Biomedicine

Lecture 13:
Nervous System
PART 1
Learning Outcomes

In today’s topic you will learn:

- The structure, function & physiology of the nervous system
- Nerve impulses & neurotransmitters
- Neurotransmitters and their activities
- Common degenerative & neurotransmitter pathologies of the nervous system
The Nervous System

• A network of fibres which permeate the body, co-ordinating voluntary and involuntary actions.
• Transmits signals between different parts of the body.
• Responds to changes within the internal and external environment for a fast effect.
• Works alongside the endocrine system to maintain homeostasis.
• Two parts:
  1. Central nervous system (CNS)
     a) Brain
     b) Spinal cord
  2. Peripheral nervous system (PNS)
     • Peripheral nerves (all nerves not in the CNS)
Functions

1. Sensory:
   • Detects internal and external environmental changes.
   • Information is carried by sensory (afferent) neurons.

2. Integrative:
   • Processes sensory information, analyse store and cause perception.
   • Carried by interneurons.

3. Motor:
   • Produces a response to sensory information (perception) to effect change.
   • Impulse carried by motor (efferent) neurons.
Functions

PERIPHERAL NERVOUS SYSTEM

CENTRAL NERVOUS SYSTEM
(Brain and spinal cord)

PERIPHERAL NERVOUS SYSTEM

SENSORY OR AFFERENT NEURONE

Internal environment (autonomic) e.g.:
- chemoreceptors
- baroreceptors
- osmoreceptors

Sensory receptors

Senses:
- sight
- hearing
- smell
- taste

MOTOR OR EFFERENT NEURONE

Effector organs

Somatic (voluntary):
- skeletal muscle

Autonomic (involuntary):
- cardiac muscle
- smooth muscle
- glands

Sympathetic division
Parasympathetic division
Organisation

Nervous System

Central Nervous System (CNS)

Brain
Receives and processes sensory information, initiates responses, stores, memories, generates thoughts and emotions

Spinal cord
Conducts signals to and from the brain, controls reflex activities

Peripheral Nervous System (PNS)

Motor Neurons
CNS to muscles and glands

Sensory Neurons
Sensory organs to CNS

Somatic Nervous System
Controls voluntary movements

Autonomic Nervous System
Controls involuntary responses

Sympathetic Division
“Fight or Flight”

Parasympathetic Division
“Rest or Digest”
Autonomic nervous system

- Works automatically – ‘auto-pilot’ to maintain homeostasis.
- Affects organs, glands, cardiac and smooth muscles.

Two branches:
1. Sympathetic
   - ‘fight or flight’ response
   - Thoraco-lumbar innervation

2. Parasympathetic
   - ‘rest and digest’
   - Cranio-sacral innervation
Autonomic nervous system

CONTROLS:
• Rate and force of heart-beat.
• Gland activity.
• Vessel diameter – vasoconstriction / vasodilation.
• Bronchi - bronchoconstriction / dilation.
• Pupillary constriction / dilation.
Autonomic nervous system

Sympathetic & parasympathetic generally have opposite effects:
Enteric nervous system

- ‘Brain’ of the GIT.
- Functions autonomously / independently BUT mostly regulated by the autonomic NS.
- Interacts extensively with the CNS.
- Links with the CNS via the sympathetic (pre-vertebral ganglia) and parasympathetic (Vagus) NS.
  - CNS allows outside information to reach the gut

Sensory neurons monitor chemical changes (via chemo-receptors) in the GI tract and stretching (stretch receptors) of its walls.
Motor neurons govern motility and secretions of the GI tract and associated glands.
Interneurons connect the 2 plexuses.
The Brain in Your Gut

The gut’s brain, known as the enteric nervous system, is located in sheaths of tissue lining the esophagus, stomach, small intestine and colon.

**Small Intestine Cross Section**

**Submucosal Plexus**
Layer contains sensory cells that communicate with the myenteric plexus and motor fibers that stimulate the secretion of fluids into the lumen.

**Myenteric Plexus**
Layer contains the neurons responsible for regulating the enzyme output of adjacent organs.

**Lumen**
No nerves actually enter this area, where digestion occurs. The brains in the head and gut have to monitor conditions in the lumen across the lining of the bowel.

Mesentery
Attaches the bowel to the body wall and contains major arteries, veins, lymphatics and external nerves.
Two types of nerve cells:

1. **NEURONS** - process and transmit information, electrically excitable.
2. **NEUROGLIA (GLIAL CELLS)** nourish, support and protect neurons.
Nerves & neurons

**Nerve:** a bundle of one or more neurons.

**Neuron:**
- Consists of:
  - Cell body
  - Dendrites
  - Axon
  - Myelin sheath
  - Terminal endings
- Possess electrical excitability - the ability to make an ‘action potential’ (nerve impulse):
  - **Nerve stimulus:** anything able to trigger the generation of an action potential in a neuron.
  - Can be from the internal or external environment.
Neurons

Cell body:
- Size and shape varies.
- Consists of a nucleus and typical cell organelles.
- Cell bodies are known collectively as grey matter of the NS.
- Collections of cell bodies are referred to as:
  - Nuclei / Centres in the CNS
  - Ganglia in the PNS

Axons:
Determine nerve length: vary from <1mm (CNS) to ~1m (sciatic nerve)
Carries nerve impulses towards another neuron, away from the body. Covered by a membrane called the axolemma.
The ends are called axon terminals.
Axon bundles are called:
- Tracts in the CNS
- Nerves in the PNS
Neurons

Dendrites:
• ‘little trees’.
• The receiving end of a neuron.

Axons and dendrites make up the white matter of the nervous system

Myelin sheath:
• A multi-layered lipid & protein covering around the axons.
• Formed by neuroglia / glial cells (Schwann cells / oligodendrocytes) in the embryo.
• Myelination continues through childhood and peaks in adolescence.
• Covered axons are termed myelinated.

FUNCTIONS:
• Insulates the axon (covers the axolemma).
• Regeneration of axons in the peripheral NS.
• Increases speed of nerve impulse conduction (by forming gaps).
Neuroglia

- Also known as ‘glia’ or ‘glial cells’.
- Cells that surround and bind the neurons.
- Far smaller than neurons and 3-50 times as many.
- Can multiply and divide (unlike neurons).
- After trauma, glia will fill spaces left by damaged neurons (important concept when considering malignant growth of tumours).

FUNCTIONS:
- Surround neurons and hold them in place.
- Supply nutrients and oxygen to neurons.
- Insulate one neuron from another.
- Destroy pathogens and remove dead neurons.
Neuroglia

TYPES:
Six types:
• 4 found in the CNS: astrocytes, oligodendrocytes, microglia and ependymal cells.
• 2 found in the PNS: Neurolemmocytes (Schwann cells), and satellite cells.

Astrocytes
• Star-shaped with branching processes.
• Hold neurons to their blood supply.
• Contribute to the blood brain barrier (BBB).
• Found in the CNS.

Oligodendrocytes
• Smaller than astrocytes.
• Found in the CNS.
• Form and maintain myelin in the CNS.
• Similar role to Schwann cells in PNS.
**Neuroglia**

**Microglia**
- Derived from monocytes and migrate before birth.
- Found near blood vessels.
- Phagocytic – clean up.
- Mobile in the brain and multiply when the brain is damaged.
- Found in the CNS.

**Ependymal Cells**
- Endothelial & epithelial cells which line the walls of the:
  - 4 Ventricles of the cerebrum
  - Central canal of spinal cord
- Make cerebrospinal fluid (CSF).
- Beat their cilia to circulate CSF.
- Found in the CNS.
Schwann Cells & Myelination

- **Schwann cells** produce myelin sheaths around neurons in the PNS – specifically the axons.
- **Myelin sheath** is a multi-layered lipid and protein covering which **insulates** the axon and increases **speed of nerve impulse** conduction.
- Covered axons are termed **myelinated**.
- Dendrite connections & most myelination is finished by 3 years of age – malnutrition in infancy can cause irreversible damage.
- **Node of Ranvier** is the gap between the Schwann cells along a neuron – they increase the speed of nerve impulse transmission.
Satellite Cells

Support neurons around the ganglia (cell bodies) of the PNS.
Activity

Please label the parts of a neuron in the your hand-out ‘Diagrams of the Nervous System’ hand-out.
Nerve impulses

• Neurons are electrically excitable - they communicate with each other.
• There are 2 types of electrical signal:
  1. Graded potential – short distance communication
  2. Action potential – long distance communication
• Are able to occur due to 2 characteristics of a cell:
  1. Specific ion channels can open and close due to stimuli, changing the potential & creating an electrical current.
  2. An electrical difference across the membrane of the cell known as the resting potential.
Nerve impulses - ion channels

- These are transport channels for ions created by trans-membrane proteins within the neuronal membranes.
- When ion channels open they allow specific ions to move through the membrane across an electrochemical concentration gradient e.g. Na\(^+\) channels allow Na\(^+\) through and K\(^+\) channels allow K\(^+\) through.
- Channels open in response to a stimulus which changes the permeability of the neuron membrane to Na\(^+\) & K\(^+\).
- There are 4 categories of stimulus causing ion channels to open:
  1. Voltage
  2. Chemicals (hormones, neurotransmitters etc.)
  3. Mechanical pressure
  4. Light (photoreceptors of the eye)
Nerve impulses – resting potential

- Neurons at rest possess an electrical difference / gradient across the cell membrane.
- This is created by a build up of negative ions on the inside of the cell membrane and positive ions on the other side of the cell membrane in the extra cellular fluid.
- Separation of charges creates potential energy.
- This resting potential is approximately -70mV.
- Cells exhibiting a membrane potential are said to be polarised (meaning charged).

Section of an axon during the resting potential.
The extracellular fluid is rich in Na⁺ and Cl⁻ ions and carries a positive charge. The intracellular fluid is rich in K⁺ and large negative ions which cannot leave the cell. The cell thus carries a negative charge inside.

As the Na⁺ and Cl⁻ try to move back to equalise the charge, the separation of charges (polarity) is MAINTAINED by the sodium-potassium pump which pumps 3 Na⁺ out for every 2 K⁺ it pumps back in.

This separation of charges creates a potential energy of -70 mV.
Nerve impulses – action potential

• An **action potential** is the formation of a nerve impulse / signal / excitation.
• It is a series of events which **decrease and reverse the membrane potential** and then restore it to its resting state.
• Occurs in 2 phases:

1. **Depolarisation** – the negative membrane potential (-70mV) becomes less negative, reaches zero, and then becomes positive.

2. **Repolarisation** – the membrane is then restored to its resting potential of –70mV.
Depolarisation:
- Triggered by stimulation of a nerve ending.
- Depolarisation must reach a threshold value in order to generate an action potential.
- $\text{Na}^+$ channels open allowing $\text{Na}^+$ to flood into the cell.
- Positive charge builds up inside the cell.
Nerve impulses – action potential

Repolarisation:
- K⁺ channels open much more slowly than Na⁺ channels so just as the Na⁺ channels are closing the K⁺ ones open.
- This allows K⁺ to flood out of the cell, restoring the membrane potential to −70mV.
Nerve impulses – action potential

Refractory period:
- Period of time after repolarisation in which a nerve cannot generate another action potential because Na\(^+\) and K\(^+\) are on the wrong sides of the membrane!
- During this period the Na-K pump pumps Na\(^+\) out and some K\(^+\) back into the cell to restore the resting potential.
- **Absolute refractory period** - even a strong impulse cannot generate an action potential.
- **Relative refractory period** - larger than normal stimulus needed to generate an action potential.
Nerve impulses – action potential

1. Low level Na+ and fixed level K+ are maintained by Na/K Pumps.

- Sodium gates open, overriding the Na/K pumps.

- Na/K pumps pump out the Na+, resetting the resting potential (refractory period).

- K+ gates open just as the Na+ gates close.

- If Na+ floods fast enough, an Action Potential is achieved (depolarisation).

Na+ inflow slows, K+ outflow increases (repolarisation).
Nerve impulses – conduction

Conduction is the movement of the nerve impulse along the neuron / nerve.

Unmyelinated neuron / nerve:
- No myelin sheath around the nerve.
- The membrane becomes depolarised in a continuous conduction away from the cell body down the axon.
- In one direction only.
- Following depolarisation the membrane is in a refractory state.
Nerve impulses – conduction

Myelinated neuron / nerve:

- Myelinated axons - the myelin is an insulator, preventing the ionic currents from crossing the membranes.
- Instead, at the Nodes of Ranvier, there are high concentrations of Na⁺ gates hence the current appears to jump from node to node (saltatory conduction).
- These ‘leaps’ may cover long intervals, leading to much faster current in myelinated nerves.
- These nerves are vital for rapid response reactions (reflexes).
Nerve impulses – conduction

Myelinated neuron / nerve:

- Saltatory conduction is also far more energy efficient, with less ATP needed for the sodium-potassium pumps.
- Basic speed of conduction in a nerve is dictated by the width of the nerve - the thicker, the faster.
- Nerves also propagate action potentials slower at lower temperatures.
Nerve impulses – conduction

Continuous Conduction
- Unmyelinated
- Step-by-step depolarisation spread
- Far slower
- Less energy efficient

Saltatory Conduction
- Myelinated
- ‘Leaps’ of depolarisation
- Far faster
- More energy efficient

Local anaesthetics
They block the Na⁺ gates from opening therefore stopping an action potential from being formed, inhibiting the nerve from being able to transmit the pain message.

Although these can be very useful, it is important to be aware that pain is an important function of the body bringing awareness that there is an issue. Anaesthetics are not resolving the issue, but suppressing symptoms. Long-term use can be harmful.
Neurons are NOT continuous - they have spaces / gaps between them called **synapses**.  
The tips of axon terminals are called **synaptic end bulbs**.  
The SPACE between the synaptic end bulbs and the next neuron in **chemical synapses** is the **synaptic cleft**.  
The **nerve impulse** is carried across the synaptic cleft by nerve messengers – **neurotransmitters**.  
In **electrical synapses**, CONNECTIONS between the synaptic end bulbs and the next neuron are **gap junctions**.
Synapses - chemical transmission

The release of the chemical messenger (neurotransmitter) across the synaptic cleft occurs in the following stages:

1. Nerve impulse arrives at the end bulb.
2. Depolarisation phase causes calcium channels to open, sending calcium into the synaptic bulb.
3. Increase of concentration of calcium ions causes exocytosis of synaptic vesicles: neurotransmitters are released into the synaptic cleft.
4. The neurotransmitters diffuse across the synapse and bind to receptors on the post-synaptic neuron.
5. This opens the ion channels on the post-synaptic neuron allowing ions to flow across the membrane.
6. The change in ions creates a post-synaptic potential, that then triggers an action potential in the post-synaptic nerve.
Synapses - chemical transmission
A neurotransmitter is a chemical messenger (nerve messengers) used to create synaptic transmission – transmission of a nerve impulse from one nerve to another.

- The number of neurotransmitters is still unknown, but >100 have been identified so far.
- Molecules are considered neurotransmitters if:
  1. It exists in the end bulb / synapse;
  2. Is released in response to Ca;
  3. There are receptors for it in the post-synaptic neuron.
• Neurotransmitters are broadly categorised into 3 types:
  1. Amino Acids e.g. GABA & taurine, aspartate, glutamate.
  2. Peptides e.g. vasopressin, somatostatin.
  3. Monoamines e.g. norepinephrine, epinephrine, dopamine (catecholamines), serotonin & melatonin.
• There is a 4th group which consists of all the unique molecules making up neurotransmitters e.g. acetylcholine.
Synapses - neurotransmitters

Excitatory or inhibitory (neurotransmitters):

• **EXCITATORY** - cause the ligand-gated ion channels to open – depolarisation of the post-synaptic neuron occurs.

• **INHIBITORY** - cause the ligand-gated ion channels to close – hyperpolarisation of the post-synaptic neuron occurs.

Removal of neurotransmitters:
Following a nerve impulse the neurotransmitters need to be removed for the process to be able to start again – this can occur by:

1. **Diffusion** out of the cleft into surrounding tissues / circulation.
2. **Degradation / destruction** by enzymes.
3. **Recycling / re-uptake** by terminal bulb.
Acetylcholine (ACh):
**Primary action:** excitatory (inhibitory in the vagus nerve).
**Location:** CNS, neuromuscular junction, parasympathetic NS.
**Role:** muscle contractions, cognition.
**Removal:** degraded / inactivated / broken down by enzyme acetylcholinesterase.
**Associated disorders & drugs:** Alzheimer’s (associated with deficiency of ACh), botulinum (‘botox’) blocks Ach.

Glutamate / Aspartate:
**Type of neurotransmitter:** amino acid
**Primary action:** excitatory.
**Location:** CNS (most common excitatory neurotransmitter in the brain).
**Role:** excitatory neurotransmitter in the brain.
**Removal:** re-uptake.
Synapses - neurotransmitters

Gamma-aminobutyric acid (GABA) & Glycine:
Type of neurotransmitter: amino acid.
Primary action: inhibitory.
Location: CNS (most common inhibitory neurotransmitter).
Removal: re-uptake.

Adrenaline / noradrenaline (epinephrine / norepinephrine):
Type of neurotransmitter: catecholamine.
Produced by: tyrosine.
Primary action: excitatory.
Location: sympathetic NS, motor neurons, brain & adrenal medulla (hormones).
Removal: re-uptake or degradation by enzymes monoamine oxidase (MAO) & catechol-oxygen-methyl transferase (COMT).
Synapses - neurotransmitters

Dopamine:
Type of neurotransmitter: catecholamine.
Primary action: excitatory / inhibitory.
Location: brain (primarily substantia nigra - area of the brain which regulates muscle tone and some aspects of movement & co-ordination).
Removal: re-uptake or degradation by enzymes monoamine oxidase (MAO) & catechol-oxygen-methyl transferase (COMT).
Associated disorders: Parkinson’s disease.

Serotonin (5-Hydroxytryptamine):
Produced by: tryptophan.
Location: brainstem, digestive tract, blood platelets & pineal gland.
Removal: re-uptake (blocked by SSRIs) or degradation by enzyme monoamine oxidase (MAO).
Synapses - neurotransmitters

Monoamine oxidase (MAO) enzyme:
Found in neurons and astrocytes
Catalyses the breakdown of some neurotransmitters:
• Serotonin
• Epinephrine
• Norepinephrine
• Dopamine

Catechol-oxygen-methyl transferase:
Catalyses the breakdown of:
• Epinephrine
• Norepinephrine
• Dopamine

COMT inhibitors are found in green tea
Synapses - neurotransmitters

Nitric Oxide:
- Formed from arginine.
- Formed on demand (not synthesised in advance or stored in vesicles).
- Highly reactive signalling molecule & free radical (combines with O₂ & H₂O to form nitrites & nitrates).
- **Role**: vasodilation - used for angina, lowering B.P. & erections in males.

Neuropeptides:
- Small proteins acting as neurotransmitters and sometimes as hormones.
- Common neuropeptides: enkephalins, endorphins, dynorphins & substance P.
- **Role**: may act as neuromodulators - substances that do not propagate nerve impulses directly, but instead exert regulatory effects on many extra-synaptic receptors.
- Many help inhibit pain perception (analgesic), whilst substance P enhances the feeling of pain.
Activity

Please label the parts of a synapse in the your hand-out ‘Diagrams of the Nervous System’ hand-out.
Nerve - sensation

TOUCH:
- Touch excites a graded potential in the sensory nerve in your fingers.
- The graded potential triggers the axon of a sensory neuron to form an ‘action potential’ which travels into the CNS.
- Neurotransmitters are released at nerve synapses where there are interneurons.
- Perception occurs in the brain and you can recognise touch.
Activity

Working in pairs & on a piece of paper produce a diagram to show what happens in a nerve cell when an action potential occurs. Include details:

- Resting potential
- Depolarisation
- Repolarisation
- Refractory phases

Also include diagrams to show the difference between continuous and saltatory conduction.
Nerve regeneration

• Also known as NEUROREGENERATION.
• ONLY in the peripheral nervous system (PNS) - the central nervous system (CNS) cannot (amphibians CNS can regenerate).
• The PNS and CNS have two distinct types of glial cells:
  • Schwann cells in PNS
  • Oligodendrocytes and astrocytes in CNS - CNS glial cells inhibit re-growth (especially astrocytes).
• Plasticity: ability for the NS to change – sprouting new dendrites, changes in synaptic contacts.
• Neurons have limited powers of regeneration - in the PNS if the Schwann cell and cell body are intact, and scarring doesn’t happen too quickly, regeneration may occur.
• In the CNS, cut axons do not regenerate, but new neurons have been shown to be formed in times of e.g. starvation to increase brain capacity to find food.
Nerve regeneration
Bell’s Palsy:
• A demyelinating condition.
• A type of mononeuropathy - damage to a single nerve or nerve group.

CAUSES:
Mostly viral e.g. Herpes virus, surgery, injury.

SIGNS & SYMPTOMS (Sx):
Sudden weakness or paralysis of the muscles in one side of the face, usually unilateral but may be bilateral.

ALLOPATHIC TREATMENT (Rx):
Depending on the cause e.g. Acyclovir, cortisone.

ALTERNATIVE TREATMENT (Rx):
Treat the cause: herbs & nutrients to support nerve function, antiviral, immune support, anti-inflammatory diet. acupuncture, homeopathy.

PROGNOSIS:
Usually good i.e. TEMPORARY.
Neuritis:
• Also known as GUILLAIN-BARRE SYNDROME (GBS).
• A demyelinating condition.
• AIDP = acute inflammatory demyelinating polyneuropathy.
• Widespread, autoimmune disorder causing acute inflammation and demyelination of peripheral nerves usually triggered 1-3 weeks after a respiratory tract infection.

CAUSES:
Auto-immune.

SIGNS & SYMPTOMS (Sx):
Sudden, acute, progressive, bilateral, ascending paralysis.

ALLOPATHIC TREATMENT (Rx):
An emergency - respirator, intensive care.

COMPLICATION:
Death by heart or respiratory failure.
Nervous system - pathologies

Multiple sclerosis (MS):
• A demyelinating condition - progressive demyelination of neurons (CNS: brain & spinal cord) & damage to the myelin sheath causing impulse conduction and communication between nerves being disturbed.
• Increased risk with family history.
• M:F 1:2.
• Usually ~20-50 years of age.

CAUSES:
Unknown: maybe viral or autoimmune. Environmental, genetic and dietary risk factors have been found.

SIGNS & SYMPTOMS (Sx):
‘Disease of the thousand faces’: includes sensory, motor & visual degeneration, leading to: numbness, burning, tingling, blurred vision, progressive paralysis. Can be progressive or relapsing - remitting.
Multiple sclerosis (MS):

PROGNOSIS: progressive conditions have a poor prognosis.

DIAGNOSIS:
NO definite test, MRI scan, CSF analysis.

DIFFERENTIAL DIAGNOSIS (DDx):
Herniated disk, sore eyes, Herpes zoster (shingles), candidiasis, mercury poisoning, motor-neuron disease (early stages).

ALLOPATHIC TREATMENT (Rx):
Constant symptoms are therapy resistant. Corticoids, interferon, physiotherapy.

ALTERNATIVE TREATMENT (Rx):
Lifestyle & diet are important! A diet low in saturated fat has shown to help, an anti-inflammatory diet. Herbal medicine for autoimmune & antiviral conditions. Low-grade exercise (not strenuous!).

Nervous system - pathologies

Main symptoms of Multiple sclerosis

Central:
- Fatigue
- Cognitive impairment
- Depression
- Unstable mood

Visual:
- Nystagmus
- Optic neuritis
- Diplopia

Speech:
- Dysarthria

Throat:
- Dysphagia

Musculoskeletal:
- Weakness
- Spasms
- Aesthetic

Sensation:
- Pain
- Hypoesthesia
- Paraesthesia

Bowel:
- Incontinence
- Diarrhea or constipation

Urinary:
- Incontinence
- Frequency or retention

Motor neuron disease:
- Progressive degeneration of motor neurons in brain stem, spinal cord & motor cortex (involved in the planning, control and execution of voluntary motor functions).
- Age of onset is typically >40yrs (highest incidence occurring between 50-70yrs).
- Forms of motor neurone disease include:
  - Amyotrophic lateral sclerosis (ALS) – most common form (sometimes called Lou Gehrig's disease).
  - Primary lateral sclerosis (PLS).
  - Progressive muscular atrophy (PMA).
  - Progressive bulbar & pseudobulbar palsy (PBP).

CAUSES:
Unknown, suspected genetic link & toxic environmental factor as well as trauma.

SIGNS & SYMPTOMS (Sx) of ALS - the most common form: Main feature is muscle weakness.
Nervous system - pathologies

Motor Neurone Disease (MND)

CST (upper motor neurone – UMN)

α-motoneurone
(lower motor neurone - LMN)

Motor cortex

Muscle fibre

Nervous system - pathologies

Motor neuron disease:
SIGNS & SYMPTOMS (Sx) of ALS:
**Early:** progressive muscle weakness / twitching / jerking (at rest) of the hands, arms & shoulders. (progressive bulbar palsy the 1st symptoms may appear in the muscles around the mouth, face & throat).
**Late:** affects the legs & feet (dragging the leg & tripping), voice changes & speech is also affected (slurred speech), swallowing difficulties.

ALLOPATHIC TREATMENT (Rx):
Currently no cure, specialist treatment & care.

ALTERNATIVE TREATMENT (Rx):
An anti-inflammatory diet, herbs & nutrients to support nerve function.

PROGNOSIS:
Death by respiratory failure - typically, within 3-5yrs (but sometimes over 20yrs).
Alzheimer’s disease:
Neurodegenerative disease of the cerebral cortex associated with abnormal protein deposition, destroying neurons that secrete acetylcholine. It is the most common type of dementia.

CAUSE:
Unknown, genetic link, aluminium toxicity.

SIGNS & SYMPTOMS (Sx):
Starts with the inability to incorporate new knowledge despite the retention of old information. Eventually leads to dementia.

ALLOPATHIC TREATMENT (Rx):
Medication to improve the symptoms & slow down the development. Psychological treatments such as cognitive stimulation.
Nervous system - pathologies

Alzheimer’s disease:

ALTERNATIVE TREATMENT (Rx):
Nutrition is essential in prevention and support of this condition - deficiencies need to addressed, antioxidants & other nerve supporting nutrients. Herbs such as Ginkgo and Turmeric can be very helpful. Regular exercise.

PROGNOSIS:
People with Alzheimer's disease live for around 8 to 10 years on average after they start to develop symptoms.

Ginkgo biloba

- New study finds high aluminium levels in the brain of an Alzheimer’s victim, following eight years of occupational exposure to aluminium dust. Scientists conclude that this case suggests your olfactory system & lungs play a prominent role in the accumulation of Aluminium in your Brain.
  - Aluminium is Neurotoxic, targeting your CNS, which can lead to serious immunological and neurodegenerative disorders.
- Small amounts of Aluminium inhaled through your nose & mouth and absorbed from VACCINES, can accumulate over time, especially in your bones & brain.
- Recent studies show that aluminium contamination in food, drugs & consumer products is much worse than previously thought. (See Footnotes for more info. on Aluminium)
- Analysis of body Mineral Levels & Toxins can be carried out through Hair Mineral Checks at: http://www.mineralcheck.com/
Nervous system - pathologies

Parkinson's disease:
Slow, progressive neurological disorder associated with the degeneration of neurons in various parts of the brain, primarily the dopaminergic neurons in the substantia nigra. Affecting approx. 1% of individuals older than 60 years.

CAUSES:
Idiopathic, genetic link, TOXIC environmental factors: carbon monoxide or manganese poisoning, exposure to pesticides & herbicides. May develop after encephalitis.

PATHOPHYSIOLOGY:
There are 2 major neuropathological findings in the medical model:
1. Degeneration of dopamine generating neurons in the substantia nigra (midbrain) causing dopamine deficiency leaving patients less able to direct or control their movement.
2. Accumulation of abnormal proteins (called Lewy Bodies), in the cerebral neurons.
Parkinson’s disease:
SIGNS & SYMPTOMS (Sx):
The 3 cardinal signs:
1. Bradykinesia: low voice, shuffling steps.
2. Resting tremors: jerky movements.

ALLOPATHIC TREATMENT (Rx):
Dopamine replacement (Levodopa / L-DOPA) - side effects can worsen symptoms, embryological stem cells.

ALTERNATIVE TREATMENT (Rx):
Anti-inflammatory, anti-oxidant & mitochondria support through diet, nutritional supplements, herbs. Acupuncture & homeopathy can also be very helpful.
Huntington’s disease / chorea:

- Inherited neurodegenerative disorder affecting brain / basal ganglia which affects muscle co-ordination and some cognitive functions.
- The most common genetic cause of abnormal involuntary writhing movements called chorea.
- Inherited lack of GABA.
ACTIVITY

Please remember to have a look at the **glossary** for this topic. Understanding the words and their derivation will help you understand and memorise them!

Please also look at the **prefixes & suffixes** to help you understand the medical terminology used in this lecture – you may be tested on this.