Primary membrane potential establishing:

- Higher prevalence of K+ leak channels - greater efflux of cations, more negative inside.

- Nernst equation calculates membrane potential using ratio between intracellular and extracellular concentration.
  - For cations, [K+]out / [K+]in, for anions, [A-]in / [A-]out
  - Aggregate all ions into single calculation - Goldman-katz equation

Myelination

- Transmission speed: 10m/s unmyelinated, 150m/s myelinated
- Oligodendrocyte in CNS, schwann cells in PNS
- Nodes of ranvier can be depolarised
- Saltatory conduction

Action potential characterstics:

- All/nothing
- Unidirecitonal

Neurotransmitter release:

- Depolarisation - voltage gated Ca channel open - Ca influx
- NT-vesicle transported along F-actin towards presynatic membrane.
- Fusion of vesicle with synaptic membrane via t-SNARE v-SNARE interaction.
  - v-SNARE: synaptobrevin (Vesicle associated membrane protein VAMP)
  - t-SNARE: SNAP25 & syntaxin
  - Chaperone protein: UNC15, synaptotagmin
- Release of NT
  - NT could be: monoamine(dopamine, serotonin) , amino acid(glutamate, glycine, GABA)

Excitatory postsynaptic potential/current (EPSP/EPSC):

EPSP is shown as a upwards deflection: depolarisation in membrane potential.

EPSC is shown as a downward deflection: influx of positive cations, efflux of negative anions

Two types of post-synaptic receptors: Ionotropic and metabotropic

- Ionotropic: Ligand binding induce influx of ions (e.g. AMPA, NMDA, GABA)
- Metabotropic: ligand binding induce intracellular signalling

Glutamate:

- Ionotropic: AMPA, NMDA. Antagonist: CNQX, DNQX
- Metatropic: mGluR. Antagonist: MK801, APV

Acetylcholine:

- Ionotropic: Nicotinic AchR
- Metabotropic: Muscarinic AchR

GABA:

- Ionotropic: GABAa
- Metabotropic: GABAb

NMDA magnesium block: A strong depolarisation is needed to remove the Mg2+ ion blocking the channel

Current-Voltage curve

X-axis: membrane potential

Y-axis: current through the channel at the voltage. Positive influx - negative, chlorine influx - positive

Slope of the curve is determined by the conductance of the channel.

Slope cross the x-axis at the reversal potential, where the direction of ion flow changes.

Determinants of amplitudes of voltage change:

- Ohm's law: V=I*R.
    - If current is high - large amount of neurotransmittors / high conductance of channel /
    - If resistamce is high - number of open channels

Metabotropic receptors ~50ms compared to 1ms in ionotropic

General mechanism: Binding of ligand leads to conformation change of the receptor. α-β-γ subunit exchange GDP for GTP, become active, α-subunit dissociate from β-γ subunit, α-GTP bind and activate downstream proteins, triggering cascade, β-γ also activate downstream proteins.

E.g. β-adrenergic receptors: binding of adrenaline lead to exchange of GDP to GTP on G-protein complex, α-subunit bind and activate adenylyl cyclase, catalysing ATP - cAMP, activating downstrem PKA, activating downstream proteins. Hydrolysis of GTP-GDP cause dissociation and inactivation of α subunit.

Signal integration

Spatial and temporal integration

- Spatial integration: amplitude of the signal influenced by length of the axon, distance between signals (influenced by dendritic filtering)
- Temporal integration: amplitude of the signal influenced by time difference, time for signal to reach maximum amplitude (capacitance). Capacitance determined by surface area.

Synaptic plasticity: Long term potentiation is the strengthening of the synapse following a period of high frequency stimulation. Increased signal transmission efficiency, e.g. more expression of NMDA receptor.