

### CLINICAL INTRODUCTION TO NEUROLOGY. MEDICAL HISTORY + BEDSIDE NEUROLOGICAL EVALUATION: MOTOR SYSTEM.

Eva Vlčková, Department of Neurology, University Hospital Brno Most of the pictures from: Ambler Z, Bednarik J, Ruzicka E. Clinical Neurology – General Part. Praha:. Triton 2008.



## NEUROLOGY



- a branch of medicine dealing with **DISORDERS OF THE NERVOUS SYSTEM**
- deals with the diagnosis and treatment of conditions and disease involving:
  - CENTRAL AND PERIPHERAL nervous systems

including their coverings, blood vessels

- MUSCLES

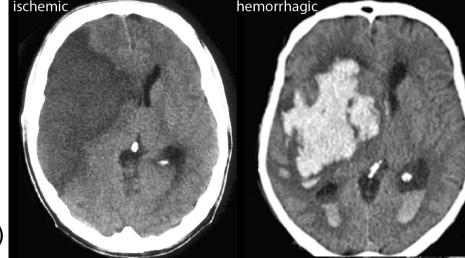
including neuromuscular junction

- $\rightarrow$  NO STETOSCOPE  $\odot$
- → NEUROLOGICAL HAMMER



# **DISEASES OR CONDITIONS**

- Stroke (ischemic or hemorrhagic)
- Epilepsy (<u>https://el.lf1.cuni.cz/epilepsie/default/Videa/video8.html</u>)
- Parkinson's disease (https://el.lf1.cuni.cz/pn/default/kazuistiky/kazuistika1.html)
- Multiple sclerosis
- Brain/spinal cord tumors
- Migraine + other headaches
- Carpal tunnel syndrome
- Neuromuscular diseases
- Spine + spinal cord diseases
- 3 Department of Neurology, University Hospital Brno



## **MEDICAL HISTORY – PRESENT ILLNESS**

- getting information <u>from the patient</u> or in some cases <u>from other observer</u> (important in seizures, dementia, aphasia...)
- Start with the PATIENT'S CHIEF COMPLAINT ("what brings you to see me?") these data focus attention on particular questions to be addressed in taking the history and clinical examination

### HISTORY OF PRESENT ILLNESS <u>including a history of development of</u> <u>particular symptoms:</u>

- mode of onset
- *duration* a *and progression* are critical in investigating the etiology!

(sudden onset x paroxysmal episodes x exacerbations and remissions x fast or slow progression)

 $M \vdash 1$ 

### - CHARACTERISTICS OF THE SYMPTOMS

(intensity – VAS, Likert scale, localisation, what relieves/makes the symptom worse)

# **MEDICAL HISTORY – OTHER DATA**

#### – history of **MEDICAL ILLNESSES AND PREVIOUS SURGICAL PROCEDURES**:

- neurological system is affected by *many non-neurological diseases* (DM)
- adversely <u>neurological diseases may involve the function of many systems</u> (Parkinson disease, diabetic neuropathy)
- Neurological presentation may be a <u>part of multiorgan involvement</u> (sarciodosis, vasculitis, mitochondrial diseases, storage diseases)
- current (and sometimes previous) <u>MEDICATIONS AND ALLERGIES</u>

previous – chemotherapy, izoniazid, neuroleptic agents

current – hypolipidemics, corticoid hormones, neuroleptics, opioids, hypnotics....

# **MEDICAL HISTORY – OTHER DATA**

#### - *timing of developmental milestones* (sitting, walk, first words)

in infants and young children

in adults whose illness started during childhood also the

#### – personal and social history

- occupation marital status
  - excessive stress at home, in school or in the workplace
- did the patient ever *use of alcohol, tobacco?*
- or did he used any other prescription or illegal drugs (dependence)?

#### – <u>family history</u>

(cave! misinterpreting symptoms and sings! – consequence of aging, family secret...)  $M \models D$ 

## **CLINICAL NEUROLOGICAL EXAMINATION**

- FULL NEUROLOGICAL EXAMINATION tests in detail every central nervous system

region, peripheral nerve, muscle, sensory modality and reflex

 $- \rightarrow too lengthy to perform in practice.$ 

#### – in practice: FOCUSED NEUROLOGICAL EXAMINATION

to examine in detail the neurological *functions that are relevant to the history* 

#### - + then SCREENING NEUROLOGICAL EXAMINATION

to *briefly check remaining parts* of the nervous system

<u>both presence and absence of abnormalities may be of diagnostic importance</u>
 (separation of hemiparesis arising from spinal cord and right cerebral cortex lesion....)

## **SCREENING NEUROLOGICAL EXAMINATION**

### = QUICK EVALUATION OF:

- mental status
- cranial nerves
- motor system
  - strength
  - muscle tone and tendon reflexes
  - presence of involuntary movements and postures
- coordination
- gait and balance
- sensation

Please, look at particular tests at:

https://el.lf1.cuni.cz/neuronorma/

 $M \vdash D$ 

## **NEUROLOGICAL EXAMINATION**

### - STARTS ALREADY DURING THE INTERVIEW:

- mental status, memory disturbace
- changes of facial expresion or mimics

hypomimia

ptosis

- rattern of speech (dysarthria, aphasia),
- presence of abnormal involuntary movements

MUNI MED



## **MOTOR SYSTEM**

10 Department of neurology, University Hospital Brno

# **MOTOR PATHWAYS**

- 1. <u>(central) motoneuron</u> (central paresis)=
   GYRUS PRECENTRALIS
- → PYRAMIDAL TRACT
  - Branches towards cranial nerves nuclei

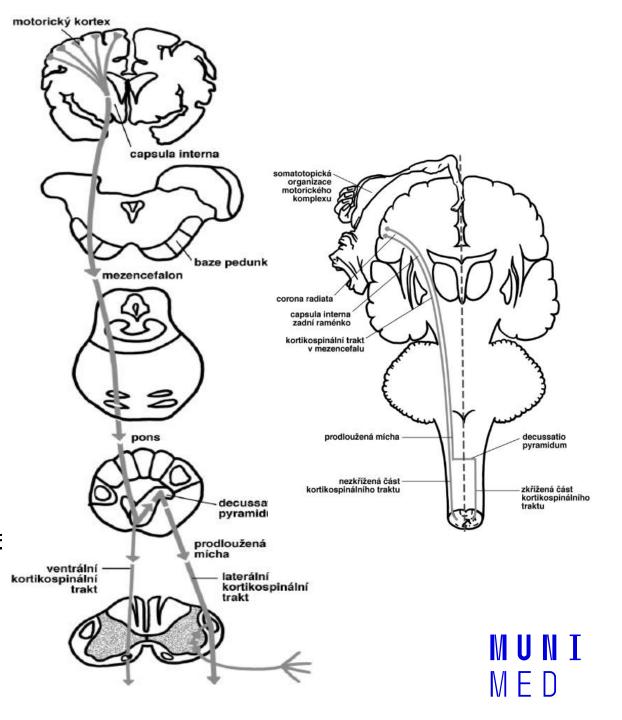
#### – CROSS OVER IN THE MEDULLA LEVEL

- resulting in muscles being controlled by the opposite side of the brain
- Function: voluntary movements, inhibiton of spontaneous spinal aktivity (gamma system)
- 2. (peripheral) motoneuron (peripheral paresis)=

#### ANTERIOR HORN OF THE SPINAL CORD

(or cranial nerve nuclei)

→ PERIPHERAL NERVE → MUSCLE



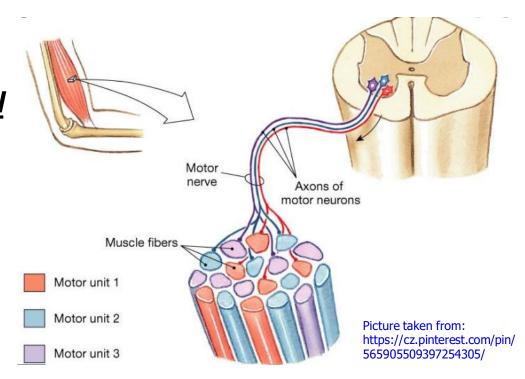
# MOTOR UNIT (MU)

= is made up of a motor neuron + the skeletal muscle fibers innervated by that motor neuron's axonal terminals (3-200).

-All muscle fibres in a motor unit are of the same fibre type.
-In a muscle, particular MUPs are intertwined with the others

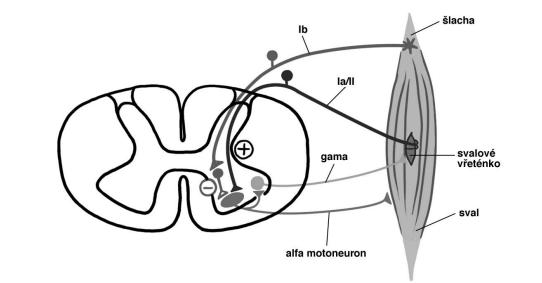
Contraction of single motor unit = a <u>FASCICULATION</u> - a small, local, involuntary muscle contraction and immediate relaxation which may be visible under the skin. In deeper areas, they can be detected by EMG. <u>Small rapid flickering or vermicular twitching</u> <u>https://www.youtube.com/watch?v=xuwdvBXcr30</u>

myelinová pochva axon presynaptický terminální útvar svnaptická ----štěrbina ganglion zadního kořene vezikuly Ach axon motoneuron myelinová pochva



# **MOTOR SYSTEM**

- Consists of 2 basic types of movements:
- <u>VOLUNTARY MOVEMENTS</u>
  - EASY (locomotion, rythmic movements)
  - COMPLICATED (piano playing)



 $N/ \vdash D$ 

- <u>REFLEX RESPONSES</u> = fast, stereotypic, involuntary, evoked by a stimulus
  - A part of many voluntary movements
  - E.g. Maintain muscle tone, relax antagonists during agonist contraction

#### <u>ABNORMAL MOTOR FUCTION RESULTS TO MUSCLE WEAKNESS = PARESIS</u> =

loss of voluntary movements (reflex movements may be perserved)

### **MOTOR IMPAIRMENT**

#### = MUSCLE WEAKNESS = PARESIS

#### = Partial or complete loss of voluntary movements. Usually refers to limb(s).

- Reflex responses may be preserved!!!! (deep tendon reflexes, Babinsky sign or even tripple flection respose in central paresis)

#### - ACCORDING TO THE SEVERITY:

**Complete** = <u>PLEGIA</u> (paralysis in which all voluntary movement is lost) Incomplete = <u>PARESIS</u> (further classification: mild, moderate, severe)

- BASED ON THE ETIOLOGY:

**CENTRAL** (spastic, ev. initially flaccid) **PERIPHERAL** (flaccid)

mixed

psychogenic (functional weakness)

## **EXAMINATION OF THE LIMBS/TRUNK**

Separate testing of each limb:

– Presence OF INVOLUNTARY MOVEMENTS, ABNORMAL LIMB POSITION

(pain release, flexion, extension)

- ACTIVE MOVEMENTS in particular segments or joints (pasive motility?)

Paretic signs + Power of main muscle groups

#### **– DEEP TENDON REFLEXES, EXTEROCEPTIVE REFLEXES**

- Plantar responses or other ABNORMAL REFLEXES
- MUSCLE MASS (atrophy x (pseudo- hypertrophy))
- MUSCLE TONE in response to passive flexion and extension

**– COORDINATION** 

MUNI Med

## **ACTIVE MOVEMENT TESTING**

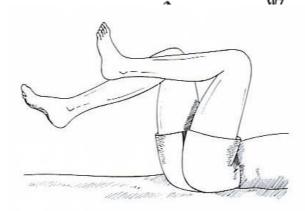
- PARETIC SIGNS = signs of paresis (Not much used in English sources)
- FUNCTIONAL TESTS OF MUSCLE ENDURANCE, reflect global impairment of muscle

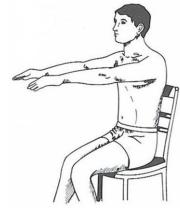
strength in particular extremity, not only the dysfunction of pyramidal tracks

 UE: <u>Mingazzini</u> – holding the extended arms raising forward, eyes closed (15 seconds or more) (decrease?)

**Duffour** (pronator drift) – supination in the same position (pronation?)

 LE: <u>Mingazzini</u> – in lying position, lower extremities flexed in hipps and knees (90 degrees angle)





### **ACTIVE MOVEMENT TESTING**

- Shoulders abduction
- Elbow flection, extension
- Wrist flection, extension
- Make a pinch
- Hip flection
- Knee extension, flection
- Ankle dorsal and plantar extension
- Movements of the big toe

### ACTIVE MOVEMENT IN EACH BIG JOINT

MUNI MED

## WEAKNESS (MUSCLE STRENGTH)

### 5-POINT MEDICAL RESEARCH COUNCIL (MRC) GRADING SCALE

- Grade 5 represents normal strength.
- Grade 4 = ability to move the limb only against gravity and resistance, but not full strength: represents "weakness" somewhere between 3 and 5.
   Covers such a large range, that should be expanded into mild, moderate, or severe: 4+, 4, and 4–
- Grade 3 = ability to move the limb only against gravity (not against resistance)
- Grade 2 = active movement only with gravity eliminated
- Grade 1 = is just a flicker or trace of contraction (visible contraction without visible joint movement).

[]/| |− | ]

- Grade 0 = no contraction

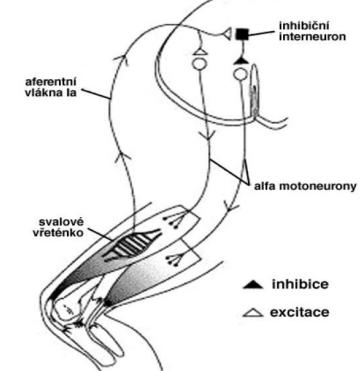
# **DEEP TENDON REFLEXES**

- Require the intact **REFLEX ARC**
- <u>PRINCPLE = Blow upon the tendon with a hammer</u>
- $\rightarrow$  short muscle stretching
- $\rightarrow$  leads to muscle contraction of the same muscle
- PROPRIOCEPTIVE REFLEXES (receptor inside the muscle)
- Quantification:

19

GRADE	Reflex
0 ()	Absent
1 (-)	Decreased (hypoactive)
2 (N)	Normal
3 (+)	Increased (hyperactive) without clonus
4 (++)	Hyperactive with clonus
https://www.youtube.com/watch?v=PPPgTg3L6k4 (1:25)	





## **DEEP TENDON REFLEXES**

### **PROPRIOCEPTIVE REFLEXES (DTR)**

#### **General interpretation:**

- Decreased DTR = periheral paresis (flaccid)
- Increase DTR = central paresis (spastic)

- In physiological conditions: <u>symmetrical</u> and intraindividually ± <u>stable</u>
- Note interindividual differences
- Note possible asymmetry
- Note **possible changes** during the patient follow-up (decrease? Increase?) IN T

 $N/ \vdash D$ 

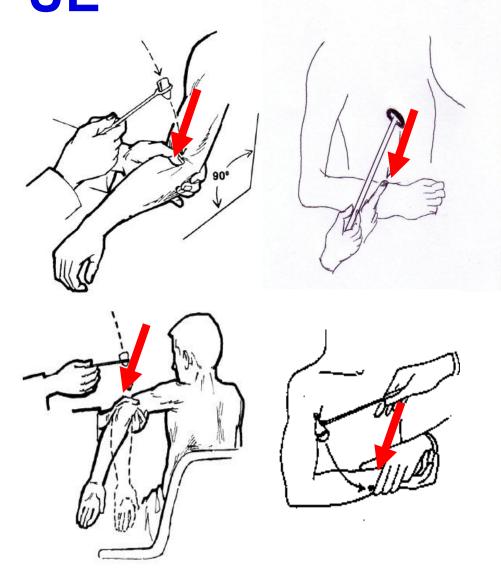
### **DEEP TENDON REFLEXES - UE**

Biceps reflex (C5-6): reflex contraction of the biceps muscle and jerk of the forearm.

**Styloradial (pronatinon)** (**C6**) (periosteal): tapping the processus styloideus radii (elbow in flexed in 90 degrees and semiproned forearm) leads to the slight forearm pronation

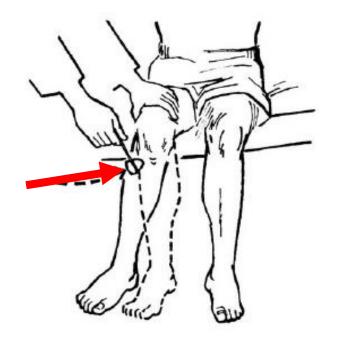
**Triceps reflex** (C6-8, mainly **<u>C7</u>**): tapping the triceps tendon while the forearm is hanging loose at a right angle to the arm causes the forearm extension.

Reflex of finger flectors (C8): tapping the ligamentum carpi transversum leads to slight flection of the fingers



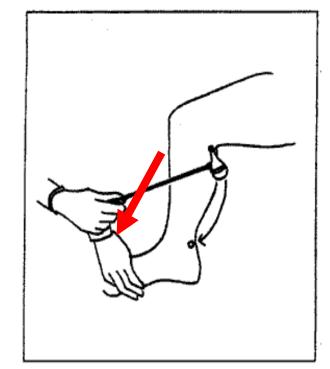
### **DEEP TENDON REFLEXES - LE**

Patellar reflex L2-L4 (knee-jerk) = striking the patellar tendon with a hammer just below the patella causes the quadriceps femoris contraction and shank extension



### Ankle jerk reflex S1/S2 (Achilles reflex)

occurs when the Achilles tendon is tapped while the foot is dorsi-flexed leading to the jerking of the foot towards its plantar surface



### **EXTROCEPTIVE REFLEXES**

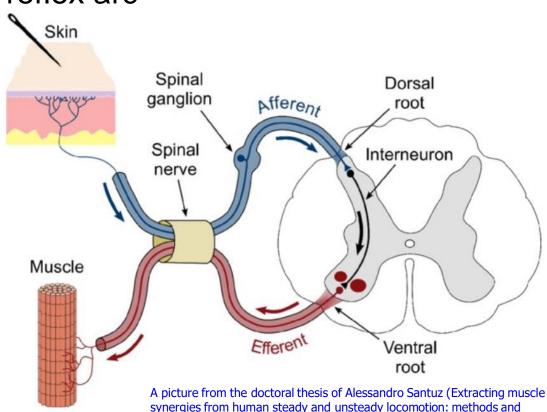
#### - The receptor transduces the signal from EXTERNAL ENVIRONMENT

- = evoked by touch, pain, pressure, vision, hearing, smell stimuli
- There are *intercalated interneurons* in the reflex arc

### = POLYSYNAPTIC REFLEX

– Response = muscle contraction

### - DECREASED BOTH IN CENTRAL AND PERIPHERAL NERVE LESIONS



experiments), source: Research Gate

## **EXTEROCEPTIVE (CUTANEOUS) REFLEXES**

#### **Superficial abdominal reflexes**:

- epigastric (T6-9)
- mesogastric (T9-11)
- hypogastric (T11-L1)

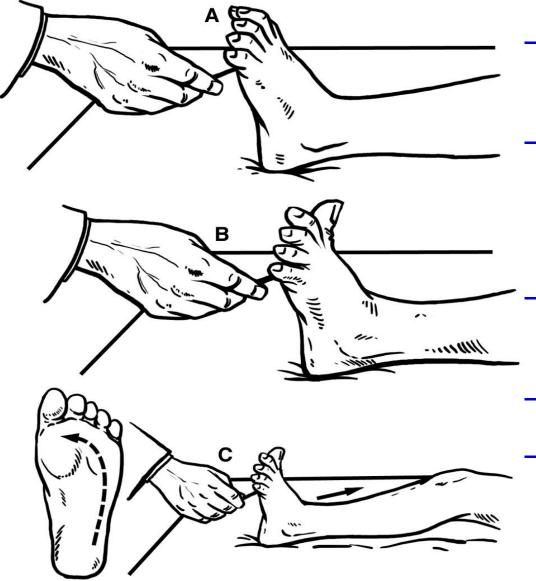
(ipsilateral abdominal wall contraction)

<u>Cremasteric reflex (L1)</u> – leads to cremaster muscle contraction and pull up the ipsilateral testicle

<u>**Plantar reflex**</u> – physiological response = flection of the foot and toes



### **ABNORMAL REFLEXES (EXTENSION)**



- "ATAVISTIC REFLEXES"(physiological in early infancy)

Later reapearance is a sign of abnormity

 Elicited by scratching the skin (not painful!) of the bottom of the foot along its lateral aspect from the heel forward

- A = <u>Plantar reflex</u> (normal response = flection of the foot and toes)

 $M \vdash I$ 

- B = Babinski sign (extensor plantar response)
- C= Triple flexion response (a spinal reflex

characterized by hip and knee flexion

accompanied by ankle dorsiflexion)

### **ABNORMAL REFLEXES - LE**

- Other abnormal reflexes with **<u>EXTENSOR RESPONSE</u>**:
  - <u>Roch</u> scratching the skin of lateral aspect of the foot from the heel forward to the half of the distance hell-fingers
  - <u>Chaddock</u> scratching the skin below the external ankle
  - Oppenheim stimulation of the skin of anterior aspect of the shank (crura)

 $N/ \vdash D$ 

by the examinator thumb and second finger

- <u>Gordon</u> distal calf massage
- Abnormal reflexes with *FLEXOR RESPONSE*:
  - <u>Rossolimo</u>: tapping the balls of the fingers (from plantar aspect) by hammer produces their flection

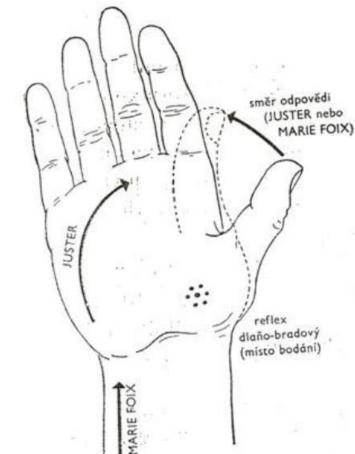
## **ABNORMAL REFLEXES - UE**

#### – JUSTER PHENOMENON (cutaneous or superficial reflex)

- counterpart of an extensor plantar reflex, a positive proof of pyramidal tract dysfunction)
- Elicited by <u>scratching the skin</u> of the palm along its lateral aspect (<u>antithenar side</u>) in the distal direction
   + along the metacarpo-phalagneal joints to the thumb.

#### – Normal reaction = no reaction

- Slow tonic slight adductuion of the thumb with slight opposition represents abnormity
  - = sign of pyramidal dysfunction



# **ABNORMAL REFLEXES - UE**

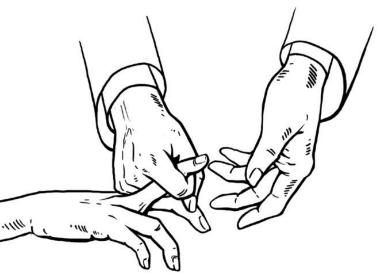


- TRÖMNER'S SIGN - with the fingers of the patient partially

flexed, the tapping of the volar aspect of the tip of the middle or index finger

<u>https://www.youtube.com/watch?v=59Tw9hbbAZE</u>

- <u>A HOFFMANN REFLEX</u> evoked by flicking a <u>dorsal</u> aspect of the relaxed finger tip with the fingers held in semiflexion
- <u>https://www.youtube.com/watch?v=PPPgTq3L6k4</u> (2:10)
  - positive response in both of them
    - = flexion of all four fingers and thumb;
  - simply implies increased muscle tone
  - not a direct counterpart of an extensor plantar reflex,
     which is positive proof of pyramidal tract dysfunction



### **OTHER ATAVISTIC REFLEXES**

- A <u>SUCK REFLEX</u> elicited by stimulation of the lips; pathological response = sucking movements of the lips, tongue, and jaw.
- The PALMOMENTAL REFLEX is an ipsilateral contraction of the mentalis and orbicularis oris after stimulation of the thenar region of the hand. This reflex is present in 20% to 25% of healthy adults in their thirties and forties.
- The <u>GRASP REFLEX</u> (physiological in infants, sign of deliberation of frontal lobes) patient grasps the examinator fingers inserted into his/her hands (palms). When being asked, patient does not know why he/she performed the grasp maneuver. Alternatively: is elicited by stroking the palm of the patient's hand. The reflex is present if the patient's fingers flex or the hand closes.

 $N/ \vdash D$ 

### MUSCLE MASS, ATROPHY

Takes weaks to month (years) to develop (not present in very acute lesions)







Muscle hypotrophy or atrophy is a sign of <u>peripheral</u> <u>motor neuron lesion</u> (flaccid paresis)

In central motor neuron lesion (spastic paresis), <u>only mild</u> atrophy develops <u>due to inactivity (</u>0.5–0.6% of MUNI total **muscle** mass per day).

## **MUSCLE TONE**

#### - MUSCLE'S RESISTANCE TO PASSIVE STRETCH DURING RESTING STATE

- **DECREASED** (*flaccidity*) in lower motor neuron/ peripheral nerve disseases
- **INCREASED** in central nervous system disease: *spasticity or rigidity*

#### - **SPASTICITY** is **caused by stretch reflex exaggration** and accordingly is dependent on stretch rate:

- if the muscle is slowly stretched, tone may be normal
- if the muscle is stretched more rapidly, increasing amounts of resistance occur
- $\Rightarrow$ <u>rate-sensitive</u>, preferential involvement of extensors
- \_ "clasp knife" or <u>pocket knife</u> fenomenon
- central motor neuron dysfunction/lesion/disease
- RIGIDITY: increased muscle tone, not depending on the rate of movement.
  - found equally in both extensors and flexors (leadpipe rigidity, cogwheel phenomenon) II U II
  - caused by <u>extrapyramidal disease</u> (or side effect of antidopaminergic drugs)



 $M \vdash D$ 

### FLACCID VS. SPASTIC PARESIS (CENTRAL VS. PERIPHERAL)

- The term "<u>flaccid" indicates the absence of spasticity</u> or other signs of disordered central nervous system motor tracts such as hyperreflexia, clonus, or extensor plantar responses or other abnormal reflexes
- <u>SPASTIC PARESIS = CENTRAL</u> (upper motor neuron dysfunction)
- FLACCID PARESIS = PERIPHERAL (lower motor neuron dysfunction)
  - Following the sudden development of central paresis (stroke...), the central paresis is flaccid for few days (weeks?)

 $N/I \vdash I$ 

### FLACCID VS. SPASTIC PARESIS (CENTRAL VS. PERIPHERAL)

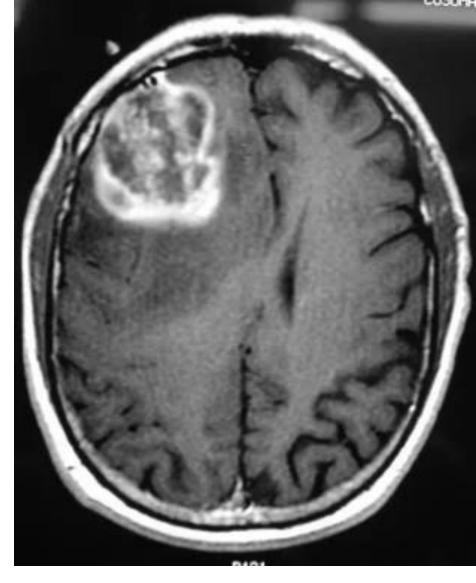
	SPASTIC PARESIS	FLACCID PARESIS
MUSCLE STRENGTH	$\downarrow\downarrow$	$\downarrow\downarrow$
PARETIC SIGNS	POSITIVE	POSITIVE
DEEP TENDON REFLEXES	$\uparrow \uparrow$	$\downarrow\downarrow$
EXTEROCEPTIVE REFLEXES	$\downarrow\downarrow$	$\downarrow\downarrow$
ABNORMAL REFLEXES	Present	Absent
MUSCLE MASS	Without pronaunced atrophies (only mild as a result of inactivity)	Atrophies (take some time to develop)
MUSCLE TONE	<b>†</b> †	$\downarrow\downarrow$
DISTRIBUTION	Hemiparesis, paraparesis, sometimes monoparesis	Particular nerve/root/plexus. Para-/ quadriparesis

## **CASE REPORT 1**

- Patient MD, man, 49 years old
- No regular medication, no important medical history
- For 2 months slow progression of headache (increasing intensity) and weaknes of left arm and leg and slight articulation problems
- decline of psychomotor speed according to his relatives
- Clinically: decreases muscle strength in left extremities, increased deep tendon reflexes, spasticity (↑ muscle tone), Babinsky sign

#### – = <u>CENTRAL (SPASTIC) HEMIPARESIS</u> (+ dysartria +

 $\downarrow$  PM speed) in patient with brain tumor (MR  $\rightarrow$ )



T1-weighted axial gadoliniumenhanced MRI demonstrates an enhancing tumor of the right frontal lobe – glioblastoma multiforme

MUNT

MED

## **CASE REPORT 2**

- TR, women, 73 years old
- Diabetes mellitus type II (25 years)
- BMI 31, hypertension, hypercholesterolemia...
- For 10 years <u>slow progression of sensory</u>
   <u>symptoms</u> (numbness in feet, speading
   proximally) and <u>later motor weakness</u> in distal
   parts of her lower legs.
- Peroneal (and calf) atrophies (→), ↓ muscle tone, ↓ deep tendon reflexies, peroneal paresis, stocking-like dysesthesia

### PERIPHERAL PARESIS – FLACCID in patient with severe diabetic polyneuropathy



## **OTHER TYPES OF PARESIS**

- FLACCID CENTRAL PARESIS – initially following acute-onset central motor neuron

lesion (it takes few hours or days to develop spasticity)

- Muscle tone and proprioceptive (deep tendon) reflexies are decreased
- Abnormal reflexes (Babinsky) are frequently negative at the initial phase
- No atrophies, no fasciculation
- Note the distribution (hemi?, para? mono?)
- •
- MIXED upper and lower motor neuron lesion (e.g. Amyotrophic lateral sclerosis)
  - Muscle tone and proprioceptive (deep tendon) reflexies may be ↓ or ↑ (more frequently ↑)

 $N/I \vdash II$ 

- Abnormal reflexes (Babinsky) frequently positive
- Fasciculations and atrophies
- No sensory distrubance!!! (unless the association with other neurological diseases occurs)

#### **CASE REPORT 3**

#### EF, woman, 63 years old

- Medical history: Diabetes mellitus type 2 (for 17 years), arterial hypertension, hypercholesterolemia, smoking 20 cigarettes per day since the age of 20 → many vascular risk factors
- Present illness: During the dinner, she realized a sudden onset of impaired speach, which was not fluent. The patient was not able to express what she wanted, but was able to understand quite well. Furthermore, the right sided weakness also developed within few minutes.
- The symptoms remained, so she called emergency and was brought to the emergency department.
- Clinical findings: <u>NONFLUENT (BROCCAS') APHASIA AND RIGHT</u>
   <u>SIDED FLACCID HEMIPARESIS</u> (decreased muscle strength and positive paretic signs, decreased proprioceptive reflexes, negative Babisky sign, no atrophies, no fasciculations).
- Acute CT = negative = ACUTE ISCHEMIC STROKE
- The patient treated with systemic thrombolysis with partial effect (regression of the symptoms, but not complete)
- Subacute MR confirmed the ischemic region in corresponding area.



Acute CT: no clear abnormities



Subacute MR – ischemic region in left posterior F lobe.

#### **PSYCHOGENNIC PARESIS (FUNCTIONAL WEAKNESS, NON-ORGANIC PARALYSIS)**

- weakness of an arm or leg without evidence of damage or a disease of the nervous system
- psychogennic trigger
- a manifestation of 'dissociative motor disorder' or <u>CONVERSION DISORDER</u>
- or **FEIGNING** (the patient is just pretending the symptom)
- both INTERNALLY INCONSISTENT and incongruent with any neurological disease.
- Only "weakness" (decreased strength + paretic signs note Dufour!)
- without any other neurological symptoms
  - Normal reflexes (both proprio- and exteroceptive), normal muscle tone
  - No abnormal reflexes (Babinsky)
  - No atrophies, no fasciculations
  - Fluctuating (unstable) weakness severity, not respecting anatomic distributions
  - Frequent atypical features (predominant plantar flexion weakness, not able to flex UE in the elbow...)

#### **BESIDES THE PYRAMIDAL TRACT, THE SPINAL MOTOR ACTIVITY IS REGULATED BY:**

- <u>SUBCORTICOSPINAL (extrapyramidal) TRACTS</u> (vestibulospinal, reticulospinal, rubrospinal, cerebellospinal...). The most important roles play:
- <u>CEREBELLUM</u> main function <u>= coordinates</u> voluntary movements such as posture, balance, limb movements, and speech, resulting in <u>smooth and balanced muscular</u>
   <u>activity</u>. Cerebellar cortex <u>receives information from the sensory</u> and vestibular systems, the spinal cord, and other parts of the brain incl. cortex. These data are used for movement regulation.
- <u>EXTRAPYRAMIDAL SYSTEM</u> (= motor structures of frontal premotor cortex, <u>basal ganglia</u> and brainstem, outside of the corticospinal pyramidal tracts). Mutually interconnected in several <u>circuits.</u> Basal ganglia appear to be important in the process <u>of initiation of</u> <u>movement</u> and the <u>maintenance of stereotyped movements</u> once they are initiated: postural control, resting muscle tone, automatic associated movements (e.g., swinging the arms while walking), and possibly emotional motor expression (e.g., smiling, frowning, laughing, crying, etc.)

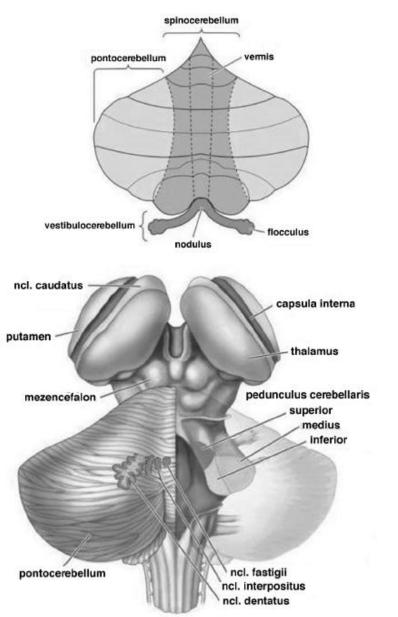
#### Located in the **POSTERIOR CRANIAL FOSSA**

- It is separated from the overlying cerebrum by a layer of dura mater (called tentorium cerebelli)
- two hemispheres + a narrow midline zone (the vermis)
- Superficial cortex (grey matter), white matter in the depth

#### **FUNCTION**

- Important role in the *motor control*
- contributes to <u>coordination</u>, precision control of movements, and accurate timing of precise movements
- Responsible for <u>muscle synergy</u>
- Modifies the muscle tone
- Upright body position, balance maintenance
- <u>HEMISPHERES</u> coordinate limb movements
- <u>VERMIS</u> coordinates trunk movements

#### CEREBELLUM



# **COORDINATION (CEREBELLUM): TERMS**

- **<u>ATAXIA</u>** denotes a syndrome of imbalance and incoordination involving gait, limbs + speech
- lack of voluntary coordination of muscle movements
- usually results from a disorder of the cerebellum or its connections
- <u>DYSMETRIA</u> refers to a lack of coordination of movement typified by the undershoot or overshoot of intended position with the hand, arm, leg, or eye. It is a type of ataxia.
   <u>HYPERMETRIA and HYPOMETRIA</u> refer, respectively, to overshooting and undershooting the intended position

<u>DYSDIADOCHOKINESIA</u>, <u>DYSDIADOCHOKINESIS</u>, (from Greek dys "bad", diadocho "receive", kinesia "movement) – a term for an impaired ability to perform rapid, alternating movements (i.e. diadochokinesia). Complete inability is called adiadochokinesia

 $N/ \vdash D$ 

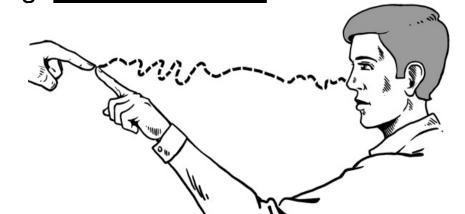
## **TESTS FOR CEREBELLAR FUNCTION**

– <u>RAPID ALTERNATING AND REPETITIVE MOVEMENTS</u> (supination – pronation – symmetry?) (ADIADOCHOKINESIA?)

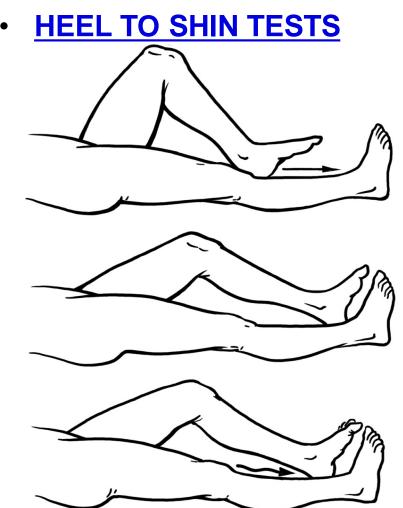
- FINGER TO NOSE / HEEL TO SHIN TESTS (see next slide)
- <u>STEWART-HOLMES TEST:</u> The subjects are asked to perform a strong isometric biceps contraction (UE flexed in elbows) against resistance, performed by examiner. Sudden release of the resistance, the healthy patient is able to stop his hand immediately, the patient with cerebellar dysfunction may not be able to stop his hand fast enought (prevent hitting the face!)
- WALKING, AND RUNNING note abnormal gait (e.g. waddling, wide based, tiptoed).
- NOTE INVOLUNTARY MOVEMENTS (e.g., tremor, dystonia, chorea, athetosis, tics, myoclonus) and conditions under which they are enhanced or suppressed.
   M U N
   M F D

# **EVALUATION OF ATAXIA AND DYSMETRIA**

- UPPER EXTREMITIES
- FINGER TO NOSE TEST
  - Eyes opened
- OR ONLY TOUCH OF THE NOSE BY THE
   INDEX FINGER eyes opened or closed
- Missing of the goal: <u>DYSMETRIA</u>
- Overshooting: <u>HYPERMETRIA</u>

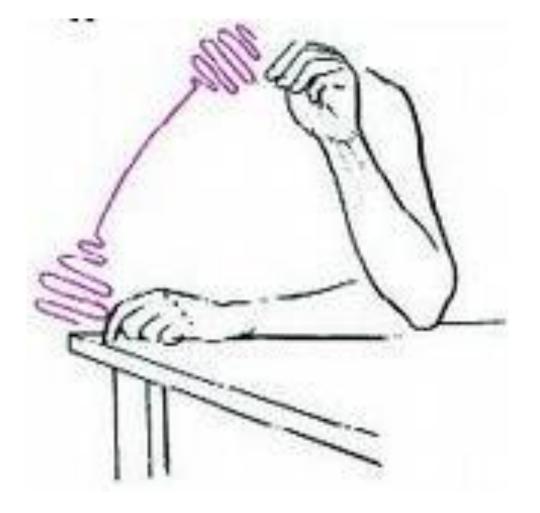


• LOWER EXTREMITIES



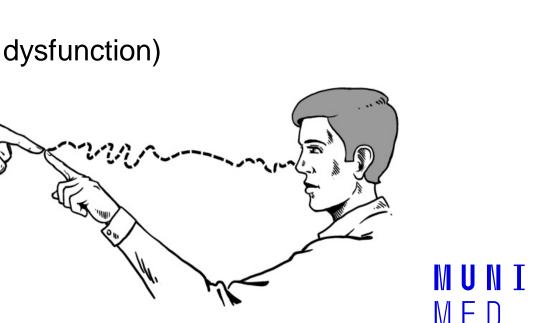
#### **ABNORMAL MOVEMENT: CEREBELLAR TREMOR**

- involuntary movement caused by alternating contractions of opposing muscle groups
- <u>ACTION</u> (only during the movement, minimal or no tremor at rest)
- <u>INTENTIONAL</u>=
  - the amplitude increases before the goal
  - patrly increased also at the beginning of the movement
- <u>Atactic = GROSS, IRREGULAR</u>



# **CEREBELLAR SYNDROMES**

- The result of the dysfunction of the cerebellum or its pathways
   IPSILATERAL!!!
- PALLEOCEREBELLAR SYNDROME (vermis dysfunction)
  - Dyscoordination of the stance and gait (a broad-based gait, "drunken sailor")
  - The ataxia of whole the body including axial muscles.
- <u>NEOCEREBELLAR SYNDROME</u> (hemispheral dysfunction)
  - The lack of coordination of limb muscles
  - <u>ataxia</u>
  - <u>dysmetria</u>
  - dysdiadochokinesia
  - hypotonia (decreased muscle tone),
- <sup>45</sup> <u>intention tremor</u>





# EXTRAPYRAMIDAL SYSTEM

- <u>EXTRAPYRAMIDAL SYSTEM</u> is a neural network that is part of the motor system that causes involuntary reflexes and movement, and modulation (tuning) of movement (i.e. coordination).
- The disorders of extrapyramidal system usually cause **TWO TYPES OF SYMPTOMS**:
- 1. <u>PARKINSONISM (PARKINSON'S SYNDROME)</u> characterized by tremor, hypokinesia, rigidity, and postural instability
- 2. abnormal involuntary movements, i.e. <u>HYPERKINESIAS OR DYSKINESIAS</u>, terms used interchangeably
  - are usually evident when a patient is at rest,
  - are frequently increased by action or stress,
  - and disappear during sleep (with some exceptions e.g. myoclonus can persist in sleep).

## HYPERKINESIAS

- <u>CHOREA</u> delineates brief, irregular contractions and movements (that, although rapid, are not as lightninglike as myoclonic jerks). The jerks affect individual muscles as random events that seem to flow from one muscle to another. They are not repetitive or rhythmic.
- presumably related to disorders of the caudate nucleus (sometimes not)
- <u>BALLISM</u> is a form of chorea in which the choreic jerks are of large amplitude, producing flinging movements of the affected limbs.
- often related to lesions of the subthalamic nucleus.
- The term <u>MYOCLONUS</u> refers to ultrabrief, shock like movements that may arise from muscle contractions or inhibitions (negative myoclonus)
- TREMORS are rhythmic oscillatory movements. They result from alternating contractions of opposing muscle groups (e.g., parkinsonian tremor at rest) or from simultaneous contractions of agonist and antagonist muscles (e.g., essential tremor). Tremors usually have a fixed frequency, although the rate may appear irregular.

## DYSTONIA

**<u>DYSTONIA</u>** is a syndrome of **sustained muscle contraction** that frequently causes **twisting and repetitive movements or abnormal postures**.

- Dystonic movements may be slow and twisting or quite rapid, resembling the shocklike jerks of myoclonus.
- There may be <u>additional rhythmic movements</u>, especially when the patient attempts to actively resist the involuntary movement.
- If the <u>patient is asked to relax</u> and allow the limb to move as it pleases, <u>the abnormal dystonic</u> posturing usually becomes evident, and the rhythmic dystonic tremor lessens.
- The varied nature of these movements often causes the misdiagnosis of dystonia as some other type of movement disorder.
- The movements typically are <u>aggravated by stress and anxiety</u> and are improved by rest.
- Patients often discover a variety of peculiar maneuvers (<u>sensory tricks</u>) that they can use to lessen or even completely abate the dystonic movements and postures
- Typical examples: cervical dystonia, blepharospasm, task-specific dystonia (graphospasm)

# **GAIT AND BALANCE**

- <u>SPONTANEOUS GAIT</u> should be observed; stance, base, cadence, arm swing,
   tandem gait (straight line), toe + heel walking, walking backward eventually hopping on one foot should be noted
- Postural stability should be assessed by the <u>PULL TEST</u> (examiner abruptly pulls the patient off balance – usually backwards - while being ready to catch the patient in the event of a fall)
- Examination of the stand stability: **<u>ROMBERG TEST</u>**. Stand with eyes open and then close
- -I = normal stand,
- II = stand with both feet together,
- III = + eyes closed,
- IV = + rotation of the head to the right or
- V = to the left

# MUNI MED

# TAKE LET'S COFFEE P BREAK