Spasticity After Stroke

Its Occurrence and Association With Motor Impairments and Activity Limitations

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Background and Purpose—There is no consensus concerning the number of patients developing spasticity or the relationship between spasticity and disabilities after acute stroke. The aim of the present study was to describe the extent to which spasticity occurs and is associated with disabilities (motor impairments and activity limitations).

Methods—Ninety-five patients with first-ever stroke were examined initially (mean, 5.4 days) and 3 months after stroke with the Modified Ashworth Scale for spasticity; self-reported muscle stiffness; tendon reflexes; Birgitta Lindmark motor performance; Nine Hole Peg Test for manual dexterity; Rivermead Mobility Index; Get-Up and Go test; and Barthel Index.

Results—Of the 95 patients studied, 64 were hemiparetic, 18 were spastic, 6 reported muscle stiffness, and 18 had increased tendon reflexes 3 months after stroke. Patients who were nonspastic (n=77) had statistically significantly better motor and activity scores than spastic patients (n=18). However, the correlations between muscle tone and disability scores were low, and severe disabilities were seen in almost the same number of nonspastic as spastic patients.

Conclusions—Although spasticity seems to contribute to disabilities after stroke, spasticity was present in only 19% of the patients investigated 3 months after stroke. Severe disabilities were seen in almost the same number of nonspastic as spastic patients. These findings indicate that the focus on spasticity in stroke rehabilitation is out of step with its clinical importance. Careful and continual evaluation to establish the cause of the patient's disabilities is essential before a decision is made on the most proper rehabilitation approach. **(*Stroke*. 2004;35:134-140.)**

Key Words: motor activity ■ muscle spasticity ■ paresis ■ prevalence ■ stroke

Stroke is characterized by sudden onset of clinical signs related to the site in the brain where the morbid process occurs.[1] Damage to the pyramidal tract and its accompanying parapyramidal (corticoreticulospinal) fibers gives rise to the upper motor neuron syndrome,[2] including positive and negative features. Positive features include spasticity and abnormal postures, features that are not normally present. Negative features include those that have been lost such as strength and dexterity. Adaptive features, including physiological, mechanical, and functional changes in muscles and other soft tissues, might also develop.[3]

Initially, some 80% of all patients with stroke experience motor impairments of the contralateral limb(s), ie, hemiparesis.[4] In the early literature, abnormal reflexes associated with spasticity were considered to be the major determinant of these motor impairments.[5,6] A recent study, conducted in a clinical setting, has reported that 39% of patients with first-ever stroke are spastic after 12 months.[7]

Spasticity was described by Lance[2] in 1980 as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper excitability of the stretch reflex." In 1990, Lance[8] reiterated this definition and added that "spasticity does not include impaired voluntary movement and an abnormal posture." In contradiction to Lance's definition of spasticity, the tonic stretch reflex has been shown to have a low correlation with the phasic stretch reflex (tendon jerks).[9] It has also been recognized that increased muscle tone, ie, increased resistance to passive stretch, is due not only to increased reflex activity but also, and perhaps more, to intrinsic changes of the muscles.[3,10] Electromyography (EMG) studies have shown that the reflex-mediated increase in muscle tone reaches its maximum between 1 and 3 months after stroke.[9,11] After 3 months, the eventual increased resistance to passive stretch is proposed to be due to intrinsic changes of the muscles.[11]

Bobath[5] and Brunnström,[6] pioneers of modern physiotherapy, considered spasticity and hemiparesis after stroke to

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almost always coexist. It has now been recognized that hemiparesis after stroke may occur without spasticity.[3] In an EMG-controlled setting, 24 hemiparetic patients were examined within 13 months after first-ever stroke. One half of the patients had increased resistance to passive stretch associated with muscle contracture, but only in a subgroup of the patients (n=5) was the resistance to passive stretch linked to neural components (the tonic stretch reflex).[3] Nevertheless, great attention is still paid to rehabilitation techniques based on the assumption that abnormal reflexes are the main purpose of hemiparesis after stroke.[12]

The exact influence of spasticity on motor impairments and activity limitations in stroke patients is difficult to assess because the degree of spasticity may change according to the position of the subject and the task being performed.[13] It has also been suggested that the hypertonicity of leg extensor muscles enables hemiparetic patients to support their body during locomotion.[14]

There is no consensus concerning the number of patients developing spasticity or the actual relationship between spasticity and disabilities after acute stroke. Few studies cover the subject, and as far as we know, no study has been performed 1 to 3 months after stroke, ie, when eventual spasticity reaches its maximum.

The aim of the present study was to describe to what extent spasticity occurs and is associated with disabilities (motor impairments and activity limitations)[15] initially and 3 months after first-ever stroke.

Subjects and Methods

The patients were consecutively recruited from the Stroke Unit at Danderyd Hospital in the Stockholm area during 10 months (weekends and public holidays not included) in 2001. We included all patients living in Stockholm with an acute first-ever stroke (subarachnoid hemorrhage and cerebellar lesions excluded) with no other diagnosis affecting muscle tone who were conscious and agreed to participate in the study.

One hundred nine patients met the inclusion criteria. Four patients were excluded because of recurrent stroke, and 4 died within 3 months. Six patients could not be assessed after 3 months. Of these, 5 claimed to be fully recovered and declined to participate further in the study; the sixth patient could not be located. Among these 14 patients, mean age was 77±7.5 years (range, 64 to 90 years), 11 were hemiparetic, and 2 were spastic initially after stroke. Thus, 95 patients, 60 women and 35 men, with a mean age of 78±9.5 years (range, 44 to 93 years) completed the study.

The patients were assessed initially (mean, 5.4 days) after acute stroke. All patients were followed up, either in the hospital or in their residence, 3 months after stroke onset with reference to the variables below. Three specially trained physiotherapists conducted the tests.

Muscle Tone Assessments

Spasticity was assessed by the Modified Ashworth Scale (MAS).[16] The scale grades the resistance of a relaxed limb to rapid passive stretch in 6 stages. Zero relates to normal or lowered muscle tone and 4 relates to a state in which passive movement of the affected limb is impossible. In the present study, we tested arm adductors, elbow flexors and extensors, wrist flexors and extensors, and finger flexors with the patient in a sitting position if possible; we also tested hip adductors, knee flexors and extensors, and plantar flexors in patients in the supine position. The MAS is considered fairly reliable.[17]

Self-reported muscle stiffness was assessed by asking the patients if they experienced increased muscle stiffness somewhere in the body.

Tendon reflexes were tested on the biceps and triceps of the upper extremities and the patellar and tendocalcaneous on the lower extremities with a reflex hammer. Plantar flexor tone was additionally assessed by counting number of clonic beats.

Motor Assessments

Motor performance was assessed by the Birgitta Lindmark Motor Assessment (BL),[18] parts 1 and 2 of a total of 7. The BL is considered reliable, valid, and sensitive.[18–20]

For patients who could not actively participate in the tests, motor performance of the affected arm, hand, and leg was assessed by the Scandinavian Stroke Scale (SSS)[21] only to determine whether the patient was hemiparetic or not. The SSS is considered reliable and valid.[22]

Activity Assessments

Manual dexterity was assessed by the Nine Hole Peg Test (NHPT).[23] The standardized equipment consists of 9 pegs (7-mm diameter, 32-mm length), a 100×100×10 container for the pegs, and a wooden board slightly smaller than the container with 9 holes slightly wider than the pegs placed 32 mm apart. The patient was asked to pick up the pegs 1 at a time and put them into the holes as fast as possible using only 1 hand and starting with the unaffected hand or, if not affected on either side, with the dominant hand. Reference values for the right and the left hands[24] were subtracted from the measured values. Then, the sum of the nonaffected hand was subtracted from the sum of the affected hand, and the difference was used to establish the side difference. The NHPT is considered reliable and valid.[23]

Mobility was assessed by the Rivermead Mobility Index (RMI).[25] Patients with <4 points of 15 are considered severely disabled.[26] The RMI is considered reliable, valid, and sensitive.[25,27]

Gait was assessed by the Get-Up and Go test (GUG)[28] at 3 months to evaluate the patient's risk of falling during gait. One point indicates normal gait; 5 points indicate severely abnormal gait. Inability to walk was also registered. The GUG is considered reliable and valid.[28]

Activities of daily living (ADL) were assessed by the Barthel Index (BI).[29] Patients with <35 points of 100 are considered severely disabled.[26,30] The BI is considered reliable, valid, and sensitive.[31]

The clinical scales are presented in Table 1.

To avoid subtle side differences without clinical significance, the patients were regarded as hemiparetic only if they fulfilled ≥1 of the following criteria: ≥5-point difference in scores between the affected and nonaffected side of the upper or lower extremities on the BL active movements; ≥2-point difference in scores between the affected and nonaffected side of the upper or lower extremities on the BL rapid movements, ≥5-second difference in scores between the affected and nonaffected hands on the NHPT, and ≤12 points on the SSS items for motor performance.

Ethics

The procedures in the present study were in accordance with the ethical standards of the responsible committee. Patients were given information saying that participation was voluntary and that they could choose not to participate at any time without having to give a reason.

Statistical Analysis

Descriptive statistics were used to present the number of patients with spasticity according to the MAS, self-reported muscle stiffness, hyperreflexia, and clonic beats and to present number of patients with hemiparesis and severe disabilities. Mann-Whitney *U* test was used for between-group comparisons. Spearman rank-order correlations were used to establish the relationships between muscle tone (spasticity according to the MAS, self-reported muscle stiffness, hyperreflexia, and clonic beats) and the BL, NHPT, RMI, GUG, and BI. Correlation coefficients (positive or, when reversed scales, negative) <0.5 are considered low, those between 0.5 and 0.75 are considered moderate to good, and those >0.75 are considered high.[32]

TABLE 1. Clinical Scales: Scale Value Range and Normal Score

Clinical Scale	Scale Value Range	Normal Score
MAS, spasticity	0, 1, 1+, 2, 3, 4	0*
Self-reported muscle stiffness	Yes/no	No
Tendon reflexes	Increased/not increased	Not increased
Clonic beats in plantar flexors	Present/not present	Not present
BL motor assessment:		
Active movements, upper extremities	0–57 for each side, 19 subtests	57 for each side
Active movements, lower extremities	0–36 for each side, 12 subtests	36 for each side
Rapid movements, upper extremities	0–6 for each side, 2 subtests	6 for each side
Rapid movements, lower extremities	0–6 for each side, 2 subtests	6 for each side
SSS, unilateral motor performance	0–18 (arm, hand, leg)	18 for affected side
NHPT, manual dexterity	Time (s)	<5 s side difference†
RMI, mobility	0–15	15
GUG, gait (risk of falling)	1–5	1
BI, ADL	0–100	100

*If >0 on the MAS, patient was considered spastic.
†See Subjects and Methods.

Significance level was set at $P<0.05$. Data were analyzed by use of Statistica 5.1 for Windows.

Results

Of all 95 patients, 77 (81%) were initially hemiparetic, and 20 (21%) were spastic. Of the 77 hemiparetic patients, 20 (26%) were spastic. Six patients were spastic in both the upper and lower extremity, 13 in the upper extremity only, and 1 in the lower extremity only. The highest estimated MAS scores were 0 (n=75), 1 (n=10), 1+ (n=7), 2 (n=3), 3 (n=0), and 4 (n=0). Among the spastic patients, 14 showed hyperreflexia (7 in both the upper and lower extremity, 7 in the upper extremity only). Of these, 3 also showed clonic beats, and 3 reported the experience of muscle stiffness.

Of all 95 patients, 64 (67%) were hemiparetic 3 months after stroke, and 18 (19%) were spastic. Of the 64 hemiparetic patients, 18 (28%) were spastic. Ten patients were spastic in both the upper and lower extremity, 7 in the upper extremity only, and 1 in the lower extremity only. The highest estimated MAS scores were 0 (n=77), 1 (n=8), 1+ (n=5), 2 (n=4), 3 (n=1), and 4 (n=0). Among the spastic patients, 12 showed hyperreflexia (all in the upper extremity and 3 also in the lower extremity). Of these, 7 also showed clonic beats, and 6 reported muscle stiffness.

The numbers of patients with spasticity, self-reported muscle stiffness, hyperreflexia, and clonic beats are shown in Figure 1. Comparisons between the spastic and nonspastic patients with reference to the motor and activity tests are shown in Table 2.

Correlations between muscle tone and the motor and activity scores were overall low ($r<0.5$ $P<0.05$), except for the initial upper-extremity MAS and BL active movements scores ($r=0.51$ $P<0.001$) and for the 3-month upper-extremity MAS and BL active movements scores ($r=0.64$ $P<0.001$), rapid movements scores ($r=0.54$ $P<0.001$), and NHPT scores ($r=0.59$ $P<0.001$).

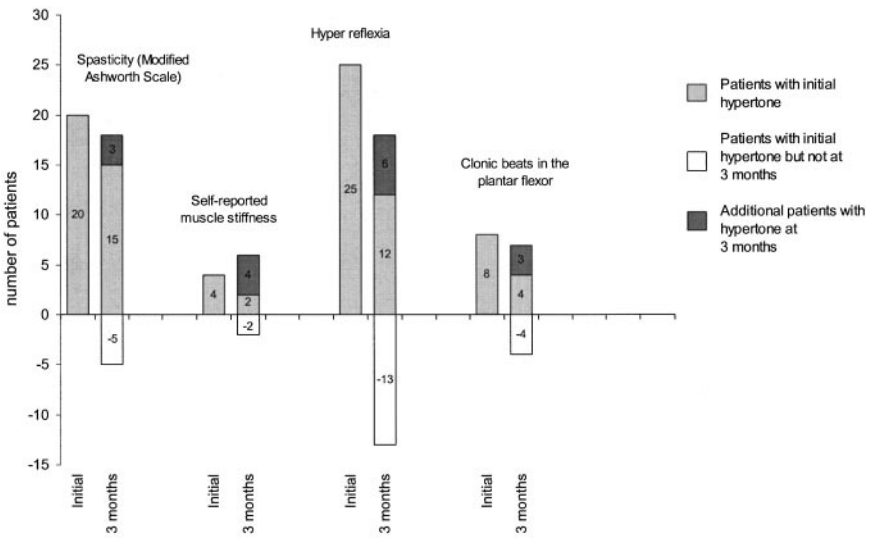

Figure 1. Muscular hypertonia (spasticity, self-reported muscle stiffness, hyperreflexia, and clonic beats in the plantar flexors) among 95 investigated patients.

TABLE 2. Comparison Between Spastic and Nonspastic Patients According to BL Active and Rapid Movements in the Upper and Lower Extremities, NHPT, RMI, GUG, and BI Initially and 3 Months After Stroke

	Median Scores (IQR)					
	UE			LE		
	Spastic	Nonspastic	*P*	Spastic	Nonspastic	*P*
BLa-UE						
Initially	0 (0–12)	56 (33–57)	<0.001	*	*	*
At 3 mo	3 (0–30)	57 (55–57)	<0.001	*	*	*
BLa-LE						
Initially	*	*	*	2 (0–10)	31.5 (19–36)	<0.001
At 3 mo	*	*	*	12 (2–31)	36 (34–36)	<0.001
BLr-UE						
Initially	0 (0–2)	4 (3–4)	<0.001	*	*	*
At 3 mo	0 (0–4)	5 (4–6)	<0.001	*	*	*
BLr-LE						
Initially	*	*	*	0 (0–2)	4 (2–6)	<0.001
At 3 mo	*	*	*	0 (0–2)	5.5 (4–6)	<0.001
NHPT						
Initially	†	†	†	*	*	*
At 3 mo	>60 (>60)	6 (18–3)	<0.001	*	*	*
RMI						
Initially	1 (0–2)	5 (2–13)	<0.001	1 (0–2)	4 (1–13)	<0.05
At 3 mo	2 (1–7)	12.5 (8–14)	<0.001	2 (2–7)	12 (8–14)	<0.001
GUG						
Initially	*	*	*	*	*	*
At 3 mo	>5 (>5–3)	2 (2–1)	<0.001	>5 (>5–3)	2 (3–1)	<0.001
BI						
Initially	10 (5–40)	70 (35–100)	<0.001	10 (5–40)	65 (15–100)	<0.05
At 3 mo	35 (25–80)	100 (85–100)	<0.001	37.5 (35–80)	100 (77.5–100)	<0.001

IQR indicates interquartile range; UE, upper extremity; LE, lower extremity; BLa, BL active; and BLr, BL rapid.
*Not applicable.
†Not calculable (n=5 in the spastic group).

Hemiparesis and severe disabilities 3 months after stroke among patients with and without spasticity in the upper and lower extremities, respectively, are shown in Figure 2.

Discussion

In the present study, we focused on the occurrence of spasticity and its association with motor impairments and activity limitations initially and 3 months after first-ever stroke. Of the 95 patients studied, 21% were initially spastic according to the MAS; 3 months after stroke, 19% were spastic. One third of the spastic patients experienced muscle stiffness. Spasticity was more frequent in the upper than the lower extremities. Three months after stroke, the patients who were nonspastic (n=77) had statistically significantly better motor and activity scores than patients who were spastic (n=18). However, the correlation between muscle tone and the motor and activity scores was overall low, and severe motor and activity problems were seen in almost the same number of nonspastic as spastic patients.

Consistent with earlier findings,[4] 81% of the stroke patients seen in the present study were initially hemiparetic. Three months after stroke, 67% were still hemiparetic, and 19% were spastic. In a resent study from the United Kingdom, 23 of 59 patients (39%) in a clinical setting were spastic 12 months after first-ever stroke.[7] Because of the "late" follow-up, not only neural components but also adaptive features such as intrinsic changes of the muscles may have contributed to the number of spastic patients in that study. The relatively low incidence of spasticity among the hemiparetic patients (28%) in the present study was in accordance with those of O'Dwyer and coworkers,[3] who found EMG-verified spasticity in only 21% of the hemiparetic stroke patients assessed 13 months after stroke.

It is well recognized that spasticity after stroke may interfere with motor and activity performance, cause pain, and lead to secondary complications.[31,33] Initially, the limb may be flaccid and then tone is supposed to emerge, followed by increasing spasticity.[31] The use of a neurodevelopmental approach, focusing on normalizing tone and movement patterns, is widespread and claims that inhibition of spasticity should result in an improved motor function.[34,35] Some studies have reported reduced spasticity and increased activ-

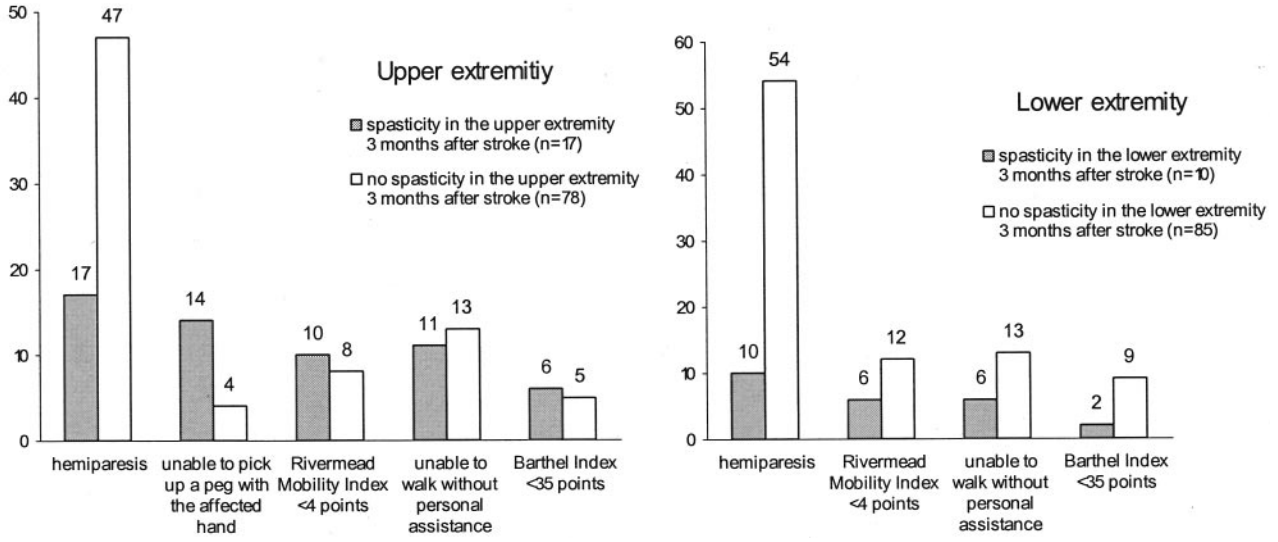

Figure 2. Hemiparesis and severe disabilities 3 months after stroke among patients with and without spasticity in the upper and lower extremities, respectively.

ity performance after botulinum toxin injections in stroke patients.[36,37] However, there is no evidence that suppression of spasticity by either physiotherapy or medication results in parallel improvements in motor function.[35] Controlled outcome studies have also failed to demonstrate the superiority of any treatment approach in stroke rehabilitation.[38]

In the present study, we excluded patients with recurrent stroke or diseases affecting muscle tone. This was to ensure, as far as possible, that the eventual increased resistance to passive stretch reflected an increased tonic stretch reflex resulting from the present stroke rather than from neural or soft tissues changes caused by earlier stroke events or other neurological deficits. Because spasticity after stroke has been shown to reach its peak 1 to 3 months after onset,[9,11] we chose a 3-month follow-up. We found spastic patients at 3 months who were not initially spastic, as well as patients with normal muscle tone at 3 months who were initially spastic and/or had increased tendon reflexes (Figure 1). Initial transient cerebral edema and circulation disturbances may be reasonable explanations for this latter phenomenon, thus emphasizing the need for continual evaluation of these patients.

The MAS is often used in clinical practice and research to measure spasticity.[39] The MAS measures resistance to passive stretch, ie, both the tonic stretch reflex and possible intrinsic changes of the muscles; thus, it can be criticized for only reflecting muscle tone of a relaxed limb and for not giving information about activated muscles. We also measured the tendon jerk reflexes, ie, the phasic stretch reflex. In accordance with earlier findings,[9] we found that not all patients with an increased resistance to stretch (>0 on the MAS) showed increased tendon jerks and vice versa. It has also been recognized that the tonic stretch reflex is of greater clinical significance than the phasic stretch reflex.[3] Additionally, we found only 6 patients who experienced muscle stiffness 3 months after stroke, thus emphasizing the low incidence of spasticity/muscle stiffness among the patients in the present study.

In summary, spasticity seems to contribute to motor impairments and activity limitations and may be a severe problem for some patients after stroke. However, most patients (81%) in the present study were nonspastic, and among hemiparetic patients, only 28% were spastic 3 months after stroke. Also, severe motor and activity problems were seen in almost the same number of nonspastic as spastic patients. Our findings support the opinion of O'Dwyer and coworkers[3] that the focus on spasticity in stroke rehabilitation is out of step with its clinical importance. Careful and continual evaluation to establish the causes of a patient's disabilities is essential before a decision is made on the most proper rehabilitation approach.

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Editorial Comment

Spasticity After Stroke: What's the Catch?

Motor deficits are the most common impairment acutely after stroke and persist in nearly half of all patients.[1,2] Although much focus is on hemiparesis in this setting, injury to the motor system does not produce a homogenous clinical syndrome. Instead, weakness may be accompanied by other negative findings such as slowness and fatigue and by positive findings such as synkinesia and spasticity.

Spasticity is a state of increased tone with exaggerated reflexes resulting from upper motor neuron injury. It is a condition of many contrasts. Reduced activity in one area, the descending motor tracts, results in increased activity in another area, the skeletal muscles. Spasticity is common across neurological conditions, yet accurate measurement is difficult. It is associated with weakness, yet its maintenance is critical to function in some patients. Importantly, spasticity remains a key dividing point among major schools of physiotherapy, with some aiming to inhibit[3] and others aiming to encourage[4] spasticity and its accompanying motor abnormalities. The medical system expends substantial resources to reduce spasticity with methods that include botulinum toxin injection, intrathecal medication, oral pharmacological agents, and physical/occupational therapy. Yet, limited information is available on its prevalence and significance after stroke. Indeed, in a recent review, Barnes[5] noted the limited availability of quality data on the prevalence of spasticity after stroke.

Some of those data are now available. Sommerfeld et al[6] studied consecutive patients with a first stroke over a 10-month period. Among 95 patients assessed a mean of 5 days after stroke, 21% had spasticity and 81% had hemiparesis. Three months later, 19% were spastic and 67% were hemiparetic. Of note, only 28% of the hemiparetic patients had spasticity. A weakness of the study is that the authors provided limited detail as to precisely which muscles were affected by spasticity. Values for spasticity prevalence in this study are not likely an underestimation, because any measurable increase in tone was considered to constitute spasticity. Study results suggest that although spasticity is associated with greater deficits

and disability, it is present in a minority of stroke patients and in a minority of hemiparetic stroke patients.

The indications for reducing spasticity after stroke remain a topic of ongoing investigation. A compelling argument can be made for treating spasticity after stroke in certain specific instances, eg, when the goal is to prevent an incipient contracture or to reduce a regional pain syndrome such as that associated with a hemiplegic shoulder.[7–9] However, improvement in overall coordinated movement or in disability after stroke as a general response to reduction of spasticity remains to be firmly established.[10] Indeed, treatments targeting spasticity have often had difficulty demonstrating functional benefit.[11] Dobkin[8] recently noted in this context, "With the exception of lessening painful or disruptive spasms and dystonic postures, drugs in general do not decrease impairments or lessen disabilities."

A range of additional studies is needed to refine guidelines for treating spasticity after stroke. As with so many aspects of stroke, response to spasticity-related therapy may be maximum in a subset of patients or may be realized in performance of a subset of motor tasks.[12] The effects of such therapy may be best measured not by general neurological outcome scales but rather by the use of end points most relevant to effects of spasticity.[13] Newer instrumentation-based methods might also improve measurement of spasticity.[14,15] Clinical trials may further clarify the utility of specific approaches to reduce spasticity. The study by Sommerfeld et al,[6] by providing quality data on the prevalence and functional significance of spasticity after stroke, is an important step.

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