Toward an epidemiology of poststroke spasticity

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Toward an epidemiology of poststroke spasticity

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ABSTRACT

Poststroke spasticity (PSS)-related disability is emerging as a significant health issue for stroke survivors. There is a need for predictors and early identification of PSS in order to minimize complications and maladaptation from spasticity. Reviewing the literature on stroke and upper motor neuron syndrome, spasticity, contracture, and increased muscle tone measured with the Modified Ashworth Scale and the Tone Assessment Scale provided data on the dynamic time course of PSS. Prevalence estimates of PSS were highly variable, ranging from 4% to 42.6%, with the prevalence of disabling spasticity ranging from 2% to 13%. Data on phases of the PSS continuum revealed evidence of PSS in 4% to 27% of those in the early time course (1–4 weeks poststroke), 19% to 26.7% of those in the postacute phase (1–3 months poststroke), and 17% to 42.6% of those in the chronic phase (>3 months poststroke). Data also identified key risk factors associated with the development of spasticity, including lower Barthel Index scores, severe degree of paresis, stroke-related pain, and sensory deficits. Although such indices could be regarded as predictors of PSS and thus enable early identification and treatment, the different measures of PSS used in those studies limit the strength of the findings. To optimize evaluation in the different phases of care, the best possible assessment of PSS would make use of a combination of indicators for clinical impairment, motor performance, activity level, quality of life, and patient-reported outcome measures. Applying these recommended measures, as well as increasing our knowledge of the physiologic predictors of PSS, will enable us to perform clinical and epidemiologic studies that will facilitate identification and early, multimodal treatment.
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GLOSSARY

AS = Ashworth Scale; **BI** = Barthel Index; **EQ-5D** = a standardized measure of health status; **MAS** = Modified Ashworth Scale; **PSS** = poststroke spasticity; **REPAS** = REsistance to PASsive movement; **TAS** = Tone Assessment Scale; **UMNS** = upper motor neuron syndrome.

Stroke-related disability has emerged as a health problem that causes major impairment and significant socioeconomic consequences for patients as well as society.[1,2] The burden of stroke has long-lasting and profound effects on the patient, with the greatest impact attributable to impaired neurologic function. More than two-thirds of stroke survivors develop poststroke sequelae, including impaired motor function and poststroke spasticity (PSS).[3,4] These impairments have a significant impact on a stroke survivor's daily life, impeding basic tasks such as eating and self-care. Additionally, such disabilities place a significant burden on caregivers of stroke survivors.

The term *spasticity* as a defined clinical entity was coined by J.W. Lance in the 1980s as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflex (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex as one component of the upper motor neuron syndrome (UMNS)."[5] In clinical practice, spasticity is used to describe a combination of symptoms and clinical signs after lesion formation in sensorimotor brain

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areas and tracts in the CNS. Consequently, one or more clinical signs or symptoms of UMNS, including spasticity, may be present in the acute, postacute, and chronic phases after stroke and may affect functional motor recovery in postacute rehabilitation.[6]

To understand the differing rates of spasticity reported in epidemiologic studies, it is important to understand the role of contracture in the disturbance of passive stretch in UMNS. It has long been noted that secondary structural changes occur in such muscles. These amount to spastic contractures that are the result of degenerative changes and functional alterations of passive and possibly contractile properties of muscle.[7] Such muscle contractures may develop as secondary complications in patients with stroke and may in turn interfere with functional motor recovery. Findings have suggested, however, that spasticity itself is not the cause of contracture, but contractures may actually potentiate the degree of spasticity.[8] Furthermore, it has been shown that reduced activity or immobilization due to paresis results in soft-tissue contractures, and changes in muscle contractile properties further aggravate motor impairment, leading to increased spastic paresis.[9] As this review will demonstrate, there is an apparent lack of consensus about the disabling consequences of spasticity and contracture that makes it difficult for clinicians to judge the impact of PSS on disability and motor recovery after stroke. In addition, clinical and physiologic data on spasticity after stroke are limited because of the lack of reliable, valid, and sensitive measures that can be easily implemented in epidemiologic research. Therefore, in this review, we summarize the current epidemiologic data for PSS, briefly address the risk factors associated with PSS development, and discuss the implications and limitations that may result from using different measures of PSS.

PREVALENCE AND TIME COURSE OF PSS Data on PSS are limited because of the heterogeneity across clinical studies and a lack of population-based research. This review was based on a survey using PubMed to identify relevant data sources; the following MeSH terms were used: upper motor neuron syndrome OR UMNS OR spasticity OR hypertonicity OR contracture AND stroke OR cerebrovascular accident AND epidemiology OR prevalence OR incidence OR survival OR mortality OR morbidity. The search was limited to human studies and publications written in English. No restrictions were made on publication date, and the search resulted in 81 PubMed "hits." As a next step, our search included studies on prevalence data for increased muscle tone measured with the Modified Ashworth Scale (MAS) and/or the Tone Assessment Scale (TAS), which resulted in 11 hits. The results were sorted by the criteria of population-based vs non–population-based studies (e.g., community recruits, rehabilitation clinics), with no restrictions made based on methodology. The results were further limited to articles that did not describe certain select populations such as veterans, children, or institutionalized patients. Additionally, the reference list from each article was examined for other studies not identified through the PubMed searches.

From the available literature search on increased muscle tone, one study suggests that for increased muscle tone and reflex exaggeration, spasticity reaches its peak 3 months after the initial event.[10] The mechanisms underlying increased spastic muscle tone may not remain constant over the immediate, acute, and chronic stages of poststroke recovery; therefore, the time course of spasticity is important to consider in PSS evaluation. For the purpose of this review, we selected studies reporting prevalence data on PSS in developed countries after first-ever stroke according to the established clinical pathways of early (1–4 weeks), postacute (1–3 months), and chronic (>3 months) phases of the continuum of stroke care (table). These clinical phases correspond with management phases of stroke for acute treatment at the stroke unit, followed by inpatient or outpatient postacute specialized neurorehabilitation, and finally, by community care services.

Early phase (1–4 weeks). The early time course of PSS has been evaluated in several recent studies, which report a growing incidence with time. A study by Lundström et al.[11] (2010) assessed 49 patients with first-ever stroke using the MAS, the National Institutes of Health Stroke Scale, and the modified Rankin Scale, as well as clinical examination to identify other positive signs of UMNS, and found that spasticity (MAS score $\geq$1) was present in only 4% (2 of 49 patients) at days 2 to 10. A study published in 2004 by Sommerfeld et al.[12] included 109 patients; however, 4 patients were excluded because of recurrent stroke, 4 died within 3 months, and 6 could not be assessed after 3 months and were thus excluded. The remaining patients were assessed using the MAS and tendon reflexes and showed an increase in spasticity to 21% (20 of 95 patients; MAS score >0) in the first week (mean 5.4 days) after first-ever stroke.[12] Additionally, 3% (3 of 95 patients) showed moderate spasticity (MAS score = 2).[12] Wissel et al.[13] (2010) evaluated 103 patients after stroke using the MAS (all limbs), pain, paresis (Medical Research Council scale), the Barthel

Table Time course of poststroke spasticity[6,11–18]

Time poststroke	Spasticity diagnosis	Prevalence of spasticity	Setting/sample size of patients poststroke	Study
Early phase				
2–10 d	MAS score ≥1	4%	Hospital stroke unit, n = 49	Lundström et al.[11] (2010)
1 wk	MAS score >0	21%	Hospital stroke unit, n = 95	Sommerfeld et al.[12] (2004)
2 wk	MAS score >0	20.2%–24.5%	(1) Hospital stroke unit, n = 94	(1) Wissel et al.[13] (2010)
			(2) Hospital stroke unit, n = 109	(2) Welmer et al.[14] (2010)
4 wk	MAS score ≥1	27%	Hospital stroke unit, n = 48	Lundström et al.[11] (2010)
Postacute phase				
Median 6 wk	MAS score > 0	26.7%	Hospital stroke unit, n = 86	Wissel et al.[13] (2010)
3 mo	MAS score >0	19%	(1) Hospital stroke unit, n = 95	(1) Welmer et al.[14] (2010)
			(2) Hospital stroke unit, n = 95	(2) Sommerfeld et al.[12] (2004)
Chronic phase				
>3–6 mo	(1) MAS score >0	21.7%–42.6%	(1) Hospital stroke unit, n = 83	(1) Wissel et al.[13] (2010)
	(2, 3) MAS score ≥1		(2) Hospital stroke unit, n = 47	(2) Lundström et al.[11] (2010)
			(3) Hospital stroke unit, n = 211	(3) Urban et al.[16] (2010)
12 mo	MAS score >0	27%	Follow-up study of stroke survivors, n = 106	Watkins et al.[17] (2002)
	TAS score >0	36%		
	MAS and TAS	38%		
12 mo	TAS score >0	36%	Follow-up study of stroke survivors, n = 106	Leathley et al.[18] (2004)
12 mo	MAS score ≥1	17%	Participants of a national stroke registry, n = 140	Lundström et al.[15] (2008)
18 mo	MAS score >0	20%	Hospital stroke unit, n = 66	Welmer et al.[6,14] (2006 and 2010)

Abbreviations: MAS = Modified Ashworth Scale; TAS = Tone Assessment Scale.

Index (BI), and quality-of-life score (EQ-5D, a standardized instrument for use as a measure of health outcome). Nine patients were excluded for preexisting spasticity (median MAS score = 2). Of the remaining 94 patients, results showed that in the first 2 weeks after first-ever stroke, 24.5% (23 patients) developed increased muscle tone (MAS score >0). Using the same cohort as Sommerfeld et al., a study by Welmer et al.[14] published in 2010 reevaluated 109 patients with first-ever stroke using MAS (all limbs) and the occurrence of plantar flexor clonus. Up to 2 weeks after stroke, 20.2% (22 of 109 patients) showed spasticity (MAS score >0), and this PSS seemed most prevalent in the antigravity muscles that controlled voluntary movements. By 4 weeks, the Lundström et al. 2010 study showed further increase in muscle tone in 27% (13 of 48 patients; MAS score ≥1) and disabling spasticity—spasticity having a clinically significant impact on motor function, activity performance, or social life such that intervention (e.g., intensive physiotherapy, orthosis, pharmacologic treatment) should be offered—in 2% (1 patient).[11,15]

Postacute phase (1–3 months). The previous studies also evaluated the prevalence of PSS in the postacute phase. At a median of 6 weeks after first-ever stroke, 26.7% (23 of 86 patients) from the Wissel et al.[13] 2010 study were found to have increased muscle tone (MAS score >0), and 9.3% (8 of 86 patients) showed severe spasticity (MAS score ≥3). The study by Sommerfeld et al.[12] showed a slightly lower prevalence of spasticity by 3 months: 19% (18 of 95 patients; MAS score >0), with 5% of patients exhibiting moderate (n = 4, MAS score = 2) to severe (n = 1, MAS score ≥3) spasticity. Using the same cohort at 3 months, Welmer et al.[14] (2010) showed that in the 19% exhibiting spasticity (18 of 95 patients; MAS score >0), PSS was again most common in the antigravity muscles controlling voluntary movements.

Chronic phase (>3 months). After 3 months, PSS had similar prevalences in the various studies. In the 2010 study by Wissel et al.,[13] 21.7% (18 of 83 patients) were found to have increased muscle tone (MAS score >0) at a median of 4 months (between 3 and 6 months), with 9.6% (8 of 83 patients) showing severe spasticity (MAS score ≥3). The Lundström et al.[11] 2010 study showed increased muscle tone in 23% (11 of 47 patients; MAS score ≥1) at 6 months, with disabling spasticity present in 13% (6 of 47 patients). A study by Urban et al.[16] published in 2010 evaluated 211 patients 6 months after first-ever stroke using the MAS (all limbs), Medical Research Council scale, BI, and EQ-5D. Results showed that 42.6% (90 of 211 patients) had increased muscle tone (MAS score ≥1),

with 15.6% exhibiting severe muscle tone increase (MAS score ≥3). Additionally, although the prevalence of PSS did not differ between the upper and lower limbs, higher degrees of spasticity (MAS score ≥3) were more frequently observed in the upper limbs (18.9%) than in the lower limbs (5.5%).

Several studies further investigated the chronic phase of PSS from 12 months and beyond. In the study by Watkins et al.[17] published in 2002, 106 patients with stroke (of whom 34% had recurrent stroke) were assessed with the MAS and the TAS 12 months after stroke. The prevalence of spasticity (MAS score >0; TAS score >0) was 27% (29 of 106 patients) with the MAS and 36% (38 of 106 patients) with the TAS. When spasticity was assessed with both the MAS and the TAS, the prevalence increased to 38% (40 of 106 patients). In a study by Leathley et al.[18] (2004) that evaluated the same cohort as Watkins et al. with the TAS 1 year after the stroke event, 36% (38 of 106 patients) were categorized as having spasticity (TAS score >0), and 20% (21 of 106 patients) were categorized as having severe spasticity. A longitudinal study by Lundström et al.[15] (2008) followed 140 patients with first-ever stroke for 12 months, evaluating the MAS, the modified Rankin Scale, and the BI. One year after stroke, a lower prevalence of spasticity (MAS score ≥1) of 17% was determined and only 4% of patients exhibited disabling spasticity. A 2009 study by Lundström et al.[19] using the same cohort as their 2008 study identified spasticity in 15% (11 of 72 patients) without pain. In the group of patients who had stroke-related pain, spasticity was found in 41% (12 of 29 patients). Additionally, spasticity was found in 5% (2 of 39 patients) with pain not related to stroke.

In the chronic phase 18 months after stroke, the Welmer et al.[6,14] studies (2006 and 2010) showed that 20% (13 of 66 patients) displayed spasticity, defined as MAS score >0. Addressing the time course of PSS progression, of the 13 patients showing spasticity at the 18-month follow-up, 10 had shown spasticity in the first 1 to 2 weeks, and all 13 had done so at 3 months after stroke. There was also an increase in the severity of spasticity in some muscles between the early and 3-month phases, possibly explained by neural changes, and a further increase in spasticity between 3 and 18 months, possibly caused by intrinsic muscle changes.

Risk factors associated with development of PSS. Given the potential disability associated with the development of PSS, there is an incentive for early intervention or selective treatment that may reduce or possibly prevent the development of spasticity after stroke. Such early intervention would be greatly helped by the ability to identify risk factors associated with the development of spasticity to more quickly identify those patients who might benefit from treatment and effective stroke management.[18] Numerous studies have focused on identifying such risk factors and are summarized below.

In the development of PSS, strong positive correlations have been made between lower BI scores and degree of paresis. In the study by Leathley et al.,[18] for example, lower BI scores and early arm and leg weakness were shown to be significant predictors of abnormal muscle tone. Results also showed that left-sided weakness and a history of smoking were significant predictors of more severe spasticity. The study by Urban et al.[16] revealed that a severe degree of paresis at stroke onset was a predictor for the development of spasticity. Furthermore, in the Lundström et al.[11] 2010 study, severe paresis of the arm observed in patients 2 to 10 days after stroke onset was associated with a higher risk for spasticity at 1 month (odds ratio = 10; 95% confidence interval 2.1–48.4). In the 2010 study by Wissel et al.,[13] severe paresis and any paresis in the affected limb have also been identified as risk factors for the development of permanent spasticity, along with MAS score ≥2 in at least 1 joint within 6 weeks after stroke, more than 2 joints affected by increased muscle tone, hemispasticity within 6 weeks after stroke, and lower BI scores at baseline.

Stroke-related pain and sensory deficits may be additional risk factors associated with the development of PSS. In the Lundström et al.[19] 2009 study, the prevalence of stroke-related pain was estimated to be 21% (29 of 140 patients); among these patients, 41% (12 of 29 patients) developed spasticity. However, the results also show that spasticity itself was not enough to account for stroke-related pain, which was associated with sensorimotor impairments and depression.[19] Several mechanisms have been proposed for the relationship between spasticity and pain, one of which is that spasticity may cause abnormal loading and strain on muscles and ligaments, resulting in a risk for nociceptive pain. It is also possible that the spinal reflexes involved in spasticity may be enhanced by pain. Additionally, spasticity and pain may be involved in the same nervous lesion because of overlapping neuronal networks at the spinal and cerebral levels. Nonetheless, it is important to remember that these mechanisms require the presence of neurologic defects such as sensory disturbances.[19] Regarding sensory deficits and PSS, stroke patients with hemihypesthesia in particular have been shown to be affected by spasticity of the upper and lower limbs ($p \leq 0.001$ vs patients without hemihypesthesia).[16] Thus, taken together, there is a strong positive correlation between PSS development and the degree of destruction or disorganization of the central sensorimotor system.

Implications and limitations of different spasticity measures. The process of measuring spasticity is critical to the clinical diagnosis and management of PSS.

Our data indicate that prevalence estimates and the clinical course for PSS vary greatly; this heterogeneity among studies is attributable to several factors, including differences in methods or instruments used in assessment of spasticity, and differences in the time between stroke onset and assessment. Additional limitations on generalizability occur because the studies have been restricted to clinical centers. This does not represent the real-world population, because those in a clinic setting may reflect the most severe population and/or the population who has access to care. Because population-based data for PSS are sparse at best, the prevalence and clinical course of PSS are only partially understood.

Additional restrictions result from the limitations of assessment tools used for measuring spasticity. The current spasticity assessment scales include categorization by a) judgment of muscle tone (resistance to passive stretch); b) passive range of motion, motor performance, mobility measures, and posture; and c) other clinical phenomena such as tendon reflexes, clonus, and spasms.[20] Although spasticity measures are frequently used in clinical and research settings, they rate phenomena that invariably have a large intra- and interrater variability. Especially for use in population studies, measures of spasticity need to be reliable, valid, and sensitive in addition to reflecting functionality in the most objective way possible. Although a detailed examination of all PSS measurement tools is outside the scope of this review, we think that a critical discussion of the validity, reliability, and limitations of identified key scales is necessary to enable harmonization of assessment tools for future research in this area.

The Ashworth Scale (AS) and the MAS are widely used single-item ordinal scales for the measurement of resistance to passive motion.[20] These scales have variable intra- and interrater reliability with no factors clearly accounting for such variance. It is possible that the lack of standard protocols for positioning, performance, and scoring may contribute to this variability. Because spasticity is a velocity-dependent phenomenon, scales used to assess spasticity should include control of the velocity of passive movement. Because the MAS may not account for differences in velocity, it is less reliable for use as a clinical measure of spasticity.[21] Another scale focusing on muscle tone is the TAS, a 12-item summated rating scale incorporating resistance to passive movement, resting posture, and associated reactions (specifically, 6 + 3 + 3 items) in the assessment of spasticity in different muscle groups. This global assessment may be advantageous in evaluating spasticity treatment; however, such a summary score may not be valid for comparisons among different summated scores (e.g., 1 + 2 + 1 does not necessarily equal 3 + 0 + 1).[20]

REPAS (REsistance to PASsive movement) is a summary rating scale for the resistance to passive movement that shows significant validity and interrater and test–retest reliability.[22] This scale was created to improve the reliability of the AS and the MAS by providing detailed guidelines for the performance of different passive joint motions and scoring during patient evaluation, allowing for internal consistency and reliability across raters and over time. The efficacy of this summary scale is exhibited in a REPAS assessment of 33 neurologic patients with central paresis, where it demonstrated high internal consistency with no significant difference between raters or with test repetition (correlation coefficients: 0.87–0.97) and substantial reliability in arm and leg subtests (correlation coefficients: arm subtest 0.63–0.98, leg subtest 0.56–0.96).[22] Thus, this scale features the potential for more reliable and precise clinical assessments of spasticity and possibly improved therapeutic effects as well.

For muscle changes and contracture over time, passive range of motion is suggested for initial and follow-up assessments of spasticity-related reductions in range of motion.[20] However, in the clinical assessment of muscle contracture, it is important to standardize the force applied, so as not to exceed the magnitude of force normally sufficient to stretch the muscles, as well as to standardize the positions of the joints.[8] Without these limitations, comparisons across subjects or with the normal population cannot be made.

These assessments for muscle tone and contracture represent measures of impairment level only. It is recommended that impairment assessments be combined with tests of motor performance, activity level, patient-reported outcome measures, and quality-of-life scales. One recommended quality-of-life scale is the Stroke Impact Scale-16, which is a stroke-specific, psychometrically robust, and comprehensive outcome measure designed for the assessment of a wide range of physical function limitations and the measurement of stroke-related deficits.[23,24] In a recent study conducted to determine how PSS severity affects physical function and health-related quality of life, the Stroke Impact Scale-16 was shown to correlate with severity of spasticity—with lower scores indicating worse function and increasing severity of spasticity.[25]

Motor performance and kinematic measurements may also enhance the processes of determining functional recovery after stroke. Such kinematic studies analyzing fine motor tasks and utilizing movement strategies and measures include movement analysis, the Action Research Arm Test, the Brunnstrom scale, and the Fugl-Meyer arm score.[26] Although these motor performance measures help elucidate the manner in which patients achieve functional recovery after stroke, their value in assessing the different components remains limited. Because the role of spasticity in disability is complex, the precise correlations among spasticity, movement coordination, and disability after stroke are not fully established.[27] Additionally, any functional

improvements observed may be a result of becoming more familiar with the experimental test procedure. The adaptive and compensatory behaviors that patients develop because of their lack of motor control can also complicate assessment of improvement in motor performance measures because they potentially inhibit the return to normal neurologic functioning.[26] Nonetheless, despite their limitations, the combination of functional and psychometric measurements of impairment has proven to be useful in the evaluation of early, postacute, and chronic phases of spasticity care.

The setting and timing in which spasticity measures are used in the continuum of poststroke care also have a significant impact on the results obtained. The ease and expediency of utilizing certain subjective, indirect clinical scales such as the AS or the MAS have resulted in their widespread and effective use in clinic-based assessments of PSS. For epidemiologic studies, however, simplified patient-reported questionnaires are required to identify appropriate study patients. Although clinic-based studies are limited in their representation of the general population, their benefit is the combination of common clinical, functional, and more detailed neurophysiologic measures that directly assess muscle activity. A study by Malhotra et al.,[28] for example, showed that such neurophysiologic studies may be more sensitive and useful in quantifying and classifying spasticity in routine and research practice. To complete our understanding of PSS, however, particularly regarding global estimates of spasticity, there is a current need for population-based data that are generated from tools that can identify patients with spasticity within the general population.

Additionally, the study time point has a significant effect on the comparability across multiple data sources. Determining the time course of PSS and the appropriate management of spasticity depends on findings in the various stages of development of the disability. However, without strict time-point definitions for the assessment of spasticity in the early, postacute, and chronic phases after stroke onset, corroboration of results from different studies is difficult. Thus, global standardization of PSS assessment in the early, postacute, and chronic stages would enable more precise elucidation of the mechanisms underlying spasticity and aid in the development of effective treatment and management strategies.

CONCLUSION Spasticity is emerging as a significant health issue for stroke survivors, with substantial impact on the continuum of poststroke care and recovery.[3] A targeted literature search for stroke and UMNS, spasticity, contracture, and published research that assessed muscle tone measured with the MAS and the TAS was performed to elicit reliable data on the dynamic time course of PSS. This literature search identified key risk factors associated with the development of spasticity, including lower BI scores, degree of paresis, stroke-related pain, and sensory deficits. It was difficult to quantify the prevalence of PSS, based on the heterogeneity across studies and the lack of population-based data. Current estimates have been obtained from selected cohorts of clinical studies; the prevalence of PSS ranged from 4% to 42.6%, and the prevalence of disabling spasticity ranged from 2% to 13%. The prevalence data on the different phases of the PSS continuum revealed spasticity rates of 4% to 27% in the early time course (1–4 weeks), 19% to 26.7% in the postacute phase (1–3 months), and 17% to 42.6% in the chronic phase (>3 months).[6,11-19] The relatively wide range of these estimates is notable, and several factors may have contributed to the lack of precision. Foremost, sample sizes for the summarized studies were relatively small, and the majority of study patients were recruited from acute hospitals and followed up in rehabilitation units. In some studies, this may have resulted in the exclusion of very severely and/or very mildly affected individuals, thereby limiting the ability to generalize results to a broader group of poststroke patients.

There is an evident lack of robust epidemiologic data for PSS. Regarding further research in this area, the field would benefit from studies that are population-based, cross-sectional surveys or serial longitudinal assessments of large stroke cohorts. Cohort studies of patients with stroke should ideally include a follow-up of all patients, rather than limiting participants to recruits from rehabilitation and/or clinic populations, to avoid survivor effects and improve generalizability. Consistent assessment of spasticity is also critical, which could be improved with the use of valid and reliable assessment tools that would allow for comparison with previously published studies. Additionally, to optimize understanding of the clinical course and document the natural history of motor recovery, assessments at multiple time points—beginning immediately after stroke and continuing for at least 6 months—are important. Finally, spasticity evaluation should assess specific joints in addition to an overall body or global estimate and should present findings based on severity strata.

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