MEDICAL UNIVERSITY – PLEVEN

DEPARTMENT OF NEUROLOGY

CLASS 5: Motor activity – methods of examination. Muscle power. Muscle tone. Upper and lower motor neuron lesion. Bulbar and pseudobulbar palsy.

Pyramidal system /Cortico-spinal tract/

- Consists of upper /corticospinal and corticonuclear tracts/ motor neuron and lower motor neuron /neurons in the anterior horns of the spinal cord/
- Motor cortex in precentral gyrus, known as Brodman's area 4:
  - is a narrow cortical field, located in front of the central fissure
  - has a somatotopical arrangement – this is a somatotopic order of a homunculus standing on its face, corresponding to that of a sensory homunculus in the postcental gyrus

Upper motor neuron:

- 31-60% of its fibers originate in Brodmann area 4 /primary motor cortex/, 29% originate from area 6 /premotor cortex and supplementary motor cortex/, 20-40% from area 3, 1,2, 5 and 7 /primary and associative cortex/.
- Only 3-4% of the axons in the pyramidal tract originate from giant Betz cells, Vth layer of Brodmann's area 4
- The premotor area occupies the larger lateral surface of area 6 and is associated with planning of the motor act, stabilization of the proximal muscles of the limbs during realization of the motor act.
- The supplementary motor cortex occupying the medial interhemispheric part of area 6 is connected with initiation of movements, autonomous responses.

Two tracts of pyramidal system:

Corticospinal tract:

- The fibres of corticospinal tract starts from the motor cortex, form corona radiata, converge through the corona radiata towards the internal capsule. They pass through the internal capsule in somatotopic order /anteriorly the fibre for arm, after that these for the trunk and posteriorly those for legs/. The fibres of corticospinal tract pass through the anterior two-thirds of the posterior limb of the internal capsule.
- The fibres of corticospinal tract enter the midportion of crus cerebri of the midbrain in somatotopic order. After that they descend through the base of the pons, radiating between the pontine nuclei. They continue downwards to the anterior surface of medulla oblongata where they form the pyramids. At the lower end of medulla oblongata 80-85% of the fibres of each pyramidal tract cross to the contralateral side in so called pyramidal decussation and form the lateral corticospinal tract. The other motor fibres 15-20% uncrossed descend in the anterior funiculus and form anterior corticospinal tract and connect the anterior horns on the cervical and thoracic spine.

Table 1: Main differences in the descending corticospinal tracts on the level of the spinal cord

Descending corticospinal tracts	Function	Origin	End	Location in spinal cord
Lateral corticospinal tract	Fine motor function (controls distal muscles)	Motor and premotor cortex	Anterior horns cells	Lateral columns (crossed at pyramidal decussation)
Anterior corticospinal tract	Postural motor function (controls proximal and axial muscles)	Motor and premotor cortex	Anterior horns cells	Anterior columns (uncrossed until spinal cord)

Corticonuclear (or known also as corticobulbar) tract

- The fibres of the corticonuclear tract representing the face and head pass through the genu of the internal capsule and terminates in the motor nuclei of cranial nerves
- The fibres originates from the cortical motor area where the face is represented, pass through the genu of the internal capsule and end either directly or indirectly by interneurons on the motor nuclei of cranial nerves.
- The nuclei of CN V, X, XI and upper part of the nucleus of CN VII have bilateral innervation due to partial supranuclear decussation of the fibres.
- The lower part of the nucleus of CN VII and nucleus of CN XII have only one-sided, contralateral innervation from the pyramidal tract.
- The nuclei of CN III, IV and VI do not receive pyramidal tract projections.

Lower motor neuron

- These neurons form the final common pathway to the skeletal muscles.
- Alpha motor neurons in the anterior horns of the spinal cord innervate extrafusal muscle fibres, responsible for muscle contractions. Their axons leave the spinal cord ventrally, form ventral roots and terminate on the neuromuscular junctions. Each alfa motor neuron controls a group of several muscle fibres, forming a motor unit, up to 10-20 motor units in fine motor muscles and up to 1500 in the large muscles. The large motor units are responsible for maintaining the posture, the small ones – for fine movements.
- Gamma motor neurons are small motor neurons, innervating intrafusal muscle fibers. Their impulses increase tension on the muscles spindles, influencing its sensitivity to overall muscle stretch.

Muscle strength (or muscle power)

Muscle strength is a kinetic muscle activity measured by the strength of clonic muscle contraction. It depends on the functional status of the pyramidal system, lower motor neuron and that of the

striated muscles. The term used for reduced muscle strength is paresis. The complete absence of active movement is called paralysis or plegia.

Table 2. Terms used for motor deficit in clinical practice

monoparesis	weakness of one limb
monoplegia	paralysis of the muscles of one limb
hemiparesis	weakness of both limbs on one side of the body
hemiplegia	paralysis of both limbs on one side of the body
paraparesis	weakness of both legs or hands
paraplegia	paralysis of both legs or hands
quadriparesis	weakness of four limbs
quadriplegia	paralysis of four limbs

Table 3. Grading of muscle strength on Medical Research Council Scale

Grade	Muscle strength
5	Active movement against gravity and full resistance
4	Active movement against gravity and some resistance
3	Active movement against gravity
2	Active movement of a part of the body when gravity is eliminated
1	No active movement of a part of the body is noted. Only a trace of contraction is evident.
0	No muscle contraction is detectable

A way of examination of static muscle strength – the patient is asked to resist the examiner's attempt to move a fixed part of the examined's limb.

A way of examination of dynamic muscle strength – the patient is asked to perform an active movement against the examiner's resistance.

Examples for examination of the muscle strength

For upper limbs:

– Abduction of the shoulder – the patient extends outstretched upper arm to the side against resistance of the examiner (test for deltoid muscle)

- Extension of the forearm – the patient is trying to straighten out his flexed forearm against resistant (test for triceps brachii muscle)

- Abduction of fingers – the patient is trying to spread the fingers against the attempt of the examiner to keep them together (test for interossei muscles and ulnar nerve)

For lower limbs:

- Adduction of the hip – after separating the extended legs the patient is trying to bring the knees together against the examiner's hands (test for adductor muscle)

- Extension of the knee – with partially flexed knee the patient is trying to do extension against the resistance of the examiner (test for quadriceps muscle)
- Plantar flexion of the foot – the patient is trying to push the hand of the examiner, placed on his foot (test for gastrocnemius muscle)

Table 4. Tests for examination of muscle power

Hip flexion - Hip flexed against resistance - Femoral nerve	Hip extension - Patient attempts to keep heels on bed against resistance - Inferior gluteal nerve
Hip abduction - Patient lying on back tries to abduct the leg against resistance - Superior gluteal nerve	Hip adduction - Patient lying on back tries to pull knees together against resistance - Obturator nerve
Knee flexion - Patient pulls heel towards the buttock and tries to maintain this position against resisitance - Sciatic nerve	Knee extension - Patient tries to extend knee resistance - Femoral nerve
Dorsiflexion - Patient dorsiflexes the ankle against resistance - Patient has difficulties in walking on heels - Deep peroneal nerve	Plantarflexion - Patient plantareflexes the ankle against resistance - Patient has difficulties in walking on toes - Tibial nerve

Tests for examination of latent paresis are important for clinical practice – methods for testing the slightest disorders of muscle strength.

Mingazzini-Strumpel's test – the patient is lying with closed eyes, outstretched in maximal extension and supination arms and elevated legs. In positive test - the latent hemiparesis is demonstrated by pronation of the arm and gradually lower position of the leg in comparison with the first position

Barre's leg test – the patient is lying in recumbent ventral position, his both legs are maximally flexed in the knee joints. In this position the affected leg starts to fall gradually and to increase the angle at the knee joint.

A syndrome of the upper motor neuron lesion – clinical signs:

- Decreased muscle strength (mono- or hemi-paresis/plegia)
- Muscle hypertonia - spastic muscle tone
- Exaggerated tendon reflexes
- Decreased or absent superficial reflexes (abdominal, cremasteric)
- Pathological reflexes – plantar response - extensor
- Absence of degenerative wasting and fasciculation

A syndrome of the lower motor neuron lesion – clinical signs:

- Muscle weakness in one or several muscles
- Muscle hypotonia
- Decreased or absent tendon reflexes
- Normal superficial reflexes
- Wasting and fasciculations (involuntary, spontaneous visible contractions of a group of muscle fibres, common in disorders affecting anterior horn neurons)

Upper motor neuron syndrome	Lower motor neuron syndrome
1.Reduced muscular strength (paresis) or complete loss of muscular strength (plegia) in the limbs	1. Decreased strength (paresis to plegia) in individual muscles or muscle groups
2. Exaggerated deep tendon reflexes	2. Hyporeflexia or areflexia
3. Abdominal skin areflexia	3. Muscular hypotonia to atonia
4. Positive pathological reflexes	4. Hypotrophy of the affected muscles
5. Spastic increase in muscle tone	5. Muscular fasciculations
6. Absence of muscular atrophy or hypotrophy	6. EMG indications of peripheral motor neuron damage
7. Presence of pathological synkinesias	
8. Absence of electromyographic (EMG) changes	

Table 5. Clinical feature according to the location of the lesion of UMN or LMN

Location of a lesion of UMN or LMN	Clinical features
A lesion in motor cortex - area 4	Monoparesis of arm or leg Focal epileptic seizure Caused by tumor, trauma or stroke
A subcortical lesion in motor area	Contralateral monoparesis of the hand or leg Caused by tumors or stroke
An internal capsule lesion	Contralateral spastic hemiplegia Contralateral paralysis of facial muscles innervated by facial and hypoglossal nerves Caused by stroke
A peduncule lesion	Contralateral spastic hemiplegia Ipsilateral lesion of oculomotor nerve Weber's syndrome
A lesion in the pons	Contralateral hemiplegia

	Ipsilateral lesion of abducens or facial nerves Millard-Gubler's or Foville's syndromes
A cervical spine lesion	Ipsilateral spastic hemiplegia without involvement of facial or tongue muscles Caused by spinal cord trauma, multiple sclerosis, amyotrophic lateral sclerosis
A thoracic spine lesion	Ipsilateral spastic monoplegia of the legs Caused by spinal cord trauma, multiple sclerosis, amyotrophic lateral sclerosis
An anterior horn lesion	Ipsilateral flaccid paresis or paralysis Fasciculations in the affected muscles Caused by amyotrophic lateral sclerosis, poliomyelitis, transverse myelitis, intramedullary tumors, hematomyelia
An anterior root lesion	Ipsilateral flaccid paresis Caused by a prolapse of intervertebral disk

Muscle tone. Disorders in muscle tone

Muscle tone is a measure of normal resistance while the muscle is stretched passively. It consists of static and dynamic components.

The **static muscle tone** depends on non-reflexive mechanisms like elasticity of the muscles, changes in the joints and length of muscles determining the tension at rest and maintaining the body posture at rest.

The **dynamic muscle tone** depends on the change of the length of the muscle and its stretching speed which is determined by reflex mechanisms and myotatic slow-stretch reflex.

Suprasegmental control of the muscle tone:

- The primary, supplementary motor, premotor and postcentral sensory cerebral cortex control the muscle tone via extrapyramidal efferent fibres
- The brain stem structures are:
- The vestibulospinal system includes lateral vestibular nucleus and lateral vestibulospinal tract functioning under the suppressive control of vestibulocerebellum.
- The reticulospinal system (reticular formation and reticulospinal tract) consists of two parts – excitatory area that can increase the muscle tone and supresssive area via the spinal cord neurons decreases the muscle tone.
- The cerebellum decreases via the frontal lobe and increase via the neocerebellum the muscle tone through projections to the reticular formation and vestibulospinal system.
- The basal ganglia control the activity of the segmental motor and interneurons via the thalamus, motor cortex and coticospinal tract. The reticular part of substancia nigra, pallidum and thalamic nuclei have an important influence on muscle tone.

Segmental control of muscle tone – The muscle tone depends on the activity of alpha, gamma motor neurons and interneurons in the anterior horns of the spinal cord.

Muscle tone examination:

- The patient is lying and relaxed as much as possible in a room with an optimal temperature.
- The examination begins with the neck muscles, continues with the upper limbs and then with lower limbs.
- Slowly stretching of particular muscles, achieved by passive movements (flexion or extension, abduction or adduction). The examiner has to evaluate the resistance felt during the movement (dynamic tone).
- The examiner palpates the muscle groups of the upper and the lower limbs to feel their tonic tension, informative for static tone.

Disorders in muscle tone

Muscle hypertonia can be divided on local – muscle spasms and generalized – spasticity and rigidity

Spasticity – increased tone, caused by lesions of upper motor neuron. It represents the resistance to passive stretch of muscles, which affects different muscle groups to a different extent. It is characterized by speed dependent increase of the tonic muscle stretch reflex and pathologically exaggerated deep tendon reflexes due to hyperexcitability of the stretch reflex.

Decortication – caused by a damage above the nucleus ruber especially a damage of the cortical control on this nucleus and its influence on the motor neuron via the rubrospinal tract. Its clinical manifestation is increased muscle tone in the upper limb flexors and in the lower limb extensors. Typical example is Wernicke-Mann posture, characterized by contralaterally to the side of the lesion flexed forearm and extended leg.

Decerebration – caused by a damage above the vestibular nuclei. Releasing from its control the result is increased muscle tone in extensors and decreased one in flexors of upper and lower limbs.

Rigidity is an increased plastic resistance of the muscles to passive stretching which remains constant throughout the whole muscle stretching act. It is caused by affected basal ganglia. Rigidity is relatively the same in flexor and extensor muscle groups and affects the distal, proximal and axial muscles. It is slightly prevalent in flexors groups, explaining the flexor posture of the patients. Rigidity indicates extrapyramidal dysfunction. It is the most common symptom of Parkinson's syndrome. It could be found in degenerative diseases – Parkinson's disease, strionigral degeneration, progressive supranuclear paralysis, multiple systemic atrophy), tumors (primary or metastatic), vascular (poststroke), metabolic disease (Wilson's disease, Fahr's disease), intoxication (neuroleptics).

"Lead pipe" rigidity manifests as a resistance through the whole stretching act similar to folding a lead pipe. It occurs in Parkinson's disease, mesencephalic damage, tetanus.

The combination between rigidity and tremor is known as **''cogwheel phenomenon'' or "Negro's phenomenon"**, clinically manifested in Parkinson's disease.

Hypotonia is a reduced resistance of the muscles to passive stretching. Hypotonic muscles are abnormally soft on palpation. Muscle hypotonia can be caused by a lesion of lower motor neuron, cerebellar diseases, in the acute stage of stroke or intoxication – anticonvulsants, neuroleptics, lithtium, antidepressants, lead, etc.

Bulbar and Pseudobulbar palsy

Bulbar palsy	Pseudobulbar palsy
1. Dysarthria	1. Dysarthria
2. Dysphonia	2Dysphonia
3. Dysphagia	3.Dysphagia
4. Paralysis of the soft palate homolateraly to the lesion	4.Glossal paresis
5. The uvula is diverted to the healthy side	5.Paresis of the vocal cords
6. Lingual hemiatrophy	6.Bilateral paralysis of the soft palate
7. Fasciculations in the affected lingual side exist in nuclear lesion of n. hypoglossus	7.Atrophy and fasciculation of the tongue are not found
8. There is disturbed sensation of the pharyngeal homolateral posterior wall, the tonsils and the lingual root.	8.Oral automatism reflexes are positive
9. Pharyngeal reflex is absent	9.Pharyngeal reflex is preserved
10. Taste perception in the rearmost lingual one third is impaired	10.Mandibular reflex is abnormally brisk