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Occurence and Clinical Predictors of Spasticity After Ischemic Stroke

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Background and Purpose—There is currently no consensus on (1) the percentage of patients who develop spasticity after ischemic stroke, (2) the relation between spasticity and initial clinical findings after acute stroke, and (3) the impact of spasticity on activities of daily living and health-related quality of life.

Methods—In a prospective cohort study, 301 consecutive patients with clinical signs of central paresis due to a first-ever ischemic stroke were examined in the acute stage and 6 months later. At both times, the degree and pattern of paresis and muscle tone, the Barthel Index, and the EQ-5D score, a standardized instrument of health-related quality of life, were evaluated. Spasticity was assessed on the Modified Ashworth Scale and defined as Modified Ashworth Scale >1 in any of the examined joints.

Results—Two hundred eleven patients (70.1%) were reassessed after 6 months. Of these, 42.6% (n=90) had developed spasticity. A more severe degree of spasticity (Modified Ashworth Scale ≥ 3) was observed in 15.6% of all patients. The prevalence of spasticity did not differ between upper and lower limbs, but in the upper limb muscles, higher degrees of spasticity (Modified Ashworth Scale ≥ 3) were more frequently (18.9%) observed than in the lower limbs (5.5%). Regression analysis used to test the differences between upper and lower limbs showed that patients with more severe paresis in the proximal and distal limb muscles had a higher risk for developing spasticity ($P \leq 0.001$). Spasticity of the upper and lower limb was more frequent in patients with hemihypesthesia than in patients without sensory deficits ($P \leq 0.001$). Patients with spasticity showed a lower Barthel Index and EQ-5D score compared with the group without spasticity.

Conclusions—Spasticity was present in 42.6% of patients with initial central paresis. However, severe spasticity was relatively rare. Predictors for the development of spasticity were a severe degree of paresis and hemihypesthesia at stroke onset. **(*Stroke*. 2010;41:2016-2020.)**

Key Words: epidemiology ■ rehabilitation ■ stroke care ■ spasticity

Stroke is a leading cause of morbidity and mortality in Western countries,[1] and motor deficits are the most common impairment after acute stroke and persist in nearly half of all patients.[2] Damage to the pyramidal tract and its accompanying parapyramidal (corticoreticulospinal) fibers results in the upper motor neuron syndrome, which has positive and negative features. Negative components include the loss of strength and dexterity; positive features include spasticity and abnormal postures, characteristics that are not normally present. Spasticity is a state of increased muscle tone with exaggerated reflexes. Although muscle weakness and the loss of dexterity are important factors of motor function disability in these patients, the contribution of muscle spasticity is often significant. The clinical evaluation of spasticity includes an assessment of the velocity-dependent increase in resistance to passive movements as the key element of spasticity, as defined by Lance.[3] Despite its high incidence, there is currently no consensus on (1) the number of patients who develop spasticity after ischemic motor stroke and (2) the relation between spasticity and initial clinical findings after acute stroke, especially in regard to the distribution, pattern, and degree of paresis in different muscle groups. The aim of the present study was therefore to investigate the prevalence of spasticity after motor stroke and to identify clinical predictors of subsequent spasticity.

Subjects and Methods

In a prospective, longitudinal study, all stroke patients admitted to the stroke unit at the Department of Neurology of the University Hospital Mainz were consecutively recruited during a 22-month

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period. This department is exclusively responsible for the neurologic care of a region with ≈600 000 inhabitants, and all stroke patients without any selection were accepted. These were patients with acute symptoms of stroke within the last 24 hours and/or fluctuating or progressive symptoms over a longer period of time according to the recommendations of the German Stroke Society. The presence of stroke was determined by an experienced neurologist after exclusion of other differential diagnoses. Included were all patients with clinical signs of a manifest or latent central paresis (limb monoparesis or hemiparesis) due to a first-ever ischemic stroke. Exclusion criteria were previous brain lesions independent of their etiology, severe aphasia and/or motor neglect that precluded grading of paresis, transient ischemic attack, other stroke etiologies, relevant brain lesions on magnetic resonance imaging or computed tomography independent of etiology, and other neurologic diseases. A total of 1484 patients were screened. Of these, 301 patients fulfilled the inclusion and exclusion criteria and were initially assessed within 5 days after acute stroke.

Of the group of 301 patients, 211 (70.1%) were reassessed after 6 months (±1 week) in their homes. Of the remaining 90 patients, 41 were unavailable for reassessment, 24 had died, 16 refused reassessment, and 9 had a recurrent stroke within this period and were therefore excluded from reassessment. The data of 211 patients obtained at the time of the initial examination and at follow-up were available for analysis.

Muscle Tone and Motor Assessment

Each patient was examined in the acute stage and 6 months later (T.W. and M.U.) by the same person. Thus, no interrater variability needs to be considered. At the time of examination, no patient used oral antispastic drugs or had been treated with botulinum toxin.

Spasticity was assessed on the Modified Ashworth Scale (MAS).[4] 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 5 relates to a state in which passive movement of the affected limb is impossible. Evaluation of spasticity included passive flexion and extension movements of different joints of the upper and lower limbs. In the present study, we tested arm abductors and 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 and extensors in patients in the supine position. Any spasticity was defined as an MAS score ≥1 for any of the passive movements performed. The MAS is considered to provide fairly reliable and reproducible results.[5] Furthermore, a detailed history was obtained, and we observed related activities during examination to capture any activity-related manifestations of spasticity.

Motor performance was assessed by testing muscle power on the British Medical Research Council (BMRC) scale (grades 0 to 5) for all muscles that were also tested for muscle tone.[6] For statistical analysis, we modified this scale and defined no paresis as grade 0 (BMRC grade 5), slight paresis as grade 1 (BMRC grade 4), moderate paresis as grade 2 (BMRC grades 3 and 2), and severe paresis as grade 3 (BMRC grades 1 and 0).[7] The grading of muscle strength was carried out for proximal and distal muscle groups. The proximal group included the shoulder and elbow for the upper limb and hip and knee for the lower limb; the distal group included hand and finger for the upper limb and foot for the lower limb.

Assessment of Activities of Daily Living and Quality of Life

Activities of daily living were assessed with the Barthel Index (BI).[8] The BI is considered to be reliable, valid, and sensitive.[9] Quality of life was assessed by the EQ-5D score with a visual analog scale ranging from 0 (worst imaginable health state) to 100 (best imaginable health state), developed by the EuroQuol Group.[10] The score has been shown to be reliable and valid.[11]

Table 1. Stroke Onset Characteristics of Participants (N=211)

Characteristics	Value
Age, mean (SD), median, interquartile range, y	68 (13), 70, 59–78
Sex	
Male, n (%)	131 (62)
Female, n (%)	80 (38)
NIHSS score, mean (SD)	6.6 (4.6)
Length of stay in hospital, d (SD)	10.4 (4.3)
Length of stay on stroke unit, d (SD)	4.2 (1.6)
Discharged to	
Rehabilitation clinic, n (%)	158 (74.9)
Home, n (%)	53 (25.1)

NIHSS indicates National Institutes of Health Stroke Scale.

Statistical Analysis

Analysis was performed with SPSS version 15.0.1. Bar graphs and tables demonstrate the prevalence and degree of spasticity and the degree of paresis in the cohort. Differences between groups were tested with the χ^2 test for categorical variables and the Kruskal-Wallis and Mann-Whitney U tests for continuous variables. When expected values were <5, Fisher exact test was used. A multiple logistic-regression analysis was performed to investigate the potential influence between factors identified in the univariate analysis associated with spasticity. Logistic-regression analysis with a stepwise, forward, likelihood-ratio variable selection was used. CIs at the 95% level were calculated for the odds ratio. All probability values are 2 sided. Because these analyses were not adjusted for multiple testing, the results cannot be considered significant at any level, and probability values were calculated for the purpose of description only.

Ethics

The present study was approved by the local ethics committee (LÄK Rheinland-Pfalz).

Results

Demographic data of the 211 patients reexamined 6 months after stroke are summarized in Table 1. The mean age of the 211 patients who completed the study was 68±13 years (range, 17 to 96 years; normally distributed Kolmogorov-Smirnov test, $z=1.357$, $P=0.05$), and there were 131 men and 80 women. The National Institutes of Health Stroke Scale score at the time of admission was 6.6±4.6 (range, 1 to 24). With regard to the data from the 90 patients not included, this group did not differ from the study group regarding age (68±15 years; range, 33 to 92 years) or the distribution and severity of paresis.

Patients stayed for 4.2±1.6 days on the stroke unit. The total hospital stay was 10.4±4.3 days (range, 2 to 25 days). After discharge from hospital, 158 patients (74.9%) were referred to a rehabilitation clinic, and 53 patients (25.1%) returned to their homes. Thirty-seven patients (17.5%) of the latter group received further physiotherapy in their homes. More patients with spasticity had a longer stay in a rehabilitation clinic (<2 weeks, 3.9%; 3 to 4 weeks, 42.9%; >5 weeks, 53.2%) than did those patients without spasticity (<2 weeks, 4.9%; 3 to 4 weeks, 59.3%; >5 weeks, 35.8%).

Of the 211 patients evaluated after 6 months, 199 were at home and 12 were in a nursing home. The 211 patients included in the study had different degrees of paresis in the

Table 2. Degree of Initial Paresis of the Proximal and Distal Upper and Lower Limbs

Paresis/Limb	n	Percent
Degree of paresis, distal upper limb		
0	6	2.8
1	75	35.5
2	88	41.7
3	42	19.9
Total	211	100.0
Degree of paresis, proximal upper limb		
0	4	1.9
1	96	45.5
2	69	32.7
3	42	19.9
Total	211	100.0
Degree of paresis, distal lower limb		
0	8	3.8
1	111	52.6
2	61	28.9
3	31	14.7
Total	211	100.0
Degree of paresis, proximal upper limb		
1	119	56.4
2	59	28.0
3	33	15.6
Total	211	100.0

Paresis was graded as follows: 0=no paresis, 1=slight paresis; 2=moderate paresis, and 3=severe paresis.

upper and lower limbs after stroke; most commonly observed was a slight or moderate degree of paresis (Table 2). Upper limb muscles were generally more affected than lower limb muscles, whereas the degree of paresis was more or less equally distributed in the distal and proximal muscles of the upper and lower limbs.

Occurrence of Spasticity

Of the 211 patients with initial central paresis, 42.6% (n=90) had developed spasticity by 6 months after stroke. Spasticity was present in both the upper and lower limbs in 27.0% (n=57), in the upper limb only in 8.5% (n=18), and in the lower limb only in 7.1% (n=15) of patients.

Severity and Distribution of Spasticity

Most patients showed only a slight increase in muscle tone (Table 3). A more severe degree of spasticity (MAS ≥3) was observed in only 15.6% of all patients with spasticity. Although the prevalence of spasticity did not differ between upper and lower limbs, higher degrees of spasticity (MAS ≥3) were more frequent in upper limb (18.9%) than in lower limb (5.5%) muscles. However, disabling spastic postures of the hand and fingers, such as a clenched hand, were persistent in only 2 patients and transient in another 8. The degree of spasticity differed considerably between the antagonists of the different joints. The muscles responsible for an antigravity posture generally showed a higher degree of spasticity (Table 4).

Table 3. MAS Score of the Upper and Lower Limbs 6 Months After Ischemic Stroke

MAS/Limb	n	Cumulative Percentage
Mean MAS score, upper limbs		
≤0.99	31	41.9
1–1.99	18	66.2
2–2.99	11	81.1
3–3.99	10	94.6
≥4	4	100
Total	74	
Mean MAS score, lower limbs		
≤0.99	39	53.4
1–1.99	23	84.9
2–2.99	7	94.5
3–3.9	3	98.6
≥4	1	100
Total	73	

Relation Between Spasticity and Other Variables

Logistic-regression analysis confirmed an interaction between the degree of paresis during the acute stage in the proximal and distal muscles of the upper and lower limbs and the development of spasticity ($P \leq 0.001$). In comparison with patients with a severe paresis, patients with moderate paresis (odds ratio=0.23; 95% CI, 0.10 to 0.54; $P=0.001$) and mild paresis (odds ratio=0.15; 95% CI, 0.07 to 0.35; $P \leq 0.001$) showed a decreased risk for spasticity in the upper limbs. Differences between patients with moderate paresis and mild paresis in regard to spasticity score were not determined ($P>0.05$, Mann-Whitney U test). Patients with hemihypesthesia showed an increased risk for the development of spasticity (odds ratio=2.27; 95% CI, 1.21 to 4.28; $P=0.011$). Included in this analysis was only the sensation to light touch,

Table 4. MAS Score of Different Joints of the Upper and Lower Limbs

Joints	MAS
Upper limb	
Shoulder adductor	1.99
Shoulder abductor	0.23
Elbow extensor	1.41
Elbow flexor	1.94
Wrist extensor	0.66
Wrist flexor	1.86
Finger extensor	0.48
Finger flexor	1.23
Lower limb	
Hip extensor	0.49
Hip flexor	0.31
Knee extensor	0.70
Knee flexor	1.37
Ankle extensor	0.11
Ankle flexor	1.64

whereas other qualities such as joint motion, vibration, or heat and pain sensation were not systematically tested. An influence of age, sex, or the affected hemisphere was not identified.

The median BI was 80 (mean, 71.19; interquartile range, 50 to 100) in the acute phase (n=156) and 100 (mean, 90.38; interquartile range, 90 to 100) 6 months later (n=156). In patients with spasticity, a lower BI was found 6 months later (median, 90; mean, 82.47; interquartile range, 65 to 100) compared with patients without spasticity (n=81) (median, 100; mean, 97.72; interquartile range, 100 to 100; Mann-Whitney *U* test, $P<0.001$). Patients with spasticity of the upper and lower limb had a lower BI (median, 85) compared with patients with spasticity in 1 limb only (median, 100) and patients without spasticity (median, 100; Mann-Whitney *U* test, $P=0.001$).

The mean EQ-5D score (n=155) in the acute stage was 49.6 (median, 50.0) and 58.3 (median, 60.0) 6 months later. Patients with spasticity (n=75) showed a lower mean score of 53.6 (median, 50) compared with patients without spasticity (m=80; mean, 62.7; median, 61.50; Mann-Whitney *U* test, $P<0.001$).

Discussion

In this prospective, longitudinal study, we investigated a large cohort of acute stroke patients and focused on the prevalence of spasticity 6 months after stroke. We restricted our analysis to patients with initial limb paresis independent of its severity and excluded patients with nonmotor strokes and transient ischemic attack. Of a large number of screened stroke patients (N=1484), only 301 fulfilled the inclusion and exclusion criteria. Two hundred eleven patients (70.1%) were available for a face-to-face reexamination 6 months later. The remaining 90 patients were excluded due to recurrent stroke, death, unavailability, or refusal of reassessment. Of the cohort of 211 patients, 90 (42.6%) showed spasticity in at least 1 limb.

In a previous study including 95 patients, the prevalence of spasticity was 19% for the entire group of patients.[12] Sixty-four of the 95 patients had initial hemiparesis, and 18 (28%) developed spasticity 3 months later.[12] The lower prevalence of spasticity compared with our results might be due to the smaller number of patients included, as well as the inclusion of patients with transient ischemic attack and stroke etiologies other than ischemic stroke, a shorter interval between acute stroke and reexamination, and the nonidentity of examiners performing the initial examination and the reexamination.

In another study that reexamined 83 patients 16 weeks (median) after ischemic or hemorrhagic stroke, 21.7% of patients developed a spastic increase of muscle tone in at least 1 joint.[13] Weakness was present in 62% of patients, and higher MAS scores were observed in those patients with more severe paresis at the first examination at 6 days (median) after stroke.

In 2 other publications, the 12-month poststroke prevalence of spasticity was reported to be 38%[14] and 17%[15] for all stroke patients (N=106 and 140, respectively). The reexamination rate in the study of Lundström et al[15] was 86%. In the study of Watkins et al,[14] the reexamination rate was extremely low (39% compared with 70% in our study), in addition to which patients who had had a previous stroke were also included. Furthermore, it is not apparent from the study design whether only ambulatory patients were reexamined, an additional factor contributing to an underestimation of the true prevalence rate of spasticity.

The studies of Watkins et al[14] and Lundström et al[15] did not differentiate between initial motor or nonmotor strokes regarding the prevalence of spasticity. The reported prevalence rates are therefore not suitable for a direct comparison with our data. Furthermore, both studies also included patients with intracerebral bleeding, and the examiners at the initial examination and reexamination were not identical. Furthermore, owing to the late follow-up of 12 months, a substantial number of patients with severe hemiparesis might have been lost to follow-up, causing the prevalence rate to be underestimated. After 1 year, spasticity may be due not only to damage to neural components but also to adaptive features such as intrinsic changes of the muscle.[16,17] Corresponding to results obtained by other studies, the presence of spasticity was not influenced by sex, age, or the affected hemisphere.[12,14,15]

The degree of spasticity in our cohort was generally low to moderate, showing an MAS $\geq$3 in 18.9% and in 5.5% of patients with spasticity in the upper and lower limbs, respectively. A clinically relevant degree of spasticity (MAS $\geq$3) thus occurred in only 15.6% of all patients with ischemic motor stroke and spasticity. The severity of spasticity is relatively similar to the findings of Watkins et al,[14] who reported an MAS $\geq$3 in 22.6% and in 17.1% of patients with upper and lower limb spasticity, respectively. Disabling spastic postures of the hand and fingers, such as a clenched hand, were persistent in only 2 patients and transitory in 8 patients of our study. The finding that only a small number of patients developed some degree of spasticity suggests that botulinum toxin treatment might represent a therapeutic option. In a previous study of patients with wrist and finger spasticity after stroke, patients with Ashworth Scores of 3 and 4 (4 and 5 on the MAS) for wrist flexors and of $\geq$2 (3 on the MAS) for finger flexors in fact received botulinum toxin therapy. Aspects of treatment are, however, beyond the scope of this study. Although the majority of patients in our study participated in a rehabilitation program after stroke, we did not have the opportunity to survey the rehabilitation interventions in which the patients participated. Interestingly, none of these patients had been treated with intramuscular injections of botulinum toxin, and no patient was taking oral antispastic drugs at the time of examination.

Although there was no difference in the prevalence of spasticity between the upper and lower limbs, spasticity was more severe in upper limb than in lower limb muscles. The distribution of spasticity in the different joints corresponded to the antigravity posture, which constitutes the typical spasticity pattern of poststroke hemiparesis.[18] However, the mechanisms underlying the antigravity posture are still obscure. Hypothetically, an impaired vestibulospinal function mediating otolith contributions to postural control is relevant for the development of the antigravity posture.[19]

We further analyzed the topical distribution of the initial paresis with a view to the later prevalence of spasticity. We were able to demonstrate for the upper and lower limbs that

patients with severe paresis in the acute stage had higher spasticity scores than did patients with a slight paresis, which confirms previous observations.[7,15] These findings strongly suggest the need for thorough follow-up and increased awareness of the development of spasticity in patients with severe paresis.

An additional finding of this study is that patients with hemihypesthesia are more often affected by spasticity of the upper and lower limb than are patients without sensory deficits (Fisher test $P \leq 0.001$). However, when only 1 limb showed spastic muscle tone, no relation between sensory findings and spasticity was detected. This observation does not necessarily imply a causal relation, but it might be attributable to the close topographic relation to the damaged pyramidal tract fibers. Similar to the findings reported by Lundström et al[15] and Watkins et al,[14] we did not identify an influence of sex and age on the occurrence of spasticity. We were further able to demonstrate that the presence of spasticity had an impact on quality of life, as reflected in the EQ-5D score, as well as on activities of daily living, as shown by the BI.

However, the present study has some limitations. Owing to a number of strict exclusion criteria, only 301 of 1484 screened patients were included, which introduced a selection bias. Another potential selection bias is the loss of 29.9% of patients to follow-up. The follow-up interval of 6 months was determined from previous studies.[20,21] Thus, one has to consider that the described percentage of patients with spasticity is true only at this time. We are unaware of studies that have prospectively investigated the development of spasticity over a longer period of time at short intervals, so the time course of spasticity is still unknown.

In summary, spasticity is a frequent occurrence after ischemic stroke, although in most cases, it does not lead to a substantial increase in muscle tone. Initial severe paresis and hemihypesthesia were associated with a higher risk for the development of spasticity.

Disclosures

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