**Upper Motor Neurons and Lower Motor Neurons Dr. Gary Mumaugh – Campbellsville University**

**Upper and Lower Motor Neurons**

* **Upper motor neurons (UMN):** have their cell bodies located in the motor cortex and synapse with lower motor neurons
* 2 tracts of UMN:
  + Corticospinal tract **-** from motor cortex to spinal cord
    - UMNs are located in motor cortex (primary motor cortex, premotor cortex, supplementary motor area)
    - They give off axonsthat travel through thecorona radiata**,** internal capsule**,** midbrain, pons, medulla to end up in the
    - Spinal cord: UMN synapse with LMN
  + Corticobulbar tract: from motor cortex to cranial nerves nuclei
    - UMNs are located in the motor cortex (primary motor cortex, premotor cortex, supplementary motor area)
    - They give off axons that travel through the corona radiata**,** Internal capsule**,** midbrain, pons and medulla
    - UMNs synapse on LMN located in cranial nerves nuclei
* **Diagram

  Description automatically generatedLower Motor Neurons (LMN):** they have their cell bodies either in the spinal cord or the cranial nerve nuclei, and are directed towards skeletal muscles
* 2 destinations of LMN:
  + Anterior grey horn – continuation of the corticospinal tract
  + LMNs then continue towards skeletal muscles of the body, where they elicit voluntary movements
  + Specific cranial nerves nuclei – continuation of the corticobulbar tract
  + Relevant nuclei
    - Trigeminal (CN V) nucleus – mastication muscles
    - Facial (CN V) nucleus – facial expression muscles  (mimetic)
    - Nucleus ambiguous (CN IX, X, XI) – muscles of pharynx, larynx, uvula, palate
    - Relevant for speech, swallowing and articulation
    - Hypoglossal (CN XII) nucleus – tongue muscles
  + Damage to these LMNs leads to bulbar palsy

**Causes of UMN and LMN Lesions**

* **UMN Lesions**
  + UMN lesions can be caused by
    - Stroke (CVA) – both ischemic and hemorrhagic
    - Demyelination
    - Multiple sclerosis, Freiderich’s Ataxia, B12 deficiency
    - Amyotrophic Lateral Sclerosis (ALS) – motor neuron disease
  + Damages the cells body of the UMN through free radical accumulation
* **LMN Lesions**
  + LMN lesions can be caused by
    - Viruses: Poliomyelitis, West Nile Virus
    - Spinal muscle atrophy
    - Cauda equina syndrome – damage to the axon, caused by a disk herniation of the lumbar and sacral portions of the spine
    - Diabetic neuropathy
    - Botulism
    - ALS

**Muscle Mass**

* Refers to muscle mass changes in different types of lesions
* UMN Lesions
  + Decreased protein synthesis in the muscle cell will cause
    - Disuse Atrophy
    - 15-20% loss of muscle mass
* LMN Lesions
  + Increased protein synthesis in the muscle cell will cause
    - Denervation Atrophy
    - 75-80% loss of muscle mass

**UMN Lesions**

* UMN lesions can involve damage anywhere from cortex to spinal cord.
* Motor cortex controls voluntary movements:
  + In case of damage: no connection between cortex and spinal cord (where LMNs are located) → no control over skeletal muscles → they start atrophying
  + **Disuse Atrophy**: decrease of 15-20% in muscle mass

**LMN Lesions**

* LMNs release Acetylcholine (Ach) at neuromuscular junction → Ach can bind to 2 types of receptors
  + Nicotinic receptors: when activated, elicit AP in muscle cell → contraction
  + Muscarinic receptors: begin intracellular cascade → production of transcription factors → up-regulation of protein synthesis in muscle cell
* In case of damage: decreased Ach release, which has 2 effects
  + No muscle contraction
  + No intracellular pathway for transcription factors
    - Decreased protein synthesisand increased protein degradation
    - Muscles start atrophying
      * **Denervation atrophy** – decrease of 70-80% of muscle mass

**Fasciculations**

* Fasciculation are involuntary pathological contractions.
  + A muscle twitch is an involuntary contraction, but it's usually benign.
* Only seen in **LMNs lesions**: especially evident in the tongue
* LMNs lesions cause reduced Ach release
* In physiological situations:
  + Ach binds to nicotinic receptors → open Ligand- gated Sodium channels → Na+ flows into the cell → depolarization → AP → contraction
* In pathological situations:
  + Decreased **Ach** reduces receptor binding (the muscle thinks there are not enough receptors on the membrane) > increases receptor production > increases sensitivity to stimuli> they respond to a tapping of the muscle > involuntary muscle contraction - Fasciculation
* Fasciculations can be seen on a **EMG**: there called **fibrillations**

**UMN Lesions**

* In case of **UMNs lesions**: no stimulation of medullary reticulospinal nuclei which decreases inhibition of LMN
* LMN are more active and has two effects
  + ↑ motor neuron activity: ↑ contractions
    - Increases muscle tone
    - **Hypertonia**
  + ↑ motor neuron activity: ↑ sensitivity to mechanical stimuli
    - Creates a stronger and bigger reflex - **Hyperreflexia**
    - Produce **spastic paralysis: decrease in strength**
      * NOTE: spasticity is very different from rigidity

**Spasticity vs. Rigidity**

* Spasticity
  + Velocity-dependent: increased resistance with higher velocity
  + Resistance only in one direction
  + Weakness
  + Clasp-knife phenomenon: the muscle makes resistance until eventually it gives out and goes slack
* Rigidity
  + Velocity-independent
  + Resistance in both direction
  + No significant weakness
  + Lead-pipe rigidity: arm is rigid throughout the whole movement
  + Seen in Parkinson’s: characterized by tremors

**LMN Lesions**

* Decreased motor neuron activity causing decreased stimulation of muscle fibers
* Decreased muscle tone – Hypotonia
* Decreased sensitivity of muscle spindles which causes
  + Decreased reflexive strength and muscle movement
  + Decreased DTR – Hyporeflexia
* Muscle strength is a flacid paralysis

**Tests to diagnose UMN vs. LMN lesions**

* There are several special test that can be performed to test the presence of damage to motor neurons, however they result positive (pathological) exclusively in UMNs lesions.
* **Babinski sign:** 
  + Run the tip of your reflex hammer of the plantar surface of the foot, from heel to toes, moving laterally to medially
  + In physiological situations, it creates reflex that activates the plantar flexors: the toes curl downwards.
    - At the same time, UMNs send inhibitory signals to dorsiflexor.
  + In pathological situations: decreased inhibition of dorsiflexor → dorsiflexor overcome activity of plantar flexors
    - When stroking the bottom of the foot, the big toe dorsiflex and the other toes fan out
  + Positive Babinski sign is also seen in babies (<1yo):
    - Their corticospinal tract isn’t fully developed, it lacks myelination → the toes dorsiflex and the sign is positive
    - Not pathological, it disappears as they grow older.
* **Pronator drift** 
  + Ask the patient to stand with their arms extended and supinated, their eyes closed. Tap on the arms.
  + If a lesion of UMN is present, there will be an increase in pronators activity and decrease of supinator activity → the arms start drifting downwards and pronate at the same time

**Wrap Up of Upper Motor Neuron vs. Lower Motor Neuron Lesions**

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| --- | --- | --- |
|  | **UMN** | **LMN** |
| Muscle Mass | Decreased muscle mass 15-20% | Decreased muscle mass 75-80% |
| Type of Atrophy | Disuse Atrophy | Denervation Atrophy |
| Muscle Strength | Spastic Paralysis | Flacid Paralysis |
| Muscle Tone | Increased Muscle Tone  Hypertonia | Decreased Muscle Tone  Hypotonia |
| DTR | Increased - Hyperreflexive | Decreased - Hyporeflexive |
| Fasciculations | Absent | Present Because of decreased AcH |
| Fibrillations | Absent | Present Because of decreased AcH |
| Special Tests | Present - Positive | Absent - Negative |