-gated Na channel is formed by association of 4 main (α) subunits

A) Absolute refractor period (second AP cannot occur under any circumstance)
B) Relative refractory period (a stronger-than-normal stimulus may evoke an AP)
1. Axons of many types of neurones are “insulated” with multiple layers of myelin.
2. Bare “gaps” between the insulated parts are known as nodes of Ranvier.
3. This is known as saltatory conduction. AP “jump” from one node to the next as an electrical field, rather than a wave of Na+ channel openings.
4. This greatly accelerates AP propagation and saves much energy.

Speed of conductance depends on the degree of myelinisation. “Thick” vs “thin” axons.
1. Axons of different types of central and peripheral neurones have different degree of myelinisation.
2. Axons with thick myelin insulation are generally faster and can conduct at many tens of meters/second. Un-myelinated (or almost un-myelinated) axons can be as slow as 1 m/sec.
3. Loss of myelin occurs in many diseases, for example multiple sclerosis. This may be fatal.

Summary
1. Features of AP
2. Ionic basis of AP
3. Basic mechanism of AP propagation
4. Saltatory conduction and the role of myelin

What happens at “the end” - action potentials lead to release of transmitter from pre-synaptic terminals.