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صدق الله العظيم

الآيه(32) سوره البقره

SCREENING OF NEUROMOTOR SYSTEM

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INTRODUCTION

- The control of movement includes both **conscious** and **unconscious processing** utilizing a **vast and complex array of neurological structures.**
- review the **neuromotor concepts of human movement** that *underlie the motor examination in a patient with neurological pathology*, focusing on motor manifestations of the nervous system, and to describe specific neuromotor examination tests/ measures organized according to the Guide to Physical Therapist Practice, including **range of motion**, **muscle performance**, and **motor function** (APTA, 2015).
- Normal functions of **the neurological** and **musculoskeletal systems** ***enable voluntary controlled movement***. Impairments of these neuromotor functions frequently contribute to limitations in functional activity.

WHERE IS IT?

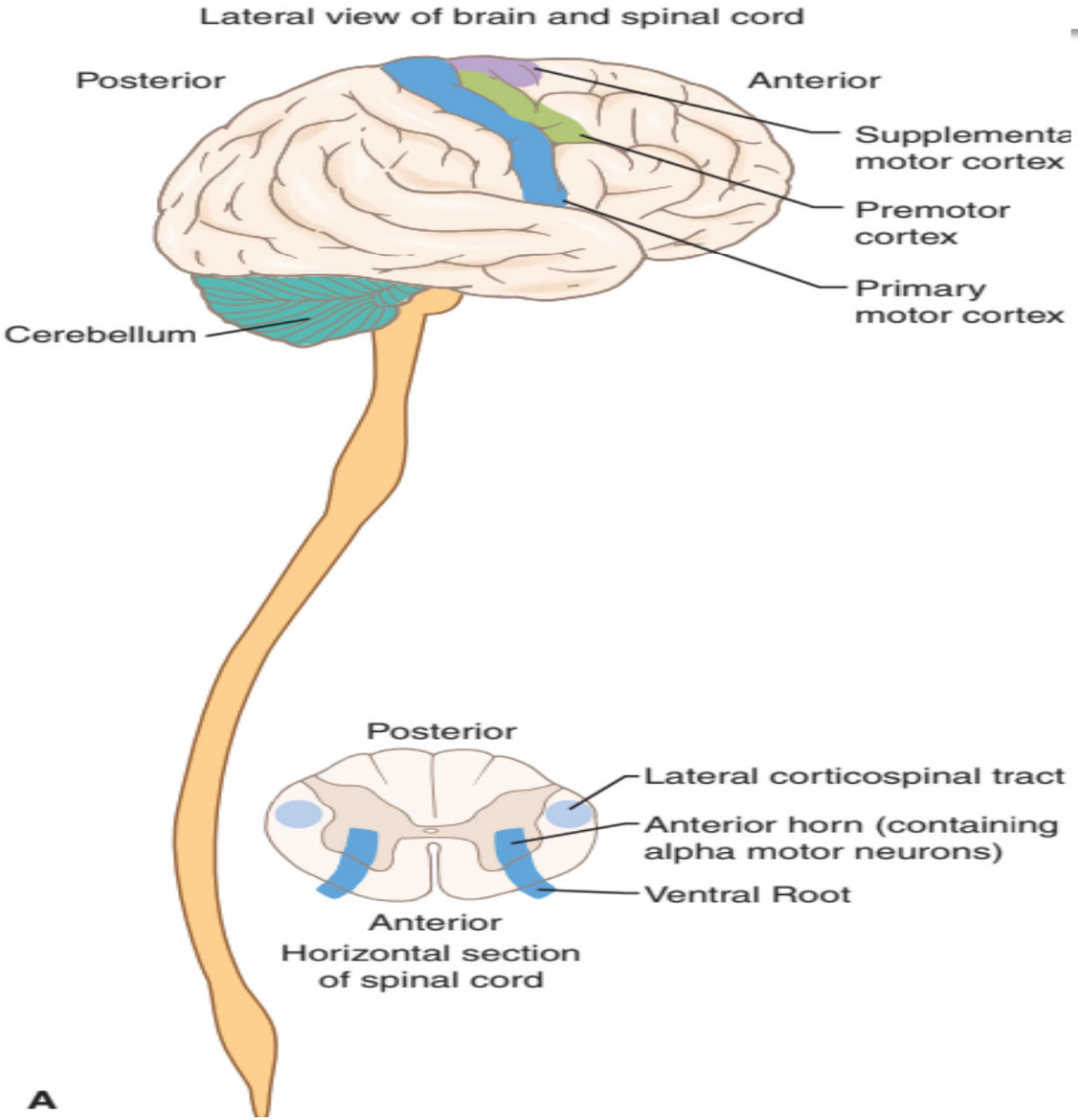


FIGURE 6-1 **A.** Motor areas of the CNS showing the primary **motor cortex** in the precentral gyrus, **premotor cortex** in the superior frontal lobe just anterior to the precentral gyrus, **supplementary motor cortex** medial and anterior to the premotor area including part of the medial hemisphere surface, and the cerebellar cortex.

NEUROMOTOR SYSTEM FUNCTION:
ANATOMICAL PERSPECTIVES:

Areas of the Motor Cortex: primary motor cortex(precentral gyrus),

The motor cortex, with **a key role in voluntary movement,** includes three portions: the primary, premotor, and supplemental areas.

- **primary motor cortex(precentral gyrus), located in the most posterior gyrus of the frontal lobe**

✓ houses a representation of the different muscle groups of the body organized regionally **(Figure 6-1A: Where Is It?).**

✓ **The specific organization of the primary motor cortex is known as the motor homunculus**

✓ illustrates the idea that body regions over which the brain exerts more motor control, including *hands* and *face*, are represented as shown in the right half (motor cortex) of Figure 6-1B by *disproportionately larger areas of cortex*.

✓ Areas with less motor control are disproportionately smaller in the homunculus.

primary motor cortex(precentral gyrus),

A larger representation in the homunculus shape , + a larger number of cortex cells,

allows

More selective activation of motor units because *there is a smaller ratio of muscle fibers to motor neurons*,

results in

specific discrete movements + better fine-motor control of the muscles in a region.

primary motor cortex(precentral gyrus),

Conversely,

areas with less representation within the motor cortex,

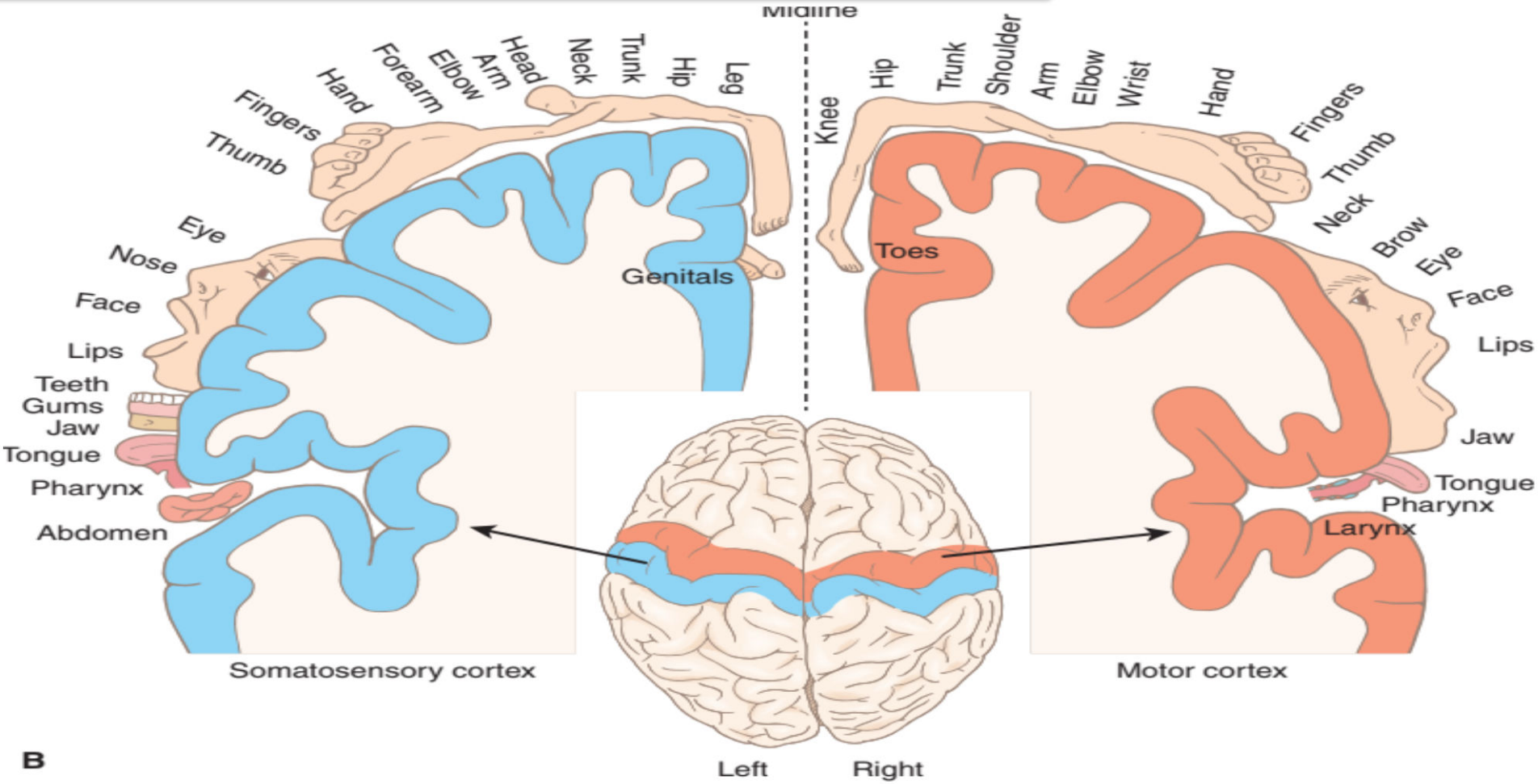
↓

and therefore **a higher ratio of muscle fibers to motor neurons,**

↓

have relatively **less precise motor control** (Figure 6-1B).

The primary motor cortex also controls absolute force & movement velocity (Evarts, 1968).



- FIGURE 6-1—cont'd B. **The motor homunculus** of the precentral gyrus illustrates the fact that parts of the body with the greatest motor *control have a greater proportion of motor cortex controlling movement of that region.*

premotor cortex

- ✓ lies adjacent and anterior to the superior portion of the primary motor cortex on the superolateral frontal lobe (see Figure 6-1A).
- ✓ **function:** provides *activation of muscles that are either directly involved* or *support a specific activity such as limb positioning of the humerus & forearm of UL* that allows the hands to perform more discrete functions including object manipulation.

The premotor cortex relies on input from other areas such as vision centers; premotor activity has been shown to increase during tasks guided by vision (Mushiake, 1991).

The premotor area may either send signals to the primary motor cortex, the basal ganglia, or other extrapyramidal structures such as reticulospinal neurons and thus **serves as the primary cortical influence upon these structures** (Campbell, 2005).

The premotor area most commonly activates the primary motor cortex indirectly by activating the **basal ganglia** to in turn activate the **thalamus**, which finally activates the **primary motor cortex**.

supplementary motor area

- ✓ the most anterior motor cortex area, lying medial and anterior to the premotor area (Figure 6-1A).
- ✓ Blood flow to the supplementary area does not increase during simple finger movements but does increase during mental rehearsal and actual performance of complex finger movements (Roland, 1980). This suggests that the **supplementary area participates in the assembly of a motor scheme or program, particularly bilateral muscle actions.**
- ✓ In fact, both the supplementary and premotor areas become active during **planning of movement (**Bear, 2007).
- ✓ **also assists the premotor area in attaining trunk and extremity positions** while the primary cortex area enacts more specific discrete movements during functional activities.

Extrapyramidal System/Subcortical Motor Nuclei

- **The extrapyramidal system of motor control** is *technically all (CNS) motor structures external to the pyramidal system, with the exception of the cerebellum*, and includes pallidal, thalamopallidal, basal ganglion, and striatal levels (Campbell, 2005) (see Figure 6-1C).

- Extrapyramidal motor control is **involuntary** and considered by some to contain older phylogenic structures (Campbell, 2005).

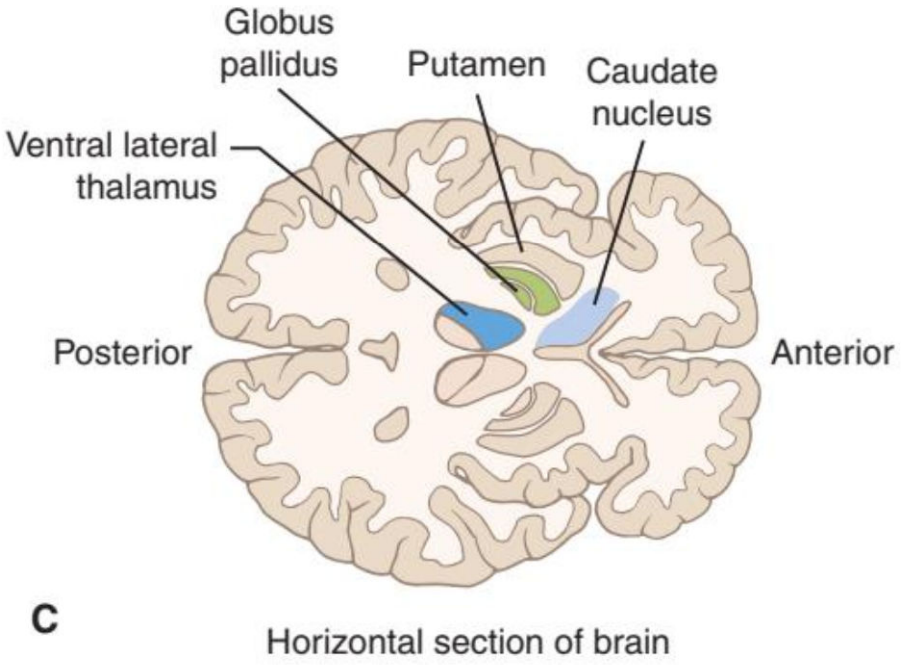


FIGURE 6-1—C. Deep CNS structures with primary motor function, which are not part of the pyramidal or corticospinal direct pathway, including parts of **the basal ganglia (BG)** (caudate and putamen nuclei *as primary BG input* and globus pallidus *as primary BG output*) and **the ventral lateral thalamus.** Damage to any of these structures will likely result in significant motor impairment.

basal ganglia + **dorsal thalamus** are the ***major subcortical input areas to the premotor & supplemental motor areas***.

- Other subcortical areas that influence motor function include the **substania nigra** (considered part of the basal ganglia), red nucleus, **vestibular nuclei**, and the **reticular nuclei**.
- The **basal ganglia** (Figure 6-1C) contain the specific of the **caudate**, **putamen**, **subthalamic nucleus**, and the **globus pallidus**.

At rest, the globus pallidus tonically inhibits VL nucleus of the thalamus (Fig. 6-2A).

When the motor cortex is active (Fig. 6-2B),

excitatory input

the striatum (caudate & putamen)

inhibits the globus pallidus,

disallowing inhibition of the VL nucleus of the thalamus (Figure 6-1C)

excitatory input

supplemental motor cortex,

- ✓ *completing a complex positive feedback loop* that not only assists motor function but also
- ✓ sends information to areas of the brain that involve *memory* & cognitive function

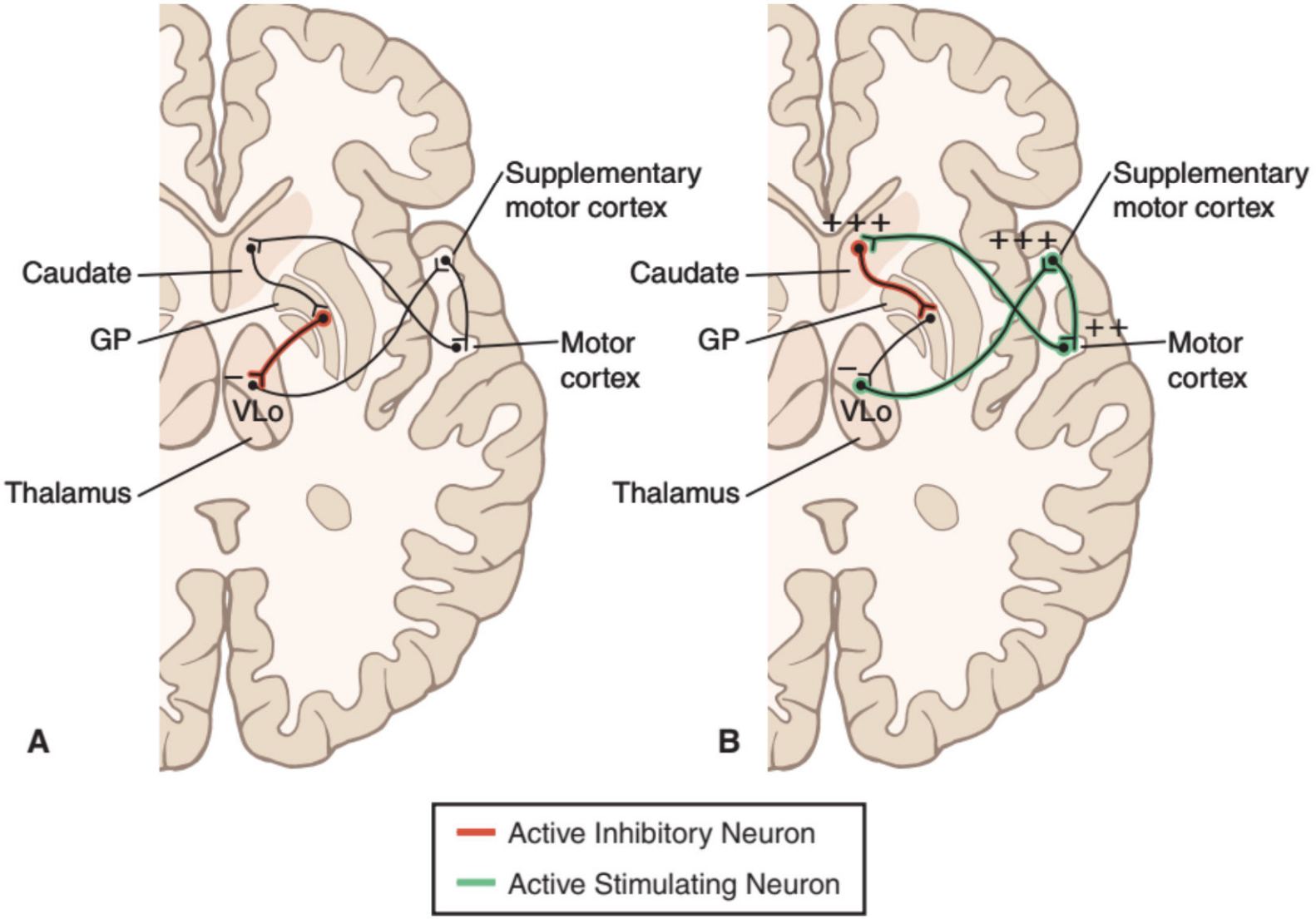


FIGURE 6-2 The basal ganglia circuits described by Bear (2007) applied to an anatomical illustration. **A.** With the motor cortex at rest, and not stimulating the basal ganglia, the globus pallidus sends tonic inhibition to VL thalamus to ***decrease motor output of the basal ganglia***.
B. When the motor cortex sends stimulation to the basal ganglia, the inhibition from the striatum (caudate or globus pallidus) decreases the globus pallidus inhibition of VL thalamus, allowing an *increase in stimulation of the thalamus to:* the supplementary motor cortex and then primary motor cortex.

The basal ganglia function to

produce crude subconscious movements

1) that *operate in the background*

and

2) *refine more discrete skilled voluntary & automatic movements*.

The substantia nigra

- located within the cerebral peduncles of the midbrain.
- Intricately and reciprocally connected with the basal ganglia,
- **functions:**

✓ **control subconscious movements**.

✓It is also part of the **Dopaminergic modulatory system** and is partially responsible for the **initiation of movement in response to the environment**

(Bear, 2007).

The red nucleus,

- *a collection of cell bodies in the midbrain*, and deep to the substantia nigra, is a *subcortical area that operates similarly to the corticospinal tract*.
- **function:**
  - ✓ **provide an accessory route & modulation of motor input to the spinal cord.**

- Direct connections exist from the **primary motor cortex to the red nucleus via the corticorubral tract.**
- **The red nucleus sends signals to the spinal cord via the rubrospinal tract**, which crosses to the contralateral side and runs a course parallel to the corticospinal tract down the lateral columns of the spinal cord.
- Connections also exist between the red nucleus and the cerebellum.

The vestibular and reticular nuclei

- **Reticular nuclei** are named for their primary location in the brainstem and are divided into two major groups: **pontine** & **medullary**.

I- Pontine nuclei are *highly excitable* and *receive excitatory signals* from the cerebellum, vestibular nuclei, and other areas of the brainstem.

II- medullary reticular nuclei receive input from motor pathways, primarily the corticospinal and rubrospinal pathways

vestibular & pontine reticular nuclei
activate + maintain
tone of antigravity musculature.

medullary reticular nuclei are
antagonistic to the pontine reticular nuclei
inhibit the antigravity muscles when certain movements of antigravity limbs are desired.

The Cerebellum

mainly **refines movement by processing a number of unconscious senses** related to *muscle length* and *joint* and *body segment positions*.

- **receives afferent information from**

✓vestibular system,

✓brainstem,

✓spinalcord.

- **sends information to**

✓reticular formation,

✓vestibular nuclei & tracts,

important structure for balance and coordination.

The Cerebellum

support of equilibrium reactions + regulation of muscle tone & posture

cerebellum processes unconscious proprioception by coordinating afferent signals, potentiating them by

- **--comparison of actual movement with planned movement,**

then

--passing signals on to other structures.

refining muscle activation

smooth and synchronous voluntary movements

coordination between opposing muscle groups (one shortens while the opposite lengthens).

Overall, the cerebellum serves to receive, modulate, and integrate the information it receives.

DESCENDING PATHWAYS

Signals arising from the motor cortex travel to the spinal cord

direct

lateral corticospinal (pyramidal tract)
carries signals directly
from the cortex
to alpha motor neurons of the spinal cord

indirect neuronal tracts

- bidirectional connections with the extrapyramidal motor system, including:

basal ganglia,
cerebellum,
brainstem, and
other nuclei from the brainstem

DESCENDING PATHWAYS

lateral corticospinal (pyramidal tract)

- ✓ **80% to 90% of them decussate to the opposite side at the inferior medulla** (as the pyramidal decussation) to become the lateral corticospinal tract, which descends in the *posterior aspect of the lateral funiculus near the posterior horn .*
- ✓ **The fibers that remain ipsilateral travel in the ventral** (anterior) corticospinal tract primarily arising from the supplemental motor area and affecting postural movements.

Some of the largest & fastest conducting nerve fibers in the entire nervous system are found in the corticospinal tract.

One corticospinal fiber *innervates more than one alpha motor neuron in the spinal cord.*

This relationship, known as signal **divergence**, allows *amplification of the corticospinal tract's activity* (Campbell, 2005).

❑ **Divergence:**

✓ increases the ratio of alpha motor neurons to excitatory descending pathway fibers

✓ particularly prevalent in large muscle groups that affect posture and perform gross movements.

❑ **Divergence also occurs at the neuromuscular level**

- concerning the ratio of alpha motor neurons to skeletal muscle fibers.
- Example: *soleus of the lower leg compared with muscles of the face or hands*.

rubrospinal tract

- **Terminal connections of the rubrospinal tract connect with the gray matter of the spinal cord:**
- **Along with fibers of the corticospinal tract**
- or
- **directly connect with alpha motor neurons.**

Together, the **rubrospinal** + **corticospinal tracts** are considered the ***lateral motor system of the cord.***

The ventromedial pathways

- are involved in **postural control and are controlled by the brainstem.**
- Four descending tracts:

vestibulospinal +	keep the head postured during dynamic activities.
tectospinal tract	keep the head postured during dynamic activities.
Reticulospinal tracts,	**Dampen** antigravity muscle reflexes of the spinal cord depending on particular needs during motor activities.
, medullary and pontine	**Facilitate** antigravity muscle reflexes of the spinal cord depending on particular needs during motor activities.

CLINICAL MANIFESTATION OF CNS MOTOR PATHOLOGIES

Negative neurological signs	Positive neurological signs
weakness	spasticity
hypotonia	rigidity
sensory loss	increased deep tendon reflexes (DTRs)
Visual loss	hyperesthesia (pins and needles)
vestibular hypofunction	dystonia
memory loss/confusion	tremor

BOX 6-1 Examples of Pathological Disorders Affecting Motor Areas of the Central Nervous System and Related Distribution of Motor Effects

CVA-Anterior Cerebral Artery: Contralateral motor symptoms **primarily in lower extremity.**

CVA-Middle Cerebral Artery: Contralateral motor symptoms **primarily in face, upper extremity and trunk; lower extremity may be involved** if **infarction of deep subcortical white matter** (corona radiata, posterior limb of internal capsule) takes place.

CVA-Hemorrhage: Contralateral motor symptoms depending on location of hemorrhage.

Traumatic Brain Injury: Variable motor symptom distribution depending on lesion location & extent; **could be bilateral, but not necessarily symmetrical.**

Brain Tumors: Contralateral motor symptoms specific to CNS area affected.

Dementia: Decreased movement and activity level.

Cerebral Palsy: Motor symptom distribution most often **hemiplegia, diplegia,** or **quadriplegia.**

CLINICAL MANIFESTATION OF CNS MOTOR PATHOLOGIES

- *Contralateral motor symptoms* result from **motor cortex disorders** because of decussation of motor pathways.
- The paralysis of cerebrovascular accident **(CVA) primarily affects distal muscles and joints compared with proximal joints,** which *adversely influences the ability to produce movements requiring finer volitional control* (Campbell, 2005).
- ✓ Following CVA, spasticity appears after a period of flaccidity and is more pronounced with damage of increasing severity and location

BOX 6-2 Common Contralateral Motor Manifestations of Pyramidal System Damage

- Paresis
- Impaired motor control (lack of isolated movement and impaired timing)
- Possible hypotonia (initially)
- Spastic hypertonia and hyperreflexia (typically develops later)

CLINICAL MANIFESTATION OF CNS MOTOR PATHOLOGIES

premotor cortex + primary cortex lesions

result in

severe spasticity in experimental models.

However, experimental models suggest that **spasticity** associated with damage to the motor cortex may actually arise from **extrapyramidal dysfunction,** including *imbalanced inhibition from the brainstem* (Campbell, 2005).

Supplemental motor area lesions

result in

Movement deficits during activities requiring **bilateral coordinated actions of distal extremities** such as the bimanual tasks (Bear, 2007).

CLINICAL MANIFESTATION OF CNS MOTOR PATHOLOGIES

Lesions in the extrapyramidal areas

- result in signs and symptoms generally referred to as "movement disorders" . (see Box 6-3) and include neurological conditions such as **Parkinson** and **Huntington diseases.**
- The signs of Parkinson disease include bradykinesia, disturbances in muscle tone, loss of "automatic movements," abnormal movements, and difficulty initiating movement (Campbell, 2005). Rigid hypertonicity of muscles, particularly cogwheel rigidity, and hyperkinesias in the form of resting tremors are clinical manifestations of Parkinson disease.

BOX 6-3 Common Motor Manifestations That May Occur Following Extrapyramidal System Damage

- **Dystonia**
- **Rigid hypertonicity** (perhaps **cogwheel**)
- **Hyperkinesias** (**resting tremors**)
- **Hypokinesias** (**bradykinesia**)
- **Impaired motor control** (**initiation of movement**)
- **Involuntary movements**

CLINICAL MANIFESTATION OF CNS MOTOR PATHOLOGIES

Damage to the cerebellum

BOX 6-4 Common Motor Manifestations of Cerebellar Damage

- **Muscle incoordination** (ataxia; see Box 6-5)
- **Hypotonia** (low muscle tone)
- **Asthenia** (general decrease in strength or loss of energy)
- **Diminished postural equilibrium**
- **Nystagmus** (involuntary rhythmic eye movements)
- **Speech disturbance** (ataxic)

BOX 6-5 Terms for Specific Components of Ataxia/Incoordination

- Adiadochokinesia
- Ataxia
- Dysmetria
- Dysdiadochokinesia
- Hyperkinesias
- Speech disturbances
- Hypermetria (past-pointing)

CLINICAL MANIFESTATION OF UPPER & LOWER-MOTOR NEURON LESIONS

Neurons
cells of the nervous system
functionally

upper-motor neurons ← → **Lower-motor neurons**

- **Essentially, all neurons originating in the CNS, *except* *those in the anterior horn of the spinal cord*, *are considered to be* *upper-motor neurons*.**
- ***Lower-motor neurons*** include:
  - ✓ cell bodies of motor neurons in the anterior horn of the spinal cord, or
  - ✓ motor neurons of the brainstem that contribute to cranial nerve motor functions and their axonal projections toward the periphery to innervate muscle.

"final common pathway" : *axons of lower-motor neurons that exit the CNS with fibers* (in cranial nerves & peripheral spinal nerves) that *directly stimulate motor units of muscles*.

CLINICAL MANIFESTATION OF CNS MOTOR PATHOLOGIES

UMNL

UMNL

- **Any pathology that affects upper-motor neurons in the brain and spinal cord.**
- **Clinical manifestations of an UMN lesion include the positive neurological signs:**

1) spastic hypertonia (or spasticity) and

2) hyperreflexia, which occur because of:

✓ *loss of cerebral control over voluntary & reflexive motor activity* and

- because *normal inhibitory influences from higher motor structures are discontinued*.

- *Without inhibition of intact lower-motor centers, aberrant reflexes are unregulated, causing distinctive clinical manifestations.*
- **UMN syndrome also typically includes the negative neurological signs** of

1) paralysis,
2) impaired motor control movement, or
3) muscle weakness.

UMN lesion manifestations are detailed in Table 6-1.

TABLE 6-1 Common Manifestations of Upper-Motor Neuron Lesions
(From damage to corticobulbar, corticospinal, corticorubrospinal, and corticoreticulospinal systems—cell bodies or tracts)

Hemiplegia/Hemiparesis	• **Internal capsule lesions** result in *lower face paralysis* along with *paralysis/weakness of the upper extremity & lower extremity on the contralateral side.*
Spastic hypertonicity	• After days to weeks, spasticity (increased resistance to passive elongation that is velocity-dependent) develops accompanied by **hyperactive DTRs**, possibly due to loss of the normal inhibitory influence of these tracts on alpha-motoneurons. • *Firm resistance to passive movement, especially in UE flexors and LE extensors. The tone will often suddenly yield in the midst of the passive elongation* **(i.e., clasp knife)**.
Hyperreflexia	• Increased (hyperactive) DTRs, especially at wrist and sometimes at ankle.
Clonus	• *Rhythmic oscillation of a joint*, especially the ankle, is initiated by *sudden, passive stretch of the spastic muscle* (i.e., sudden dorsiflexion stretches the plantar flexors). Via the stretch reflex, this results in plantar flexion, which in turn stretches the dorsiflexors, beginning the cycle again.
Babinski sign	• Stroking the lateral plantar aspect of the foot results in hyperextension of the big toe and splaying abduction of the other toes.

LMNL

- **Damage that occurs to the "final common pathway".**
- typically express just the opposite of UMN lesions and prevent activation of the muscles.
- **Partial LMN lesions, with some motor units spared,** *cause the negative neurological signs of* ***weakness*** *and* ***hypotonia*****.**
- But **if all motor neurons innervating a particular muscle are affected,** the *negative neurological sign of* ***complete paralysis*** (i.e., cessation of voluntary muscle activity) *will occur along with* ***flaccidity***.
- LMN lesion manifestations are found in Table 6-2.

TABLE 6-2 Common Manifestations of Lower-Motor Neuron Lesions
(From damage to brainstem motor cranial nerves or anterior horn cells—cell bodies or peripheral nerve fibers)

Paralysis/Paresis	• Loss of motor function.
	• Loss of coordination in muscle action.
	• (Decrease of motor function and decreased coordination if the LMN lesion is only partial)
Atrophy	• Secondary decrease in muscle mass and bulk with evident bony prominences.
	• Loss of automatic responses.
Abnormal palpation	• Soft, flabby muscle; lacks bulk.

Vicarious movements	• Compensatory or trick movements:
	  • Ex: muscle substitution (long head bicep, long head tricep, clavicular pec major, supraspinatus, lat rotators can all abduct the humerus if deltoid is out).
	  • Ex: this phenomenon may actually be helpful and useful to support compensatory actions such as tenodesis effect = flexion of the fingers passively occurs as the wrist is extended, especially in permanent losses such as individuals with quadriplegia.

Hyporeflexia	• Decreased or absent DTR.
Fasciculations	• Muscle twitches could indicate denervation injury.

PRINCIPLES OF NEUROMOTOR EXAMINATION

- **The general sequence of a motor examination should include initial observation of :**

✓ patient's posture, position, movement and intralimb coordination, a detailed history and review of systems, and finally a comprehensive, patient centered, physical examination to document the specific problems of the individual patient.

✓ It is also important to realize factors that may influence and affect the motor examination. Several of these factors include the patient's level of cognition and arousal, visual and sensory function, communication, the ability and willingness to cooperate, their anxiety level, the timing of the examination following injury or disease, and the influence of pharmacological agents (see Table 6-3) (Brown, 1991).

TABLE 6-3 Examples of Pharmacological Agents That May Impact Motor Function and Possible Effects

PHARMACOLOGICAL AGENT OR CLASS	POSSIBLE EFFECT ON MOTOR BODY FUNCTION
Antipsychotics	Extrapyramidal symptoms including tardive dyskinesia, pseudoparkinsonism, akathisia (restlessness), dystonia, and neuroleptic malignant syndrome, and sedation.
Baclofen	Muscle weakness.
Benzodiazepines	Sedation and ataxia (including Diazepam).
Dantrolene sodium	Generalized weakness.
Diruetics	Muscle weakness and/or fatigue.
Lithium	Sedation, lethargy, and muscle weakness.
Levodopa	Long-term use (3 months to 3 years) associated with dyskinesias such as ***ballismus,*** dystonia, myoclonus, tics, tremors, and choreoathetoid movements.
Tricyclics	Sedation, lethargy, and muscle weakness (Including Amoxapine = Ascendin: possible ataxia or tardive dyskinesia).

Adapted from Ciccone CD. *Pharmacology in Rehabilitation.* 4th edition. Philadelphia, PA: F.A. Davis Co.; 2007.

MUSCLE TONE

Muscle tone is the *amount of inherent neuromuscular activity present even in a resting muscle* and is

- **detected by** the *response, specifically the amount of resistance, to passive elongation or stretch of the muscle being tested* (Masi, 2008).
- Damage to several neuroanatomic regions can result in abnormal expressions of muscle tone.

✓ **corticospinal (pyramidal) system in the cerebrum or the brainstem or the motor pathways of the spinal cord** can result in **spasticity**.

✓ **extrapyramidal elements**, including basal ganglia often results in **rigidity** or **dystonia**.

For example, **damage to the substantia nigra**, either traumatic from traumatic brain injury or degenerative as in Parkinson disease, can result in rigidity.

✓ **Damage to the vestibular centers, the cerebellum, anterior horn cells, peripheral nerves, neuromuscular junction, or the muscle itself** may result in **decreased muscle tone**.

TABLE 6-7 Description of Variations in Muscle Tone

TONE STATE:	FLACCID	HYPOTONIA	NORMAL	SPASTIC HYPERTONIA	RIGID HYPERTONIA
Description of muscle resistance to passive elongation:	Complete absence of resistance to elongation	Decreased resistance to elongation	Mild and appropriate resistance to elongation	Increased resistance to elongation that increases with faster stretch **(velocity-dependent)**, usually more predominant in muscle on one side of affected joints in **stroke/TBI** (or in both agonists and antagonists in affected areas following **SCI**), and more obvious toward the end of the range when the muscle is on maximal stretch	Increased resistance to elongation that does not increase with faster stretch (present even at slow speeds); often present in muscles on both sides of the joint and present throughout the range **(though it may show a "cog-wheel" pattern)**
Reflexes (DTRs)	Absent	Diminished (hyporeflexia)	Normal	Increased (hyperreflexia) particularly with spasticity	May be increased, but often dampened by activity of opposing muscle group
Common Medical Conditions	• Acute CVA • Spinal shock in SCI • Polio (complete) • Some spinal muscular atrophy • Peripheral nerve injury • Guilllain Barré	• Some genetic disorders (Down syndrome, Angelman syndrome) • Polio (partial) • Cerebellar disorders		• Stroke (CVA) • TBI • SCI • Multiple sclerosis	• Parkinson disease • Basal ganglia trauma or stroke

- **Hypertonia (hypertonicity): a positive neurological sign,** is *a muscle state with increased resistance in a muscle during passive elongation of that target muscle.*

hypotonia (hypotonicity), a negative neurological sign, is *a muscle tone state in which resistance is less than normal during passive elongation of a muscle*. (Campbell, 2005).

factors can influence the clinical presentation of muscle tone, including:

- ✓ the temperature of the muscle,
- ✓ the emotional & behavioral state of the patient,
- ✓ the speed of passive movement (faster speeds may more likely elicit activity of stretch reflexes),
- ✓ cooperation of the patient ***(most important to control)***. *(The patient must be able to voluntarily relax the*
- ✓ *limb being tested to allow optimal muscle tone testing.)*

COMMON CLINICAL PRESENTATIONS ASSOCIATED WITH SPASTIC MUSCLE TONE AS PART OF THE UPPER-MOTOR NEURON SYNDROME INCLUDE

- **clasp-knife resistance,**

(the passive stretch results in initial resistance followed by sudden release as seen when opening a spring loaded pocketknife.فتح مطواه)

- **scissoring gait,**

(a motor control problem observed in patients with hip adductor spasticity associated with *excessive abnormal synergy adduction of the leg in swing phase*.)

- **HYPERREFLEXIA**
- **clonus**, and
- **Babinski and Chaddock signs** (Campbell, 2005).

COMMON CLINICAL PRESENTATIONS ASSOCIATED WITH SPASTIC MUSCLE TONE AS PART OF THE UPPER-MOTOR NEURON SYNDROME INCLUDE

✓ Clonus

def/ A rhythmic involuntary contraction & relaxation alternating between agonist and antagonist muscle groups that occurs in response to sudden passive movement of the ankle (most common), wrist, or patella (Campbell, 2005).

Test for clonus by rapidly & forcefully pushing the joint up into end-range + maintaining pressure at end-range.

- **Observe** for resulting reflexive movement, counting the "beats" or oscillations of the movement.
- **Document** clonus by noting the number of "beats."

(If the clonus does not subside with maintained pressure at end-range, documenting "sustained clonus" may be appropriate. In spasticity, DTRs as previously described will be hyperflexive.)

- **Babinski sign**

observed by great toe extension + toe splaying (Figure 6-7) ***as a result of*** noxious stroking of the ventral aspect of the foot (starting at the base of the fifth metatarsal, sliding up the foot to the metatarsal head and then medially across to the first metatarsal head) (Campbell, 2005).

- The Babinski sign has a sensitivity of only 35% but specificity of 77% for *predicting UMN weakness* (Miller, 2005).
- Mass toe flexion or withdrawal of the foot and leg is considered *a normal response to Babinski testing*.

Chaddock signs

similar to the Babinski sign ***but*** is great toe extension with or without splaying of toes that ***occurs in response to*** noxious stimulation of the dorsolateral aspect of the foot inferior to the malleolus (Figure 6-8) as opposed to the ventral surface (Campbell, 2005).

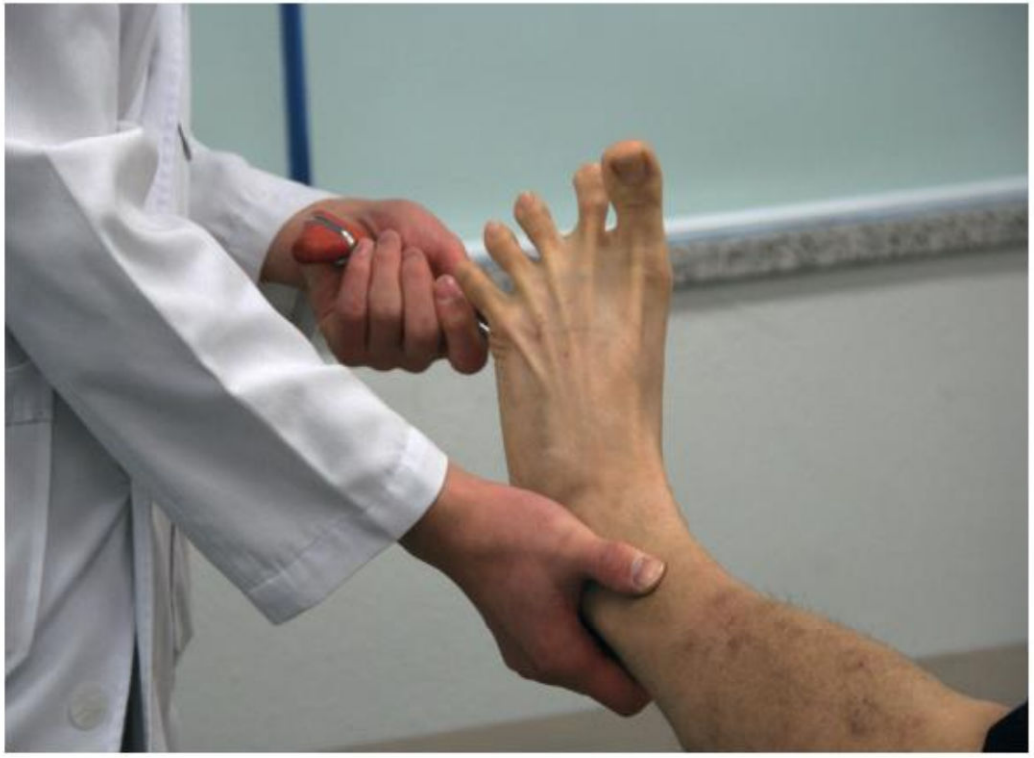


FIGURE 6-7 Babinski sign with great toe extension and *splaying of other toes* in response to stroking the plantar surface of the lateral foot (along the length of the 5th metatarsal) and then along the metatarsal heads.

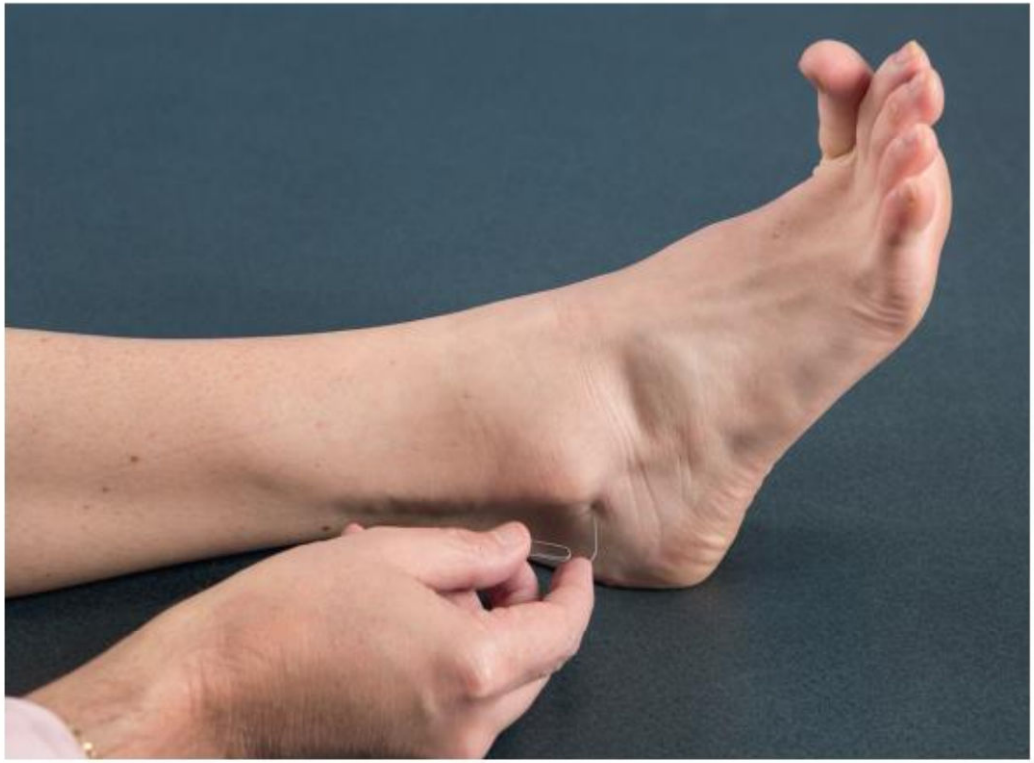


FIGURE 6-8 Chaddock sign with *toe extension*, with or without *splaying of the toes*, in response to an inframalleolar noxious stimulus.

TONE ASSESSMENT

- ***easier and more typically tested in*** *the extremities than the trunk*, although abnormal tone is just as likely in proximal musculature following certain injuries or disease.
- ***Muscle tone examination is often difficult***, and interrater reliability has been called into question because of relative subjectivity in traditional methods of tone assessment (Campbell, 2005).
- ***The traditional method*** for detecting abnormal muscle tone is **carrying a muscle through passive elongation while the therapist "feels" or assesses how much resistance the muscle generates opposing the passive elongation.**

❖ By comparing the amount of resistance to that felt when moving the limb of an individual without impairment, the therapist can determine generally whether the tone is decreased, normal, or increased.

TONE ASSESSMENT

- In formulating an intervention plan, the therapist will have to

✓ ***determine the type of abnormal tone present*** (i.e., flaccidity, spasticity, rigidity, etc.) and

✓ consider whether or not the ***abnormal muscle tone is truly limiting the individual's function and activity or not.***

✓ documentation to ***specify the specific type of abnormal tone instead of making a generic and indeterminate statement*** such as **"Billy has tone in his right biceps."**

✓ ***Finally compare resistance in agonist versus antagonist at each joint and compare the two sides***, especially when unilateral symptoms are expected.

This comparison between limbs and joints could help provide insight in the patient's baseline level of tone before the neurological event.

PROCEDURES FOR MEASURING MUSCLE TONE

- **first begin to observe their body segment posture and movement.**
- **Observe whether the muscles are bulked or atrophied a**nd whether there appears to be any tension in the muscle at rest or during action.

For example, - *a limb hanging limply in a gravity-dependent position may have flaccidity or severe hypotonia.*

- A limb held in an unusual antigravity posture might, among other impairments, be related to hypertonia that you could definitively test.

- **By definition, muscle tone is tested by passively elongating the target muscle,**

✓ at both **slow** & **fast** velocities,

✓ it is important to **position the patient for optimal stability** + to **hold the patient's limb using bony prominences** as much as possible to minimize contact with the potentially abnormal muscle.

PROCEDURES FOR MEASURING MUSCLE TONE

- **For example, as you test muscle tone in the elbow flexors and extensors,**

your proximal hand could firmly stabilize at the elbow with contact over the medial and lateral epicondyles while your distal hand uses contact over the styloid processes to provide the motion at the elbow joint.

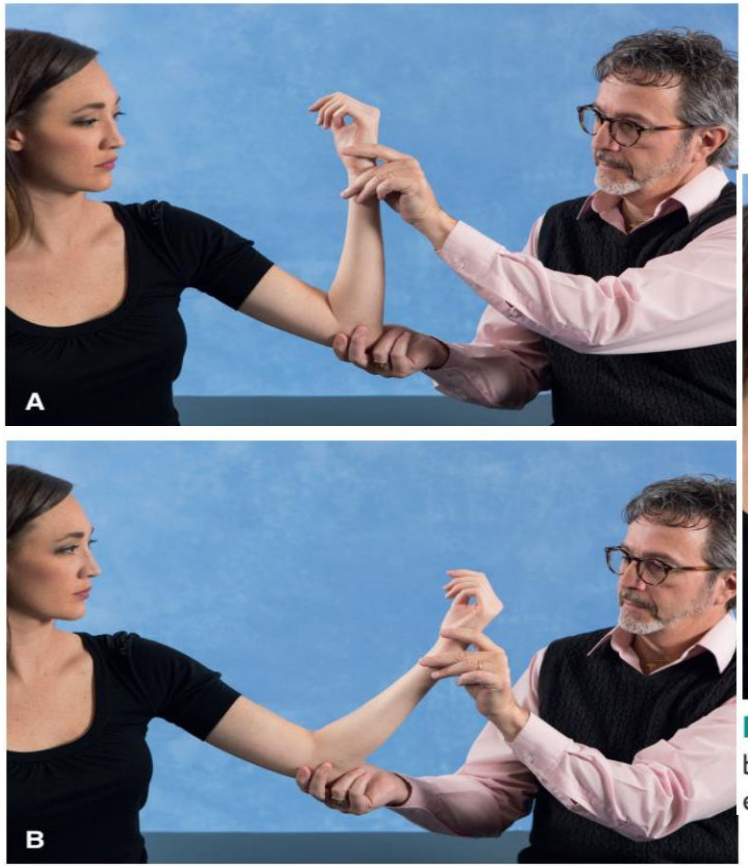


FIGURE 6-9 **A., B.,** and **C.** Testing muscle tone of the biceps by passive elongation of the biceps, moving the arm into elbow extension.

PROCEDURES FOR MEASURING MUSCLE TONE

For tone testing at the wrist joint,

you could contact over the styloid processes to stabilize at the wrist

while contact at the lateral 2nd metacarpal head and the medial 5th metacarpal head will allow you to control movement at the wrist joint.

In assessing muscle tone, it is important to determine muscle resistance to passive, not active, movement or elongation **while the patient is relaxed** (Campbell, 2005).

- **You should also move the extremity through a full ROM and at a variety of speeds** (as will be emphasized in the following section for testing spasticity).
- As you perform the passive elongation, **use your proprioceptive awareness to determine the "feel" or resistance of the muscle throughout the movement**.

PROCEDURES FOR MEASURING MUSCLE TONE

- For spasticity, **note particularly where, in the range, the resistance is first felt** as this is important for grading spasticity in commonly used tests and measures.
- When testing muscle tone in individuals with cerebral disorders (e.g., CVA, traumatic brain injury [TBI], cerebral palsy [CP]), **it is important to observe for and recognize the influence of tonic reflexes that may have reoccurred or are persistent related to the pathology.**

✓ **Keep the head in midline, not rotated,** to *avoid the influence of a recurrent asymmetrical tonic neck reflex (ATNR), which would cause increased tone in elbow extensors and knee extensors of the side toward which the neck is rotated and increased elbow flexor tone in the opposite arm.*

✓ **Test muscle tone in sidelying or in sitting, rather than in prone or supine,** to *avoid the influence of a recurrent tonic labyrinthine response, which would cause increased flexor tone while the individual is in prone and increased extensor tone while the individual is in supine.*

PROCEDURES FOR MEASURING MUSCLE TONE

tests assess general muscle tone detected in passive elongation testing,

- **Arm-Dropping Test**

is **sitting** with the arms adducted and resting comfortably. With the patient in the sitting position, quickly elevate the patient's arms to approximately 90 degrees of shoulder flexion or abduction, then immediately drop the arms of the patient.

- Hypertonicity causes relative *decreased rate of return to the starting position*
- whereas hypotonicity causes an *increased rate of return to the resting position* compared with the uninvolved extremity.

(Campbell, 2005)

- ✓ it is advisable to assess **bilateral upper extremities simultaneously**.
- ✓ **Supine is an alternate test position** for patients that cannot maintain sitting during the Arm-Dropping Test; the set-up movement is passive horizontal adduction instead of abduction.

PROCEDURES FOR MEASURING MUSCLE TONE

❑ **Pronator Positioning Test**

elevate the arms of the patient as high as possible into shoulder **flexion,** preferably to an overhead position. **External rotation** of the humerus normally accompanies overhead elevation, and **supination** of the forearm should accompany external rotation of the humerus when the elbow is **extended (Neumann, 2009).** However, patients with **hypertonicity** display *excessive forearm pronation in the elevated humeral positions when the elbow is extended* (Figure 6-10).

✓ **The classic clinical presentation or positive sign** for the Pronator Positioning Test is that the *patient's palms face outward in the elevated humeral position with the elbows extended* (Campbell, 2005).

FIGURE 6-10 Pronator-positioning test with the patient's forearm in excessive pronation while the patient tries to maintain humeral elevation and elbow extension.

PROCEDURES FOR MEASURING MUSCLE TONE

Pronator Drift Test:

begins with the patient's **eyes closed in a standing position** with the forearms in **supination** and the **elbows, wrists, and fingers in extension.**

- ✓ Patients with mild increased muscle tone **"drift"** into slight forearm pronation and elbow, wrist, and finger flexion.
- ✓ This test is sensitive to discern mild increases in muscle tone that may not otherwise be discernable (Campbell, 2005).

PROCEDURES FOR MEASURING MUSCLE TONE

Lower Extremity Pendulum Tests

require the patient to sit on a plinth high enough to allow the lower extremities to dangle without touching the floor. Passively move the patient's relaxed lower extremity from a position of knee flexion into knee extension then suddenly release (Figure 6-11).

- ✓Normally, the lower extremities should swing as a pendulum several times. Rigidity or spasticity causes the LL to swing with diminished movement or not at all (Campbell, 2005).
- ✓This test may also be performed as a general tonal assessment for hypertonicity or hypotonicity resulting in increased and decreased swing, respectively, of the lower extremity.

FIGURE 6-11 **A., B., C.,** and **D.** Lower Extremity Pendulum Test for muscle tone in the knee extensors. **A.** With the patient seated and extra room under the table for leg swing, the therapist releases the leg from a position of full knee extension. **B.** The leg passively swings down into knee flexion. **C.** Then the leg swings forward toward knee extension. **D.** The leg swings back into partial knee flexion and then alternates between extension and flexion movements with dampening of each cycle.

PROCEDURES FOR MEASURING MUSCLE TONE

- **DTR testing**

DEF/ striking a muscle tendon to cause a sudden stretch elongation of the muscle, allowing observation for an appropriate reflexive contraction of the same muscle.

- ✓ ***As part of DTR testing,*** it is important to assure that the muscle being tested is either on **stretch** or has **a partial contraction** before striking the tendon.
- ✓ It is essential that *the patient isn't actively contracting the opposing muscle group during test*.
- ✓ Because of the variability of tone and reflex sensitivity even among individuals without neurological impairment, it is most important to check for symmetry in the tested locations, especially in unilateral disorders.
- ✓ With some **smaller tendons**, such as the biceps brachialis, *you can place your thumb over the tendon then strike your thumb* (Figure 6-12A) to provide opportunities for you to feel subtle biceps contractions that might not be strong enough to elicit elbow movement.
- ✓ If a person has absent or minimal response to the Patellar DTR test, **the Jendrassik maneuver** (Figure 6-13) may be used to heighten reflex reaction by increasing upper extremity tension by clasping hands and pulling them against each other (Nardone, 2008).

FIGURE 6-12 **A.** Measuring DTRs can sometimes be accomplished by placing your thumb over a discrete smaller tendon, then striking your thumb with the reflex hammer as illustrated in this photograph of elbow DTR testing of biceps. **B.** The patellar (quadriceps) muscle is also a common location for testing DTRs.

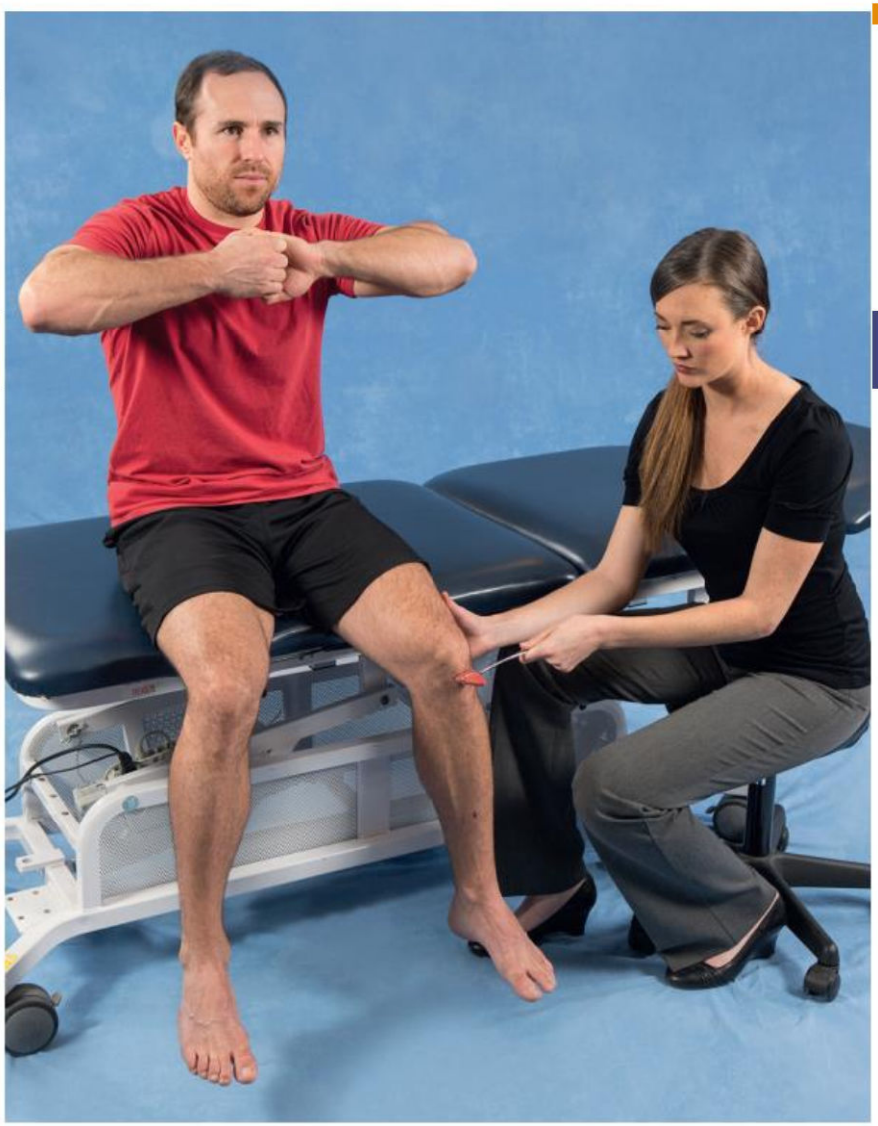


FIGURE 6-13 The Jendrassik maneuver, with aggressive clasping of the hands with self-imposed resistance, can sometimes help to heighten a DTR that is decreased and difficult to observe.

TABLE 6-8 Deep Tendon Reflex Assessment

Biceps Tendon (tendon lies in midline of anterior surface of arm superior to the cubital fossa)	With the patient sitting, support the patient's forearm with your forearm and position patient's elbow in less than 90-degree flexion. Palpate tendon with thumb and press thumb on tendon to add extra stretch. Then tap over your thumb and feel and observe for reaction. The reaction may vary from slight tension in tendon felt by thumb to visible movement of muscle belly or obvious elbow flexion movement. If there is difficulty in eliciting the DTR, the reaction can be heightened by ***increasing tension in lower extremities during test*** (e.g., crossing ankles with self-resistance between the lower extremities; ***modified lower extremity Jendrassik maneuver***).
Triceps Tendon	With the patient sitting, support the humerus into abduction with internal rotation allowing the elbow to passively flex. The triceps tendon should be accessible above the tip of the olecranon; therefore, tap the tendon directly with the reflex hammer. Observe the reaction that may include no response, slight tricep muscle belly contraction, or visible elbow extension movement.
Patellar Tendon (tendon lies inferior to inferior border of the patella and superior to tibial tuberosity)	With the patient sitting with knee at 90 degrees, without foot contact to floor, palpate the tendon. Tap the tendon directly with a reflex hammer and observe for reaction that may vary from no reaction, to a slight quadriceps muscle belly contraction, to gross knee extension movement.
Achilles Tendon (tendon lies inferior to gastrocnemius/ soleus muscles and superior to calcaneous	With patient sitting, passively dorsiflex the patient's ankle slightly, palpate tendon in midline of posterior surface of the ankle, and tap it with the reflex hammer and observe for reaction. Reaction may vary from no response, to a slight contraction of the plantar flexor muscles, to gross plantar flexion movement of the ankle.

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

Flaccidity

- It is *a complete lack of muscle tone typically associated with complete LMN lesions when the lesion disrupts activation of the motor units of the particular muscle.*
- **LMN lesions** can affect either *cell bodies located in AHCs* or *peripheral motor fibers of PN*.
- Flaccidity may also occur **temporarily** early after UMN lesions of the cerebrum and brainstem such as **cerebral shock** following **CVA** with the flaccidity of the affected regions lasting for days to weeks (Campbell, 2005).
- Temporary flaccidity of affected muscle groups following **traumatic SCI** is called **spinal shock** and typically lasts for 24 hours, though it may last for up to 4 to 6 weeks.
- *An absence of reflex activity*, **areflexia**, is an expression of flaccidity. Flaccidity is associated with an absence of voluntary muscle activity, complete paralysis with no possibility of activation, as the affected motor neurons are unable to propagate an effective signal to the muscle or muscle group.

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

Flaccidity (Clinically)

Hypotonia is the state in which *resistance to passive elongation of the muscle is decreased below normal levels*; in other words, it is easier than usual to move a joint through PROM.

- **Hypotonia is most often found in:**

✓ a variety of genetic disorders (e.g., Down syndrome and Angelman syndrome),

✓ motor units to a muscle are damaged (e.g., polio),

✓ neuromotor junction dysfunction (e.g., myasthenia gravis), and

✓ muscle disorders (e.g., muscular dystrophy).

✓ disorders affecting the cerebellum when it is typically also accompanied by ataxia,

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

hypertonicity

two common forms of hypertonicity or increased muscle tone: spastic and rigid.

Spasticity (spastic hypertonia)

associated with a UMN injury (damage to the pyramidal or extrapyramidal tracts).

a key characteristic of the increased resistance to passive elongation is that the increased muscle tone is **velocity dependent**.

- ✓ Faster movements increase the resistance to stretch via an uninhibited or increased monosynaptic stretch reflex.
- ✓ In the milder grades of spasticity, you may not detect any increased muscle tone when moving the joint very slowly but notice an obvious increase when you move the limb more quickly.

The increased excitability of the stretch reflex occurs from decreasing its threshold for activation or by **diminished inhibitory control over spinal reflexes** (Campbell, 2005) as *damaged pyramidal tracts are unable to effectively inhibit reflexive muscle tone and normal stretch reflexes in lower-motor centers*.

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

hypertonicity

Spasticity from cerebral lesions	Spasticity in an individual with SCI
❑ affects muscle groups on one particular side of the joint predominantly ❑ the distribution most often includes the *flexors of the upper extremities* + the *extensors of the lower extremities*.	disruption of the corticospinal pathway may affect *opposing muscle groups equally in the affected regions of the body*.

Spastic resistance decreases as tension increases in the affected muscle, such when it is stretched at the end of its available ROM.

✓ The decrease in spastic resistance **resulting from** muscle elongation may be due to **activity of the Golgi tendon organ** (GTO) (Nielsen, 2007)

Long-term spasticity can minimize ability to move into the elongated range and can **result in adaptive shortening of the spastic muscle group**, *which may cause permanent decreases in available ROM*.

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

hypertonicity

Loss of joint ROM or flexibility due to spasticity
differs from
muscle tightness or inflexibility.

- ✓ **The major difference is related to movement velocity.**
- ✓ **Another difference is what is felt at the beginning and end of the available ROM.**
  - *During passive assessment of a spastic muscle, the clinician perceives velocity-dependent increased resistance.*
  - *Resistance associated with muscle tightness or inflexibility, however, increases as the joint moves toward the end of the available range but is not velocity dependent and therefore increases even during very slow speeds of muscle elongation.*

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

Modified Ashworth Scale (MAS)

- a scale specifically developed to rate the degree of spasticity (Bohannon, 1987; Lehmann, 1989;)
- Generally, the MAS grade (detail shown in Table 6-9) is determined based on the degree of resistance, what portion of the joint range exhibits the increased resistance, and how easily the resistance can be overcome.

TABLE 6-9 Modified Ashworth Scale For Grading "Spasticity"

GRADE	DESCRIPTION
0	No increase in muscle tone (no spasticity).
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance but only at the end of the ROM when the affected part(s) is moved in flexion or extension.
1 +	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance detected throughout the remainder (less than half) of the ROM.
2	More marked increase in muscle tone detected through most of the ROM but affected part(s) are easily moved.
3	Considerable increase in muscle tone, passive movement difficult.
4	Affected part(s) rigid in flexion and extension.

Adapted from Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. *Phys Ther.* 1987;67(2): 206–207. Data from Table 1 found on p. 207.

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

The Tardieu Scale

- originally described in 1954 (Tardieu, 1954) as a passive measure of spasticity and adapted as the Modified Tardieu Scale (Boyd, 1999)
- administered by *moving the client's limb passively through the available ROM* and *noting the precise point in range where resistance is first met or catches* compared with the end of the available ROM

TABLE 6-10 Modified Tardieu Scale For Grading "Spasticity"

GRADE	DESCRIPTION
Zero	**No resistance** throughout the course of the passive movement.
1	**Slight resistance** throughout the course of the passive movement, with **no** clear catch at precise angle.
2	**Clear catch** at precise angle, interrupting the passive movement, **followed by** release.
3	**Fatigable clonus** (<10 seconds when maintaining pressure occurring at precise angle).
4	**Infatigable clonus** (>10 seconds when main-taining pressure occurring at precise angle).

From: Boyd RN, Graham HK. Objective measurement of clinical findings in the use of botulinum toxin type A for the management of children with cerebral palsy. *Eur J Neurol.* 1999;6(suppl. 4):S23–S35.

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

The Tardieu Scale

- **Specifically, you should passively stretch the muscle at three different speeds:**

(1) as slow as possible,

(2) at the speed a limb falls under gravity, and

(3) as fast as possible (greater than the speed of a limb falls under gravity) to elicit the stretch reflex.

- ❑ During each movement, **use a goniometer** to measure the point at which the muscle catch (resistance) first occurs and the end of the available range.
- ❑ The angle of catch of the high-velocity movement is subtracted from the angle of catch of the slowest possible movement to acquire **a Tardieu spasticity angle**, which indicates where the movement is influenced by the spasticity.

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

The Tardieu Scale

- **The Modified Tardieu Scale (MTS)** was introduced by *adding standardized limb placement and alignment (e.g., supine as the starting position in the lower extremity)* to the previous technique (Boyd, 2002; Boyd, 1999).
- **The MTS** is able to differentiate between neural limitations that are velocity dependent (spasticity) and passive stiffness (soft tissue limitations) while the Ashworth Scale cannot (Boyd, 2002; Damiano, 2002),
- repeating measures can be used to detect change in spasticity related to medical management with botulinum toxin A in children with CP with good testretest reliability (Boyd, 1998).

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

The Triple Spasticity Scale (TSS) (Li, 2014)

- It measures spasticity on the basis of tonic and phasic stretch reflexes, and evaluates passive resistance and dynamic muscle length.
-

For the first item,

- ✓ perform a slow stretch "R2" (less than 5 degrees/sec) through full range and
- ✓ fast stretch "R1" (as fast as possible) of the target muscle and
- ✓ assess for increased resistance with the fast stretch (compared with a slow stretch)

0 = *no increased resistance*, **1** = *mild increase*, **2** = *moderate increase*, **3** = *severe increase*, and **4** = *extremely severe increase*.

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

The Triple Spasticity Scale (TSS) (Li, 2014)

- **For the second item,**

assess the degree of clonus:

0 = no clonus;

1 = fatigable, (a clonus less than 10s);

2 = infatigable, (a clonus greater than10s).

- **for the last item,**

✓**assess dynamic muscle length (R1-R2)**

✓by **noting the angle** at which the increased resistance is *detected for each speed* and

✓**calculate** the difference in the two angles, converting to this

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

The Triple Spasticity Scale (TSS) (Li, 2014)

- **five-point scale:**

0 = angle difference between R1 and R2 is 0,

1 = angle difference is <1/4 full ROM,

2 = angle difference is ≥1/4 and <1/2 full ROM,

3 = angle difference is ≥1/2 and <3/4 full ROM,

or 4 = angle difference is ≥ 3/4 full ROM.

"**mild** (0 to 2),
moderate (3 to 5), or
severe spasticity (6 to 8)

- **in the muscles in which clonus coul not be elicited";**

mild (0 to 3),
moderate (4 to 6), or
severe spasticity (7 to 10) in the muscles in which **clonus** could be elicited"

(Li, 2014).

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

The Myotonometer (Neurogenic Technologies Inc., Missoula,

- developed to provide a quantitative assessment of muscle stiffness.
- Because muscle stiffness is thought to correlate with muscle tone, the Myotonometer has been validated as a *reliable quantitative muscle tone assessment tool*.
- It is unclear how muscle stiffness, a property inherent in the muscle itself (which presumably is not velocity dependent), *correlates with abnormal muscle tone* such as spasticity (which by definition is velocity dependent).

FIGURE 6-14 **A.** Myotonometer equipment. **B.** The myotonometer applied to a pediatric patient.

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

Rigidity or rigid hypertonia,

- a form of hypertonicity, is associated with several neurological disorders, particularly Parkinson disease.
- **Clinical manifestations of rigidity,**

✓detected during the passive elongation testing procedure for muscle tone,

✓ differ from spasticity in that **muscle groups are commonly affected** **on both sides of the joint** (opposing muscle groups, e.g., flexors and extensors, are both significantly affected),

✓**the resistance is present throughout the ROM**, and it may be present **even at slow speeds.**

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

Rigidity or rigid hypertonia,

- **In Parkinson disease, cogwheel rigidity** is sometimes felt in which the increased muscle tone is characterized by *multiple occasions of catch and release throughout the range along with rigidity and superimposed tremor*.
- Some common types of rigidity are **extrapyramidal**, **voluntary** and **involuntary**, **reflex**, **catatonic**, and **myotonic**.

TABLE 6-11 Decerebrate Rigidity versus Decorticate Rigidity

TYPE	CAUSE	MANIFESTATION
Decerebrate Rigidity	Occurs from disruption of excitatory input to medullary reticular nuclei causing disinhibition + overactivity of pontine reticular nuclei.	**Spasticity of the antigravity (extensor) muscles of the entire body:** static, fixed posturing as extension of upper extremity and lower extremity (*equates to 2 points for Best Motor Response on Glascow Coma Scale*).
Decorticate Rigidity	Occurs from disruption of influence from the cerebral cortex *without disruption of the red nucleus or basal ganglia.*	**Spasticity of the flexor muscles of the upper extremity** *(static flexion posturing)* and **the extensors of the lower extremity** *(static extension posturing)* (*equates to 3 points for Best Motor Response on Glascow Coma Scale*).

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

Rigidity or rigid hypertonia,

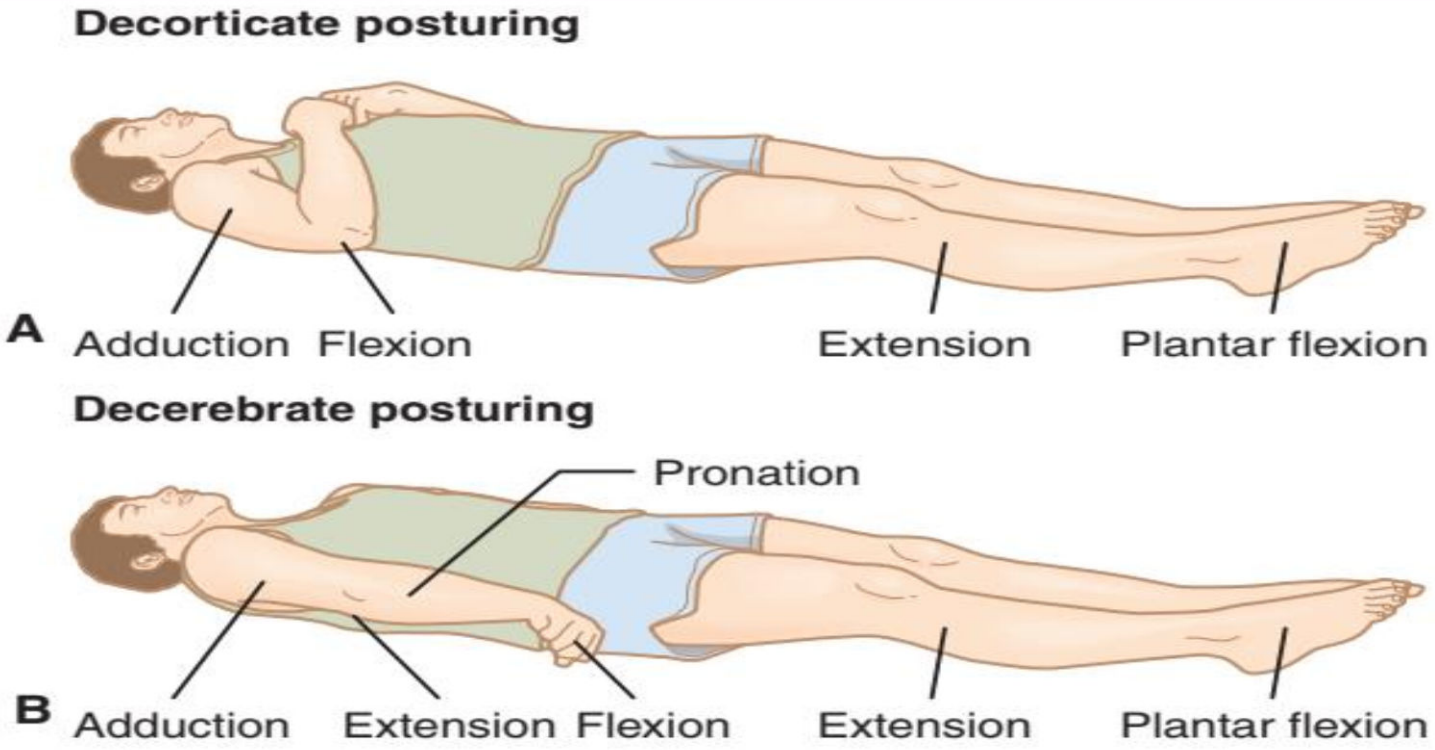


FIGURE 6-15 Characteristic posturing of **A.** decorticate and **B.** decerebrate rigidity.

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

Rigidity or rigid hypertonia,

- ✓ These postures are most often associated with very severe cerebral injury and decreased levels of consciousness, usually **coma**.
- ✓ The terms decorticate rigidity and decerebrate rigidity are **misnomers**, as they could be considered the most extreme forms of spasticity (see Modified Ashworth Scale, grade 4) with hypertonia predominant on one side of the joint that fixes the limb in the static posture described.
- ✓ On the Glascow Coma Scale, decerebrate posturing (score 2 of the possible 5 points for the Best Motor Response item) is considered more severe than decorticate posturing (score 3 of the possible 5 points for the Best Motor Response item).

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

rigidity assessment:

Unified Parkinson's Disease Rating Scale (UPDRS) Motor Examination section (Fahn, 1987).

- Most commonly used In clinical and research settings,

TABLE 6-12 Qualifiers for Rating Rigidity Using the UPDRS Motor Examination

SCORE	DESCRIPTION
Zero	Rigidity is absent.
1	Slight rigidity or only with muscle activation.
2	Mild/moderate rigidity.
3	Marked rigidity, full ROM.
4	Severe rigidity for five regions (neck, right upper extremity, left upper extremity, right lower extremity, and left lower extremity).

(Fahn, 1987)

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

rigidity assessment:

the Shoulder-Shaking Test

- facing the standing patient, and place your hands on the patient's shoulders.
- Move the shoulders alternately back and forth to rotate the trunk with varying speed and force.
- ✓ This will result in **passive swinging of the patient's arms**, which may be diminished in one arm in unilateral Parkinson. Rigidity results in diminished range of movement in the affected body segments.

Starting at a speed that causes equal pendulous movements on each side, gradually and slowly reduce the force and range of the trunk movements. As the pendulous movements decrease, you may reach a point where only one arm swings. The arm with decreased swing is the one with the greater rigidity.

(Campbell, 2005).

OBSERVATIONS FOR SPECIFIC TYPES OF ABNORMAL MUSCLE TONE

rigidity assessment:

Head-Dropping Test

- occurs in supine with the patient's eyes closed.
- There should not be a pillow between the patient's head and the plinth.
- Place a hand on the plinth under the patient's occiput while the other hand raises the head off the plinth. Complete the test by dropping the head into the hand under the occiput .

Rigidity causes the head to lower more slowly into the awaiting examiner's hand where as without rigidity the head falls more rapidly (Campbell, 2005).

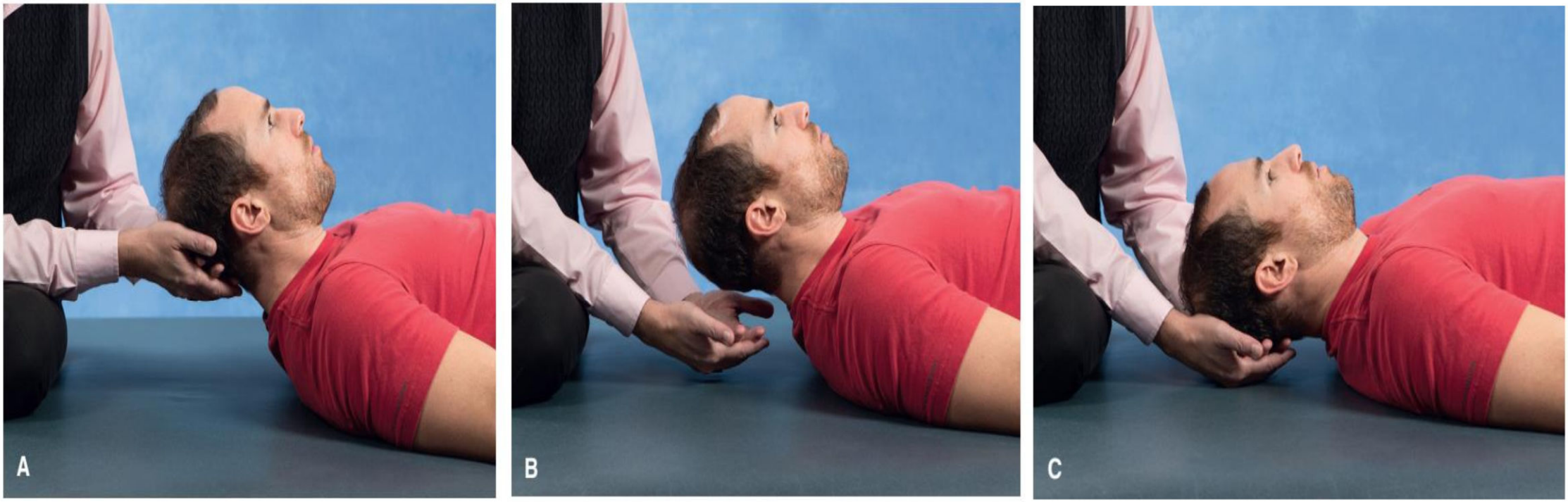


FIGURE 6-16 **Head-Dropping Test:** A photo series showing A. the patient's head supported by the therapist's hand while the other hand waits below nearer the mat surface, B. sudden release of the supporting hand, and C. the patient's head dropsgently into the therapist's lower hand.

MEASURING MOTOR FUNCTION

Motor Control

- **Muscle strength alone** does not fully **define proper motor function as force gradation** and the **timing of activation** often determine whether a muscle interacts appropriately with other muscles or muscle groups during functional activities (Shumway-Cook, 2012).
- **An earlier definition provided by Shumway-Cook (2001)** emphasized **"the study of the nature and cause of movement,** including stabilizing the body in space (MC-stability)+ moving the body in space(MC-movement)."
- *will discuss details for observation and measurement of both motor control* **for movement**, *which includes motor* ***coordination*** *in movement, and motor control* **for stability.**

DEF / motor control is the ability of **the nervous system to regulate the motor system** *with* **appropriate activation or relaxation** *during all* ***normal functional activities.***

MEASURING MOTOR FUNCTION

Motor Control

- Motor control occurs from the collective influences of **cognition** or **volition**, **cerebral motor plans (sometimes subconscious)**, available **ROM**, **muscle strength**, **muscle tone**, **environmental conditions**, and **task characteristics** that **influence quality of movement** including velocity, timing, and accuracy.
- The degree of motor control ultimately affects **balance** and each movement of every **functional activity.**

MEASURING MOTOR FUNCTION

Motor Control

- An individual who has spasticity, for example, may or may not demonstrate deficits in motor control movement. Of necessity, for any muscle group being examined, **the therapist must test separately :**

1) for **muscle tone** (a passive examination on the patient's part) and

2) **motor control** (an active measure on the patient's part).

✓Also realize that the term **postural control** is not synonymous with **motor control.**

postural control is most often used to refer to balance or equilibrium in maintaining an upright posture .

3) It is also essential to understand that the biomechanical joint stability inherent in normal ligaments and joint capsules is also ***distinct from the dynamic neuromotor joint stability that results from motor control stability***.

TABLE 6-13 Observable Characteristics of Three Aspects of Motor Control

	RELATED TO MOVEMENT		RELATED TO STABILITY
	MC-Movement	***Motor Coordination***	***MC-Stability***
General Description of Components	**Isolated Control/Selective Motor Control:** Ability to activate and regulate *similarly acting* target muscles for *movement* at a single joint without unintended movement of other muscle groups. **Timing and Sequence:** Ability to activate muscles with appropriate sequencing (coordination between muscles at multiple joints that are cooperating to complete a complex movement) and timing within the activity/task, and capability to perform movement at a variety of speeds. **Initiation and Cessation:** Ability to start (initiate) and stop (cease) a particular muscle action, including grading on and grading off of muscle action.	**Coordination between *opposing* muscle groups at a joint during *movement*** (one grading on while the opposing group is appropriately grading off).	when a joint or segment is *steady and unwavering* and **should not move.**
Common Abnormal Expressions	**Isolated Movement:** Abnormal movement synergies (lack of isolated movements) **Timing and Sequence:** Bradykinesia **Impaired initiation or cessation**	**Ataxia** (incoordination).	**Joint instability** or segment instability, particularly during closed-chain or weight-bearing actions.

MEASURING MOTOR FUNCTION

Motor Control for Movement (MC-movement)

- also be called movement control.
- **To test for MC-movement, ask the person to volitionally move the joint or body segment being tested** and **observe the quality of the movement**.

1) *able to perform an isolated movemen* termed **selective motor control** (Voorman, 2007).
2) also be **smooth, steady, and flowing continuously without disruptions**, jerks, spurts, or starts throughout the entire range of active motion as the motor units of the involved muscle fire in a precise and accurate sequence and magnitude.
3) **controlled initiation and cessation of movement** allows the start and end of the motion to be gradual in timing and smooth at beginning and end.
4) **ability to voluntarily move the joint or segment at a variety of velocities**, moving slowly when desired, but moving quickly when necessary.

MEASURING MOTOR FUNCTION

5) able to fractionate movement as a sign of good MC-movement.

Fractionation

is the ability of the individual, through motor control, to move the target joint through very small fragments of the available range, as small as 1 or 2 degrees, even with large gross muscle groups (by activating a small fraction of the total motor units of a muscle or group).

After **cerebral injuries** such as **CVA** and traumatic brain injury, abnormal MC-movement is often observed *as* ***the lack of isolated voluntary movement*** *even though the limb can often* ***move with poor control***.

- commonly called an abnormal synergy of movement,

Although the person can move, the movement lacks accuracy + efficiency and therefore contributes minimally to the individual's functional ability + activity.

MEASURING MOTOR FUNCTION

Motor Control for Movement (MC-movement)

TABLE 6-14 Typical Abnormal Synergies for Lower Extremity (LE) and Upper Extremity (UE)

Typical Flexor Abnormal Synergy of UE Movement:

Scapula: Elevation and retraction
Shoulder: Abduction and external rotation **or** adduction and internal rotation
Elbow: Flexion
Forearm: Supination
Wrist/Digits: Position varies, **but** usually flexion

Typical Extensor Abnormal Synergy of LE Movement:

Pelvic: Elevation and retraction
Hip: Adduction and extension
Knee: Extension
Ankle: Plantar flexion
Forefoot: Usually inversion

Typical Extensor Abnormal Synergy of UE Movement:

Scapula: Downward rotation and protraction
Shoulder: Internal rotation and adduction
Elbow: Extension
Forearm: Pronation
Wrist/Hand/Digits: Position varies

Typical Flexion Abnormal Synergy of LE Movement:

Pelvic: Elevation
Hip: Flexion
Knee: Flexion
Ankle: Dorsiflexion
Forefoot: Usually eversion

MEASURING MOTOR FUNCTION

Motor Control for Movement (MC-movement)

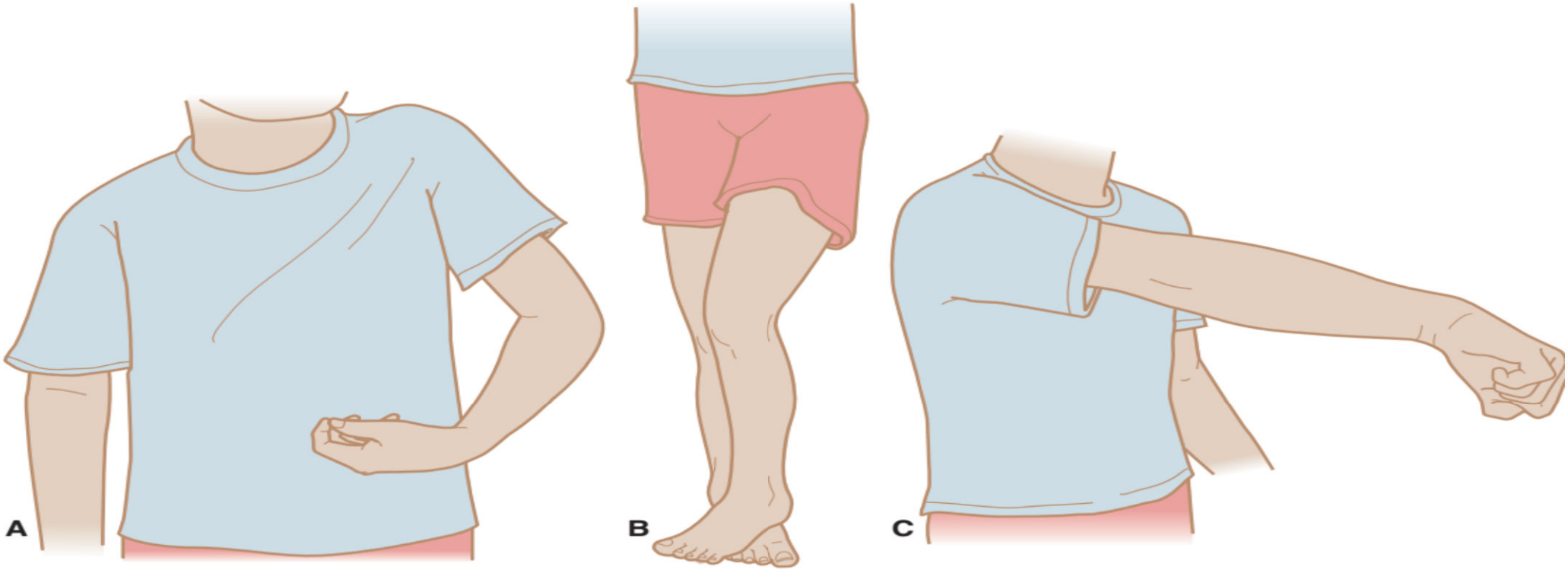


FIGURE 6-18 Examples of the most common abnormal synergies of upper extremity and lower extremity. **A.** Upper extremity flexion synergy (left arm in this patient) commonly happens during intended flexion or antigravity movements of the upper extremity. **B.** Lower extremity extension synergy (left leg in this patient) commonly happens during intended extension of the lower extremity, for example, during the terminal portion of swing phase. **C.** Upper extremity extension synergy (right arm in this patient) that may occur during intended elevation of the arm as opposed to intended flexion of the arm.

MEASURING MOTOR FUNCTION

Motor Control for Movement (MC-movement)

In 1966, Brunnstrom (1966) first described six stages of recovery following CVA and included **a description of characteristic motor control at each stage** with specific observations defined separately for upper and lower extremities (see Table 6-15).

- **(Stage 1)** Often, the patient initially exhibits flaccidity with complete absence of volitional movement.
- **(Stage 2)** progresses to weak abnormal synergies with small range movement,
- **(Stage 3)** then stronger, more obvious abnormal synergies.
- **(Stage 4)** Volitional movement is first observed in Stage 4, especially in proximal muscle groups (see specifics in Table 6-22).
- **In Stages 5 and 6,** active movements become progressively more complex with an increased number of isolated/selective movements outside of synergy.

Progressive changes in muscle tone were also described across the six stages (see Table 6-16) with progressive development and increase of spasticity in Stages 1 to 3 with a gradual diminution of spasticity from Stage 4 to 6 (Brunnstrom, 1966).

TABLE 6-15 Brunnstrom Stages of Recovery Following Stroke for the Upper and Lower Extremities

BRUNNSTROM STAGE	TONE	MOTOR CONTROL	SPECIFIC UE ISOLATED/ SELECTIVE MOVEMENTS ON AFFECTED SIDE	SPECIFIC LE ISOLATED/ SELECTIVE MOVEMENTS ON AFFECTED SIDE
Stage 1 • **Flaccid Tone** • **No Movement**	Flaccid; little or no resistance to passive movement	No active movement	None	None
Stage 2 • Increasing Spasticity • Weak Synergies	Slight spasticity	Weak synergies **+** weak associated reactions	None	None
Stage 3 • Peak of Spasticity • Basic Synergies	Spasticity, may be severe	**Voluntary movement within basic synergies**; demonstrates small but determinable joint movement	None	None
Stage 4 • Spasticity Decreasing • Earliest Isolated Motor Control	Spasticity begins to decrease	**Active movement begins to occur outside of basic limb synergies**	(1) **Hand to sacral region** (involves difficult combina-tion of internal rotation,extension and adduction of the arm) (2) **Elevation of arm to forward-horizontal position with elbow fully extended** (3) **Pronation/supination with elbow flexed to 90 degrees**	(1) Knee flexion beyond 90° sliding foot back-ward on floor (2) Dorsiflexion of ankle without lifting entire foot off floor
Stage 5 • Spasticity Decreasing • More Isolated Motor Control	Spasticity continues to decrease	**Able to perform more difficult movement combinations**	(1) Shoulder abduction to 90° with elbow extended (2) Shoulder flexion past 90° with elbow extended (3) Pronation/supination with elbow extended	(1) Knee flexion with hip extension in standing (2) Ankle dorsiflexion in standing with foot in position of short step
Stage 6 • No Spasticity • Selective Motor Control	No spasticity with passive movement but ***tone may increase during faster move-ment velocities***	**Movements are generally selective**,but may require performance at **decreased velocities** with **diminished coordination**	Generally selective with decreased speed and decreased coordination	(1) Hip abduction beyond pelvic elevation in standing (2) Isolated knee inter-nal and external rotation with ankle inversion and eversion

*Patients may progress through all stages or remain/arrest in any one stage. The extent of each manifestation and the duration of the various stages are dependent on the severity of the stroke and the age of the patient.
(Adapted from Brunnstrom, 1966)

MEASURING MOTOR FUNCTION

Motor Control for Stability (MC-stability)

- the ability of the brain to regulate muscles to precisely keep joints or body segments from moving at times when they should not move(see MC-stability column of Table 6-13).

This action of the brain, working through the muscles,
is very different from the stability that the ligaments or skeleton provide.

Motor control stabilization is often required in **closed chain activities** or **weight-bearing phases of tasks** and most often **occurs through cocontraction** or **simultaneous contraction of opposing muscle groups** so ***the joint will not move in either direction.***

- **MC-stability, particularly examination, is a neglected concept in the evidence-based literature.**
- Table 6-16 provides a summary table of the most common examples of impaired MC-stability, organized by body regions, which can result from specific neurological pathologies.

MEASURING MOTOR FUNCTION

Motor Control for Stability (MC-stability)

TABLE 6-16 Common Examples of Impaired MC-Stability

Scapulothoracic Instability:

- Following stroke, TBI, or CP (and others), **scapular winging** may occur because of an inability to control scapular positioning on the trunk related to *decreased motor control in serratus anterior.*

Glenohumeral Instability:

- In the UE, glenohumeral instability can result in **shoulder subluxation** (and subsequent pain syndromes related to the musculoskeletal consequences of prolonged subluxation), *accentuated by gravity in sitting and upright positions.*

Elbow Instability:

- **Inability to stabilize the elbow in extension** will *severely limit UE weight-bearing through an extended arm* and *the ability to push with the arms.*

Wrist Instability:

- Impaired MC-stability in the wrist/hand complex can **impair grasp.**

Trunk Instability:

- Lack of stability in intraspinal segments and abdominal muscles can result in an **abnormal posture or alignment of the trunk** ; can also result in the trunk being an *insufficient base from which the upper extremities and lower extremities can move.*

MEASURING MOTOR FUNCTION

TABLE 6-16 Common Examples of Impaired MC-Stability

Lumbopelvic Instability:

- Inability to stabilize the pelvis on the trunk can result in problems with maintaining stability of the pelvis (and **a level position**) *from which lower extremity swing phase operates.*

Hip Instability:

- If the femoral head does not stabilize against the pelvic acetabulum, the hip can go into sudden flexion or extension, or even more commonly, during weight-bearing, a Trendelenberg gait can occur during stance phase on the affected side, as the opposite side of the pelvis drops because of insufficient control from the hip abductors.

Knee Instability:

- Instability of the knee during the stance phase of gait can result in either **genu recurvatum** (sudden snapping of the knee into hyperextension during the stance phase of gait), locked knee extension in midstance as a biomechanical mechanism to provide stability in absence of control, or an unstable knee that buckles in midstance full weight-bearing.

Ankle Instability:

- Instability in the sagittal plane allows **plantar flexion to occur during swing** (any amount of plantar flexion is too much during the swing phase of gait) with **foot drop or toe-drag**; **subtalar instability** results in *a medial/lateral wobble of the ankle during weight acceptance or midstance phases of gait.*

MEASURING MOTOR FUNCTION

- **The composite effect of decreased MC-stability of the hip, knee, and ankle on the affected side following a stroke will be obvious in gait** *as the decreased stability will result in obvious asymmetry with decreased weightbearing on the affected side.*

person being unable to trust the affected limb
for
weight-support.

a decreased single-support phase
on the **affected side**

a decreased step time & **step length**
on the **unaffected side**

MEASURING MOTOR FUNCTION

Depending on the task,
the **muscles at a particular joint**
may have *to switch or alternate between the two forms of motor control*.

- **The gait cycle** is a perfect example of body segments and specific joints that cyclically alternate between **motor control stability** *(required in the joints of the weight-bearing stance-phase leg)* and **motor control movement** *(necessary for the precise movement of the swing phase leg)*.

Another interesting combination can be seen in the **swing phase as the trunk & pelvis must simultaneously maintain motor control stability** *with the motor control movement of the hip, knee, and ankle* to allow the step to take place with skill and precision.

The same concept applies to an **upper extremity reach task** as the **trunk** & **proximal UL** must maintain MC-stability while the more distal joints of the arm fluidly carry out MC-movement.

So in any functional activity,
therapists must understand and observe
which joints need to have MC-stability and **which need to have MC-movement**
and
whether *some body segments require* **MC-stability part of the time**
and
MC-movement part of the time throughout the activity

Motor Coordination (MC-coordination)

- Def/ a specific subset of MC-movement that specifically focuses on the motor control interactions and cooperation between opposing muscle groups during a movement (agonists and antagonistic).

Coordinated movements

are

controlled, smooth, and precise

because of the cooperation between opposing muscle groups, largely mediated through action of the **cerebellum.** (Schmitz, 2014)

Coordination assessments

- **nonequilibrium examinations**
- **Equilibrium examinations**

Motor Coordination (MC-coordination)

Impairment of coordination is called **incoordination** or **ataxia**

observed as the following specific signs:

(1) intention tremor, (2) dysmetria, (3) astasia,

(4) dysdiadochokinesia, (5) dysynergia, (6) decomposition,
(7) overcompensation.

Note that all these specific signs occur during intended active movement and not when the limb is at rest. (each term is defined in Table 6-17):

TABLE 6-17 Coordination Examination: Abnormal Signs of Ataxia/Incoordination

ATAXIC SIGN	DEFINITION
Ataxia/Incoordination	**An umbrella term** including all aspects of incoordination listed here. Abnormally inaccurate or uncoordinated volitional movement of skeletal muscle.
Intention Tremor	A specific component of ataxia: tremor occurs **during voluntary** (intended) movement, often in a *back-and-forth direction in the plane of intended movement*.
Dysmetria	A specific component of ataxia: the **inability to judge the distance to a target during movement;** results in either *stopping short of the target* **or** *pass-pointing*; inability to correctly position limbs relative to one another.
Hypermetria (pass-pointing)	A specific component of ataxia: **reaching past, or overshooting, the intended target.**
Astasia	A specific component of ataxia: an **inability to maintain standing that results from incoordination during weight-bearing.**
Dysdiadochokinesia	A specific component of ataxia: **decreased ability to perform a rapidly alternating movement** (e.g., forearm pronation/supination; wrist flexion/extension; ankle dorsiflexion/plantar flexion).
Adiadochokinesia	A specific component of ataxia: **inability to perform rapidly alternating movements.**
Dysynergia	A specific component of ataxia: the **incoordination of antagonist muscle groups.**
Decomposition	A specific component of ataxia: a **movement is abnormally broken down into its component** parts instead of a smooth, fluid, multijoint movement.
Overcompensation	A specific component of ataxia: **in attempting to correct a dysmetric, ataxic error (over- or undershooting), the individual overcorrects and again passes the target.**

TESTS/MEASURES FOR:

- MOTOR CONTROL-MOVEMENT
-MOTOR CONTROL STABILITY
- MOTOR COORDINATION

TESTS/MEASURES FOR MOTOR CONTROL-MOVEMENT

simply ask the patient to actively move the limb

and carefully observe,

one joint/muscle action at a time, for

smoothness, initiation, cessation, fractionation, performance at a variety of speeds, and the ability to isolate movement

at the specific joint being tested.

Then record your observations, including the specific abnormal muscle group combination the patient demonstrates.

TESTS/MEASURES FOR MOTOR CONTROL-MOVEMENT

a method to quantify the extent of isolated MC-movement and track improvement over time:

by *measuring the portion of available joint range through which the individual can actively perform an isolated movement.*

- ✓ You should stop the movement for angle measurement at the first observed sign of an abnormal synergy combination.
- ✓ helpful to document the position in the available range at which the isolated movement can be initiated, (which will most often be at the end of range).

As the individual's MC-movement improves during the period of stroke recovery and rehabilitation, the quantity of controlled isolated movements (measured as degrees of available range through which selective control is observed) will increase.

TESTS/MEASURES FOR MOTOR CONTROL-MOVEMENT

1) Finger Extension/Grasp Release

- Grasp and release are essential components of hand function.
- While **maintaining grasp** is an *example of MC-stability*, **release of grasp** is an *example of MC-movement.*
- **Active release of grasp** is one of the *items in the Upper Extremity Motor Score section of the Fugl-Meyer Assessment* (Fugl-Meyer, 1975).

- After stroke, individuals often have difficulty releasing an object because of **limited ability to initiate isolation of finger extension movement.**

✓ Finger extension/grasp release, defined as the ability to actively release a mass flexion grasp as specified by the Fugl-Meyer item, is **a significant predictor of motor recovery** (as measured by the Wolf Motor Function Test) (Fritz, 2005).

2) The Trost Selective Motor Control Test

- a basic general assessment system to document isolated movement ability for a specific muscle group, generally using an absent/impaired/normal rating system.
- **It does not specify a specific joint or motion** and can therefore be applied at any joint/segment.

"**0** (no selective, only synergistic movement),

1 (diminished selective movement [the first range of movement selective and later on, during the movement, no selective movement]), and

2 (full selective movement during extension of the knee and dorsiflexion of the ankle)."

3) The Boyd and Graham Selective Motor Control Test

- a five-point scale (0 to 4) related to active isolated MC-movement specifically of ankle dorsiflexion + how much muscle substitution occurs.
- **Because isolated ankle dorsiflexion is accomplished by the tibialis anterior muscle**, *the scale assigns lower ratings if the ankle movement is accomplished primarily by involvement from extensor hallicus longus (Figure 6-19) or extensor digitorum longus* **while higher ratings are used when these muscle groups are not utilized.**

- ✓ Have the person short-sit on a flat surface with hips flexed and knees comfortably extended.
- ✓ Ask the individual to dorsiflex to a target over the foot, one foot at a time, and observe and document overall muscle activity.

3) The Boyd and Graham Selective Motor Control Test

- **grade of 0** is assigned for "No movement when asked to dorsiflex the foot";
- **grade of 1** for "Limited dorsiflexion using mainly Ext Hall Longus (EHL) and/or Ext Digitorum Longus (EDL)";
- **grade of 2** for "Dorsiflexion using EHL, EDL, and some Tibialis Anterior (TA)";
- **gradeof 3** for "Dorsiflexion achieved using mainly TA activity, but accompanied by hip and/or knee flexion"; and
- **grade of 4** for "Isolated selective dorsiflexion achieved, through available range, using a balance of TA activity without hip and knee flexion"

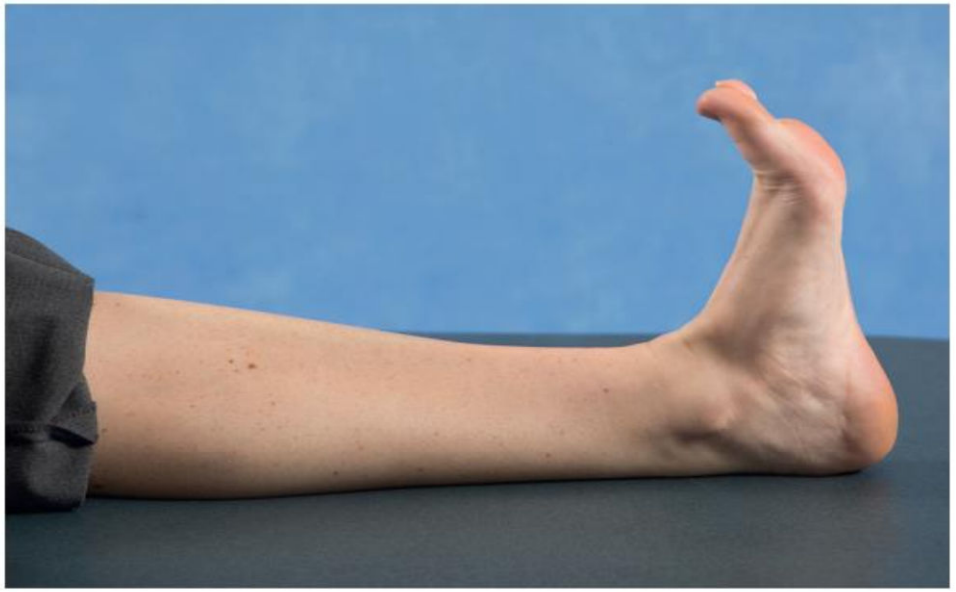


FIGURE 6-19 An example of a deficit in isolated movement (selective control) for **ankle dorsiflexion** using the **Boyd and Graham Selective Motor Control Test**. While attempting to perform isolated ankle dorsiflexion, the extensor hallicus longus in the test leg is observed with major contribution to the active ankle dorsiflexion in this person who is *unable to perform selective ankle dorsiflexion on the affected side*.

- While this specific scale is for isolated control of ankle dorsiflexion, a similar scale could be developed for any specific muscle group using an understanding of muscle actions that commonly occur as abnormal synergies with the target muscle.

✓ For example, ***elbow flexion*** *may be* ***accompanied by synergistic*** *shoulder flexion & internal rotation* + *wrist & finger flexion*.

TESTS/MEASURES FOR MOTOR CONTROL STABILITY

1) Motricity Index Pincer Grip Item

- ***Item 1 of the Arm section of the Motricity Index*** specifically tests the patient's ability to grasp a 2.5-cm cube between the thumb and forefinger using **a pincer grip** (Collen, 1990).

✓ The patient is scored in six increments depending on how well the individual performs the task.

✓ means of tracking a patient's progress in therapy.

2) L-Shaped Jig With Calibrated Rule

- L-shaped thermoplastic jig with an embedded 21-cm tape measure has been described as a means of **measuring impairment of MC-stability of the glenohumeral joint** (Hayes, 1989).

✓ The **acromion** and **a mark 20 cm above the olecranon** served as landmarks.

FIGURE 6-20 **Using a transparent ruler to measure glenohumeral subluxation of the shoulder using a method based on Hayes (1989).**
A. Mark a point on the skin, along the lateral humerus, 20 cm above the olecranon. **B.** With the arm hanging in a gravity-dependent position, measure the distance between two landmarks (the acromion and the point you marked). **C.** Support the arm under the flexed elbow to reduce the subluxation and remeasure the distance between the two points. ***The subluxation is documented as the difference between the two measures.***

TESTS/MEASURES FOR MOTOR COORDINATION

- During each requested movement, observe for **smooth fluid movement with continuity throughout the movement** as evidence that opposing muscle groups are cooperating, one contracting and shortening while the opposing group relaxes and elongates.

A generic ordinal grading system is designed to provide standardization;

1 = *the patient cannot perform the intended activity,*
2 = *the patient has a severe impairment,*
3 = *associated with moderate impairment,*
4 = minimal impairment,
5 = *normal*.

MOTOR COORDINATION UPPER EXTREMITY TESTS

❑ Tests of motor coordination of the upper extremity may involve either:

I. mass (large movements) or II- smaller (finite movement) tasks.

mass (large movements)	smaller (finite movement) task
finger-to-finger test, ○ touching their index fingers together; **combining** *the finger-to-nose test* with the *finger-to-therapist's finger* such that the *patient alternates touching their nose with touching the therapist's finger*; ○ pointing and pass-pointing test; ○ drawing geometrical shapes in the air such as figure eight or circle.	○ tapping the hand or fingers in a specified pattern, ○ rapid pronation and supination, ○ the rebound test, ○ rapid fist development, ○ handwriting tasks, and ○ finger opposition

Remember to test both right and left sides for a built-in comparison, *which is especially useful in conditions where one side of the body is affected.*

MOTOR COORDINATION UPPER EXTREMITY TESTS

Three types of tremor with cerebellar or somatosensory ataxia (Ropper, 2009)

Intention tremor

(see Table 6-17)

- ✓ occurs only during intended voluntary movement

(think of it as the opposing muscle groups struggling against each other in the planes of intended movement)

- ✓ frequency of 3 to 5 Hz.

Postural tremor

- presents when a patient is asked to adopt and maintain a posture or maintain a limb against gravity, for instance, **holding an arm in 90-degree abduction**.
- **only present when** the patient is attempting to maintain a position.
- typical frequency of postural tremor is about **3 Hz.**
- Both the agonist and antagonist muscles contribute to the tremor.
- **disappears** if the limb is supported proximally.

Titubation

- a rhythmic tremor mainly of the head and/or upper trunk
- Primarily present in anteroposterior plane
- a frequency of **3 to 4 Hz**

finger-to-nose-to-finger test

- ✓ assesses nonequilibrium coordination of the upper extremities.
- ✓ precision movements of the upper extremity to bring the patient's index finger into contact with either the patient's own nose, the therapist's finger, or alternating between the two (Figure 6-22).
- ✓ **To begin the finger-to-nose test:**

seat the patient with their arms abducted to 90 degrees + ask the patient to flex one elbow at a time + touch the tip of their nose with their index finger.

- ✓ Have the patient alternate upper extremities at various speeds
- ✓ with and without their eyes open.

Observe for dysmetria, pastpointing, intention tremor, and other components of ataxia described in Table 6-17.

FIGURE 6-22 Coordination test for upper extremity: finger-to-nose. **A.** The patient will first touch their own nose with index finger of the hand to be tested. **B.** Then move the same finger to touch the therapist's finger, then repeat the *cycle between nose and finger.*

FIGURE 6-23 Coordination test for upper extremity: finger-to-finger. **A.** The patient starts with arms horizontally abducted and index fingers extended (with eyes closed). **B.** Then have the patient *touch the two index fingers together in the midline.*

To quantify the magnitude of inaccuracies during the finger-to-nose test,

- **a transparent shield** over the face with concentric rings centered around the patient's nose has been used (Notermans, 1994).
- The error is measured as the distance from the point where the patient's index finger contacts the shield to the patient's nose.

The use of stopwatch measures has also been suggested to quantify the speed of finger-to-nose movement (Swaine, 1993).

Another possibility is **measuring the frequency of errors by counting the number of missed targets in either 10, 15, or 30 seconds (errors/second)** or calculating the ratio of **errors to total attempts**.

- The test can be expanded by asking the patient to alternate between touching the patient's own nose and the therapist's index finger as targets.
- This is a more complex test requiring **visual input** to execute the task and reach the targets appropriately, especially if the therapist moves the finger to varying locations.

Another version includes the finger-to-finger test where

finger-to-finger test

- the patient touches the two index fingers together (Figure 6-23).

✓Beginning in a position with the patient's arms abducted to 90 degrees with full elbow extension, the patient is then instructed to move both upper extremities into horizontal adduction until the tips of each index finger touch in midline.

The pointing and pass-pointing test

- requires you to face the patient as you both place your arms in 90 degrees of shoulder flexion allowing index fingers to approximate.

✓ Next, while maintaining the position of your upper extremities in 90 degrees of shoulder flexion, have the patient move into full shoulder elevation causing the patient's index fingers to point toward the ceiling.

✓ The patient then returns their upper extremities back to the beginning position.

Tapping the hand or fingers in a specified movement

- may be quantified by the use of sophisticated equipment that records the number of taps or
- you may employ less-sophisticated tasks such as observation of the patient during **tapping of a table surface or the ipsilateral knee**, which may be **timed.**

The rebound test

- **requires isometric tension in a muscle group, which is suddenly released.**

For example,
gradually build up resistance against a muscle group such as the elbow flexors or the shoulder flexors, *then abruptly release without warning.*

Normally, the **cerebellum rapidly detects the change in velocity at the joint and the antagonistic muscle group activates *to prevent excessive movement*** *as a reflection of appropriate processing of proprioceptive information.*

An abnormal response is observed as the *limb moves excessively in the direction of the tested muscle group's action.*

Dysdiadochokinesia

Rapidly alternating movements

- Test rapid forearm pronation and supination with the patient's shoulder in adduction, the elbow in 90 degrees of flexion, and the wrist in neutral.
- **Other tests:**

✓ repetitive patting of the hand **(rapid wrist flexion/extension)** on a flat surface

✓ **rapid fist development** where the patient extends all fingers maximally followed by full finger flexion

✓ **finger opposition movements** involve sequential movement of the thumb to the tip of each individual finger one at a time.

upper extremity shape-drawing test

the patient draw geometrical shapes in the air such as a "figure eight" or circle.

✓ The idea is to have the patient use large movements with the hand fixed in one position throughout the movement.

✓ The idea is to have the patient use large movements with the hand fixed in one position throughout the movement.

✓ Observe the quality of the movement, including the symmetry,

Writing by hand on paper TEST

is similar to drawing shapes in the air, but the movement is much smaller and is supported.

✓ The therapist may draw an object or write a letter or words and have the patient trace with a performance of their own

Motor Coordination Lower Extremity Tests

similar to those described for the upper extremity, including mass movements or finite movements of multiple or single joints, respectively.

Mass movements

include

- ✓ the heel-to-shin test,
- ✓ touching the great toe to an examiner's finger (toe-to-finger test),
- ✓ picking up objects with the toes and moving them to another location,
- ✓ drawing geometrical shapes with the foot . (DeHaven,1969).

Smaller finite movements

include

- ✓ tapping tasks with the feet and toes.

mass movement tests

- The patient should assume a lying or sitting position. The supine position can also help minimize any confounding affects that may be due to weakness.
- Testing in standing would compromise safety and, making it an equilibrium test.

heel-to-shin test

ask the patient to sit or lie supine and to place the heel of the limb being tested along the anterior surface of the contralateral ankle. The patient then slides the calcaneus along the anterior surface of the contralateral tibia up toward the knee and then back down again, repeating this cycle several times (Figure 6-24).

Observe

the **quality of the movement**, such as the *ability to make smooth movements or to maintain a straight path* along the tibia.

You might quantify errors by

- ✓ counting the number of times the heel moves horizontally off the tibia,
- ✓ the number of times the movement stops and starts (versus continuous flow of movement), and
- ✓ the number of times that overshooting and undershooting occurs during one cycle.

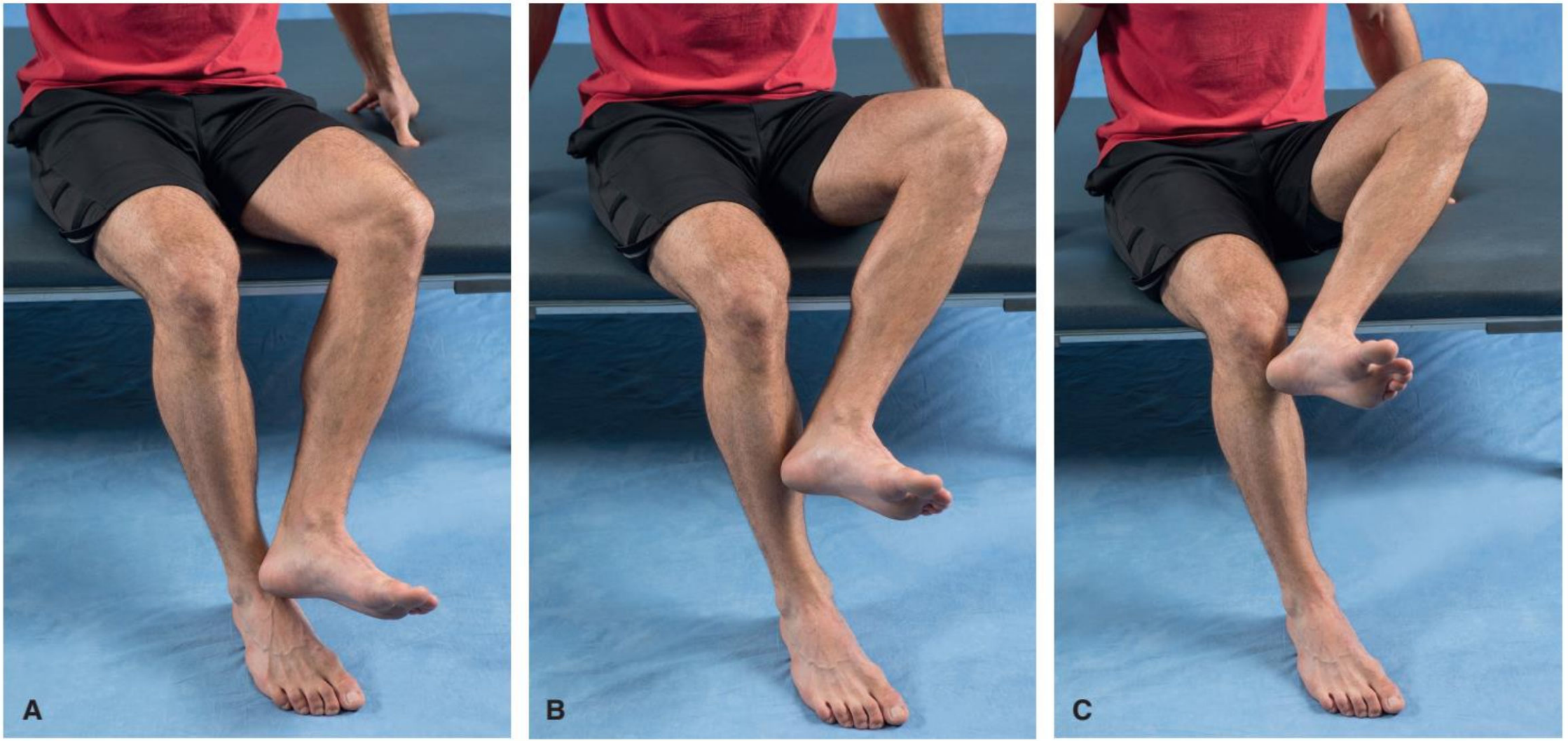


FIGURE 6-24 Coordination test for lower extremity: heel-to-shin. **A.** The patient starts with placing the heel of the test leg anterior to the ankle of the opposite leg. **B.** Then ask the patient to slide the calcaneous of the test leg from the anterior aspect of the opposite ankle along the tibia **C.** up to the anterior aspect of the opposite knee, then repeat the movement up and down along the anterior tibia, back and forth.

touch their great toe to your finger in a manner similar to the upper extremity finger-to-finger test (occur in the supine position)

- As with the upper extremity, you can modify the examination by moving your target finger to change the distance, speed, or direction required to complete the task.
- As you move the finger in close, then away from the patient's body, relatively more flexion and extension are required for the hip and knee, respectively.

use toes to pick up objects with the lower extremity and move them to another location.

requires skills similar to the toe-to-finger test but is **even more advanced** ***because*** *toe flexion is required to grasp an object such as a marble, crumpled towel, or small cylinder and move it to another location.*

Shape drawing test for lower extremity

- (i.e., asking the patient to draw geometrical shapes with the lower extremity) is similar to the upper extremity test except that the patient may not be able to use the great toe to independently draw the object as in the upper extremity assessment.
- The patient uses the great toe as if it were a writing utensil, only the toes remain fixed such that the entire lower extremity must move to perform the task.

Foot tapping

test for dysdiadochokinesia occurs with rapidly alternating movements of ankle PF & DF.

While sitting, the patient begins with the knee and hip in approximately 90 degrees of flexion and the test foot flat on the floor.

Ask the patient while maintaining heel contact with the floor to quickly dorsiflex and then plantarflex the ankle as the metatarsal heads (forefoot) alternately tap the floor.

- ✓ Tapping speeds may be increased to challenge
- ✓ **In ataxia,** the normal response of rhythmic tapping will deteriorate to erratic arrhythmic movements with no obvious cadence