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Neurophysiological methods for the assessment of spasticity: The Hoffmann reflex, the tendon reflex, and the stretch reflex

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Abstract
Purpose: To review the literature concerning neurophysiological methods to assess spasticity with respect to mechanisms and methodology, and to describe the three most commonly used methods: the Hoffmann reflex (H-reflex), the Tendon reflex (T-reflex), and the Stretch Reflex (SR).
Method: A systematic internet database search was performed to identify neurophysiological measurement methods of spasticity. A systematic exclusion procedure resulted in 185 included references, completed by additional informal search. For this paper, information about the H-, T- and stretch reflexes was extracted from these references.
Results: Although the reflexes are basically monosynaptic, there are many supraspinal pathways which modulate the responses in terms of their amplitude and latency. As a consequence the methods are sensitive to a considerable number of experimental conditions and are characterized by a moderate reliability and sensitivity. Correlations with other (i.e. biomechanical, neurophysiological or clinical) spasticity assessment parameters are moderate to poor. Standardised and broadly accepted protocols are still largely lacking preventing an effective exchange of knowledge.
Conclusions: The clinical and experimental use of the three methods is restricted due to moderate reliability and sensitivity. It is recommended to perform combined neurophysiological – biomechanical assessment of spasticity during active, functional movement.

Keywords: *Neurophysiology, phasic stretch reflex, spasticity, assessment*

Introduction

Spasticity is one of the most frequently observed phenomena after a lesion of the upper motor neuron system. Although there has been considerable discussion about its definition, spasticity is commonly agreed to be 'a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from the hyperexcitability of the stretch reflex, as one component of the upper motor neurone syndrome' [1]. Spasticity affects muscles and joints and is responsible for deformity, pain, and abnormal movement. The incidence of spasticity is not exactly known, but it likely affects over half a million people in the United States and over 12 million people all over the world [2]. Studies indicate that about 36% [3] to 38% [4] of the stroke patients develop spasticity during the first year, however another study reported only 19% of the stroke patients developed spasticity after three months [5]. The large number of patients affected, spasticity related disabilities and the subsequent costs for health care emphasize the need for accurate measurement and treatment of spasticity.

Reliable and valid spasticity assessment is indispensable for clinical and experimental purposes. In clinical practice, patients benefit from an accurate assessment since it allows medication to be individually optimized, resulting in a more meticulous decrease of negative side effects of spasticity like pain, contractures, or severe movement limitations. In research environments, proper spasticity assessment provides the opportunity of high quality

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spasticity research, the evaluation of therapeutic interventions, dose-effect studies, and so on.

Various methods have been proposed in literature for the assessment of spasticity of clinical, biomechanical, as well as neurophysiological origin. This paper will focus on the neurophysiological methods, which aim at the assessment of the electrical manifestations of the (ab)normal motor control of movements. A commonly used technique for this purpose is electromyography (EMG) with surface electrodes. EMG signals are the electrical signals which precede the mechanical activity of skeletal muscles from which several parameters can be derived to provide information about anatomical and physiological muscle properties and neuromuscular control [6]. As such, it can provide useful clinical and scientific information. Neurophysiological methods are often employed to evaluate the effects of treatment on spasticity or, more fundamentally, to investigate the different pathways involved in spasticity. However, up to now a good overview of protocol recommendations as well as the pros and cons of the different methods, methodological quality, and the quality of these methods are largely lacking, or at least unsurveyable.

This study was performed within the framework of the SPASM project (Support Programme for the Assembly of database for Spasticity Measurement) that aimed at systematically reviewing the literature on methods of measuring spasticity to identify best practice and direction for future developments. Aim of this review was to describe the methods to assess spasticity with the mechanism of the response, a detailed overview of all relevant factors that determine the properties of the response, and the behaviour of the response in spasticity. This paper gives a short overview of the results of the review process and describes three often applied neurophysiological methods in detail: the Hoffmann-reflex (H-reflex), the Tendon-reflex (T-reflex), and the Stretch reflex (SR). These methods have in common that they all describe the phasic stretch reflex although their method of evocation is different. The other neurophysiological methods will be described in a forthcoming book, Deliverable 410 of the SPASM project.

Methods

Database search and keyword definition

The first step of the literature review was aimed at obtaining literature references of all methods for the assessment of spasticity. A general search was performed several times until November 2002 in the following seven databases: Medline, Pubmed, CINAHL, EMBASE, Web of Science, Science direct, and First Search, with keywords:

1. *spas**
2. *hyperton**
3. *reflex**
4. *measur** or *assess**
5. *UMN or upper motor neuron*

The first three keywords were used separately in combination with the fourth and the fifth. This search resulted in 3793 hits. The papers were clinically, neurophysiologically, and/or biomechanically oriented in spasticity assessment.

A second search strategy was needed to filter out those references that were only neurophysiologically/electrophysiologically oriented. For this purpose, three additional sets of keywords were defined in three different areas, i.e. neuro- and/or electrophysiological methods, methodological quality of methods, and finally a direct search of the methods. Objective of the first strategy was to obtain an overview of all neurophysiological methods that have been used in literature to assess spasticity, hypertonia, or reflex excitability. Central keywords were *neurophys** or *electrophys**, in combination with *measur** or *assess**, and *spas** or *hyperton** or *reflex**, resulting in 66 hits. The second search, directed at identifying articles providing information about the methodological quality of the methods used in the assessment of spasticity, was performed with *reliab** or *sensitiv** or *valid** as keywords. Furthermore, these were combined with *neurophys** or *electrophys**, and *measur** or *assess**, leading to 22 references. The last category of keywords was defined in order to obtain information directly about the neurophysiological methods for the assessment of spasticity that might have been missed with the previous two searches. Therefore, *EMG, T-reflex* or *Tendon* and *reflex*, *Stretch* and *Reflex, TMS* or *Transcranial*, or *Motor* or *Sensory* and *Evoked* and *Potential, Flexor* and *Reflex, Pendulum* or *Wartenberg,* or *Range* and *Motion* were used as keywords. To ensure the relation with spasticity, hypertonia, or reflex excitability these words were used in combination with *spas** or *hyperton** or *reflex**, resulting in 474 references. From this selection case- and animal studies were removed. Articles written in other languages than English, Dutch, French, German, Italian, Slovenian, or Spanish were also excluded from the review. Four hundred and thirty-seven records remained.

Finally a Medline – Pubmed search was performed to identify papers in which the H-reflex and the F-wave were used for spasticity quantification. This search was performed separately from the one described above due to organizational reasons, but

the purposes were identical. For the H-reflex, keywords used were:

1. *H-reflex*
2. *reliab** or *valid** or *sensitiv**
3. *measure** or *assess**
4. *spas** or *hyperton**

The first two keywords were used in combination with the 3rd and 4th groups of keywords. The search resulted in 98 hits, of which animal studies, case studies, as well as references that were written in languages other than English, Dutch, French, German, Italian, Slovenian, or Spanish, were excluded. Based on their abstracts, 66 references were included.

For the F-wave, the search was considerably similar to that of the H-reflex. Keywords were:

1. *F-wave*
2. *reliab** or *valid** or *sensitiv**
3. *measure** or *assess**
4. *spas** or *hyperton** or *reflex**

The first two keywords were used in combination with the 3rd and 4th groups of keywords. Altogether, the F-wave search resulted in 132 records. Animal studies, case studies, as well as references that were written in languages other than English, Dutch, French, German, Italian, Slovenian, or Spanish, were excluded. Ninety-eight articles remained and were included for the F-wave review, based on their abstracts.

The results of these searches were combined in one database, which ultimately consisted of 638 records. Each reference was then read by two reviewers who judged whether it should be in- or excluded from the review. References excluded from the database were:

- duplicates;
- studies in which neurophysiological parameters were not the main outcome measures; and
- studies that did not concern spasticity measurement *and* that were not reviewing the method, *or* giving information about normal values or methodological aspects of a method.

This exclusion procedure eventuated in the inclusion of 185 references. The list was complemented with references found by extensive additional hand search of journals, books, and communication with experts.

Review procedure and strategies

Based on full-text references, the following relevant data from each selected reference were stored in an Access database:

(1) study design,
(2) demographic variables of subject population, including in- and exclusion criteria,
(3) a description of the method(s) and instruments used, including a specification of limb/muscle(s) to which the method has been applied as well as information about placement of instruments and subjects and the availability of a measurement protocol,
(4) whether or not an intervention was evaluated, and, if yes, which intervention was evaluated,
(5) information about methodological quality of the study and the method used (if available),
(6) results of the study (outcome measures and outcome values, especially with regard to data concerning reliability, validity, and sensitivity),
(7) practical information about the method (advantages and disadvantages, time and cost issues, if reported),
(8) conclusion with regard to quality of the method, and
(9) other relevant notes about the study.

This database ensured systematic, extensive data storage of each selected reference as well as the possibility for an easy-reference data exchange. The structure of the database also enabled easy data extraction per method.

Results

Nine different neurophysiological methods for spasticity assessment were found in literature. These methods were classified in four subgroups, i.e.

1. EMG responses to electrical stimuli
   - Hoffmann reflex (H-reflex)
   - F-wave
2. EMG responses to mechanical stimuli
   - Tendon reflex (T-reflex)
   - Polysynaptic responses
3. EMG responses during passive and active movements
   - EMG recordings during passive movements
     - Stretch reflex (SR)
     - Wartenberg Pendulum test
   - EMG recordings during active movements
4. Evoked Potentials.
   - Motor Evoked Potentials: Transcranial Magnetic Stimulation
   - Sensory Evoked Potentials: Lumbosacral Potentials

In this paper, the focus will be on three methods for spasticity assessment, i.e. the Hoffmann reflex (usually abbreviated as H-reflex), the Tendon reflex (usually referred to as T-reflex), and the Stretch

Reflex (SR). These methods have in common that they all describe a method of short-latency, or phasic, stretch reflex measurement.

1. The Hoffmann reflex

1.1 Mechanism of the Hoffmann-reflex. The Hoffmann reflex (H-reflex) is a low threshold, spinal reflex that can be elicited by electrical stimulation of a mixed peripheral nerve [7]. It was first shown by Piper in 1912 [8] and described in more detail by Hoffmann in 1918 [9], who studied the response in the triceps surae muscles. Since then, the reflex has been subject to several hundreds of studies [10], mainly performed at the calf muscles. The reflex is usually considered to be monosynaptic, although there is some evidence that it might be di- or trisynaptic in origin [11].

In Figure 1, a schematic presentation of the neural pathway of the H-reflex is given. Electrical stimulation of a peripheral nerve causes impulses travelling up the sensory afferent fibers (Ia) to the spinal cord via the dorsal root. Here, synaptic connections are made with α-motoneurons that belong to the same muscle. Via these motoneurons located in the ventral horn, these impulses orthodromically travel back to the muscle. A reflex response can be recorded with a delay of several ms: the H-reflex [7,12].

Recently new methods for elicitation of the reflex in the soleus muscle have been documented, for which the reader is referred to [13–16].

During his study, Hoffmann identified a second response due to direct activation of the axons of α-motoneurons. This response is called the M-wave: a direct motor response with a shorter latency compared to the H-reflex (Figure 2).

The M-wave has a higher threshold for excitation compared to the H-reflex [17]. At low stimulus intensities, the H-reflex will occur without the M-wave. When the stimulation level increases, at first the H-reflex amplitude increases and gradually the M-wave occurs. At augmenting stimulus levels, the M-wave continues to increase and the H-reflex fades away, because the stimulation blocks the reflex by producing antidromic motor depolarisation of the α-motoneuron [18]. Another explanation given in literature for this gradually dissipating response is increasing Renshaw cell inhibition with augmenting stimulus intensities [7]. When the M-wave reaches its peak, no H-reflex can be detected in the EMG [17]. The course of the H-reflex amplitude and the amplitude of the M-response at different stimulation intensities is often presented in a recruitment curve (see Figure 3).

The H-reflex is dome shaped and the M-wave has an S-shape. To be accepted as an H-reflex, a response needs to fulfil three qualifications: (1) the response can be obtained without M-response or with a very small M-response, (2) the amplitude should decrease with increasing stimulus frequency, and (3) more proximal stimulation should cause a shortened latency [19].

The H-reflex is usually expressed in the parameters latency and amplitude. The latency is the time that elapses between the stimulus (i.e. electrical) and the first deflection in the recorded signal. It represents the sum of the conduction time of the afferent and efferent impulses, plus the time for the synapse transmission in the spinal cord [20] and is best measured to the inception of the response, either positive or negative [7]. The amplitude is calculated

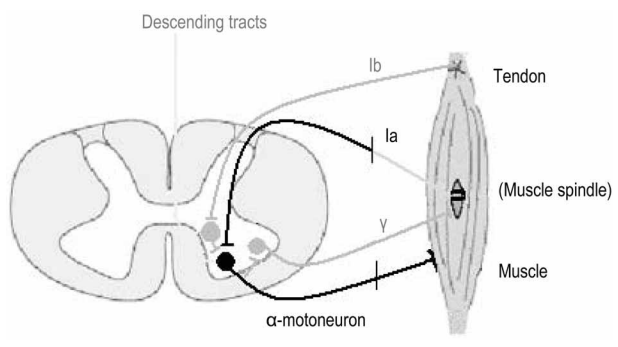
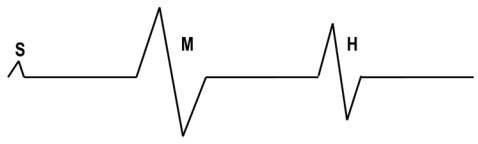
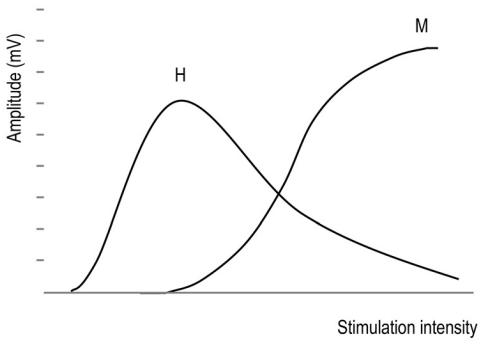


Figure 1. Neural pathway of the H-reflex (black lines). The vertical, dotted line marks stimulation site.



EMG signal
S = Stimulus
M = M-wave
H = H-reflex

Figure 2. EMG signal. S, stimulus; M, M-wave; H, H-reflex.



Recruitment curve
H = H-reflex
M = M-wave

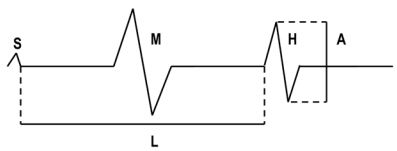
Figure 3. Recruitment curve. H, H-reflex; M, M-wave.

as the difference in mV between the positive and negative peak of the EMG signal [7] (see Figure 4).

Due to its predominantly monosynaptic character the H-reflex is clinically as well as experimentally relevant in spasticity measurement [10,21–26]. It provides information about changes in inhibition and excitability of the motoneuron pool [27], due to segmental and/or supraspinal influences (e.g. upper motor neuron lesions). These alterations can be induced by therapeutic interventions (e.g [28–35]) applied to spastic subjects. The H-reflex can also be used to study segmental and/or supraspinal influences during gait.

1.2 Methodological considerations. The factors listed below have been described in literature as affecting H-reflex measurement outcome. These factors should be kept in mind when preparing a protocol, when performing H-reflex measurement, or when analysing data.

1.2.1 The position of the subject. Changing body position implies a change in the constitution of the afferent volley because of input of muscle receptors, skin receptors, and joint receptors [36]. When measuring the H-reflex in the soleus muscle, the subject can be placed in a prone, (semi) supine, sideways, sitting or standing position. Most studies are performed with subjects in a (semi) supine position [21,22,37]. Ali and Sabbahi [25,38] compared the soleus H-reflex amplitude under several different positions in healthy participants and their results showed that reflex measurements in the soleus muscle should best be performed with the subject in (loaded) standing position or supine instead of prone. According to Hopkins and colleagues [24], measurements during both one-legged standing as well as supine position deliver reliable information about H-reflex amplitude, as long as the same protocol was used as described in that study. After comparing the H-reflex parameters during standing and sitting, Al-Jawayed *et al.* [39] concluded that subjects should best be measured in their most comfortable position because in this study no differences in H-reflex responses between lying and sitting were found.



H-reflex parameters amplitude (A) and latency (L)
S = stimulus
M = M-wave
H = H-reflex

Figure 4. H-reflex parameters amplitude (A) and latency (L). S, stimulus; M, M-wave; H, H-reflex.

The increased reliability measured during standing can be ascribed to spinal reflexes being more engaged in closed loop activity as compared to a lying position, resulting in more reliable measurements [25]. Other explanations are increased cortical control during standing compared to lying, and the larger amount of peripheral feedback during standing positions [25]. However, the size of the amplitude is reduced when measured in standing position [36,40,41] or more vertical positions of the body [42,43], but this does not account for the H-reflex latency [25].

Handcock *et al.* [44] investigated the H-reflex amplitude in quiet standing. They found a high intra-individual reliability and recommended to improve reproducibility of the measurements during a standing position by placing subjects on two digital scales to ensure that body weight is distributed evenly between the two legs.

Little is known about factors concerning subject positioning that may influence excitability when measuring the H-reflex in the upper extremity. Most commonly, measurements are performed with the subject in a sitting position [23], but also other positions have been used, e.g. Katz *et al.* [45] who performed H-reflex measurements in the upper extremity with subjects in a supine position.

1.2.2 The position of the head. The position of the head can modulate motoneuron excitability due to vestibular and neck receptor input [46,47]. Traccis *et al.* [48] who studied the influence of the position of the head on soleus motoneuron excitability, showed that the amplitude of the H-reflex increases for contralateral rotation with regard to the recording site and inhibited for ipsilateral rotation. Therefore, the position of the head should not be changed during measurement. In lying positions, the head can be strapped to a table with elastic bands to prevent movement of the head compared to the trunk. When H-reflex values as found in several studies are compared, the position of the head should be taken into consideration.

1.2.3 The position of the limbs. Soleus H-reflex measurements can be performed with the knee or ankle in flexion or extension. This limb position can influence the excitability of a motoneuron pool, but also the distance between cathode and nerve may change. For example, in healthy subjects a dorsiflexed ankle [49,50] or stretched triceps inhibits the motoneuron pool of the soleus muscle [51]. There are many different limb positions used in literature, making it hard to compare results. For reliable outcome data of a study, it is of great importance that for all subjects the position of the limb is identical.

Most H-reflex measurements in the upper extremity are performed in a sitting position, with the arm maintained in 45° of shoulder abduction and the elbow extended to within 30° of maximum (see e.g. [23]). The position of the forearm seems to be determinative for the size of the H-reflex as shown by a study of Baldissera *et al.* [52]. The H-reflex size in the flexor carpi radialis muscle was studied with the forearm in pronation, supination, and three intermediary prone-supine positions. H-reflex amplitude when measured in supination showed a reduction of about 50% compared to when measured prone [52].

1.2.4 Sensory input. Sensory input influences the excitability of the H-reflex and should therefore be kept as constant as possible. Similar to the results from Hoffmann and Koceja [53], the study of Kameyama *et al.* [54] learned that the H-reflex amplitude decreases when performed in subjects with closed eyes. When measurements are performed with eyes opened, they need to be fixed at one point in front of the subject [25]. Excitability of postural muscles is changed due to activation of the reticulospinal tract by means of an auditory stimulus [55]. A change can also be induced by activation of the vestibulo-spinal tract [56].

1.2.5 Stimulus duration and frequency. The relative recruitment of motor and sensory fibers depends on the duration of the applied electrical stimulus [57]. Stimulus pulses can be applied with different stimulus durations. In order to investigate the optimal stimulus duration, Panizza *et al.* [58] recorded the H-reflex in 10 healthy volunteers with stimulus durations varying from 0.1 to 3 ms. Results showed that stimulation shorter than 0.5 ms was not optimal because the M-wave was often activated before or simultaneously with the H-reflex. However, the duration should be relatively short, 0.5 – 1 ms, in order to activate the large sensory fibers [58].

Stimulus frequency should be 0.2 Hz to prevent effects related to the previous stimulus that can distort results [58]. Other authors recommend a frequency of one per three seconds [26]. In healthy subjects, the H-reflex size gradually falls when series of identical stimuli are applied with frequencies between 1 and 10 Hz [60]. This is called 'low-frequency depression' that mainly affects the H-reflex evoked at lower intensities of stimulation [61].

Mezzarane and Kohn [62] investigated the asymmetry in reflex depression outcome parameters between left and right leg (evoked at the same time) in 10 healthy subjects. The results indicated that there is no asymmetry in H-reflex amplitude depression of both legs with regard to mean, coefficient of variance, or time constants [62]. The depression is abnormal in patients with chronic spasticity due to spinal cord injury [63 – 65] compared to healthy subjects and acutely injured subjects [65]. This decrease in low-frequency depression is considerable at 1, 5, and 10 Hz, and absent at 0.2 Hz [65]. The suppression may be due to a decreased presynaptic inhibition caused by loss of supraspinal control [64,65] and interneuronal cells [66], or a decreased homosynaptic depression [65].

Apart from stimulus duration, the duration of the experiment also influences the amplitude of the maximal M-wave and the maximal H-reflex amplitude. After evocation of the H-reflex for about 2 h, Harburn *et al.* [67] found a slightly increased H-reflex amplitude supposed to be related to changed skin impedance. In contrast, Crone *et al.* [68] found a 20% reduced maximal H-reflex amplitude and maximal M-wave amplitude of the soleus muscle after one hour of stimulation. Based on this it can be concluded that the amplitudes of the M-wave and H-reflex cannot be held constant during a long-term stimulation of a peripheral nerve [68], and that the duration of the experiment should be restricted.

1.2.6 Post-activation depression. In healthy subjects, repetitive stimulation, slow passive stretch of the muscle, vibration applied to the Achilles tendon, or voluntary contraction results in a decreased H-reflex of the soleus muscle [69]. This is called post-activation depression, only seen in the homonymous stretched or activated muscle, which is decreased in spastic patients [12,69,70]. It indicates a depression caused by previous activity in the homonymous Ia afferents [69,71], and lasts for several seconds [69]. It is seen for reflexes evoked in activated muscles at intervals longer than 300 – 400 ms after the conditioning stimuli [70].

The mechanism of this post-activation depression is mainly unclear. Several studies have been conducted to discover the principles and pathways of this depression. It has been suggested that both fast as well as slow mechanisms are involved. Voigt and Sinkjær [72] performed a study with both healthy and complete spinal cord injured subjects, from whom the ankles were passively rotated at 0.5 Hz. There was a fast mechanism of depression (400 – 500 ms), which seemed to be caused by presynaptic inhibition. The slow mechanism (> 2 s, sometimes still found after even more than 8 s) might be due to neurotransmitter depletion, although the exact mechanism has remained unclear so far [72]. Post-activation depression is reduced in complete spinal cord injured subjects [72] and spastic subjects [70,73,74] what possibly prevents from clonus [75]. By measuring the H-reflex, inter-stimulus time intervals should be long enough to overcome this depression of amplitude size.

Maximal muscle contraction for over 30 s and the subsequent measurement of H-reflex, M-wave, and reciprocal inhibition, revealed a decreased reciprocal inhibition when the ipsilateral arm muscles had been contracted but not when the contralateral muscles were contracted or activated by tetanic stimulation. The H-reflex seemed to be changed within the first 15 s after contraction but remained unchanged after a longer period post-contraction [76]. Also, after maximal voluntary contraction has ended for about 45 s, the M-wave appeared to be unchanged as well [76]. Thus, on one hand, this post-contraction requires careful control during measurements, on the other hand post-contraction measurements can provide valuable information about long-lasting changes in reciprocal inhibition [76].

1.2.7 Electrode location. The location of the recording electrodes is of great importance for the amplitude of the H-reflex as well as reproducible H-reflex testing as was shown by several authors [8,77,79]. Although the location of the electrodes differs from study to study, the standard location for the recording electrodes according to Fisher [7,26] is medial to the tibial one-half the distance between the stimulation site and the medial malleolus with the indifferent electrode at the Achilles tendon for the measurement of the H-reflex in the soleus muscle. Maryniak and Yaworski [80] investigated 31 recording locations at the calf musculature and they found the highest response at a more distal site than the conventional midcalf recording location [80]. At this more distal site, in all 20 subjects a triphasic waveform was found. The H-reflex latency at this point is about 1.4 ms longer compared to the midcalf location. They concluded that bipolar distal recording of the H-reflex is preferred over the conventional midcalf location. In a study of Morelli *et al.* [81] triceps surae H-reflexes obtained from mid (soleus muscle) and distal (musculotendinous junction) recording sites were compared. They concluded that the distal site is an acceptable alternative. Little *et al.* [79] performed monopolar surface EMG measurements and recommended a position of electrodes 3 cm proximal and 6 cm more distal than the standard recording site, when the ankle is fixed at 90° [79]. In the European Seniam project extensive research was performed for optimal electrode locations for surface EMG recordings [82]. It was concluded that bipolar electrodes with 2 cm inter-electrode distance is optimal for most large muscles. The electrodes should preferably be placed between the motor end plate zone and the distal tendon to be least sensitive for small electrode dislocations. Based on this, specific recommendations have been developed for 31 different muscles [83].

In literature, the insensitivity of the H_{max}/M_{max} ratio for changes in electrode location has been confirmed [84] as well as denied, since Little *et al.* [79] registered an increased ratio with more distal (to the m. gastrocnemius belly) recording sites.

For H-reflex measurements with surface electrodes at the upper extremity, the standard location is at one-third of the distance from the medial epicondyle to the radial styloid [7,23,26].

1.2.8 Number of measurements. Because of all factors that may influence H-reflex measurement outcome, there is uncertainty about the number of measurements that need to be performed for reliable outcome. In the 1970s and 1980s, Hugon [85] and McIllroy and Brooke [86] recommended that at least 10 trials should be performed and averaged to obtain reliable values. Nowadays, 5 ($r = 0.862$) or 4 ($r = 0.96$) measurements are recommended by, respectively, Hopkins *et al.* [24] and Handcock *et al.* [44], when measurements are performed in a standing position. The high intra-class reliability within a test-session of the H-reflex amplitude in standing subjects was later confirmed by Earles *et al.* [87].

1.2.9 Muscle background activity. The H-reflex is affected by the level of muscular relaxation, or baseline EMG [22], especially when the amplitude is small. The amplitude of the H-reflex is positively correlated with the increase in baseline EMG activity or muscle tension [88]. Muscle background activity may be used in order to reinforce the H-reflex amplitude, to increase the integrity of the H-reflex, or to decrease the effects of habituation [89]. Reflex recording in spastic subjects under remained contraction showed a relatively smaller decrease in H-reflex latency with increasing contraction levels compared to healthy controls [90]. Also during ramp contraction, H-reflex modulation was considerably smaller compared to healthy controls [90]. The H-reflex latency is not susceptible to muscle contraction, in contrast to the Tendon-reflex latency [91].

1.2.10 Other factors important in H-reflex measurement. There are several factors that may also influence H-reflex measurement outcome. One of these factors is age, which has found to be negatively correlated with the H-reflex amplitude [10,22,92]. De Vries *et al.* [93] compared the H-reflex amplitudes between older and younger, healthy subjects, and concluded that the amplitudes were 32.7% smaller in the old. Besides this, Falco [94] concluded from their study that ageing increases the between-leg variability of the H-reflex latency. Older adults also show a longer recovery profile [92].

Other demographic variables that can affect the H-reflex are length of the arm or leg, and height [7,10,91,94,95]. The relation between age and H_{max}/M_{max} ratio is confusing. Several authors found a declined ratio [96,97] while others reported a declined M-wave amplitude but an identical H_{max}/M_{max} ratio [93] or no change in ratio at all [22]. Like age, these factors influence the latency of the H-response. When analysing measurements these factors should be taken into consideration.

The amplitude of the response can be reinforced by remote facilitation as is done for example with voluntary teeth clenching. Sugawara and Kasai [98] studied the effects of voluntary teeth clenching on a cortical and spinal level, in the m. flexor carpi radialis of 13 healthy subjects. Results showed that the facilitation resulted in increased amplitude of the H-reflex, but the latency did not significantly diminish [98]. The H-reflex amplitude of the soleus muscle is also increased due to voluntary teeth clenching [99,100]. Those manoeuvres result in a considerable facilitation of the corticospinal pathways of muscles in the hand and leg [100].

Finally, the H-reflex depends on several other factors like input from cutaneous afferents ([101], for review see [47]), and physical and mental conditions such as the state of awareness [47,102], task complexity [53], galvanic vestibular stimulation [103], mood, and infections. Several studies have shown that the H-reflex does not change under experimentally induced (muscle) pain-conditions [104 – 107].

1.3 The H-reflex in spasticity. Disturbances in several control mechanisms, or pathways, are responsible for spasticity [12], such as α-motoneuron excitability, presynaptic inhibition, reciprocal inhibition, recurrent inhibition, and polysynaptic changes. The H-reflex can be used to study some of these pathways. Several outcome values of soleus H-reflex parameters are presented in Table I.

1.3.1 α-Motoneuron excitability. Different parameters of the H-reflex indicate the excitability of the α-motoneuron pool. The most important parameters are described below.

1.3.1.1 Latency. The latency of the H-reflex reflects the excitability of the α-motoneuron pool. The m. soleus H-reflex latency can be predicted by using the formula given by Braddom and Johnson [111]: 0.46*distance between stimulation to medial malleolus + 0.1*age. The normal latency of the m. soleus H-reflex is approximately 30 ms in healthy subjects [12,84,86] while the M-wave latency evoked in the soleus is about 5 – 10 ms [17]. In the upper limb, the H-reflex latency is shorter compared to the lower limb: values of 21 ms [26], 16.6 ms [108], and 17.1 ms [109] (muscle slightly contracted) have been given for the flexor carpi radialis.

In spastic subjects, usually a decreased H-reflex latency is found, indicating higher excitability of the motoneuron pool [37], although values that approach normal levels are also found [110]. The m. flexor carpi radialis H-reflex in 53 post-stroke patients was 17.1 ms [108].

Bilateral comparison of H-reflex latencies gives an indication of differences in motoneuron excitability between both limbs by calculating a difference or a ratio, thereby reducing interindividual variability. Mean difference in latency values found in the soleus muscle of healthy adults are usually situated between 0.4 and 0.5 ms. [22,38] with a maximum of 1.5 ms in adults [109] and 1.8 ms in older subjects [94]. In the forearm, upper limit for healthy subject side-to-side latency difference is 1.0 [111] or 1.5 ms [26]. Maximum normal value of interside ratio in the calf muscles is 2 [112] or 3 [22]. In patients with a unilateral paresis or spasticity, the side-to-side latency difference exceeds the upper limits. It should be mentioned here, however, that side-to-side comparison in spastic patients may not be effective since the unaffected side in cerebral lesion patients appeared to be impaired as well, compared to a healthy control group [113]. The comparison then results in an exaggeration of the extent of reflex increase [114].

The H-reflex latency is not correlated to the Modified Ashworth Scale [108], but abnormalities of the H-reflex have shown to be well correlated with changes of the ankle jerk during clinical observations [115].

1.3.1.2 Amplitude. The amplitude of the H-reflex is an expression of α-motoneuron excitability to excitatory inputs from Ia-afferents [36,116]. The amplitude is non-linearly related to motoneuron excitability and is also subject to presynaptic inhibition of Ia afferents [117] (see Section 1.2.3). An increase in amplitude thus reflects an increase in excitability. According to the results of Ali and Sabbahi [38], the amplitude of the H-reflex would be a better parameter for the detection of physiological processes and changes in posture than the latency, although results from other authors do not agree with this statement [118,119]. The H-reflex amplitude is usually significantly increased in the affected side of spastic patients compared to healthy subjects [12,117,120] or the unaffected side [122]. During spinal shock the amplitude is significantly reduced 24 hours after injury [123], but recovers quite soon. This remarkable reduction or absence of the reflex was not confirmed by results from Hiersemenzel *et al.* [124] in the majority of subjects.

Table I. Soleus H-reflex parameters.

Author(s)	*n*	Position subjects	Parameter	Normal latency (ms)	UMN latency (ms)
[38]	20	Prone	*Latency*	mean (l) 29.6 ± 2.5 mean (r) 29.8 ± 2.6	
[38]		Standing	*Latency*	mean (l) 29.7 ± 2.7 mean (r) 29.8 ± 2.6	
[38]		Loaded standing	*Latency*	mean (l) 29.7 ± 2.7 mean (r) 29.8 ± 2.7	
[38]		Unloaded standing	*Latency*	mean (l) 29.9 ± 2.7 mean (r) 30.0 ± 2.7	
[110]	10	Prone	*Latency*	30.1 ± 2.2	
	12	Prone			31.5 ± 1.9
[339]	9	Prone	*Latency*	mean 28.2 ± 1.8	
[339]	9	Supine Sitting Standing	*Latency*	mean 31.2 ± 0.9 mean 31.1 ± 0.6 mean 30.6 ± 0.3	
[10,85,94]	**	**	*Latency*	range 27–35	
[22]	45	Prone	*Latency*	mean 29.6 ± 2.5	
[35]	3	Prone	*Latency*		mean 32.30 ± 1.01
[125]	30	Supine/Prone	*Latency*	mean	
[37]	7	Semi-supine	*Latency*	mean 30.3 ± 1.7	
[37]	10	Semi-supine	*Latency*		mean 28.6 ± 1.6
[80]	20	Prone	*Latency*	range 23.1–31.6 (mid)* range 23.7–33.5 (distal)*	
[197]	30	Prone or semi-reclined	*Latency*	mean 33.3 ± 1.7	
[331]	60	Supine	*Latency*	mean 29.6 ± 2.21 range 26.4–34.0	
[38]	20	Prone	*Side-to-side difference*	0.4 ± 0.34	
[38]		Standing	*Side-to-side difference*	0.5 ± 0.3	
[38]		Loaded standing	*Side-to-side difference*	0.4 ± 0.3	
[38]		Unloaded standing	*Side-to-side difference*	0.5 ± 0.3	
[109]	**	**	*Side-to-side difference*	maximum 1.5	
[10,85,94]	**	**	*Side-to-side difference*	< 1.2 or < 1.4 (younger adults) < 1.8 (elderly)	
[336]	40	Prone	*Side-to-side difference*	0.37 ± 0.28	
[22]	45	Prone	*Side-to-side difference*	mean 0.45 ± 0.40	
[331]	60	Supine	*Side-to-side difference*	mean 0.49 ± 0.36	
[38]	20	Prone	*Amplitude*	mean (l) 4.6 ± 2.2 mean (r) 4.0 ± 2.1	
[38]		Standing	*Amplitude*	mean (l) 3.2 ± 1.9 mean (r) 3.4 ± 2.1	
[38]		Loaded standing	*Amplitude*	mean (l) 3.5 ± 1.9 mean (r) 3.5 ± 1.9	
[38]		Unloaded standing	*Amplitude*	mean (l) 3.2 ± 1.9 mean (r) 3.0 ± 1.9	
[84]	**	**	*Amplitude*	range 5–25	
[120,121]	**	**	*Amplitude*	mean 2.6 ± 2.3	mean 4.9 ± 3.2
[116]	22	Sitting	*Amplitude*	range 0.1 ± 7.0	
[36]	9	Supine Sitting Standing	*Amplitude*	mean 5.93 ± 0.78 mean 6.32 ± 1.05 mean 6.85 ± 1.22	
[44]	18	Standing	*Amplitude*	mean 3.05 ± 0.05	
[22]	45	Prone	*Amplitude*	mean (l) 9.0 ± 4.6 mean (r) 8.6 ± 4.6	
[35]	10	Prone	*Amplitude*	mean 10.05 ± 4.87	
[35]	3	Prone	*Amplitude*		mean 6.10 ± 3.96
[57]	19	Prone	*Amplitude*	mean max (l) 1.44 ± 1.32 mean max (r) 1.52 ± 1.02	
[80]	20	Prone	*Amplitude*	mean 5.97 # range 1.8–12.8 #	

(*continued*)

Table I. (*continued*)

Author(s)	*n*	Position subjects	Parameter	Normal latency (ms)	UMN latency (ms)
[27]	120	Prone	*Amplitude*	mean max (u) 3.4 ± 2.4	mean max (a) 6.3 ± 3.6
[81]	9	Prone	*Amplitude*	maximal 6.26 (mid)⋆ maximal 5.55 (distal)⋆	
[211]	10	Prone	*Amplitude*		mean 6.72 ± 2.3
[211]	10	Prone	*Amplitude*		mean 7.21 ± 2.4
[126]	10	Prone	*Amplitude*	maximal 8.21 ± 2.79 [1] maximal 2.17 ± 2.89 [2]	
[337]	60	Supine	*Amplitude*	mean 5.2 ± 2.2 range 1.8–11.0	
[84]	**	**	H_{max}/M_{max}	mean 0.06 ± 0.38	
[120,121]	**	**	H_{max}/M_{max}	mean 0.18 ± 0.12	mean 0.50 ± 0.24
[110]	9	Prone	H_{max}/M_{max}		mean 0.60 ± 0.08
[35]	10	Prone	H_{max}/M_{max}	mean 0.44 ± 0.18	
[35]	3	Prone	H_{max}/M_{max}		mean 0.32 ± 0.22
[125]	30	Prone/Supine	H_{max}/M_{max}	0.46 ± 0.20	
[45]	10	Sideways	H_{max}/M_{max}		range 0.033–0.904
[57]	19	Prone	H_{max}/M_{max}	0.28 ± 0.19 (l) 0.29 ± 0.21 (r)	
[123]	4	**	H_{max}/M_{max}		0.67 ± 0.05 in acute SCI
[123]	8	**	H_{max}/M_{max}		0.62 ± 0.06 in chronic SCI
[123]	16	**	H_{max}/M_{max}	0.66 ± 0.07	
[37]	7	Semi-supine	H_{max}/M_{max}	mean 0.42 ± 0.17	
	10	Semi-supine			mean 0.67 ± 0.30
[131]	6	Supine	H_{max}/M_{max}	mean 0.51 ± 0.19	mean 0.70 ± 0.11
[81]	9	Prone	H_{max}/M_{max}	mean 0.66 ± 0.14 (mid)⋆ mean 0.66 ± 0.14 (distal)⋆	
[27]	120	**	H_{max}/M_{max}	mean 0.29 ± 0.14 (u)	mean 0.52 ± 0.21(a)
[300]	35	Sitting	H_{max}/M_{max}		mean 0.72 range 0.13 – 1.0
[211]	10	Prone	H_{max}/M_{max}		mean 0.66 ± 0.3
[211]	10	Prone	H_{max}/M_{max}		mean 0.90 ± 0.4
[33]	22	Sitting	H_{max}/M_{max}		mean 0.59 ± 0.05
[126]	10	Prone	H_{max}/M_{max}	maximal 0.55 ± 0.13 [1] maximal 0.56 ± 0.14 [2]	
[197]	30	Prone or semi-reclined	H_{max}/M_{max}	mean 0.38 ± 0.21	
[262]	21	Prone	H_{max}/M_{max}	mean 0.61	
[262]	39	Prone	H_{max}/M_{max}		mean 0.70
[135]	17	Semi-supine	H_{max}/M_{max}		mean 0.43 ± 0.22
[38]	20	Prone	*Side-to-side ratio*	0.73 ± 0.22**	< 0.70
		Standing		0.72 ± 0.22**	
		Loaded standing		0.74 ± 0.17**	
		Unloaded standing		0.70 ± 0.24**	
[22]	45	Prone	*Side-to-side ratio*	mean 0.74 ± 0.17 ** max 2.50**	< 0.40
[159]	52	Sitting	H_{vibr}/H_{contr}		0.48 ± 0.32
[110]	9	Prone	H_{vibr}/H_{contr}		0.30 ± 0.08
[37]	7	Semi-supine	H_{vibr}/H_{contr}	mean 0.47 ± 0.27	mean 0.48 ± 0.23
[73]	120	**	H_{vibr}/H_{contr}	mean 0.52 ± 0.09 (u)	mean 0.88 ± 0.13 (a)
[158]	6	**	H_{vibr}/H_{contr}	mean 0.47 ± 0.08	mean 0.92 ± 0.03
[97]	46	Sitting	H_{vibr}/H_{contr}	mean 0.37 ± 0.23	
	16	Sitting			mean 0.81 ± 0.15
[197]	30	Prone or semi-reclined	H_{vibr}/H_{contr}	mean 0.59 ± 0.30	
[325]	7	Prone	H_{vibr}/H_{contr}		mean 0.57 $
	5	Prone			mean 0.30 ##
[262]	21	Prone	H_{vibr}/H_{contr}	mean 0.25	
[262]	38	Prone	H_{vibr}/H_{contr}		mean 0.61

⋆'Mid' and 'distal' indicate recording sides; **ratios were computed by dividing the smaller amplitude by the larger amplitude; $incomplete SCI-patients; #optimal recording side; ##complete SCI-patients.
(l), left side; (r), right side; (a), affected side; (u), unaffected side.
[1]Recorded at day 1; [2]recorded at day 2.

A disadvantage of the amplitude parameter in general is its variability (e.g. see [79,87,125]), due to all kinds of factors as described in Section 1.2. Despite the frequently mentioned variability, several studies have been able to record the H-reflex with considerable intrasession or intersession reliability [24,25,44,126], although McIlroy and Brooke [86] found poor between-days H-reflex reliability. These reliability studies mainly aimed at investigating the soleus H-reflex, but Palmieri *et al.* [126] also included the assessment of intersession reliability of the tibial anterior and the peroneal muscles into their investigation. The H-responses of the three muscles were simultaneously invoked on two consecutive days by one stimulus applied to the sciatic nerve bifurcation of 10 healthy subjects lying in prone position. In all muscles, intraclass correlation coefficients were between 0.859 and 0.997, indicating a high reliability [126].

Considerable within-subject reliability reduces the application of normal limit absolute amplitude values [22]. The side-to-side amplitude ratio or difference might be considered to help overcome this problem. In 45 healthy subjects, Jankus *et al.* [22] performed a study in order to determine normal values for the side-to-side ratio of the H-reflex amplitude of the soleus muscle. The amplitude of the side with the smaller response is expressed as a ratio of the contralateral response amplitude. The mean value found was 0.74 with a standard deviation of 0.17. Ratio differences were considered abnormal when smaller than 0.40 (mean − 2 sd).

Analogous to the H-reflex latency, the H-reflex amplitude poorly correlates with clinical measurement of spasticity, like the Modified Ashworth Scale (MAS), muscle tone (investigated with the Ashworth scale; $r = 0.4$ for the upper and lower limbs) [127], clonus [128], Achilles tendon reflex [127], and muscle force (measured with the Medical Research Council Scale; $r = 0.4$ in both extremities) [127].

1.3.1.3 H_{max}/M_{max} ratio. As the amplitude of the H-reflex, the amplitude of the M-wave reflects motoneuron excitability. The amplitude of the M-wave is the same under all conditions, while, in contrast, the amplitude of the H-reflex is subject to facilitation and inhibition. The ratio of the maximal H-reflex amplitude and the maximal M-response amplitude gives an indication of the relation between the maximal number of motor units that can be activated through reflexes, influenced by inhibitory or excitatory input, compared to the total number of motor units [129] thus indicating the level of excitability of the motoneuron pool [130]. As in spastic subjects the size of the H amplitude increases (see Section 1.3.1.1), the H_{max}/M_{max} ratio raises as well [12,110,124,131], reflecting a larger part of the α-motoneuron pool activated by electrical stimulation of a peripheral nerve.

Taking a ratio instead of an amplitude can be considered as a normalization procedure, decreasing subject variability [87] which makes comparison between subjects [37,110] and between groups justifiable [110]. It also facilitates interpretation of motoneuron excitability. For the soleus and peroneal muscles, intraclass correlations of the H_{max}/M_{max} ratios have been shown to be remarkable in healthy subjects ($r = 0.966$ and $r = 0.975$, respectively), but the tibial anterior H_{max}/M_{max} ratio showed lower, but still strong, reliability between days (0.785) [126]. Although this looks promising, one should be aware of the fact that using intraclass correlation coefficients with standardized scores (e.g. H-reflexes across days) results in an underestimation of the true reliability [87].

The H/M ratio is a rather sensitive measure for (intervention-induced) changes in motoneuronal excitability [27,132]. It is also quite reproducible over time in spastic patients [37], but another study found quite variable ratios in healthy subjects [125]. When repeated measures are performed, one should ensure that the amplitude of the M-response is identical for all different measurements [133] since this provides relative certainty that the number of motoneurons activated is similar in all recordings, thus increasing standardisation. The increased H/M ratio is associated with increased excitability [134] and significantly correlates with the latency of the stretch reflex, the stretch reflex area, the stretch reflex threshold speed, and the total stiffness index [108]. One should be, however, careful in concluding that an increased H_{max}/M_{max} ratio is due to increased excitability of the motoneuron pool, since the increased ratio could also be due to a decreased M-wave amplitude [124].

A major disadvantage of the H_{max}/M_{max} ratio is the considerable overlap of values in spastic and normal limbs in different patients [84], narrowing its diagnostic use. Little *et al.* [79] and Levin and Hui-Chan [37], who found large inter-subject variability in the H_{max}/M_{max} ratio later confirmed this finding. Bilateral comparison of the H_{max}/M_{max} ratio could be recommended but is more time consuming, more uncomfortable, and less well tolerated since supramaximal stimulation is required to assess M_{max} [22]. The ratio is poorly correlated with clonus and the Achilles tendon reflex [128], muscle tone as assessed with the Ashworth Scale ($r = 0.3$ in both extremities), other tendon reflexes of the upper and lower limbs ($r = 0.2$ and 0.25, respectively) [127], and clinical scales, for example the Modified Ashworth Scale (MAS) [108,128,135]. More detailed, the H_{max}/M_{max} ratio was significantly correlated to the total reflex score of the lower extremity ($r = 0.392$) but not

to the Achilles tendon reflex score and the muscle tone score of ankle dorsiflexion (0.223 and 0.213, respectively).

1.3.1.4 H_{slp}/M_{slp} ratio. Funase *et al.* [136] presumed that the threshold of the H-reflex does not dependent on α-motoneuron excitability alone and therefore proposed a new method for assessing changes in α-motoneuron pool activity by hypothesising that the developmental slope of the H-reflex (H_{slp}) recruitment curve as a ratio of the developmental slope of the M-response (M_{slp}) could be used as a better parameter for evaluating the excitability of an motoneuron pool. The H_{slp} represents the relationship between the number of motoneurons activated and a given incremental raise in stimulation intensity [34]. In 22 healthy subjects, they investigated H-reflex parameters in the soleus muscle, under rest conditions and during voluntary tonic contraction of the ankle plantar flexion and dorsi flexion. Because the H_{slp} is free from the collision of the H-reflex discharge and the antidromic volley from the M-response of the α-motoneuron efferents, the H_{slp} can estimate the recruitment properties of a whole MN pool. From their results, Funase *et al.* [136] concluded that the H_{slp}/M_{slp} is a better parameter for the evaluation of MN excitability of the motoneuron pool than the H_{max}/M_{max}. This finding was confirmed by Bradnam *et al.* [34] and Higashi *et al.* [137]; H_{slp} is more sensitive than the H_{max}/M_{max} ratio since it provides information about the recruitment threshold [34]. The H_{slp}/M_{slp} ratio also is a better indicator of motoneuron excitability in spasticity than the conventional measures [137].

1.3.2 Presynaptic inhibition: H_{vibr}/H_{contr}. Presynaptic inhibition on Ia afferents reduces the release of neurotransmitters onto the motoneurons thereby weakening the effects of Ia afferents on motoneurons. Consequently, a reduction of presynaptic inhibition enlarges the excitability of the reflex arc [138]. There are several ways of measuring presynaptic inhibition, for example the methods described by Harburn *et al.* [67], Nielsen and Petersen [139] and Ørsnes *et al.* [33]. The application of a vibratory stimulus of 100 – 200 Hz with an amplitude of 1 – 2 mm to a tendon for about one minute [140 – 143], is most often applied in literature and considered an appropriate conditioning stimulus for the primary endings causing Ia afferent fibers to discharge [140]. The vibration selectively stimulates primary muscle spindle endings without involvement of other stretch receptors and a minimal antagonist-spread [144]. This stimulus switches on the spinal mechanism of presynaptic inhibition, which inhibits the transmission of neurotransmitters from Ia afferents on α-motoneurons and subsequently inhibits H-reflex amplitudes [140,145 – 150]. Optimal inhibition of the H-reflex is achieved when vibration of the Achilles tendon is applied 20 – 60 ms before H-reflex measurement [151].

The Vibratory Inhibition Index (VII) of the H-reflex can be calculated to quantify presynaptic inhibition. This index represents the percentage of H-reflex amplitude reduction induced by vibration of a muscle tendon (H_{vibr}/H_{contr} *100). In healthy, young subjects, the H_{contr} of the soleus muscle is inhibited 40% [12] to 50% [146,152] compared to the normal H-reflex amplitude. In acute stages of a CVA or spinal lesion, the VII is not altered [12], although H_{vibr} is almost completely suppressed in 'spinal shock' [146]. In chronic spastic patients the vibratory inhibition is significantly reduced (e.g. [12,31,73,96,97,110,117,154]), indicating a reduced presynaptic inhibition in upper motor neuron disease. In spastic subjects only 20% of the Ia afferentiation is inhibited by presynaptic inhibition [146,152]. Pinelli and Lorenzo [155] reported in their study a VII equal to 100 in spastic subjects. Levin and Hui-Chan [37] found a mean ratio of 47% (standard deviation 27) in seven healthy subjects. In the same study, seven spastic volunteers showed a mean value of 48% (sd 23) which was not significantly different from the control group. In the flexor carpi radialis muscle of spastic subjects, Childers *et al.* [23] found a mean ratio of 71% (sd 13). On the other hand, a return to normal values has been recorded 1 year after the onset of the lesion [12]. So, the level of increase of the VII in spastic subjects is not always identical between different studies.

Between-leg difference is another application of the VII that can be used to study differences between healthy subjects and spastic patients. In healthy, young subjects, the VII is identical in both legs [12], while in hemiplegics a difference of 15% is found between the two sides [12].

There are several advantages of using the VII. The measure provides important quantitative information about presynaptic inhibition and motoneuron excitability [23] that is highly reproducible [21,37,97] and sensitive to changes in motoneuronal excitability due to an intervention [132,157]. It has proven to correlate with hypertonia [134], and clinical spasticity scores [157,158], like the Modified Ashworth Scale [159]. However, in a considerable number of studies, VII is only moderately correlated with muscle tone (measured with the Ashworth scale; $r = 0.35$ and 0.40 in the upper and lower extremity) and muscle force (assessed with the Medical Research Council Scale; $r = 0.31$ in the upper and lower extremity) and tendon reflexes were even poorly correlated ($r = 0.2$ for both the upper and the lower limbs) with the VII [127]. A specific

advantage of the ratio over an absolute measure is that problems associated with measuring reflex amplitudes alone are avoided [23]. Finally, the VII is easily performed with electromyography equipment as found in many clinical settings [23].

Despite this, several papers still report variability, for example a large age-related spread (e.g. [145,160]) although no correlation was found with age of the patient [117]. Also, a large between-subject variability was found in a patient population [97] and some studies found a low correlation with intensity of spasticity [127,161,162] or duration of disease [127]. According to Ongerboer de Visser *et al.* [97] this variability is due to the variance of two H-reflexes (control and vibration) that used for calculation [97]. In addition, the VII does no pay attention to stimulus intensity. Therefore, they suggested a new Vibratory Index: the Cumulative Vibratory Index (CVI), which represents the ratio between the surface under the recruitment curve obtained with vibration and the surface under the recruitment curve without vibration. Final and important disadvantage is that the mechanism that induces the inhibition is not completely clear and possibly not solely due to presynaptic inhibition, at least not in the later stage of inhibition [69].

1.3.3 Reciprocal inhibition. Both Ia reciprocal inhibition as well as the less familiar non-reciprocal inhibition can be studied with the H-reflex.

1.3.3.1 Ia Reciprocal inhibition. Contraction of a muscle activates Ia interneurons that inhibit motoneuron activity of the antagonist muscle. This mechanism is called Ia reciprocal inhibition and can be assessed by H-reflex assessment during either sensory stimulation of the antagonist muscle group [163,164] or by simultaneously contracting the antagonist muscle group [130,164]. For example, feeble electrical stimulation of the peroneal nerve activates Ia inhibitory interneurons projecting on the soleus muscle [147,165]. This stimulation results in three inhibitory periods: a short latency inhibition (at interstimulus intervals of 1 – 3 ms) due to disynaptic reciprocal inhibition [166,167], and two long lasting inhibitory periods; D1 at 7 – 8 ms [164,167,168], likely due to group I fiber activation [164], and D2 with onset at 60 ms. However, these inhibitory periods have not always been duplicated in other studies [169] and furthermore, several interstimulus intervals are used to describe these inhibitory periods. Inhibition was maximal at stimulation intensity of 1.25* motor threshold of the conditioning stimuli. This level of stimulation intensity activates Ia afferents as well as multiple sensory afferents and therefore causes an optimal inhibition [164]. Stimulation/activation decreases the size of the H-reflex when measured simultaneously in the agonist muscle (e.g. [168,169]). So, in normal subjects, (maximal) contraction of the tibialis anterior muscle while the H-reflex is elicited in the soleus strongly inhibits the reflex amplitude: The soleus H-reflex appears normally with smaller amplitude and a larger onset. The size of reciprocal inhibition can also be expressed in a ratio: the H-reflex amplitude after stimulation or contraction of the antagonist muscle group divided by the H-reflex under normal conditions [170,171]. Abnormalities in Ia reciprocal inhibition have been seen in hemiplegics [172 – 174] and spinal cord injured subjects [169,171]. The inhibition is reduced [155,174] or absent in spastic subjects [12,163] indicating an enhancement of excitability of the reflex arc although some studies have reported facilitation [175,176]. It could be concluded that in spastic subjects, there are changes in Ia reciprocal inhibition, but these changes differ from muscle to muscle [130] and maybe also between patient-groups. This makes it less suitable for clinical evaluation. A confounding factor in assessing reciprocal inhibition by contracting the antagonist muscle group is that selective contraction of muscles is more difficult in spasticity, further increasing the reflex [138].

During measurement, attention should be paid at two factors: stimulus intensity and stimulus duration applied to both the agonist as well as the antagonist muscle. In the upper extremity, the amount of inhibition increases with increasing amplitude of the agonist H-reflex and with an increasing intensity of antagonist stimulation. When providing a stimulus of short duration, inhibition will not occur, while a stimulus of long duration produces inconsistent inhibition [102]. In the upper arm, finally, contralateral stimulation of the median or radial nerve also results in altered reciprocal inhibition: an increased reciprocal inhibition when the contralateral median nerve is stimulated, and a decreased reciprocal inhibition when the radial nerve is stimulated contralaterally [174]. This is probably caused by the Ia muscle afferents [174].

1.3.3.2 Non-reciprocal inhibition. Interneurons activated by Ib afferents originating from Golgi Tendon organs produce inhibition of extensor motor nuclei at the same time as facilitation of flexor motor nuclei. It has proven to be a complex mechanism. Therefore, it is better to consider 'non-reciprocal inhibition' rather than Ib inhibition [130]. The H-reflex can be used to study this phenomenon. Afferents of the medial gastrocnemius muscle send just Ib projections to the soleus MN pool. Conditioning of the soleus by a prior stimulus at low intensity to the gastrocnemius medialis nerve reduces the H-reflex amplitude, indicating the intensity of

non-reciprocal inhibition [177]. This non-reciprocal inhibition is reduced in paraplegics [178] and also reduced or replaced by facilitation in hemiplegics [179]. It seems to be better correlated with spasticity [180] and the intensity of spasticity [130] than the other pathways [130] but a lot of profound investigation is required to further work out and define this method.

1.3.4 Recurrent inhibition. Recurrent inhibition is also called Renshaw's cell inhibition. Renshaw cells are interneurons that inhibit the homonymous motoneurons and are controlled in supraspinal centers. Recurrent inhibition reduces the number of motoneurons that fire and their rate of firing [181], causing a decrease in the current flow to the muscle and a subsequent smaller response. In 1952, Magladery *et al.* [182] noted that the H-reflex amplitude was dramatically affected when the afferent nerve stimulus to elicit the H-reflex was preceded by intervals of a conditioning shock. Renshaw cell activity can be studied with the H-reflex during double-collisions: The tibial nerve is stimulated with two stimuli in which S1 is the first, submaximal stimulus (test stimulus) that is able to evoke an H-reflex, and S2 is the second, supramaximal stimulus (conditioning stimulus) that can not evoke the H-reflex when applied alone. When the two stimuli are serially administered to the nerve, with an interval of about 10 ms, a reflex (H′) can be recorded [183]. The conditioning reflex discharge collides with the antidromic volley of S2 and eliminates it [87,181]. Motoneurons that already fired due to the first (S1) stimulus will not be invaded by the antidromic volley and will be available for activation by the Ia afferent volley elicited by the second stimulus [181]. The amplitude of this resulting H′-reflex is usually lower than the H-reflex evoked after S1 alone, thereby indicating a certain amount of hyperpolarisation of the motoneuron and an activation of recurrent inhibitory pathways in normal man. Decrease of inhibition is often found in patients with spastic paraparesis or paraplegia, but not in patients with spastic hemiplegia at rest [181,184]. During contraction, the inhibition was not normally distributed anymore between the different patient-populations.

Earles *et al.* [87] investigated the reliability of the protocol as described above in nine standing subjects. The results showed high ICC estimates for trial-to-trial (0.97), but the day-to-day reliability was considerably lower (0.12) possibly due to normalization with the M-wave amplitude [87]. Despite, the authors suggested that the assessment of recurrent inhibition could be used in establishing the effectiveness of intervention programs [87]. However, it seems that recurrent inhibition only plays a subsidiary role in the existence of spasticity and the usefulness of the measurement of the H-reflex during double-collisions is therefore restricted [20,181].

1.3.5 Polysynaptic changes in α-motoneuron excitability: The H-reflex recovery curve. Polysynaptic changes in motoneuron excitability, common interneuron activity, can be observed with the H-reflex recovery curve, which was described by Magladery *et al.* [182] in 1952. Paired stimuli with different interstimulus intervals (i.s.i.) at equal strength are (usually randomly) applied to the nerve to elicit an H-reflex [12,59]. The reflex evoked by the first stimulation (if intense enough to evoke an H-reflex) is usually called H1, or conditioning H-reflex, while the reflex that occurs after double stimulation is usually labeled H2, or test H-reflex (e.g. [185]). The results are then expressed as proportions of the H-reflex that would occur without conditioning (e.g. see [186]). The H-reflex recovery curve follows a specific pattern in normal situations, although this pattern is not that obvious until 1 year of age [187]. Usually, the curve shows two periods of facilitation with background depressed reflex excitability [186]. Early facilitation occurs within the first (tens of) milliseconds. After the first period of facilitation, a subsequent period of inhibition is discovered, followed by the second facilitation phase (i.s.i. $\pm$ 100 – 400 ms). Final recovery of the curve is preceded by a second period of depression [10,47,188,189]. Three mechanisms account for the different stages in the recovery curve. Early facilitation is assumed to be caused by local excitatory postsynaptic potentials [190]. Then, Ia afferent discharge, cutaneous afferents, or supraspinal reflexes result in the late facilitation phase [191 – 194]. Finally, there is transmitter depletion resulting in the late depressed state for 2000 ms [191]. However, these three different phases are not always that evident [186,195]. Different values for the facilitation stages as found in several studies may be due to the intensity of the conditioning volley and the test volleys, and the appearance of an M-wave in the gastrocnemius muscle affecting the excitability of motoneuron of the soleus muscle [186].

The H-reflex recovery curve can be used to study the excitability of the central motoneuron pool [189] in several disorders, like syringomyelia [189], UMN lesions [181], cerebellar disorders [182], Parkinson [197], and several kinds of dystonia [59]. Abnormalities have also been found in the clinically normal, unaffected side of the body [188,189]. Magladery *et al.* [182] noted that H-recovery curves in patients with the UMN syndrome show early recovery (less than 100 ms), more complete recovery, and less late depression compared to normal subjects. This increased facilitation can be seen as an important

characteristic for spasticity [182,198–201]. Chandran *et al.* [188] found a clear pattern in a paraplegic group with decreased motoneuron excitability 1 and 2 weeks after the lesion, a normal motoneuron excitability 2 and 3 months after the lesion, and motoneuron hyperexcitability 4 and 5 months after the lesion compared to healthy subjects. Also in hemiplegics, the excitability of the motoneuronal pool was significantly depressed during the first and second weeks [188].

Kagamihara *et al.* [185] hypothesized that the typical signs of spasticity found in the recovery curve where due to other mechanisms than the increase of motoneuron excitability. The high peak (at around 250–300 ms) seen in spastic subjects may be caused by muscle afferent inflow activated by stretching the ankle extensor generated by mechanical contraction resulting from the conditioned H-reflex [185,202]. Secondly, the greater H-reflex that is found in spasticity causes the quicker recovery [185]. The level of recovery is inferior to the size of the conditioning and the test reflexes, so when the test H-reflex is expressed as a proportion of the maximal amplitude of the H-reflex the recovery rate is significantly enlarged in spastic subjects [185]. To overcome this problem, authors recommended to express the test H reflex as a proportion of M_{max}. Although they found considerable inter- and intra-variability both between as well as within days, taken as a whole the recovery curve showed quite consistent periodicity. This variability increased the need for measuring at least four conditioned H-reflexes for each i.s.i. to increase. From 400 to 1000 ms there was a gradual recovery of the H-reflex amplitude [186]. The H-reflex recovery curve is a sensitive test but can be affected by a number of factors like technical [96,199], physiological conditions, and (supra) segmental factors. With stronger stimulation, the recovery curve shows a delay of recovery from early depression and a reduction of the overall recovery [111]. With stronger stimulation, the recovery curve shows a delay of the recovery from early depression and a reduction of the overall recovery [111]. Another disadvantage is that the origin of the altered inhibition or facilitation can hardly be found out. The test may illustrate an abnormal clinical state, but its contribution to physiological studies is restricted because it does not represent a specific neurophysiological mechanism [130].

1.3.6 Other methods. The reflex action of muscle afferents on motoneurons is modulated by *reflex activation history* [203], owing to muscle thixotropy [204]; changes resulting from the mechanical effects of contraction [76]. After muscle contraction, motoneuronal excitability to corticospinal input is depressed [205] and motor cortical excitability is reduced [206]. Activation of the synapse between Ia afferents and α-motoneurons results in a decreased neurotransmitter flow and cellular changes [207], resulting in a subsequent reflex amplitude depression. According to Trimble *et al.* [208], frequency of reflex activation reduces the effectiveness of the muscle spindle afferents when the muscle length is increasing and feedback of the spindles high. Together with other segmental mechanisms, the reflex depression regulates the excitability of the motoneuronal pool [87]. Evocation of two H-reflexes with 80-ms interstimulus interval results in a so-called Paired Reflex Depression (PRD) [208], expressed as an amplitude ratio of the depressed H-reflex and the unconditioned H-reflex. Since the influence of Ib and recurrent inhibition is minimal at an interstimulus interval of 80 ms [209], this depression is assumed to indicate the influence of reflex activation history and the frequency of primary afferent feedback [87]. A reliability study of Earles *et al.* [87] in nine standing healthy subjects showed remarkable trial-to-trial reliability (0.93) over seven trials, but lower day-to-day reliability estimates (0.65). With a similar protocol as described above, this measure could be used in assessing the effectiveness of interventions in neuropathology [87].

2 The Tendon reflex

2.1 Mechanism of the Tendon reflex. The Tendon reflex (T-reflex), or phasic stretch reflex, can be considered the mechanical counterpart of the H-reflex. Tapping a distal tendon stimulates the Ia afferents that originate in the muscle spindle [210,211]. This causes impulses that travel to the spinal cord through the sensory neurons. In here, they synapse with α-motoneurons that send their impulses to the muscle inducing a short contraction (see Figure 5).

Traditionally the reflex was considered to be monosynaptic although oligosynaptic contributions to the response were found as well [11]. Tapping a tendon can result in three separate responses that can

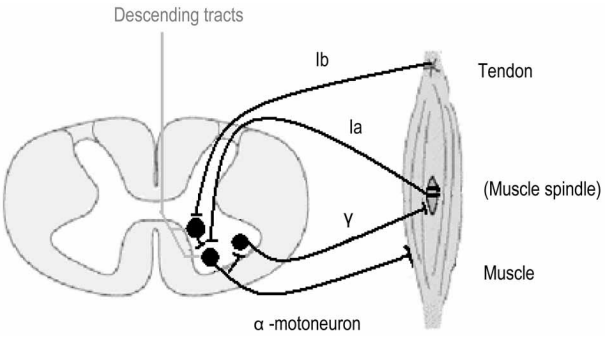


Figure 5. Neural pathway of the T-reflex (black lines).

be recorded in the muscle with surface Electromyography (sEMG): the tendon reflex (T-reflex, corresponds to the H-reflex and the stretch response), a silent period, and a subsequent long-loop reflex [212–215].

The mechanism responsible for reduced activity in the silent period has often been discussed and may be attributed to an activation of Ib inhibitory interneurons, activation of mechanoreceptors, or simply phase advancing the time of occurrence of the action potentials [51,212–214].

Like the H-reflex, major T-reflex parameters are the latency and its amplitude. The latency is the time that elapses between the stimulus (i.e. tendon tap) and the first deflection in the recorded signal. It represents the sum of the conduction time of the afferent and efferent impulses, plus the time for the synapse transmission in the spinal cord [20] and is best measured to the inception of the response, either positive or negative [26]. The amplitude is calculated as the difference in mV between the positive and negative peak [26].

Several tendons in the upper and lower extremity are suitable for evoking the reflex in healthy subjects. In general, the reflex is commonly evoked in the lower limb especially at the knee (Patellar reflex) and ankle (Achilles reflex). In the upper extremity, evoking the reflex is usually a bit more difficult, but the tendon tap can be applied to the supinator, the pronator, the biceps, the triceps, finger flexor, and the jaw tendons. The clinical significance of the T-reflex (especially of the Patellar and the Achilles) is mainly determined by its capability to assess the functional disturbance of a normal or augmented reflex arc [216,217], and evaluation of the motor system [218]. It is useful in the assessment of several neurological/neuromuscular disorders [133,217–223]. The experimental relevance of the tendon jerk is mainly based on its sensitivity for inhibiting and facilitating influences [224], for example in the acute [225], subacute and chronic stages of an upper motor neuron lesion [124]. Also therapeutically induced alterations in the reflex arc [156], the effects of training [226,227] and ageing can be investigated with the T-reflex [228]. The T-reflex is not really sensitive to psychofarmaca [229].

Although in literature the tendon jerk and H-reflex are usually approached as methods describing identical pathways, the excitability of α-motoneurons, T-reflex measurement involves different pathways as well. The T-reflex is assumed to be responsive to fusimotor tone ([145,230–232]. When the fusimotor fibers contract, the muscle spindle becomes more sensitive to stretch [210]. The fact that muscle spindles are included in the reflex loop of the tendon reflex implies that its amplitude is 2 or 3 ms longer than the H-reflex amplitude [233]. Besides this, tapping a tendon excites besides the Ia afferents also other nerve fibers, like group II afferents [27].

2.2 Factors influencing outcome. Like the H-reflex, the reflex arc of the tendon reflex is considerably susceptible to several factors. Below, the most important factors that have been described in literature will be discussed being stimulus intensity and frequency, muscle background activity, conditioning influences, maneuvers affecting reproducibility, and several other factors.

2.2.1 Stimulation intensity and frequency. The amplitude of the tendon jerk is related to the intensity of stimulation: An increasing force will induce increasing muscle stretch up to a certain level [234,235] and above that level muscle spindles are probably maximally excited so that the response cannot increase any further [234]. It is generally accepted that the threshold to evoke the tendon reflex is lower in spastic subjects than in healthy normal subjects, which means that the reflex can be evoked with lighter taps/lower stimulus intensity in more muscles [236]. The observed hyperexcitability of the stretch reflex as found in spastic patients is probably due to a decreased threshold [45,237,238] rather than an increase in hyperactivity of the stretch receptors [239]. During investigation of 10 MS patients and comparing the tapping force needed to evoke the tendon reflex with 14 healthy subjects, it appeared that the force needed was significantly lower in spastic subjects compared to healthy controls [236]. Also O'Sullivan *et al.* [240] found a decreased threshold of the biceps brachii T-reflex in children with spastic Cerebral Palsy. This threshold was significantly lower in children with quadriparesis than in children with hemiparesis.

Not only the intensity of stimulation, but also the frequency of the stimulation applied is important in tendon reflex evaluation. Repetitive stimulation to evoke the phasic reflex may induce post-activation depression [241] (see also Section 1.2.6). When inter-stimulus duration is between 1 and 10 seconds, the reflex seemed to be hardly influenced in the biceps [241].

2.2.2 Muscle background activity. The level of baseline EMG/contraction should carefully be recorded since the size of the amplitude increases [91,242] and the latency reduces at increasing contraction levels [91]. The reduction in latency may be brought about by the influence of contraction on γ- and α-motoneurons and the transmission of the afferent volley [91]. On the other hand, contraction of agonist/antagonist muscles [47] may be responsible for decreased reproducibility since this may activate complemen-

tary pathways. An advantage of measuring the reflex with a slightly contracted muscle (for example the m. tibialis anterior) is that it enables the elicitation of tendon reflexes that could not have been evoked in rest situation [91].

2.2.3 Conditioning of the T-reflex. Conditioning the Patellar T-reflex by a tap to the contralateral Patellar tendon [226], the contralateral or ipsilateral Achilles tendon, results in an increased excitability of the Patellar reflex [243]. A conditioning tap to the Achilles tendon resulted in excitatory effects on the quadriceps muscle, especially at later intervals (conditioning intervals used were 10 – 145 ms). The triceps surae muscles on the other hand, receive predominantly inhibitory effects from a conditioning stimulus to the contralateral Achilles tendon [244]. The study of Koceja and Kamen [244] revealed that the contralateral conditioning stimulus results in a long-latency facilitation for the quadriceps and a long-latency inhibition of the triceps surae, for up to 150 ms [226]. The short-latency change in motoneuron excitability due to a conditioning tendon tap may be caused by afferents of cutaneous receptors. The long-latency change in excitability may be due to supraspinal influences, although the exact mechanisms are still unclear [245].

Contralateral stimulation to the quadriceps muscle resulted in a short-latency facilitation for the younger subjects, but not for the older individuals. Ipsilateral conditioning, on the other hand, resulted in short-latency facilitation and a long-latency inhibition for both the younger as well as the older subjects, although in the older subjects the short-latency facilitation is shortened and the long-latency inhibition is prolonged compared to the healthy subjects [246].

2.2.4 Maneuvers affecting reproducibility. In the upper limb several maneuvers can be applied to increase reproducibility of the T-reflex. For example, placement of the thumb between tendon and hammer should produce more reliable results [221]. Facilitating maneuvers can influence the tendon reflex as well. For example, the Jendrassik maneuver [246] or contraction of remote muscles [247] result in an increased tendon reflex amplitude. Also, intra- and inter-individual variation has shown to be larger when the facilitating Jendrassik maneuver has been used during the elicitation of the tendon reflex [222]. Teeth crisping before tendon tap and relaxation of the other muscles [217] enhances the T-reflex and reduces the left-right asymmetry of the amplitude [248]. Anticipation is another factor that can affect tendon reflex outcome, since this can activate supraspinal pathways. To overcome this one could use different between-tap intervals.

2.2.5 Other factors that influence T-reflex outcome. The Achilles tendon reflex is different in the elderly compared to younger subjects [245], probably due to a change in α-motoneuron excitability [92,93,216,245,249 – 252]. Increased age as well as increased body height both result in prolonged latency. This does not count for the T_{vibr}/T_{contr} parameter [117]. The latency of the T-reflex also correlates with body height [91,153,216,253,254]. A study of Kuruoglu and Oh [216] in healthy subjects revealed that the Patellar tendon reflex is significantly correlated with leg and thigh length. Thus, to obtain the most reliable and comparable tendon reflex latency data, it should at least be corrected for age and height [222]. Several authors have made regression equations that offer the possibility to calculate the expected latency values for subjects based on height (see Table II).

The latency of the triceps tendon reflex, in contrast, did not show any correlations with age, height or arm length [216], and in general, the latency of the T-reflex is not strongly correlated with its amplitude or briskness [217]. Finally, tendon reflex duration is not correlated with age, height or extremity length [216].

Except for external sources of variance, the amplitude of the T-reflex may be variable due to central (interneuronal) and peripheral input [255,256]. If the balance between inhibition and facilitation is continuously changing, it will be very hard to duplicate measurements even though external variables are controlled.

2.3 T-reflex in spasticity. Several weeks after a lesion of the central nervous system that has disturbed the descending supraspinal control, an increased muscle tone develops associated with spasticity [257]. Several T-reflex parameters reflect the severity of some of the pathophysiological mechanisms or processes that have developed due to the lesion. Like the H-reflex, main pathways under investigation are α-motoneuron excitability and presynaptic inhibition, but also the excitability of the γ-motoneurons has been described with T-reflex assessment in a few studies. This section overviews the application of T-reflex to study these pathways, their advantages and disadvantages, and, eventually some data about reproducibility, sensitivity, and correlations with other methods to measure spasticity. Several outcome values of T-reflex parameters are presented in Table II.

2.3.1 α-Motoneuron excitability. Major parameters indicating the excitability of the α-motoneurons are amplitude of the T-reflex, its latency, and the T_{max}/M_{max} ratio. These parameters are described in the following sections.

Table II. T-reflex parameters.

Author(s)	*n*	Muscle (tendon stimulated)	Parameter	Normal	UMN
[120,121]	**	(Patella)	*T-amplitude*	1.6 ± 1.3	Pathology when above 4.3 mV
				2.3 ± 1.3 (J)	
[120,121]	**	(Achilles)	*T-amplitude*	0.9 ± 1.1	Pathology when above 4.1 mV
				1.7 ± 1.0 (J)	
[216]	24	Medial Gastrocnemius (Achilles)	*T-amplitude*	3.0 ± 2.0 range 0.4 – 8.8	
[216]	24	Rectus Femoris (Patella)	*T-amplitude*	1.4 ± 0.9 range 0.1 – 3.9	
[216]	24	Triceps (lat.) (Patella)	*T-amplitude*	1.3 ± 0.8 range 0.2 – 4.2	
[27]	120	Soleus (Achilles)	*T-amplitude*	2.14 ± 2.01 (u)	4.28 ± 3.41
[211]	10	Soleus (Achilles)	*T-amplitude*		6.53 ± 0.4
[211]	10	Soleus (Achilles)	*T-amplitude*		6.59 ± 3.1
[222]	102	Rectus Femoris (Patella)	*T-amplitude*	mean (rest) 1.77 ± 1.23 mean (J) 2.43 ± 1.38	
[222]	102	Soleus (Achilles)	*T-amplitude*	mean (rest) 4.05 ± 2.31 mean (J) 5.51 ± 2.65	
[222]	102	Rectus Femoris (Patella)	*T-amplitude side-ratio*	mean (rest) 0.49 mean (J) 0.60	
[222]	102	Soleus (Achilles)	*T-amplitude Side ratio*	mean (rest) 1.20 mean (J) 1.47	
[222]	102	Rectus Femoris (Patella)	*T-latency*	mean (rest) 21.0 ± 1.5 mean (J) 20.8 ± 1.5	
[222]	102	Soleus (Achilles)	*T-latency*	mean (rest) 35.2 ± 2.6 mean (J) 35.2 ± 2.6	
[245]	10	Soleus (Achilles)	*T-latency*	mean 33.5 ± 2.5[$]	
[245]	10	Soleus (Achilles)	*T-latency*	mean 39.1 ± 3.2[$$]	
[241]	16	Biceps brachii (Biceps tendon)	*T-latency*	range 16.8 – 23.4	
[222]	102	Rectus Femoris (Patella)	*T-latency side-ratio*	mean (rest) 0.40 mean (J) 0.43	
[222]	102	Soleus (Achilles)	*T-latency side-ratio*	mean (rest) 0.36 mean (J) 0.41	
[216]	24	Rectus Femoris (Patella)	*min T-latency*	17.2 ± 2.0 range 14.0 – 23.0	
[216]	24	Triceps (lat.) (Patella)	*min T-latency*	11.6 ± 1.4 range 7.2 – 15.6	
[216]	24	Medial Gastrocnemius (Achilles)	*min T-latency*	32.1 ± 3.0 range 26.0 – 41.0	
[91]	11	Soleus (Achilles)	*min T-latency*	38.2 ± 3.3	
	12			35.5 ± 3.0☆	
[91]	6	Tibialis Anterior	*min T-latency*	37.0 ± 3.9	
	12			35.75 ± 3.5(	
[216]	24	Medial Gastrocnemius (Achilles)	*T-latency Side difference*	1.0 ± 1.0 range 0.0 – 4.0	
[216]	24	Rectus Femoris (Patella)	*T-latency Side difference*	0.9 ± 1.0 range 0.0 – 4.0	
[216]	24	Triceps (lat.) (Patella)	*T-latency Side difference*	1.1 ± 0.9 range 0.0 – 2.7	
[110]	9	Soleus (Achilles)	T_{max}/M_{max}		0.37 ± 0.05
[27]	120	Soleus (Achilles)	T_{max}/M_{max}	0.17 ± 0.11 (u)	0.39 ± 0.21
[262]	21	Soleus (Achilles)	T_{max}/M_{max}	0.26	
[262]	39	Soleus (Achilles)	T_{max}/M_{max}		0.47
[117]	120	Soleus (Achilles)	T_{vibr}/T_{contr} (%)	54.61 ± 10.37 ⋆	
[117]	120	Soleus (Achilles)	T_{vibr}/T_{contr} (%)		87.4 ± 13.20#
[211]	10	Soleus (Achilles)	*T/H ratio*		1.07 ± 0.7
	10	Soleus (Achilles)	*T/H ratio*		1.02 ± 0.4
[216]	24	Medial Gastrocnemius (Achilles)	*Duration*	14.9 ± 2.5 range 10.0 ± 20.0	
[216]	24	Rectus Femoris (Patella)	*Duration*	27.6 ± 5.1 range 20.0 – 40.0	

(*continued*)

Table II. (*continued*)

Author(s)	*n*	Muscle (tendon stimulated)	Parameter	Normal	UMN
[216]	24	Triceps (lat.) (Patella)	*Duration*	16.5 ± 4.2 range 5.9 – 24.9	
[222]	102	Triceps (lat.) (Patella)	*Regression equation*	$y = 0.107x + 2.44$	
[216]	24	Triceps (lat.) (Patella)	*Regression equation*	$y = 0.12x - 2.55$	
[254]	40	Triceps (lat.) (Patella)	*Regression equation*	$y = 0.15x - 5.0$	
[222]	102	Soleus (Achilles)	*Regression equation*	$y = 0.191x + 1.99$	
[216]	24	Soleus (Achilles)	*Regression equation*	$y = 0.21x - 3.74$	
[254]	40	Soleus (Achilles)	*Regression equation*	$y = 0.12x - 10.2$	
[91]		Soleus (Achilles)	*Regression equation*	$y = 0.2558x - 5.9731$ $y = 0.2746x - 11.876$	

U, unaffected side; J, evoked during performance of Jendrassik manoeuvre.
Rest, evoked with muscles at rest.
⋆Healthy side of hemiplegics; #spastic side of hemiplegics; (evoked under weak contraction; $^{\$}$evoked in young subjects (mean age 20.9); $^{\$\$}$evoked in old subjects (mean age 74.3).

2.3.1.1 Latency. The latency indicates the excitability of the α-motoneurons of the tendon reflex arc. The number of studies describing T-reflex latency values is considerable, especially the Achilles and Patellar reflex latencies. From these studies, it can be concluded that the Achilles T-reflex latency in healthy subjects is between 30 and 40 ms. For the Patellar reflex, the time between stimulation and response is [216]. Maximal normal side-to-side differences have been reported as 1.6 ms for the biceps brachii [253], 3 ms for the triceps brachii [221], soleus, vastus lateralis and tibialis anterior [91], and 2 ms for the biceps and triceps [221]. In spasticity, the excitability of the α-motoneurons is increased so that a shorter T-reflex latency is expected in spastic patients.

In general, the latency of T-waves is quite stable. The reproducibility of the T-reflex latency of the relaxed or slightly contracted soleus, tibialis anterior, and vastus lateralis was tested in five subjects on different days and the coefficient of variation was calculated. Results showed a relatively low level of variation, means varying from 0.3 to 0.4 with standard deviations smaller or identical to 0.01 [91], indicating a high level of intersession reproducibility during rest as well as contraction. Contraction may end in a reduced response [91], but it does not result in a more reliable T-reflex latency [91]. The results were comparable with those found by Frijns *et al.* [222] who reported low intra-individual variability. Furthermore, the Achilles T-reflex latency was well correlated with the H-reflex latency recorded in the soleus [222].

2.3.1.2 Amplitude. The amplitude of the T-reflex gives an indication of the number of motoneurons that are activated by tapping a tendon with a hammer [258]. In the Achilles tendon (activity measured at the soleus muscle), mean values in normal subjects are 0.9 mV (sd 1.1 mV) [120,121]. For the Patellar reflex, mean values in healthy man are 1.6 mV (sd 1.3 mV). Enhancement of the phasic stretch reflex is feature of upper motor neuron syndrome [117,259,260], but it is not always found within the first month after stroke [114]. In a study of Cozens *et al.* [241] with healthy subjects, coefficients of variations for the biceps tendon tap amplitude were studied. Results showed that values for immediate reproducibility (i.e. response evoked and measured 4 times with inter-measurement interval of maximally 2 min, $n = 8$) were 6.2%, and for daily reproducibility (one measurement each day on three consecutive days, $n = 4$) 8.0%. In a study of Zhang *et al.* [236] the coefficient of variation for Patellar T-reflex amplitude was much lower in the MS population than in the healthy control group, indicating more reliable testing in the patient group probably due to the larger reflex responses and the consequently higher ratio of signal-noise. However, values in this study are hard to generalize since the mechanism used to evoke the T-reflex is slightly different from the conventional method (see [241]). The amplitude of the T-reflex showed a great degree of reproducibility between measurements in the study of Stam and van Crevel [254]. Other studies indicated a considerable inter-individual and intra-individual variation in T-value amplitudes, making comparison between subject populations hard [222,234,253,254].

Correlations between the T-reflex amplitude and some clinical parameters like muscle tone ($r = 0.34$ in the upper limb and $r = 0.35$ in the lower limb), muscle force ($r = 0.3$ in the upper and lower limbs), tendon reflexes as measured at a five-point scale ($r = 0.25$ and 0.33 in the upper and lower limbs, respectively) [127], and the Ashworth Scale ($r = 0.5$) [236] are insufficient. Results from a study in 120 subjects with post-stroke spasticity demonstrate a moderate correlation of the Achilles T-reflex with H-

reflex amplitude ($r = 0.7$) and a poor correlation with the H_{max}/M_{max} ratio ($r = 0.5$), maximal F-wave amplitude ($r = 0.2$) and mean F-wave amplitude ($r = 0.1$) [27]. In addition, the tendon response amplitude has shown to have some relation with the briskness of the tendon jerk [217].

2.3.1.3 T_{max}/M_{max} ratio. The maximal amplitude of the Tendon reflex (T_{max}) expressed as a proportion of the maximal amplitude of the M-wave (M_{max}) gives information about the excitability of the proportion of α-motoneurons. It likely also depends on the γ-system (the sensitivity of the muscle spindles) [261]. The normal range of the T_{max}/M_{max} ratio in healthy subjects is between 5 and 40% [161,200]. The ratio is increased in spasticity [84], resulting in a higher ratio [262,263]. In patients with spasticity due to incomplete spinal cord lesions, T_{max}/M_{max} ratios of the Achilles tendon were higher compared to those with complete lesions, and the ratio increased with increased duration of the lesion [262]. These results were less evident in the study of Brouwer and de Andrade [110]. They investigated the Achilles tendon reflex in nine MS patients and compared the results with 10 normal, healthy subjects. Although MS patients did have higher T_{max}/M_{max} ratios, the difference was not significant, presumably suggesting that abnormal fusimotor drive was not present in these patients [110].

The amplitude of the T-reflex can be increased up till 4 times in spasticity. The amplitude of the H-reflex, on the other hand, is increased up till 2 times in spasticity, indicating that the T_{max}/M_{max} ratio does not resemble the H_{max}/M_{max} ratio [51]. One explanation for this dissimilarity might be the involvement of different pathways in both reflexes [163,200]. Nevertheless, the T_{max}/M_{max} ratio was correlated with the H_{max}/M_{max} ratio ($r = 0.6$) and, although less strongly, with the amplitude of the H-reflex ($r = 0.4$) [27], but not with clinically assessed muscle tone ($r = 0.2$ and 0.22 in the upper and lower limbs, respectively) and muscle force ($r = 0.22$ in the lower limbs) [127]. The ratio of the tendon reflex amplitude and the M-wave amplitude seemed to be a better indicator of motoneuron excitability than just the absolute T-reflex amplitude [264]. Another advantage of the T_{max}/M_{max} ratio is that it is more sensitive to changes in α-motoneuron excitability due to diazepam and tetrazepam compared to some other assessment methods [156].

2.3.2 Presynaptic inhibition: $T_{vibration}/T_{control}$. Prolonged vibration of a tendon results in inhibition of the T-reflex recorded in the corresponding muscle, just as seen for the H-reflex [117,140,143,265]. A vibratory stimulus with 100 – 200 Hz and 1 – 2 mm in amplitude [144] switches on a spinal mechanism that is called presynaptic inhibition which results in a diminished α-motoneuron excitability and subsequently a smaller muscle contraction [138]. The level of presynaptic inhibition can be expressed as the T-reflex amplitude under vibration (T_{vibr}) divided by the maximal T-reflex amplitude without vibration (T_{contr}). The rationale behind this ratio given in literature is that the T-reflex reflects the same pathway as the H-reflex and with the latter presynaptic inhibition is usually expressed as H_{vibr}/H_{contr}. The verity of this assumption was inquired by a few authors. Milanov [117] compared the results of the T_{vibr}/T_{contr} and the H_{vibr}/H_{contr} of the soleus muscle and concluded that in the spastic side both ratios were significantly, and to the same degree, increased compared to the healthy side. He concluded that the T_{vibr}/T_{contr} parameter was valuable for the estimation of presynaptic inhibition [117]. These findings did not correspond with those of van Boxtel [232] who found a greater inhibited H- than T-reflex.

The strength of tendon reflex inhibition was not correlated with the clinical assessment of spasticity or duration of disease [117]. However, a quite strong correlation ($r = 0.86$) was found between the ratios of the healthy and spastic sides [117]. A disadvantage of the T_{vibr}/T_{contr} parameter is its dependency of the T-reflex amplitude on γ-motoneuronal activity that may influence the sensitivity of the muscle spindles to vibration. To overcome this problem, there should be no fusimotor activity in the muscle of study [256,257].

2.3.3 γ-Motoneuron activity: T_{max}/H_{max}. Some authors adhere to the assumption that fusimotor functioning is disturbed in spasticity and that muscle spindle and γ-activity could be studied by comparing the soleus H-reflex amplitude with the (Achilles) T-reflex amplitude (T_{max}/H_{max} ratio) [266]. The maximal amplitude of the T-reflex (T_{max}) as a ratio of the maximal H-reflex amplitude (H_{max}) has been hypothesized to indicate γ-motoneuronal excitability [73,267]. The ratio was raised [31,73,117] in 69% of the subjects with the spastic syndrome [73]. However, Burke [269] did not find an elevated ratio in spastic patients compared to healthy controls. The amplitude of the Achilles tendon reflex and its ratio with the H-reflex amplitude seemed to be quite well correlated with the Ashworth scale in one group of spastic subjects [211].

The use of this parameter to investigate spasticity is subject to considerable discussion, since theoretical information [255] concerning the contribution of γ-afferents to spasticity is lacking and there is no clear evidence supporting this view. In 1983, Burke and colleagues [51] showed that the afferent drives of both reflexes were considerably different from each other and both were influenced by mechanoreceptors

located in the soleus, skin, and other muscles. These results led to the statement that in spastic patients, an evocation of the H-reflex and the T-reflex during a sufficiently dorsiflexed ankle would result in a more affected tendon than H-reflex. This difference was usually devoted to excessive fusimotor drive in spastic patients, but appeared to be caused by the different characteristics of the afferent volleys of both reflexes [51]. Later, in 1999, Wilson *et al.* [269] assessed the contribution of disturbed fusimotor functioning to deficits in hemiparetic subjects. Results invalidated the contribution of this fusimotor activity to motor abnormalities in spasticity, at least applying to muscles at rest. In fact, discharge rates of spindle endings were similar to those found in normal subjects [269]. This finding, in combination with other publications, led to the conclusion that increased fusimotor activity is not necessarily needed to develop spasticity [269]. On the other hand, increased input from muscle spindle afferents to the spinal cord cannot result in spasticity on its own, since input is too low [269–271] concluded that motor control deficits as found in patients with hemiparetic spasticity are mainly due to central instead of peripheral (muscle spindle) pathways [269], thereby reducing the relevance of the T/H parameter in the evaluation of spasticity.

Therefore, the use of the T_{max}/H_{max} ratio is not recommended [51].

2.3.4 Other parameters. The above mentioned parameters have received considerable attention in literature. A less well known parameter of the Tendon reflex is its duration. The duration is expressed in ms and was found to be about 15 ms for the Achilles T-reflex measured in the medial gastrocnemius muscle [216].

Besides the neurophysiological assessment, T-reflex recording offers the possibility to assess biomechanical measures as well. Reflex torque, gain, contraction rate, and the reflex loop delay (defined as the latency between tapping the tendon and the onset of the reflex torque) of the tendon reflex proved to be quite useful parameters, sometimes showing higher correlations with clinical measures than did the neurophysiological parameters [236].

3 The Stretch Reflex evoked by passive movement

3.1 Mechanism of the Stretch Reflex evoked by passive movement. The Stretch Reflex (SR) evoked by passive movement is an important, more complex method to measure spasticity compared to the H- and T-reflexes. It is evoked by short muscle contraction, sinusoidal movements [272], displacement of the limb either rotation of a joint [273]. In theory, the SR can be evoked in any muscle, but in literature the joint to be examined is usually the ankle, the knee, or the elbow, although the SR can also be evoked in the extrinsic finger muscles by rotation of the metacarpophalangeal joints as was done in a study of Kamper and Rymer [274].

Passively moving the ankle and thereby stretching the slightly contacted ankle dorsiflexors can result in three peaks (M1, M2, M3) in muscle activity [242]. The first peak (M1) corresponds to the short-latency SR [275,276], is found at a latency of 30–50 ms [277], and is assumed to be monosynaptic [259] originating in Ia afferents [218] but probably also reflects muscle receptor activity [278]. This M1 response is more often subject to investigation then the M2 and M3 responses [279]. The second peak in sEMG seems to be mediated by afferents of the muscle spindles with a slower conduction velocity (group II afferents) [280], although others suggest that group Ia afferents transmitted with a considerable central delay may be responsible for this response, at least in the intrinsic hand muscles [281]. This second peak is usually defined as M2, medium-latency reflex, or 'early component of the long-latency reflex', and is larger than M1 [281]. The last deflection in EMG is caused by a long-latency response to stretch, M3, tonic SR, or also called 'the late component of the long-latency SR'. It is generally assumed that M3 does not occur in relaxed human muscles of healthy subjects when their muscles are stretched [282,283]. Confirmingly, the response was found to be absent in the first dorsal interosseus and flexor carpi radialis muscles of the majority of 77 healthy subjects [281]. The origin of the tonic SR is unclear [218], although several hypotheses have been developed over years. Supraspinal pathways [284], a long-loop transcortical pathway [285], polysynaptic spinal [286], or consecutive monosynaptic spinal reflexes due to repeated input from muscle spindles [287] are assumed to be sources of this late response. Each hypothesis suggests the influence of sensory inflow

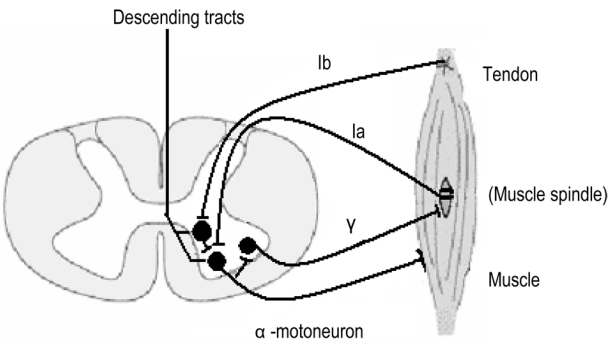


Figure 6. Neural pathways of the SR (black lines).

due to the muscular [288,289], joint, or cutaneous receptors [289,291–293] activated by stimulation. Nowadays, the long-latency SR is believed to be mainly a transcortical reflex [293]. See also Figure 6 for an overview of neural pathways involved in the SR.

Long-latency reflexes can behave in three ways. Firstly they can be absent, as in many MS patients [282], while the short-latency reflex is present and large. The absence of the long-latency reflex may reflect an inhibiting period due to the large monosynaptic response [282], or a disruption of the long-loop pathway due to the lesion. The long-latency reflex can also be normal, preceded by a slightly increased short-latency reflex. Finally, the long-latency reflex can be enlarged with a usually normal short-latency reflex [282]. The long-latency reflex seemed to be depressed or absent more often when the short-latency reflex was large in amplitude [282,290].

Responses to imposed movement provide information that is supportive in diagnosing, grading, and monitoring of diseases in the nervous system [218] and evaluation of the motor system. SR investigation is assumed to be advantageous in the quantification of spasticity or the upper motor neuron disorders and monitoring its progress (e.g. [218,221,279,282, 284,294]) and the effects of intervention (e.g. [138,151,272,295–297]). Phasic and tonic stretch reflexes can provide more insight in inhibited or excited pathways responsible for the disorder. The tonic SR contributes to muscle tone and can be estimated in clinical settings with several clinical methods as well as measured with neurophysiological methods [260].

In the ensuing text 'SR' refers to the short-latency, phasic SR, unless explicitly mentioned otherwise.

3.2 Methodological considerations. Several factors are mentioned in literature as being responsible for the changes in the SR. These factors will extensively be described below. Principal factors are the velocity of stretch, limb position, background muscle activity, and frequency of SR evocation. In addition, mechanical muscle properties and demographic characteristics contribute to the size of the SR.

3.2.1 Velocity of stretch. The stretch velocity is an important factor that influences outcome sizes, since the EMG amplitude is related to the velocity of stretch (e.g. [295]). Basically, if the velocity increases the amplitude of the EMG recordings increases as well [114,290,298–300] but also the latency is dependent on the initial velocity of stretch [37]. An increase in ramp velocity produces larger reflexes [37] until 300°/s in healthy subjects and those with Parkinson's disease [284], and Ashby and Burke [301] reported a threshold of 500°/s in spastic patients. Also, the size of the reflex is log-linearly, instead of linearly, related to the velocity of stretch, up to 300–350°/s. Above this limit physical restrictions limit joint rotation (at least in the thumb) [284]. In stroke subjects, the SR can be evoked in the ankle with movement speed of more than $20^{\circ}/s^{(1}$ [340]. For the long-latency reflex, this relation between amplitude size and velocity of stretch is more diverse [114,290].

Besides velocity acceleration affects the amplitude of the SR: Higher acceleration resulted in an increased amplitude of the reflex in the triceps of at least 30 out of 45 subjects. The magnitude did not always increase when the SR was performed with background force. This was more common when the short-latency reflex at rest was already large in magnitude [282,302].

3.2.2 Position of the limbs. Limb position influences the length excitability of a motoneuron pool. The position of the limb (or body) determines the length of the (homonymous) muscle which affects the SR [303] and this effect seems to be more widespread than just affecting the homonymous muscle [304]. For example, a dorsiflexed ankle [49] or stretched triceps inhibits the motoneuron pool of the soleus muscle [51]. Maximum isometric EMG levels of the quadriceps muscle appeared to be higher during active ankle dorsi- or plantar flexion compared with the ankle in neutral position [305]. Furthermore, quadriceps activity with the ankle in neutral position appeared to be higher than measured with the ankle in external or internal rotation [306]. Activity of the vastii lateralis and medialis, and the rectus femoris also appeared to be higher when the ankle was dorsiflexed compared to neutral position [307]. For reliable SR recording and interpretation of data, it is of great importance that for all subjects the position of the limb is identical.

3.2.3 Background muscle activity. Evoking the reflex under contraction increases the size of the SR amplitude [177,213,242] and decreases the latency of the phasic SR in healthy subjects but not in spastic subjects [260]. This relation between background EMG and reflex size causes a need to normalize the size of the response to baseline level just prior to the stretch [281,284]. Subtracting recorded EMG background activity from recorded response (see [277]) is one way to normalize data. An advantage of evoking the reflex under contraction is that it decreases the level of variability. On the other hand, evoking the reflex in totally relaxed muscles is not preferred since the exact level of motoneuron excitability cannot be measured because sub-threshold variations can not be established [284]. Finally, since the SR may not

always be evoked in relaxed muscles, especially not in healthy subjects, slight contraction of the muscle increases the probability of occurrence [276]. The influence of background EMG on reflex size outcome consequently implies careful monitoring of muscle activity with an oscilloscope.

In control subjects the onset latency decreased with increased contraction level, which is possibly due to an increase of larger diameter fibers with a higher conduction velocity, or due to decreasing synaptic delays in reflex pathways [260]. In spastic patients, the onset latency did not vary with contraction level, which may confirm the hypothesis that spasticity results in muscle changes which influences muscle mechanical properties and consequently stiffness [260].

3.2.4 Saturation and frequency of evocation. In the majority of studies it remained unclear whether the increase in reflex size was caused by an increased saturation level or by an increase in sensitivity [284]. The response should be studied in the range below the limit of saturation, since the size of the long-latency SR is saturated for disturbances just below the maximum set by the limits of joint rotation [308].

Also frequency of evoking the SR appeared to be important. Habituation and fatigue of M2 and M3 responses occur after frequent stimulation of each 5 seconds [309]. To minimize this, stretches should be delivered at frequency of maximal 0.1 Hz [309]. To prevent subjects from anticipating to stretch onset inter-stimulus intervals should be varied [238].

3.2.5 Mechanical muscle properties. Total joint-stiffness is compounded of reflex mediated and non-reflex mediated stiffness. Non-reflex stiffness is composed by a response from passive tissue (passive response) and a response from muscle fibers (intrinsic response). The intrinsic response is assumed to reflect the contractile properties of the engaged cross-bridges [310] and will stronger affect the total joint stiffness with increasing muscle force [311] and history of activation [312]. The non-reflex part of total stiffness can be measured at low velocity of the stretch since no reflex-induced muscle activation occurs during low velocities [313]. Another method is to evoke muscle contraction with electrical stimulation [90].

With stronger contractions the contribution of the reflex mediated response declines [314] and the non-reflex stiffness considerably contributes to total joint-stiffness [311]. These findings were later confirmed in healthy human subjects and revealed a reflex contribution of 50% in the ankle extensors and 35% in the ankle flexors with low to intermediate contraction levels [90].

Stroke patients showing spasticity signs appeared to have higher total-stiffness as measured by Harburn *et al.* [50]. Toft *et al.* [260] found an increased phasic EMG response in spastic patients, while the mechanical response was not enlarged [260]. Sinkjær *et al.* [315] reported a larger non-reflex stiffness in the flexors and extensors of the ankle of spastic subjects compared to healthy subjects. Spastic MS patients showed increased passive (73%) and intrinsic (79%) responses in the ankle flexors compared to normal subjects [90]. In the same muscles, the reflex mediated response appeared to be considerable decreased compared to the control group [90], which was unsuspected since the contribution of the SR was assumed to be considerable in decreased muscle control in spasticity [316]. The increased passive response was also found in the ankle extensors but was relatively higher (152% increase compared to healthy subjects) than in the ankle flexors. Furthermore, the intrinsic response and the reflex mediated response in these muscles were almost equal to those in healthy subjects. An increase of intrinsic muscles stiffness of 20% was found in hemiplegic subjects later as well [317]. Since the intrinsic muscle response reflects the contractile properties of the involved cross-bridges, increasing muscle contraction linearly resulted in increased intrinsic muscle response [90]).

The question is whether assessment of non-reflex stiffness by means of electrical stimulation is valid. Electrical stimulation first recruits larger motoneurons and successively smaller neurons with increasing stimulation intensity [90]. Mechanical stimulation at low contraction levels, reversely, first activates smaller motoneurons and larger ones at increasing contraction levels [318,319]. Furthermore, one could question the similarity of stretch responses evoked by electrical stimulation and voluntary contraction [90]. Alternatively, the non-reflex response can also be estimated by ischemia since this induces a considerable reduction in the reflex mediated component of the total response by blocking the peripheral Ia afferents [320,321]. This technique is not preferred since it is inherent to some considerable errors. Toft [90] investigated whether the reflex mediated mechanical response could also be measured using the reflex mediated EMG response. This method was successfully applied to the ankle flexors, but in other muscles generating larger reflex response, like the ankle extensors, reflex mediated EMG could not predict the mechanical reflex response [90].

The above mentioned results recommend careful evaluation of the SR in which passive and intrinsic mechanisms [50,90,236] are examined as well as muscle fiber composition since spastic paretic sub-

jects are assumed to have mainly type I muscle fibers [322].

3.2.6 Other factors influencing SR outcome. Experimentally induced muscle pain appeared to influence the SR outcome in healthy subjects, resulting in a significantly increased M1 SR amplitude, and a non-significant increase in the medium-latency EMG response (M2) ([107]. With increasing muscle background activity, this facilitation reduces or even disappears [323].

Yeo *et al.* [283] designed a study in which the tonic SR was studied in 30 healthy, but older subjects (above 45 years of age). During ramp and sinusoidal stretch of the elbow (maximal velocity of 270°/s), elbow flexors did not show any reflex activity [283]. The results justified the use of the tonic SR in the assessment of spasticity in subjects regardless of their age [283].

3.3 The SR in spasticity. One major feature of spasticity is the increased SR. The hyperexcitability of the SR can neurophysiologically be quantified in several different parameters, both neurophysiologically as well as biomechanically. This chapter presents the most important neurophysiological parameters of the SR as found in literature. For each parameter, if available, data will be presented concerning values in spasticity and healthy subjects, methodological factors, and (dis)advantages.

3.3.1 SR latency. The neurophysiological time parameter for SR threshold is the latency, which represents the sum of the conduction time of the afferent and efferent impulses, plus several ms for the synapse transmission [20]. A reduction of the SR latency could be due to a decreased reflex threshold causing motoneurons to fire earlier [238].

Normal latency values for the ankle flexor SR are between 20 and 50 ms, for M2 80 and 100 ms, and for M3 more than 100 ms. The latency of the Achilles short-latency SR was found to be reduced in hemiparetic subjects compared to controls [37]. However, Toft *et al.* [260] showed SR latencies of 45.0 ms in spastic patients and 44.0 ms in healthy controls. The peak of the SR occurred slightly later in spastic subjects (at 58.6 ms) compared to healthy controls (at 56.2 ms) [260]. In 23 spastic subjects and 20 healthy subjects, Cody *et al.* [290] studied the short- and long-latency responses in the flexor carpi radialis muscle during stretch and vibration in order to study the influence of group Ia and group II afferents. They found that in the stretch condition, in spastic subjects, the long-latency response (after 50 ms) seemed to be delayed. In the vibration condition, in both healthy as well as spastic subjects, the long-latency component of the SR was remarkably reduced [290].

Levin and Hui-Chan [37] recorded soleus SR at three different days, enabling them to calculate reproducibility of SR parameters. In nine hemiparetic subjects, interclass correlations were quite low for latency (0.44). Also, correlations with clinical measures of spasticity seemed to be quite low and variable over two testing days. The SR latency poorly correlated with the Achilles Tendon Reflex (0.13 at day one, 0.24 at day two, resistance (0.37; 0.54), clonus (0.16; 0.27), H-reflex latency (0.20; 0.13), and the vibratory inhibition index of the H-reflex (0.34; 0.01). Correlations were somewhat higher for the H_{max}/M_{max} ratio (0.54; 0.50). The SR latency showed very high and significant correlations with the onset angle of the SR (0.90; 0.92) [37].

3.3.2 SR amplitude. Almost each paper about stretch reflexes reports the reflex amplitude. The shape of the response in the ankle extensors is quite similar in both healthy subjects and spastic subjects. Toft *et al.* [260] found the SR at each contraction level, whereas the M2 peak was absent at lower contraction levels in both healthy and spastic patients. Compared to healthy subjects, spastic patients usually show a larger SR amplitude [260,275,282,324] and a reduced or absent M2/M3 response [290]. This amplitude depression of M2/M3 responses in spasticity may be brought about by motoneuronal refractoriness and other inhibitory mechanisms. Patients with vascular hemiplegia have shown increased SR amplitudes and muscle tone in the affected extremity a number of days after the infarction [325]. Sahrmann and Norton [326] studied the size of the SR in the elbow flexors m. biceps brachii and brachioradialis of upper motor neuron disease patients. Results showed a clear increase in the SR amplitude in both the biceps brachii as well as the brachioradialis muscles.

To validate the amplitude of the SR as a measure of spasticity, several authors have calculated correlation coefficients between clinical/biomechanical/other neurophysiological parameters and the SR amplitude. The correlation between the (Modified) Ashworth Scale and muscle activity during stretch of several lower leg as well as upper extremity muscles was relatively good [114,277,290,327]. This correlation was consistent over two measurements [277]. The relation between the M2 amplitude and spastic muscle tone however, was absent in the study of Cody *et al.* [290], but Berardelli and colleagues [282] did report a correlation between these two parameters. Fugl-Meyer test scores, gait performance [45,328], or pendulum test scores [329] did not correlate with reflexive EMG amplitudes in hemiplegic subjects. Furthermore, the SR amplitude was

significantly correlated with the clinical examination of tendon jerk hyperreflexia (wrist relaxed) [290], the Achilles tendon reflex score, and the total reflex score of the lower extremity, but not with the muscle tone score of ankle dorsiflexion [300]. Confirmingly, Berardelli *et al*. [282] concluded from their study that there is a relation between the tendon jerk and the short-latency reflex magnitude and rate.

Besides the above mentioned inconsistent correlations with clinical scales, SR amplitude measurement faces some other disadvantages. Major bottleneck is the inter-subject variability which causes eminent overlap between several patient groups and between patients and controls [284] and thus complicates the interpretation of results [290] and severely affects its diagnostic utility.

3.3.3 SR/M ratio. The amplitude of the SR divided by the amplitude of the M-wave (SR/M ratio) gives an indication of the proportion of the motoneuronal pool that is excited by stretching the muscle. As describe in Section 1.1, the M-wave is elicited by electrical stimulation of a mixed nerve and is the same under all conditions given a constant stimulus intensity level. Dividing the SR amplitude by the M-wave amplitude offers a method for normalization which eliminates inter-subject variance. Since the amplitude of M1 is increased in spastic subjects compared to healthy individuals and the M-wave remains stable, the SR/M ratio is significantly increased in this group [37]. Levin and Hui-Chan [37] measured the SR in the soleus muscle over three separate days in hemiparetic subjects. Interclass correlations were quite high for SR/M area (0.71). Also, the interclass correlation with the H/M ratio was remarkable (0.76; 0.75) and significant. Other clinical scales correlated only moderately to poorly with the SR/M area, like the Achilles Tendon Reflex, resistance, clonus, H-reflex latency, and the vibratory inhibition index of the H-reflex. The SR/M area was significantly related to the onset angle of the SR. Correlations with the onset latency and the duration of the reflex were weaker [37].

3.3.4 Duration of the SR. The duration of the SR gives an indication of the excitability of the motoneurons [37] and was found to be significantly increased in the nine hemiparetic subjects compared to a healthy control group [37]. The measurements were performed at three different days, so that test–retest reliability could be examined. Interclass correlations were quite low (0.20) just as with clinical measures. Only one significant correlation, with clonus (0.76), was found. SR duration was poorly correlated with the Achilles Tendon Reflex, resistance, H-reflex latency, H/M ratio, and H_{vibr}/H_{contr}. Correlations with onset angle, latency and SR/M were variable and not consistently significant [37].

3.3.5 Other parameters of the SR. Neurophysiological parameters are often presented next to the biomechanical parameters that can be extracted from SR measurement. These parameters usually describe a relation between the occurrence of the reflex and the limb angle and/or velocity of movement and are often derived from or related to neurophysiological parameters. Therefore, these parameters will shortly be mentioned, although a profound elaboration will be outside the scope of this paper.

The *SR threshold* is defined as the minimum angle under which motoneurons of the muscle are activated to cause a stretch [274,279], or as the angle at which EMG raised above 2 sd of the pre-stretch level for a certain period (see e.g. [330]) and represent the excitability of the reflex arc. In another study it has been defined as the minimal velocity at which a peak in EMG could visually be detected [300]. As such, the Achilles tendon reflex score and the muscle tone score of ankle dorsiflexion were not significantly correlated to the SR threshold [300]. Additionally, the total reflex score of the lower extremity was significantly correlated to the SR threshold. In a study of Jobin and Levin [279], the tonic SR threshold was calculated in children to investigate its validity and test–retest reliability. EMG activity was recorded from the elbow flexors and extensors of the wrist in spastic hemiparetic children with Cerebral Palsy, to establish threshold angles and velocity. The SR threshold showed to be a reliable parameter for the establishment of spasticity in children. This parameter of the SR may be more reliable than parameters solely based on EMG which in itself is variable and dependent from velocity, position, and force [331,332]. Allison and Abraham [30] reported a significant correlation between reflex threshold and the Modified Ashworth Scale (MAS). Correlations with other neurophysiological parameters were quite low for H-reflex latency, H/M ratio, and the vibratory inhibition index (VII) of the H-reflex, but significantly higher for the SR latency and the SR/M ratio. The relation with the SR duration was less strong. Interclass correlation of the onset latency in the spastic soleus muscle after stretch was substantial (0.93) [37].

The stretch reflex threshold van also be found in literature as onset angle (e.g. [37]). The onset angle was significantly lower in subjects with spastic hemiparesis compared to healthy subjects (mean $\pm$ sd: 15.5 ± 7.8 and 30.1 ± 4.7, respectively) [37]. Correlations with clinical scales however, were not strong for the Achilles Tendon Reflex, resistance, and clonus. Closely related to the

SR threshold is the *SR Threshold Speed (SRTS)*, which represents the minimal speed of joint movement needed to induce EMG activity in the joint muscles [333] and is expressed as °/s.

The reflex gain reflects the velocity sensitivity and can be defined as $EMG_{max}/^{\circ}$ [277]. It represents the change in number of motoneurons recruited per change in muscle length [279]. There is some debate about whether the reflex gain is increased or decreased in spasticity [238,277,334], although it seems to be increased [277]. Based on their findings, Powers *et al.* [230] concluded that the central disturbance as found in spasticity was a reflection of a reduced SR threshold, without a considerably increased reflex gain. The SR gain appeared to be not as reliable a predictor of spasticity as the amplitude of reflexive EMG activity given the moderate correlation (not significant) that was found between spasticity and velocity sensitivity of the SR [277,282].

Discussion

The area of neurophysiological assessment methods for spasticity has expanded considerably during the last decades, as reflected in the substantial amount of literature. Although the results of the described studies have provided a valuable source of information a negative consequence is a lack of a good overview concerning the methods, their best practice, protocols and reference values.

The aim of the review was to systematically review the spasticity measurement methods from a neurophysiological point of view and to describe the characteristics of these methods concerning background mechanism, spasticity measurement, methodological considerations, and future developments. Nine different neurophysiological measurement methods were identified with the current review procedure of which three were singled out and further elaborated in this paper: the Hoffmann reflex, the Tendon reflex, and the Stretch Reflex. A full description of all neurophysiological methods will be published in a forthcoming book (deliverable D410) from the *SPASM* project.

The Hoffmann reflex, the Tendon reflex, and the short-latency Stretch Reflex evoked by passive movement have in common that they all describe the phasic Stretch Reflex although the way of evocation of the reflex is different. As such the evoked response may assumed to be more or less similar, but the results of this review clearly indicate that one should keep in mind that they partially activate different afferent pathways and are influenced considerably by supraspinal pathways. The T-reflex and the SR are not pure representations of the α-motoneuron excitability, but are influenced by presynaptic inhibition, γ-motoneuron excitability [230], and other (descending) input as well.

There is ardent discussion between investigators about the influence of fusimotor activity in spasticity and its contribution to the phasic stretch reflex. This influence of other pathways underlines that the reflexes can not be considered as being only monosynaptic: Burke *et al.* [51] reported the oligosynaptic contributions to the T-reflex and the monosynaptic origin of the M1 response of the SR [259] was later disputed since it is very likely that it reflects muscle receptor activity as well [278]. Besides the T-reflex and SR, the monosynaptic character of the H-reflex is questionable [51] because the delay between stimulus and deflection in the EMG signal might suggest the involvement of di- or even trisynaptic pathways.

These findings implicate careful interpretation of H-reflex, T-reflex, and SR measurement outcomes. The involvement of more then one synapse easily results in more variability and a subsequent higher risk for finding low reliability values [87] and high levels of inter-session, intra-session, inter-subject, and intra-subject variability (e.g. [25,79,86,116, 335]). In line with this, presumably because of its oligosynaptic response, the T-reflex appeared to be less standardized and less reproducible compared to the H-reflex [91].

The relatively large variability is also a main cause of the lack of reference values that can discriminate between healthy subjects and patients with spasticity and thus incorporates a limited clinical use [91]. Surprisingly this variability was shown to be less dominant in spastic subjects compared to healthy controls [114]. For both the H- and the T-reflex some papers have provided normal values and even upper limit values that provide the ability to judge whether the results are in a pathological range or not. However, interchangeability between studies is limited because of differences in materials (i.e. reflex hammers), heterogeneity of subject populations, and methodological inconsistencies [222,280].

The high level of variability also affects the sensitivity of the methods to detect individual changes. In intervention studies a change in motoneuron excitability may also be due to a spectrum of factors rather than changes in inhibition or excitation induced by the intervention. Palmieri *et al.* [126] concluded that the soleus H-reflex could be used to assess changes in inhibitory or excitatory influences induced by therapeutic modalities similar for the VII of the H-reflex [156], but Milanov [117] already questioned these statements because he found an inability of the response in detecting changes in motoneuronal excitability after the application of different treatments.

Another important methodological consideration concerns the validity of the measurement method: do reflex measurements provide information about spasticity? This question can be addressed to some extend by relating parameters of the stretch reflex to parameters of other neurophysiological methods or biomechanical and clinical methods aiming at describing spasticity. Although in a few cases results were contradicting, the H-reflex, the T-reflex, and the SR parameters were moderately to poorly correlated mutually as well with other spasticity evaluation outcome parameters (see e.g. [37,108, 114,127,128,236]). One explanation for these low correlations may be that the scope of the stretch reflex is relatively small, so it provides information about a limited number of neural pathways that may be affected in spasticity [117,127] instead of reflecting a more 'complete clinical picture' of spasticity like assessed by more functional approaches. Based on this, one could question the validity of these methods in assessing spasticity. However, as shown in the review, the underlying mechanisms are now largely well known and understood. Also the effects of the method applied are well known, so it is rather clear what it measures. Actually, one could state that especially the lack of a well defined golden standard in measuring spasticity causes these low correlations.

From the above mentioned it can be concluded that the quality of the H-, T- and Stretch Reflex measurement in assessment of spasticity is restricted due to a relatively low reliability and sensitivity. Although not each fluctuation in facilitation and inhibition can be controlled, reliability and sensitivity can be increased and maybe even become acceptable if a strict protocol is used for reflex assessment. Some studies [24,25,44,126] have confirmed this and showed that a strict protocol can contribute to high inter-session and intra-session reliability levels. Although it seriously limits the clinical and experimental use, it is recommended to perform all measurements within one session, instead of several sessions [24], carefully monitoring the factors that can influence reflex measurement outcomes, and meticulous training of assessors. Elicitation of the tendon jerk, for instance, needs a lot of practice, but once skills have been acquired it can quite easily be used. T-reflex recordings are easy to perform, painless (unless overly strong tapped force is used [236]), and can extent the clinical neurological examination [217]. It is easier and equally informative compared to H-reflex measurement [219,220]. However, the H-reflex is more often used and the relevant factors have better been defined compared to the T-reflex. For example, one could imagine that the relevance of subject-, limb-, head-, and electrode positioning in T-reflex recordings is relatively equal to H-reflex recordings, but these factors have not as profoundly been investigated for the T-reflex. A major advantage of the SR over the T- and the H-reflex on the other hand, is that passive movement of the limb is a much more natural stimulus than electrical (nerve stimulation as in H-reflex or F-wave analysis) or mechanical stimulation performed with tendon tapping [114]. Although this movement still does not completely represent normal, functional movement as subjects are commonly measured in seated positions [338], it may clinically be more relevant since it represents an assemblage of pathways interacting in movement. This especially accounts for the long-latency SR which is polysynaptic. SR evaluation furthermore provides the opportunity of not only obtaining neurophysiological data but also extracting biomechanical parameters, enabling a multiple approach evaluation. In addition it enables a subdivision of the total joint stiffness into reflex- and non-reflex stiffness which makes it possible to study changes in muscle properties or mechanical changes. These changes have proven to play an important role in the stretch reflex [67,260,311,322]. Based on this, it is recommended to perform spasticity assessment with a combined neurophysiological and biomechanical approach, preferably, during functional active or passive movements because this may results in a more complete assessment and more close to the clinically related phenomena.

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