Mind, Brain & Behaviour 1

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Behavioural Neuroscience Unit:

Neurons, Glia & Mechanisms of Communication

Lecture 2

Multiple Sclerosis (MS) is an acquired neurological disorder that attacks the insulating myelin sheath that surrounds the axons of neurons.

- Begins with visual problems, numbness and weak limbs.
- Ultimately leads to paraplegia, slurred speech, issues with vision.
- Frequent attacks followed by remission and worsened symptoms.
- MS is an autoimmune disorder – the body's immune system selectively attacks the myelin surrounding the axons of neurons.
- Cause is unknown.

Withdrawal Reflex: Excitation stimulus/signal.

- The dendrites of a sensory neuron respond to a noxious stimulus in the environment (hot iron).
- This signal is sent back along the axon to terminal buttons, located in the spinal cord.
- Terminal buttons release a neurotransmitter into the synapse, exciting an interneuron which resides in the spinal cord.
- The interneuron sends a message down its axon, releasing a neurotransmitter to excite the motor neuron.
- The axon of the motor neuron joins a nerve (bundle of motor neurons), travelling to a muscle in the arm, causing the muscle to contract and pull away from the hot surface.

The role of Inhibition:

- Withdrawal Reflex comes from excitatory synapses on motor neurons in the spinal cord, but this excitation can be counteracted by inhibition arising from one's brain.
- The brain contains complex circuits of neurons that represent the emotional consequences, sending information to the spinal cord and prevents you from following through with the reflexive action.
    - Neurons within the brain sends message along axon to spinal cord.
    - Inhibitory interneuron is excited, releasing an inhibitory neurotransmitter.
    - This decreases activity of motor neuron, blocking the withdrawal reflex.
    - Illustrates the Principles of Neural Communication.

Neurons:

The basic information-processing & information-receiving unit of the Nervous System.
Neurons form complex networks within the nervous system.

- Networks are not directly connected with one another.
- Networks are separated by tiny gaps called **synapses,** across which chemical neurotransmitters are passed.

FEATURES OF A NEURON:

- **Cell Body:** Contains nucleus.
- **Dendrites:** Inputs of electrical stimulation to neuron.
- **Axon:** Carries signals (action potential) from cell body.
- **Terminal buttons:** Small knobs at the ends of axon terminals. Transmits information through neurotransmitter, passing across synaptic gap to excite or

inhibit the next neuron. May themselves send information to many other neurons via their own terminal buttons.

- **Cytoplasm:** A jelly-like substance inside the cell. Contains the **Mitochondria**, which use glucose to produce energy. Also produces **Adenosine Triphosphate (ATP) -** energy source.
- **Nucleus** contains **chromosomes,** which are composed of **DNA** - the **genes** that make up chromosomes provide recipes for making proteins.

Glial Cells:
- Support with chemical transport to and from neurons.
- Provides insulation.
- Destroys neurons that have died from old age.

Lipid Bilayer:
- Composes the cell membrane of a neuron.
- Contains complex protein molecules that regulate the entrance and exit of chemicals from neuron.
- Separates the extracellular fluid (fluid outside the cell) from the intracellular fluid (fluid inside the cell).
- Extracellular and Intracellular fluid both have different charges.
- Cell membrane is critical for transmission of information along the axon.

A signal transmitted by a neuron is an electrical process which involves the movement across the axon membrane of electrically charged molecules called **Ions.**

The inside of axon is more electrically charged than the outside.
This voltage difference between the inside and outside is known as = **Resting Membrane Potential.**
- Can be changed by injecting a certain current.
- The message that is conducted down an axon involves a brief change in the membrane potential.

Depolarisation: The inside of the neuron is negatively charged - adding a positive electrical current through the electrode causes depolarisation.

Action Potential (AP):
- A very rapid depolarisation, or fluctuation of membrane potential of an axon.
- Constitutes the basic message that is transmitted down an axon from the cell body to terminal buttons.

Membrane Potential: A balance of two forces. **Resting Membrane Potential** is at -70 mV.

Diffusion: The process by which molecules distribute themselves evenly throughout a medium.
- When molecules dissolve from high concentration --> low concentration.

Electrostatic Pressure: Ions that repel or attract according to the positive or negative charge:
Types of ions:

- **Anion** – negatively charged (a particle that has gained an electron).
- **Cation** – positively charged (a particle that has lost an electron).

Sodium-Potassium Transporter:

- Protein molecules in cell membrane pump Sodium outside, and in exchange, pump Potassium inside.
- Energy is in the form of Action Potential, which is supplied by the mitochondria.

Sodium: High concentration outside the cell.
Ion exchange across the axon (Action Potential):

- Ion channels provide an opening through which ions can rapidly enter or leave the cell.
- When ion channels for sodium open, sodium ions rush into the cell.
- Ion channels specific to potassium open shortly thereafter, allowing potassium ions to rush out of the cell.

An action potential is triggered when excitatory input is passed from the terminal buttons of a **presynaptic neuron** and received by the dendrites of the **postsynaptic neuron**.

The **myelin sheath** insulates the axon, and generates the Action Potential.

- It can cause another set of sodium-potassium gates to open as a result.

All-or-none law: As soon as the threshold of resting membrane potential (-50 mV) is reached, the same Action Potential will be reached, regardless of the level of excitation.

Effects of Myelin damage in Multiple Sclerosis:

- Abnormal conduction due to this damage, causing 'demyelination' of nerve fibres.
- Impacts sodium-potassium gates in opening and closing.
    - May lose a segment of the myelin sheath.

Na+ = Sodium
Cl- = Chloride
K+ = Potassium
A- = Proteins

The Synapse

Lecture 3

Myasthenia Gravis = A synaptic transmission disorder.

- Extreme fatigability.
- Fluctuating muscle weakness - predominately proximal muscles (head, neck & trunk).
- Respiratory weakness (breathing).
- Problems chewing (Dysphagia).
- Problems talking (Dysarthria).
- Arises from a problem with synapses on muscles.
- Action Potential in nerves are normal.

Outside of cell consists of salty water. Inside of cell contains positively charged potassium.

Action Potential communication with next neuron:

- Terminal buttons release neurotransmitter.
- Neurotransmitter diffuses across gap (synaptic cleft), between **presynaptic terminal button** and dendrite or cell body of **postsynaptic membrane.**
- If neurotransmitter has an excitatory effect on the postsynaptic cell, it will **depolarise** it and generate **action potential.**
- If neurotransmitter is inhibitory, the postsynaptic cell will be **hyperpolarised** (makes cell membrane negatively charged), and inhibits action potential.
- Process is repeated for next neuron in the circuit.

STRUCTURE OF A SYNPASE:

- **Presynaptic membrane** – The membrane of the presynaptic terminal button.
- **Postsynaptic membrane** – The membrane of the postsynaptic neuron.
- **Dendritic spine** – Ridge on the dendrite of a postsynaptic neuron - terminal button from a presynaptic neuron forms a synapse.
- **Synaptic cleft** – The tiny gap between the presynaptic and postsynaptic membrane.
- **Synaptic vesicles** – Tiny balloons filled with neurotransmitter molecules - found in the release zone of the terminal button.
- **Microtubules** – Long tubes that run down the axon and guide the transport of synaptic vesicles from the soma to the axon terminal.
- **Release zone** – Part of the interior of the presynaptic membrane to which synaptic vesicles fuse in order to release their neurotransmitter into the synaptic cleft.

(1) Release of a Neurotransmitter:

- Vesicles contain neurotransmitter molecules.
- An action potential in the presynaptic cell triggers vesicles to move forward toward the release zone of cell membrane.
- Vesicles are guided toward the membrane by proteins.

(2) Release of a Neurotransmitter:

- Guiding proteins act like ropes, helping pull the vesicle & presynaptic membrane together.

(3) Release of a Neurotransmitter:

- An influx of calcium ions into the presynaptic terminal button, induces fusion of the two membranes.
- Neurotransmitter molecules are then released into the synaptic cleft.

IONTROPIC RECEPTORS: Receptors equipped with their own binding sites.

- Neurotransmitter molecules diffuse across synaptic cleft.
- Upon reaching the other side, they attach to specific binding sites of postsynaptic receptors.
- The molecules open neurotransmitter dependant ion channels in the postsynaptic cell.
- These channels, once opened, permit the flow of specific ions in and out of the postsynaptic neuron.
- When neurotransmitter locks into binding site, the channel is opened, allowing ions to move in or out.
- Physical structure of the neurotransmitter is matched to the receptor.

*As ion moves, it causes a change in the membrane potential.

Postsynaptic potentials can be either:

- **Excitatory** (increasing the likelihood that the neuron will depolarise, triggering an action potential)
- **Inhibitory** (increasing the likelihood that the neuron will hyperpolarise, and thus *not* trigger an action potential).

  *Whether a postsynaptic potential is excitatory or inhibitory is determined not by the neurotransmitter that is released into the synapse, but by the specific ion channel that the neurotransmitter opens.

The type of postsynaptic receptor determines whether the postsynaptic neuron will be excited (depolarised) or inhibited (hyperpolarised).

The combined effects of EPSPs & IPSPs is called NEURAL INTEGRATION:

NEURAL INTEGRATION: Excitatory Postsynaptic Potentials (EPSPs)

- EPSPs depolarise the postsynaptic cell membrane.
- Increases the likelihood that an action potential will be triggered in the postsynaptic neuron.

- Prior to the release of neurotransmitter molecules from the presynaptic terminal button, the membrane potential of the postsynaptic neuron is at its resting level (i.e., -70 mV).
- After neurotransmitter molecules are released from the presynaptic terminal button, they diffuse across the synaptic cleft and bind to receptors on the postsynaptic membrane.
- If the neurotransmitter binds to sodium ion channels, these will allow an inflow of sodium ions, causing a depolarising EPSP in the dendrites of the postsynaptic neuron.

NEURAL INTEGRATION: Inhibitory Postsynaptic Potentials (IPSPs)

- IPSPs hyperpolarise the postsynaptic cell membrane.
- Decreases the likelihood that an action potential will be triggered.

** IPSPs tend to cancel out the effects of EPSPs.

Classes of Neurotransmitters:

- **Glutamate:** Most common excitatory neurotransmitter in the CNS. (Main neurotransmitter associated with synapses).
- **GABA (Gamma aminobutyric acid):** Most common inhibitory neurotransmitter.
- **The Monamines (dopamine, norepinephrine, serotonin):** Are synthesised from a single amino acid - Are present in groups of neurons located mostly in the brainstem.
- **Acetylcholine:** Neurotransmitter that operates at synapse with muscles, and other parts of the CNS.

Effects of drugs on synaptic functions:

- Drugs influence the activity of the CNS by modulating the activity of the synapse.
  - AGONISTS:
    - Facilitates activity of postsynaptic neurons.
    - Increases synapse activity.
  - ANTAGONISTS:
    - Inhibits activity of postsynaptic neurons.
    - Decreases synapse activity.

Some agonistic drug actions:

L-dopa increases the synthesis of dopamine – this drug is used to treat the symptoms of Parkinson's disease.