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Five-step clinical assessment in spastic paresis

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Among the three main factors of motor impairment that emerge in chronological order following a lesion to central motor pathways, the last two antagonize movement: 1) stretch-sensitive paresis, a reduction of agonist motor unit recruitment upon voluntary command, worsened by antagonist stretch; 2) soft tissue contracture, and 3) muscle overactivity. Types of muscle overactivity include 1) spasticity, an increase in the velocity-dependent response to muscle stretch, measured at rest; 2) spastic dystonia, *i.e.*, chronic tonic muscle activity at rest, sensitive to stretch of the dystonic muscle and 3) spastic co-contraction, an inappropriate degree of antagonistic contraction during voluntary agonist command, sensitive to stretch of the co-contracting muscle. A five-step clinical assessment may closely parallel this phenomenology, in which the first four steps aim at quantifying the antagonistic potential of each muscle group. Step-1 measures passive range of motion, *i.e.*, the angle of arrest upon slow stretch of the muscle group assessed (minimizing spastic dystonia), which provides insight on soft tissue length and extensibility. Step-2 measures the angle of catch or clonus upon fast passive stretch of the muscle group assessed, which provides insight on stretch reflex excitability. Step-3 measures the range of active motion against the muscle group assessed, a net result of agonist recruitment minus the combined resistance from passive soft tissue stiffness and spastic co-contraction in the muscle group assessed. Step-4 measures the maximal frequency of rapid alternating movements along the maximal active range of motion, evaluating Step-3 performance repeatability. Step-5 evaluates active function, using for example a walking test (10 m or 2 min) for lower limb and the Modified Frenchay Scale for upper limb assessment, and perceived function through patient global subjective assessment.

KEY WORDS: Muscle contraction - Spastic paresis - Motor skills disorders.

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Central nervous system (CNS) lesions lead to the syndrome of spastic paresis when they affect central motor pathways involved in motor command execution.[1, 2] After briefly revisiting the taxonomy of motor impairment factors in spastic paresis, this article proposes a strategy of clinical assessment focused on measuring the "capacity of nuisance" of each muscle group as an antagonist to desired movements, a strategy inspired from Tardieu's teachings.[3-5] This evaluation comprises five consecutive steps, from passive soft tissue extensibility in each muscle group to global limb function.

Phenomenology of spastic paresis

Three fundamental phenomena occur after a lesion to the central motor pathways involved with motor command execution.

Stretch-sensitive paresis

A disruption of the central execution of motor command produces immediate paresis.[1] Paresis is the quantitative lack of agonist motor unit firing when attempting to generate force or movement. This involves insufficient synchronization of motor unit recruitment and/or insufficient discharge frequency of the recruited motor units. As antagonistic muscles shorten and become more spastic, the ability to recruit agonist motor units becomes increasingly sensitive to - inhibited by - antagonist stretch.[2] This phenomenon largely departs from what occurs in peripheral paresis; a typical feature of spastic paresis, it still requires systematic analysis and quantification in that context. We termed this characteristic central lesion-induced paresis, stretch-sensitive paresis.[2]

Soft tissue contracture

The relative immobilization and disuse of the paretic body parts leaves some of the muscles - and their surrounding soft tissues - immobilized in a shortened position. Gene changes and transcriptional events occur in muscle fibers as soon as a few hours after immobilization onset, leading to protein synthesis modifications.[6, 7] Such muscle plasticity initiates soft tissue contracture, which involves 1) physical shortening, an adaptation of soft tissue to its new length, involving muscles, tendons, ligaments, joint capsules, skin, vessels and nerves; 2) loss of extensibility; 3) loss of mass; 4) slow-to-fast changes in contractile muscle properties of originally slow muscles.[1, 6, 7] This process, beginning within hours of immobilization onset, only intensifies in the following days, weeks and months if insufficient prevention treatment is implemented.[2]

Muscle overactivity

Lesion- and behavior-induced plasticity then occurs within higher centers and spinal cord.[2] Following a corticospinal lesion, gene chip analysis of denervated ventral horns at spinal segments shows changes for growth factors, adhesion and guidance molecules, as well as components of synapse formation, which will foster intraspinal reorganization.[8-10] Mature descending motor tracts (brainstem descending and contralesional corticospinal pathways) thus undergo intraspinal reconnections, through local cues guiding newly formed sprouts in the denervated spinal cord.[9, 10] For example terminal arborizations of rubrospinal fibers are observed on motor neurons normally receiving only corticospinal projections.[9, 10] In parallel, brainstem descending pathways (rubro-, tecto-, reticulo-, vestibulospinal) and contralesional corticospinal pathways are increasingly recruited at higher centers – potentially *via* frontal or transcallosal disinhibition in brain lesions - to take over some of the motor command execution.[7, 8, 11, 12] Most of the brainstem descending pathways tend to be spontaneously active at rest, thus contributing to permanent, dystonic muscle activity through their newly formed motoneuronal connections.

Through the emerging local cues mentioned above, local sprouting from neighboring interneurons is also promoted at each spinal level, creating the conditions for the formation of new abnormal synapses between these interneurons and the somatic membrane of the deprived motor neurons. These new segmental or propriospinal synapses form the substratum for new abnormal or exaggerated reflex pathways.[2] The superimposition of dystonic and exaggerated reflex activity leads to overall muscle overactivity.

SPASTICITY

Spasticity has been the most commonly recognized manifestation among these gradually occurring reflex changes. Working from the endeavor by Lance *et al.*,[13] we simplified the wording of its definition as an increase in velocity-dependent stretch reflexes.[2] Spasticity is thus clinically manifested at rest by excessive responses to muscle stretch or tendon taps. Stretch-induced contraction at rest occurs at lower threshold and with increased amplitude in patients with spastic paresis compared to normal subjects. Thus, the primary triggering factor for observing and evaluating spasticity is phasic stretch (*i.e.*, movement of stretch), and the phenomenon is detected and measured at rest. Spasticity does not constitute a highly disabling form of muscle overactivity, except when clonus interferes with posture or movement, or in attempts at fast or ballistic active movements.

SPASTIC DYSTONIA

The term spastic dystonia has been coined by Denny Brown to represent the tonic, chronic mus-

cle activity present at rest in the context of spasticity.[2, 14] Spastic dystonia thus represents spontaneous overactivity, without primary triggering factor. This type of muscle overactivity is easily recognized in patients with spastic paresis at rest, as it deforms joints and body postures. Spastic dystonia thus constitutes a major cause of disfigurement and social handicap. The degree of spastic dystonia tends to be diminished by maintained stretch of the dystonic muscle.[2]

Spastic co-contraction

Spastic co-contraction is defined as an unwanted, excessive level of antagonistic muscle activity during voluntary agonist command, which is aggravated by tonic stretch of the cocontracting muscle.[2, 11] Much like spastic dystonia (present at rest), spastic co-contraction is a descending phenomenon, most likely due to misdirection of the supraspinal drive during voluntary command.[15] It may be facilitated by increased recurrent inhibition, causing loss of reciprocal inhibition to antagonists during voluntary agonist command.[2, 16, 17] Thus, the primary triggering factor for spastic co-contraction is voluntary agonist command; spastic co-contraction is thus detected and measured during voluntary effort. Spastic co-contraction is likely the most disabling form of muscle overactivity in spastic paresis, as it impedes force or movement generation, diminishes range of active motion and rapid alternating movement frequency. Spastic co-contraction is aggravated as effort increases in intensity and duration.[15, 18]

Other types of muscle overactivity

Other types of muscle overactivity develop in spastic paresis, which have not been shown to be stretch-sensitive. Pathological extra-segmental co-contraction (also known as synkinesia, associated reactions, overflow, chorea, athetosis, depending on its pattern and on the semantics utilized) is an unwanted, abnormal level of activity in muscles that are distant (at a different segmental level) from the agonist involved in the voluntary command.[2] Excessive cutaneous or nociceptive responses and other forms of inappropriate muscle recruitment during yawning, breathing or coughing are also common in spastic paresis.

A five-step clinical assessment

Following and expanding on Tardieu's proposals, and in direct opposition to assessment in peripheral paresis (MRC scale),[19] the first four steps of this assessment strategy evaluate each muscle group for its capacity to oppose movement, not for its power to generate it. Step 1 and Step 2 rate capacity to passively oppose movement, while Step 3 and Step 4 rate capacity to actively oppose movement (Appendixes 1, 2). The concept underlying this assessment strategy is that motor impairment in spastic paresis owes more to passive and active resistance from stretched muscle and soft tissue than to command weakness itself.

Each of the five assessment steps is quantitative. In the first three steps an angle is determined that is not measured based an anatomical principles, but based on zero being the angle of minimal stretch of the muscle group assessed. This point of minimal stretch of the muscle group assessed is taken as the point of reference in each movement evaluated, as it is the point where resistance starts. The first two steps of the assessment constitute what is today known in the literature as the Tardieu Scale.[20-22]

Step 1: Maximal range of passive motion (X_{V1})

Each muscle group (and associated soft tissue) is first evaluated using very slow and powerful stretch:

– the stretching movement must be as slow as possible for the examiner. Slowness serves to minimize the possibility of eliciting a stretch reflex in the maneuver, in the attempt to remain below the velocity threshold throughout the movement. The corresponding speed is called V1 (slow velocity);

– in most adult cases we recommend the stretching movement to be performed as strongly as possible for the examiner, as long as muscle and joint integrity is not jeopardized. Power serves to maximally overcome spastic dystonia so as to encounter resistance that is as close as possible to only passive soft tissue resistance.

The angle at which soft tissue resistance is no longer overcome by the examiner is defined as the passive range of motion against the muscle group assessed. The stretching maneuver is obviously interrupted if the clinician feels that soft tissue integ-

rity is threatened or if pain occurs during stretch. In the latter situation, the maximal range of passive motion cannot be ascertained. When, despite using slow and powerful stretch, the clinician is uncertain to distinguish soft tissue contracture from severe dystonia (which may typically occur in the case of an old or severe lesion), and when such distinction becomes critical for therapeutic decisions, a complementary technique may involve performing motor block with a local anesthetic. Maximal range of passive motion (X_{V1}) may be sensitive to highly aggressive stretch programs.[23]

Step-2: Angle of catch or clonus (X_{V3}) and spasticity grade (Y) – The Tardieu Scale

Each potentially opposing muscle group is then evaluated using fast stretch, *i.e.*, stretch at the fastest speed possible for the examiner (V3, fast velocity). To reliably accomplish this step, it is essential that the clinician ensures that muscle rest is obtained prior to the fast stretch maneuver. This can be accomplished, immediately prior to the fast stretch, through an inhibiting maneuver [24] or fast repetitive movements in the direction opposite to that of the stretch to be tested.

According to the Tardieu Scale, the clinician derives two parameters. The angle of catch or clonus (X_{V3}) represents the threshold to elicit the reflex. This parameter probably bears fundamental importance as it may correlate with the degree of stretch-sensitivity of the agonist paresis, although this remains to be systematically analyzed.[2] In addition, preliminary research indicates that it correlates with spastic co-contraction during phasic movements.[24] X_{V3} is also highly dependent on the length and passive extensibility of the muscle-tendon pair (estimated by X_{V1}), as the tension exerted on muscle spindle upon a given stretch angle varies according to the underlying muscle-tendon length.

The type of muscle reaction that occurs upon fast stretch at that angle defines the spasticity grade.[20-22] This parameter represents the gain of the stretch reflex.

The different situations encountered by the clinician, defined by the Tardieu Scale, may be as follows:

– no muscle contraction upon fast stretch: Y=0 ($X_{V3} = X_{V1}$);

– mild contraction occurring upon fast stretch, but at no angle sufficient to temporarily arrest passive movement (catch): Y=1 ($X_{V3} = X_{V1}$);

– contraction occurring at fast muscle stretch, with an intensity sufficient to temporarily arrest passive movement (catch) at a specific angle X_{V3}, different from X_{V1}, followed by release: Y=2 ($X_{V3} < X_{V1}$);

– contraction occurring at fast muscle stretch, sufficient to temporarily arrest passive movement (catch) at a specific angle X_{V3}, different from X_{V1}, followed by a release that is itself sufficient to elicit a second stretch reflex. As the clinician maintains pressure and depending on the movement speed during the release after the second stretch reflex, a new stretch reflex occurs, and so on until speed slows down to a point below the velocity threshold, time at which the situation exhausts. This is the case of fatigable clonus: Y=3 a ($X_{V3} < X_{V1}$);

– contraction occurring at fast muscle stretch, sufficient to temporarily arrest passive movement (catch) at a specific angle X_{V3}, different from X_{V1}, followed by a release that is itself sufficient to elicit a second stretch reflex. Depending on the movement speed during the release after the second stretch reflex as the clinician maintains pressure, a new stretch reflex occurs, but speed of release remains constantly greater than the velocity threshold; the situation persists over 10 seconds of maintained stretch. This is the case of infatigable clonus: Y=4 ($X_{V3} < X_{V1}$).

An additional situation may occur in which the sensation of release is uncertain despite movement arrest at a specific X_{V3} angle that is found to be consistently lower than X_{V1}. This case may signify that spastic dystonia has been well overcome by the first slow and strong stretching maneuver but is still sufficient to block passive movement at fast stretch. In this case of no obvious release with X_{V3} consistent and consistently smaller than X_{V1}, spasticity *per se* is unratable. For practical purposes we still propose to call the grade Y=1.5 ($X_{V3} < X_{V1}$).

The Tardieu Scale then defines the Spasticity Angle X, which is the difference X_{V1}-X_{V3}. X also expresses the threshold for the stretched reflex, but in a manner less dependent on soft tissue length (X_{V1}) than X_{V3}. X and Y correlate with the level of spastic co-contraction during phasic active movements in the hemiparetic upper limb.[25] The three parameters X_{V3}, X and Y are sensitive to local chemical blocks (using botulinum toxin or alcohol compounds).[17]

Step-3: Active range of motion

For each passive movement evaluated, the clinician asks the patient to perform an active movement against the muscle group evaluated, as far as possible along the range until the active force produced by the agonist is balanced by the combination of passive resistance and spastic co-contraction coming from the stretched antagonist. The maximal range of active motion (X_A) is thus obtained. We then define the Paresis Angle Z, which is the difference X_{V1}-X_A. Z expresses the ability for active movement within the available passive range, in a manner less dependent on soft tissue length (X_{V1}) than X_A itself.

Step-4: Rapid alternating movement frequency

The patient performs the same active movement over the maximal range as measured above, then returns to the starting position and again, as many times as possible in a fixed amount of time (*e.g.*, 15 s). The number of maximal amplitude movements performed indicates the ability of the subject to repeat fast active movements despite the likelihood of increased spastic co-contraction as fatigue sets in.[18] The ability to repeat alternating movements is required for most everyday activities (walking, writing, bringing food to mouth, articulating language) and we hypothesize that it may constitute the closest correlate to active function among these first four assessment steps.

Step-5: Active and perceived function

UPPER LIMB

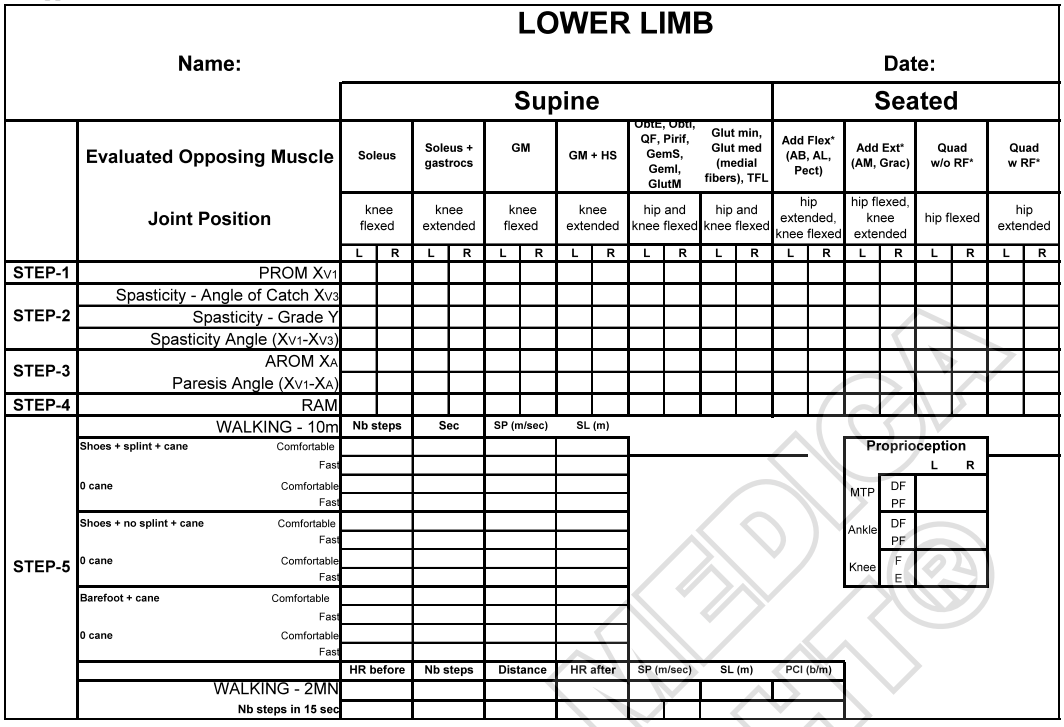
Objective assessments of active function.—Besides pure motor impairment scores such as the Fugl-Meyer that do not test real-life tasks and are time consuming,[26, 27] a large number of scales have been used to evaluate active upper limb function in various formats. Examples of widely used scales include the Frenchay Arm Test, the Rivermead Motor Assessment, the Wolf Motor Function Test, the Jebsen and Taylor test, etc.[28-32] The relevance of these scales for upper limb function may be appreciated by how closely or remotely the requested tasks relate to realistic everyday life activities for the patient population for which they are intended. Other well-known tests such as the Action Research Arm Test, the Box-and-Block, the Nine Hole Peg Test, the Purdue Pegboard Test or the TEMPA *(Test d'Evaluation de la performance des Membres Supérieurs des Personnes Âgées*) rate systematic upper limb activities that can be considered remote from everyday activities, on the line between impairment and function.[33-37] These latter tests can thus be viewed as complementary to direct real-life tasks assessments, as they may allow refining the quantification of hand dexterity, for example according to the size of the object to be grasped.

A tool we consider both simple and close to testing real-life function is the Frenchay Arm Test (FAT), which was developed to assess seven tasks of everyday living performed under clinician observation by a patient with hemiparesis and rated using a Pass/Fail system. However, the Frenchay Arm Test was characterized by a low sensitivity.[28, 38] With the purpose of improving sensitivity, reliability and validity of this functional upper limb assessment, a modified Frenchay Arm Test, termed the Modified Frenchay Scale (MFS) has been proposed (Appendix 3).[38] The MFS differs from the FAT in three points: 1) three bimanual tasks have been added, to increase their number to 6 among 10 tasks, in an attempt to more realistically reflect the way a hemiparetic patient might function in real life; 2) videotaping of the performance is used to facilitate verification and rater blinding; and 3) the categorical (pass/fail) rating system for each task in the FAT has been transformed into a 10-Interval Visual Analog Rating Scale. We use the MFS to assess real-life upper limb function, particularly in hemiparesis; this tool may be complemented by an impairment test (see above).

Subjective assessments of perceived function.—One may consider that the most important assessment is that of how the patient subjectively feels about his/her evolution or treatment effects. Patient perception can be assessed in a number of ways.

Global functional scales such as the Barthel Index, the Functional Independence Measure or the EDSS subjectively rate the ability to perform tasks without necessarily involving the limbs affected by spastic paresis, as Wade noted.[28, 39-41] Such tools may, therefore, evaluate adaptive strategies learned by patients, using for example non-paretic limbs.

The Disability Assessment Scale (DAS) was then



LOWER LIMB

Name: **Date:**

	Evaluated Opposing Muscle	Supine												Seated							
		Soleus		Soleus + gastrocs		GM		GM + HS		Obtε, Obti, QF, Pirif, GemS, Geml, GlutM		Glut min, Glut med (medial fibers), TFL		Add Flex* (AB, AL, Pect)		Add Ext* (AM, Grac)		Quad w/o RF*		Quad w RF*	
	Joint Position	knee flexed		knee extended		knee flexed		knee extended		hip and knee flexed		hip and knee flexed		hip extended, knee flexed		hip flexed, knee extended		hip flexed		hip extended	
		L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R
STEP-1	PROM X_{V1}																				
STEP-2	Spasticity - Angle of Catch X_{V3}																				
	Spasticity - Grade Y																				
	Spasticity Angle (X_{V1}-X_{V3})																				
STEP-3	AROM X_A																				
	Paresis Angle (X_{V1}-X_A)																				
STEP-4	RAM																				

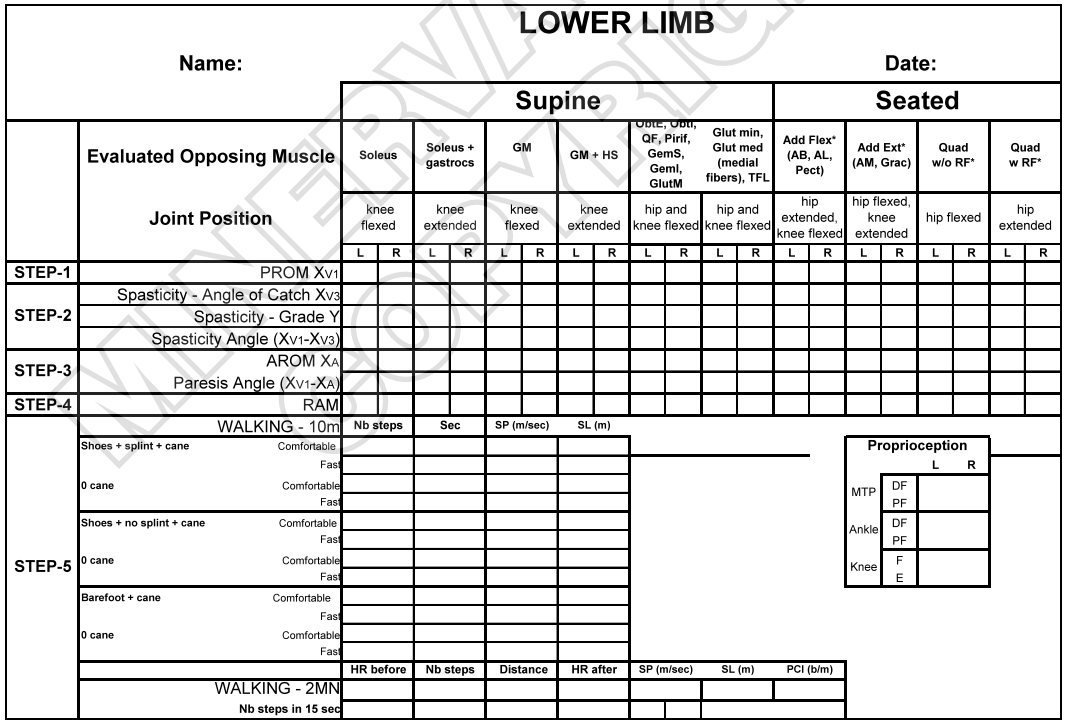
		Nb steps	Sec	SP (m/sec)	SL (m)
STEP-5	WALKING - 10m				
	Shoes + splint + cane — Comfortable				
	Fast				
	0 cane — Comfortable				
	Fast				
	Shoes + no splint + cane — Comfortable				
	Fast				
	0 cane — Comfortable				
	Fast				
	Barefoot + cane — Comfortable				
	Fast				
	0 cane — Comfortable				
	Fast				

	HR before	Nb steps	Distance	HR after	SP (m/sec)	SL (m)	PCI (b/m)
WALKING - 2MN							
Nb steps in 15 sec							

Proprioception

		L	R
MTP	DF		
	PF		
Ankle	DF		
	PF		
Knee	F		
	E		

Appendix 1.



LOWER LIMB

Name: **Date:**

	Evaluated Opposing Muscle	Supine												Seated							
		Soleus		Soleus + gastrocs		GM		GM + HS		Obtε, Obti, QF, Pirif, GemS, Geml, GlutM		Glut min, Glut med (medial fibers), TFL		Add Flex* (AB, AL, Pect)		Add Ext* (AM, Grac)		Quad w/o RF*		Quad w RF*	
	Joint Position	knee flexed		knee extended		knee flexed		knee extended		hip and knee flexed		hip and knee flexed		hip extended, knee flexed		hip flexed, knee extended		hip flexed		hip extended	
		L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R
STEP-1	PROM X_{V1}																				
STEP-2	Spasticity - Angle of Catch X_{V3}																				
	Spasticity - Grade Y																				
	Spasticity Angle (X_{V1}-X_{V3})																				
STEP-3	AROM X_A																				
	Paresis Angle (X_{V1}-X_A)																				
STEP-4	RAM																				

		Nb steps	Sec	SP (m/sec)	SL (m)
STEP-5	WALKING - 10m				
	Shoes + splint + cane — Comfortable				
	Fast				
	0 cane — Comfortable				
	Fast				
	Shoes + no splint + cane — Comfortable				
	Fast				
	0 cane — Comfortable				
	Fast				
	Barefoot + cane — Comfortable				
	Fast				
	0 cane — Comfortable				
	Fast				

	HR before	Nb steps	Distance	HR after	SP (m/sec)	SL (m)	PCI (b/m)
WALKING - 2MN							
Nb steps in 15 sec							

Proprioception

		L	R
MTP	DF		
	PF		
Ankle	DF		
	PF		
Knee	F		
	E		

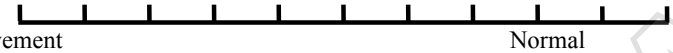
Appendix 2.

Modified Frenchay Scale (MFS)

1. Open and close jam jar using both hands (affected hand holds jar)

No movement Normal

2. Rule line with ruler using both hands (affected hand holds ruler)



No movement Normal

3. Pick up and release big bottle using affected hand

No movement Normal

4. Pick up and release small bottle using affected hand

No movement Normal

5. Pick up glass using affected hand and bring to mouth

No movement Normal

6. Clip 3 clothe-pins on paperpad edge using both hands (unaffected hand holds pad)

No movement Normal

7. Pick up comb and mimic combing using affected hand

No movement Normal

8. Put toothpaste on toothbrush using both hands (affected hand holds tube)

No movement Normal

9. Pick up knife and fork using both hands and mimic cutting on paper pad

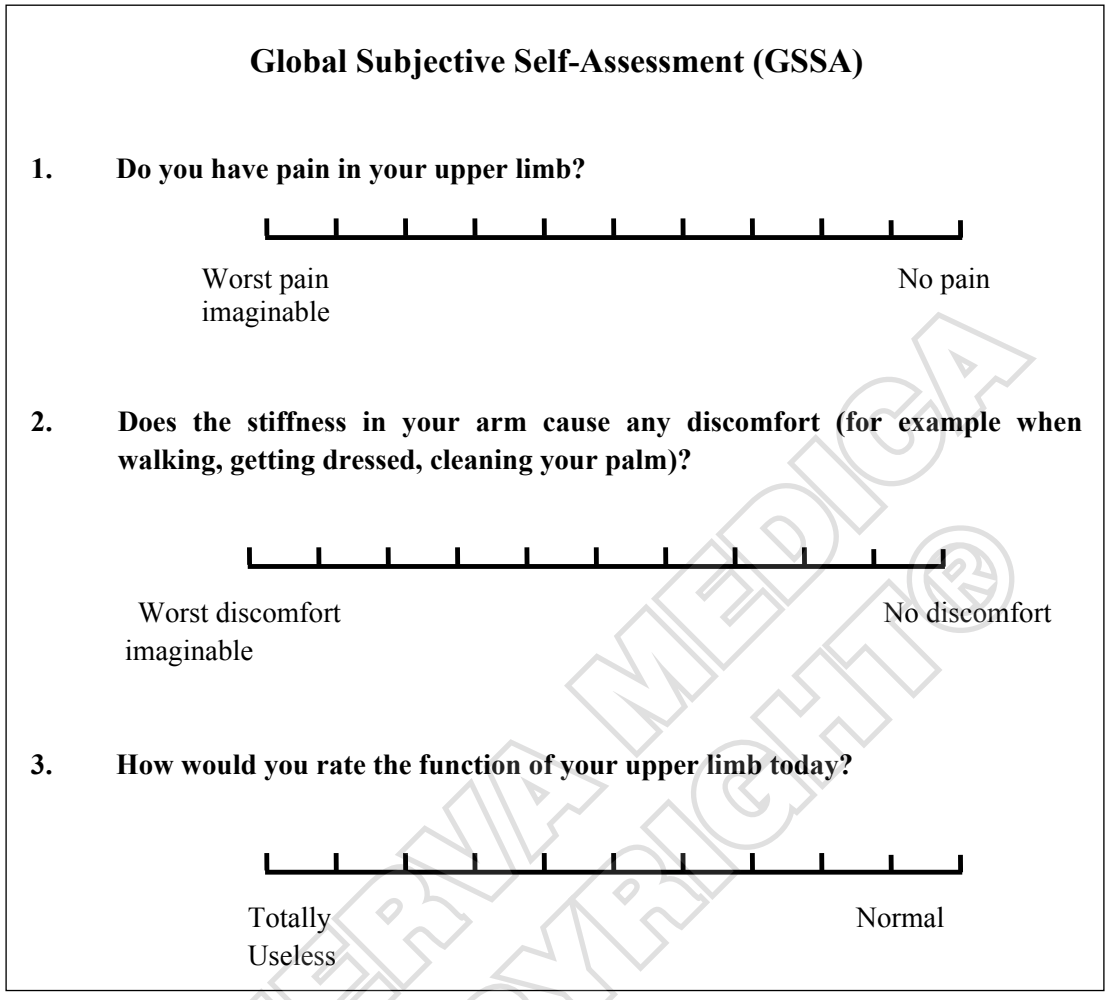
No movement Normal

10. Sweep floor with broom using both hands

No movement Normal

Note: For each task, the score 5 is used to rate a task barely accomplished.

Appendix 3.



Appendix 4.

developed to specifically evaluate function in the hemiparetic upper limb, as perceived by the patient in four pre-defined domains: limb positioning, hygiene, dressing, and pain.[42] This scale was successfully used in a recent study to demonstrate the superiority of focal treatment over a general treatment with a synaptic depressor to improve upper limb perceived function in hemiparesis.[43]

The Goal Attainment Scaling strategy is an assessment of the result of a therapeutic intervention, proposed to patients, caregivers or investigators, based on stated goals of the treatment, previously agreed upon with the patient.[44] An alternative strategy is to precisely avoid considering pre-defined goals in the assessment, as results of a therapeutic intervention often cover an unexpected scope to go beyond the specific goals that had been assumed by patient and doctor or therapist. We therefore proposed the Global Subjective Self-Assessment, in which the patient gives an answer on a Visual Analog Scale to deliberately open questions on three domains, pain, stiffness and active function in the treated limb (Appendix 4).[38]

Lower limb

Objective assessments of active function – Walking speed tests (Appendix 2).—The main function of the lower limb is walking. Walking tests are often performed over 10 meters, or in 2 mn or 6 mn endur-

ance tests. The value and validity of the 10-m walking speed as a tool to measure active lower limb function in spastic paresis are now well recognized. Walking speed correlates with most kinematic gait parameters in hemiparesis.[45] Walking speed is characterized by high inter-rater reliability when measured using a stopwatch and high concurrent validity when compared with infrared timing gate measurement procedures.[46] Walking speed and step length have excellent test-retest reliability at one week in chronic hemiparesis.[47] Comfortable 10-m walking speed has good ecological validity *vs.* walking in natural environments.[48] However, the clinician must be aware that simple extrapolation of comfortable walking speed over a short distance may overestimate the distance walked in 6 minutes.[49]

During a walking test, step length and cadence may also be measured as well as the physiological cost index, which is the speed divided by the difference between the heart rate before and after the effort.[50] The quality of the movement is difficult to assess clinically and may be completed by instrumental analysis (notably kinematic analysis) especially when surgery is considered. Tests of stair climbing performance and uneven ground walking may also be highly useful, particularly to reveal or specify qualitative gait abnormalities that can be partially masked on an "easy" flat ground.[51, 52]

Subjective assessments of perceived function.—Clinical functional scales such as the Functional Ambulation Classification or the SIP68 mobility subscale aim at evaluating how independent ambulation is in daily life. These tools are characterized by good validity in general but may not have high sensitivity to therapeutic interventions over a short period.[53, 54]

Other evaluations

Quantified proprioception.—A goniometric measurement of the threshold to detect slow movements may be performed at various joints, from distal to proximal in the upper and lower limb, in each direction from the spontaneous joint position (Appendix 2).

Combined soft tissue contracture and spastic dystonia.—Tools such as the Ashworth and the Modified Ashworth scales have been widely used in clinical trials under the initial assumption that they measured spasticity.[55, 56] These instruments actually rate a combination of soft tissue contracture, spastic dystonia, and spasticity.[21, 57]

Pain.—A number of studies indicate that pain is not a primary issue in chronic hemiparesis, compared to other domains such as active function, hygiene, cosmesis.[42, 43] However, pain may be both a cause and a consequence of muscle overactivity and become a significant issue in some cases. The Disability Assessment Scale and the Global Subjective Self-Assessment include pain in their evaluation.[38, 42] Otherwise, pain may be assessed non specifically using Visual Analog Scales.

Conclusions

We propose that the five-step clinical assessment of the antagonistic capacity of each muscle group is worth its time-consumptionmay have therapeutic value:

– when Step-1 suggests unacceptable shortening/dystonia, this may allow the clinician to focus treatment on lengthening interventions and blocking injections on the evaluated muscle group;

– when Step-3 indicates a large paresis angle - or when Step-4 shows unexpected alternating movements slowness despite a small paresis angle – with acceptable passive range of motion at Step 1, this may allow the clinician to focus treatment on agonist strengthening and blocking injections on the evaluated muscle group;

– Step-2 is used to measure treatment-induced changes in Spasticity Angle, or in the catch angle, which may serve as an indicator of how well a muscle was blocked by a focal injection.

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