

# Prevalence of spasticity following stroke and its impact on quality of life with emphasis on disability in activities of daily living. Systematic review

## *Spastyczność po udarze mózgu – częstość występowania, wpływ na jakość życia i funkcjonowanie chorego. Przegląd systematyczny*

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### Abstract

Spasticity is characterized by a velocity-dependent increase in muscle tone related to disturbed sensory-motor control of muscle tone following upper motor neuron damage. Spasticity and its clinical implications are still poorly described. There is no consensus concerning the number of patients developing spasticity or the relationship between spasticity and motor disabilities after stroke. Surprisingly, only a few studies have addressed the prevalence of spasticity following stroke. The present paper aims to review recent studies on prevalence of spasticity, its risk factors and on quality of life with emphasis on disability in activities of daily living and to relate collected data to situation in Poland.

**Key words:** spasticity, prevalence, activity of daily living, quality of life, stroke.

### Introduction

Stroke is the most common cause of upper motor neuron syndrome in adults. In the United States every year 750 000 people suffer from stroke and its incidence

### Streszczenie

Spastyczność i jej kliniczne znaczenie są wciąż mało poznane. Spastyczność cechuje się wzrostem napięcia mięśniowego zależnym od rozciągania mięśnia w wyniku utraty kontroli czuciowo-ruchowej napięcia mięśniowego w przebiegu uszkodzenia górnego neuronu ruchowego. Częstość występowania spastyczności poudarowej oraz jej wpływ na gorsze funkcjonowanie i niesprawność chorego wciąż budzą wiele kontrowersji. Co ciekawe, w bazach danych dostępnych jest jedynie kilka prac podejmujących problem częstości występowania spastyczności, czynników ryzyka jej rozwoju oraz jakości życia, ze szczególnym uwzględnieniem niesprawności w codziennym życiu chorych ze spastycznością po udarze mózgu. Celem tego przeglądu jest analiza dotychczasowych publikacji z próbą odniesienia do sytuacji w Polsce.

**Słowa kluczowe:** spastyczność, częstość występowania, funkcjonowanie, jakość życia, udar mózgu.

varies from 200 to 300 individuals per 100 000 [1]. Lower incidence is reported in Europe: 113 per 100 000 per year [2], and in Poland, with a population of 38 million inhabitants, about 60 000 patients are hospitalized because of stroke every year [3].

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Upper motor neuron (UMN) syndrome with its negative and positive features (Table 1) is a clinical manifestation of predominantly extrapyramidal (cortico-reticulo-spinal) tract damage and, to a lesser degree, pyramidal tract damage [4]. Hence, a lesion involving cortico-reticulo-spinal fibres leads to decreased inhibition (or to increased facilitation) of the spinal cord, and ultimately to spasticity [5]. In clinical practice spasticity is characterized by the 'clasp-knife' phenomenon (increasing muscle tone to some point with sudden release during passive stretching of the affected limb) contrary to e.g. parkinsonian stiffness, where increased muscle tone is rather constant during the whole range of movement [6].

Spasticity was defined by Lance in 1980 as 'a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex' [7]. In 1990, Lance reformulated the previous definition, adding that 'spasticity does not include impaired voluntary movement and an abnormal posture' [8]. In agreement with recent studies, the current definition of spasticity proposed by Pandyan *et al.* could be summarized as follows: 'spasticity is a phenomenon of disturbed sensory-motor control of muscle tone connected with upper motor neuron damage resulting in intermittent or sustained, involuntary, muscle tone hyperactivity' [9]. Spasticity is also accompanied by a decreased range of movements, abnormal postures (stigmatization), problems with hygiene, dressing, mobility, transportation and pain. If not treated, it results in muscle shortening, fibrosis, calcification and fixed contractures. Although spasticity is not the sole sign of UMN syndrome, it is one of the major factors contributing to handicap. One may expect that reducing spasticity may improve to some degree disturbed motor function and quality of life [10].

Various methods of treatment are recommended to reduce spasticity, including physiotherapy combined with pharmacological intervention. Moreover, spasticity is the only aspect of UMN syndrome which can be pharmacologically treated [11]. Botulinum neurotoxin (BoNT) injections are currently considered the first-line treatment of moderate and severe focal spasticity of upper limb after stroke [12,13].

The present paper aims to review studies on prevalence of spasticity, its risk factors and on quality of life with emphasis on disability in activities of daily living. A comprehensive review of the literature using Medline databases from 1980 until January 2010 was con-

ducted using individual and combined search terms that included 'spasticity', 'stroke', 'prevalence', 'quality of life', and 'activities in daily living'. The search was limited to English-language articles.

## Prevalence of post-stroke spasticity

Literature on the prevalence of spasticity is scarce and provides inconsistent data. Watkins *et al.* [14] examined 106 acute stroke patients (excluding subarachnoid haemorrhage and transient ischaemic attacks) among whom 36 (34%) had also suffered a stroke in the past. Twelve months after stroke patients were assessed using two muscle tone scales: the Modified Ashworth Scale (MAS) (at the elbow) and the Tone Assessment Scale (TAS) measured at the following joints: wrist, elbow, hip, knee, and ankle. Increased muscle tone was present in 29 (27%) and in 38 (36%) of the 106 patients when measured using the MAS and TAS, respectively. Forty (38%) patients were identified as having spasticity when muscle tone was measured with both assessment tools. Of 59 patients having had a first-ever stroke, 23 (39%) had spasticity. Among 36 patients with a history of a previous stroke, the prevalence of spasticity was 44%. Therefore, history of previous stroke did not significantly influence the likelihood of developing spasticity following a subsequent stroke.

Leathley *et al.* [15] assessed the same cohort of patients for spasticity using the TAS and found some spasticity (TAS score > 0) in at least one joint in 38 (36%) patients and more severe spasticity (TAS score > 2) in 21 (20%) patients. Additionally,

**Table 1.** Positive and negative features of upper motor neuron syndrome

Positive	Negative
Increased tendon reflexes with radiation	Muscle weakness
Clonus	Loss of dexterity
Babinski sign	Fatiguability
Spasticity	
Extensor spasms	
Flexor spasms	
Mass reflex	
Dyssynergic patterns of co-contraction during movement	
Associated reactions and other dyssynergic and stereotypical spastic dystonias	

potential predictors of spasticity were analysed. The following variables were included: age, sex, history of cerebrovascular disease, history of diabetes, positive history of smoking, pre-stroke Rankin score (in those patients who had stroke in the past), post-stroke confusion and arm or leg weakness (within the first 24 hours of stroke onset), side of weakness, higher cortical dysfunction (presence of at least one out of: confusion, visual neglect or aphasia), type of stroke (from computed tomography [CT] scan), Oxfordshire Community Stroke Project (OCSP) classification and the Barthel Activities of Daily Living (ADL) Index score at day 7. Those variables were selected because they were recorded routinely as part of clinical care and have been used in previous models to predict outcome after stroke [16]. The variables that had a significant association with spasticity were considered for forward stepwise logistic regression analysis to develop a predictive model for each of two outcome measures (first: presence of any spasticity; second: more severe spasticity [TAS > 2]). Lower Barthel Index (BI) score at day 7 and early arm or leg weakness were associated with any spasticity in any joint at 12 months after stroke. Lower BI score at day 7 and left-sided weakness and smoking (ever) were associated with more severe spasticity in any joint at 12 months after stroke.

Sommerfeld *et al.* [17] studied 95 Swedish patients with first-ever stroke. They were assessed at the stroke onset (mean, 5.4 days) and 3 months later using MAS for spasticity (at shoulder, elbow, wrist, fingers, hip, knee, and ankle). Additionally, self-reported muscle stiffness and tendon reflexes (with counting of clonic beats at plantar flexors) were assessed to indicate the incidence of increased muscle tone. Of all 95 patients, 77 (81%) were initially hemiparetic. Three months after stroke, 64 (67%) out of 95 patients were hemiparetic and 18 (19%) were spastic. Among the spastic patients, 14 showed hyperreflexia (7 in both the upper and lower extremity, and 7 in the upper extremity only). Of these, 3 also showed clonic beats, and 3 reported muscle stiffness.

The next study performed by Welmer *et al.* [18] was the continuation of the previous study and re-assessed 66 patients (out of 95 included by Sommerfeld *et al.* [17]) 18 months after acute stroke. Among 66 patients, 38 (58%) were hemiparetic, and 13 (20% of all patients) displayed spasticity. Ten out of these 13 patients displayed spasticity in both the upper and lower extremities, and 3 in the upper extremities alone. Of the 13 patients with spasticity 3 months after stroke and remaining in the study at the 18-month follow-up,

9 (69%) still presented with spasticity 18 months after stroke, while 4 (31%) did not.

In a recent study, Welmer *et al.* [19] in the above-presented population focused on the location and severity of spasticity in different muscle groups, in the first 1–2 weeks and at 3 and 18 months after stroke. They looked for an association between the severity of spasticity and voluntary movements (see below), and the occurrence of spasticity in younger (< 65 years) versus older (> 65 years) patients. The severity of spasticity increased over time for elbow extensors, wrist flexors and extensors, and finger flexors. When adding the MAS scores for all tested muscle groups, the severity of spasticity increased for the upper but not for the lower extremities. The differences between the numbers of younger versus older patients (in favour of younger) with spasticity tended to be statistically significant in the first 1–2 weeks and were statistically significant at 3 months but not at 18 months.

Another Swedish study on a relatively large group of patients was performed by Lundström *et al.* [20], who recruited 140 patients from the Swedish National Stroke Registry – Risk-stroke using the following inclusion criteria: (i) resident in the catchment area (not very far from the clinic), (ii) age > 18 years, (iii) first-ever stroke (cerebral infarction or intracerebral haemorrhage) and 1-year survival, as well as (iv) ability to give informed consent. Exclusion criteria were: (i) any other neurological disorder which might affect muscle tone, (ii) transient ischaemic attack (TIA) and (iii) subarachnoid haemorrhage. Spasticity was assessed by use of the MAS at the following joints: shoulder, elbow, wrist, fingers, hip, knee and ankle. Any spasticity (AS) was defined as an MAS score  $\geq 1$ . To define disabling spasticity (DS), the impact of spasticity on activities of daily living (ADL) – personal hygiene, dressing, limb position and mobility – or on pain and sleep was evaluated according to a semi-structured interview, taking into account the patients' general status with regard to ADL and social life. DS was defined as spasticity having such an impact that intervention, e.g. intensive physiotherapy, orthoses or pharmacological treatment, should be offered. The frequency of AS according to the MAS assessment (AS) in the study sample was 18% (25 of 140 patients) and the frequency of DS was 6% (8 of 140 patients). There were no gender differences for AS or DS. DS was more common in patients aged below 65, either with severe arm paresis or after haemorrhagic stroke. Patients with DS were significantly younger than patients without DS.

The largest group so far of post-stroke patients was studied by Moura *et al.* [21]. In this Brazilian study, 146 patients after ischaemic stroke (with or without history of previous stroke) were recruited. Spasticity was measured by MAS one year after stroke and its prevalence was estimated at 25%. Additionally, demographic, clinical and tomographic data were analysed and the results showed that spasticity was associated with the following variables: manual work, previous stroke, extensive lesions in CT, decrease in individual income, undergone physiotherapy, undergone physiotherapy for a longer period, pain complaints, the pain started simultaneously with the spasticity. Manual work had a relative risk of 2.9; previous stroke 3.9, and extensive lesion in CT, 3.6.

All data about prevalence of spasticity, examined populations, aetiology of stroke, time from injury to assessment and methods used for the assessment of spasticity are presented in Table 2.

### Post-stroke spasticity and its impact on quality of life with emphasis on disability in ADL

It is well recognized that spasticity after stroke may interfere with motor function and therefore with the execution of ADL, may be accompanied by pain and leads to secondary complications such as contractures and

muscle atrophy [22]. Five reported studies [14,17-20] aimed to determine the relationship between spasticity, motor impairment and functional ability.

Watkins *et al.* [14] proved that patients with spasticity were more likely to receive institutional care and had significantly lower BI scores at 12 months ( $p < 0.0001$ ).

In the study by Sommerfeld *et al.* [17], motor performance was assessed by the Birgitta Lindmark Motor Assessment (BL), and just for patients who could not actively participate in the BL tests the Scandinavian Stroke Scale (SSS) was used to determine whether the patient was hemiparetic or not. The Nine Hole Peg Test (NHPT) was used for manual dexterity. Mobility was assessed by the Rivermead Mobility Index (RMI) and Get-Up and Go test (GUG) was used to evaluate the gait. ADL were assessed by BI. Patients who were non-spastic ( $n = 77$ ) had statistically significantly better motor and activity scores than those with spasticity ( $n = 18$ ). However, the correlations between increased muscle tone and the motor and activity scores were rather low ( $r < 0.5$ ,  $p < 0.05$ ), except for the initial upper-extremity MAS and BL active movement scores ( $r = 0.51$ ,  $p < 0.001$ ), for the 3-month upper-extremity MAS and BL active movement scores ( $r = 0.54$ ,  $p < 0.001$ ), and NHPT scores ( $r = 0.59$ ,  $p < 0.001$ ).

Welmer *et al.* [18], providing 18-month follow-up of the same group of patients, found predominantly

**Table 2.** Prevalence of spasticity, characteristics of population studied with emphasis on aetiology of stroke, time from injury to evaluation and assessment methods in six cited studies

Study	Number of patients	Aetiology of stroke	Muscle tone assessment methods	Time from injury to evaluation	Prevalence of spasticity
Watkins <i>et al.</i> , 2002 [14]	106	stroke (TIA and SAH excluded)	TAS, MAS	12 months	38%
Leathley <i>et al.</i> , 2004 [15]	106	stroke (TIA and SAH excluded)	TAS	12 months	36% 21% (severe spasticity)
Sommerfeld <i>et al.</i> , 2004 [17]	95	first-ever stroke (SAH and cerebellar lesions excluded)	MAS	at onset (mean, 5.4 days) and 3 months	21% (initially) 19% (at 3 months)
Welmer <i>et al.</i> , 2006 [18]	66	first-ever stroke (SAH and cerebellar lesions excluded)	MAS	18 months	20%
Lundström <i>et al.</i> , 2008 [20]	140	first-ever stroke (cerebral infarction or intracerebral haemorrhage)	MAS	12 months	18% 6% (disabling spasticity)
Moura <i>et al.</i> , 2009 [21]	146	ischaemic stroke	MAS	12 months	25%

TAS – Tone Assessment Scale, MAS – Modified Ashworth Scale, TIA – transient ischaemic attack, SAH – subarachnoid haemorrhage

moderate to high correlations between the MAS scores and the functioning scores (BL, RMI, BI) (with correlation coefficients between 0.51 and 0.65, and  $p$ -values less than 0.05). In the most recent study [19], the correlation between the MAS for the upper extremities and the BL for the upper extremities was moderate ( $r = -0.61, p < 0.05$ ) at 3 months but the same correlation for the lower extremities was not statistically significant.

Lundström *et al.* [20] assessed impaired function using the National Institutes of Health Stroke Scale (NIHSS). Disability related to activity performance and participation was assessed using the Modified Rankin Scale (mRS) and BI. NIHSS scores were higher in spastic patients as compared to patients with no spasticity one year after stroke. All patients with spasticity exhibited some degree of paresis. Thus, there was a strong relationship between severe hand paresis and DS. The proportion of patients with dependence in everyday activities according to mRS scores and BI was greater for patients with spasticity than for patients with no spasticity.

The Health-Related Quality of Life (HRQL) was assessed in patients with spasticity only by Welmer *et al.* [18], using the Swedish Short Form 36 Health Survey Questionnaire (SF-36). It consists of 36 items grouped into the following 8 health domains: physical functioning, role limitations due to physical problems, bodily pain, general health perceptions, vitality, social functioning, role limitations due to emotional problems, and mental health. When comparing patients with and without spasticity, the latter had significantly better functioning scores and statistically significantly better scores on the physical functioning subscale of the SF-36. Correlations between MAS scores and the other SF-36 health subscales were not statistically significant. For each of the SF-36 health scales, except for bodily pain ( $p = 0.3$ ) and general health ( $p = 0.2$ ), the median scores of all the investigated patients were statistically significantly lower than scores for the general Swedish population. This may reflect general poor health status not related to spasticity itself.

## Discussion

Spasticity and its clinical implications are still poorly described. Contrary to a previous theoretical study based on the estimates of health care professionals, suggesting that the prevalence of post-stroke spasticity was

approximately 60% [23], current studies present lower rates (spasticity affects 18-38% of post-stroke patients). Three follow-up studies of a Swedish cohort [17,18,20] report a frequency of 18-21% in patients examined at stroke onset and at 3, 12, and 18 months after stroke, while two studies from the United Kingdom [14,15] report a frequency of 36-38% in patients examined at one year post-stroke. One Brazilian study reported prevalence of spasticity at the level of 25%, as assessed one year after stroke. Among the hemiparetic patients, 26-36% exhibited spasticity. These findings are in accordance with those of O'Dwyer *et al.*, who found electromyographically-verified spasticity in 21% of hemiparetic patients assessed 13 months after stroke [22].

The discrepancies between the UK, Swedish and Brazilian studies might be related to a few but important variables. Primarily, the TAS as the outcome measure was used in the two UK studies [14,15] for assessing muscle tone (wrist, elbow, hip, knee, ankle), while the MAS was only used at the elbow. The Swedish and Brazilian cohorts were examined using the MAS as a primary spasticity measure. Different methods implied different definitions of any spasticity (AS): TAS > 0 or MAS > 0; the distribution of scores for the MAS at the elbow and the TAS at each joint, however, did not reveal significant divergence [14].

The MAS is the most commonly used scale to assess tone in clinical practice as well as in research studies [24]. Recognized limitations of the scale include the most important one – it captures only one feature of spasticity, i.e. resistance to passive movement; it does not differentiate the effects of reflex hyperexcitability from those of biomechanical factors. This may explain why not all of the spastic patients had hyperreflexia and only one-third of the spastic patients experienced muscle stiffness [17]. Although MAS measures the spasticity only, which cannot be translated into functional disability, it is the most widely used scale, with rather good inter-rater reliability, and to our opinion should be used in further studies to make comparisons possible.

The use of different methods of assessment might be the main source of the difference in spasticity prevalence between the studies. It may also be the reason for the discrepancy between the studies that assessed the prevalence of severe spasticity (called disabling spasticity, DS). This is a practically important issue because this group of patients should be treated more intensively, and this approach is more expensive. Lundström *et al.* [20] distinguished the subgroup of patients with disabling spasticity from patients with any degree of spas-

ticity (AS). Its prevalence was surprisingly very low (6%). Leathley *et al.* [15] found a larger group of patients (21%) with severe spasticity (TAS > 2). DS was defined as spasticity having such an impact on ADL that intervention, e.g. intensive physiotherapy, orthoses or pharmacological treatment, should be offered. It also means the influence of subjective impression. Therefore, the definition of severe spasticity may influence the different number of patients identified in the two studies.

Another problem may emerge as the two different cohorts of patients were included into those studies. The UK and Brazilian studies recruited consecutively 106 and 146 patients, with the first-ever stroke and with a history of preceding strokes in the past, while the Swedish studies investigated two populations of 95 and 140 subjects with first-ever stroke only. The difference was not significant (39% of cases after first-ever stroke vs. 44% of patients with stroke history) among 106 subjects assessed by Watkins *et al.* [14] but the groups were probably too small to make final conclusions. Nevertheless, Moura *et al.* noted that the risk of spasticity development in those who had previous stroke is 3.9 times greater, which is in agreement with an earlier report [25].

Finally, national health care systems may also influence the final results. Easy access to rehabilitation following the acute phase of stroke may result in a lower rate of spasticity, specially severe or disabling spasticity.

Spasticity was more frequent in younger patients (under 65 years) at 12 months [20], as well as at onset of stroke (mean, 5.4 days) and 3 months [19] after stroke. This observation is difficult to explain. Aging is related to the decrease in reflexes, as seen in the tendon reflexes and in the tonic reflexes [26]. This might be a possible explanation for the differences between younger and older patients found in the presented studies. This might be influenced by an underestimation in the oldest age group (> 85 years) because of loss of those with more severe conditions. The difference was also noted, however, when the youngest group was compared with the intermediate age groups (65-74 years) [20].

Spasticity was more common in upper extremities than in lower extremities [17-20]. The severity of upper extremity spasticity was associated with the severity of impaired upper extremity voluntary movements, but this was not the case for the lower extremities [19]. These differences between the upper and lower extremities may be explained by differences in supraspinal control as the upper limb normally functions predominantly under voluntary control whilst movements of the lower limb are influenced

to a larger extent by spinal locomotor centres [27]. Additionally, spasticity was the most common in muscles resisting gravity, i.e. the arm flexors and leg extensors, which is in accordance with a previous report [28].

Only Lundström *et al.* [20] found positive correlations between haemorrhagic stroke and occurrence of spasticity. It may be related to the more common localization of haemorrhagic strokes, deeply in the hemisphere, including the basal ganglia. Disabling spasticity was strongly correlated with severe paresis. Both lesion size and its location may influence the degree of motor impairment and spasticity. It seems to be pathophysiologically reasonable, considering that central networks regulating motor control and muscle tone are structurally and functionally closely related [20]. This is in accordance with the study of Moura *et al.*, where spasticity was noted more frequently among patients with extensive lesions as seen on CT and lesions affecting more than one cerebral lobe [21].

To organize early and effective rehabilitation and treatment of spasticity, knowledge about predictive factors is necessary. It has already been said that spasticity was more common in the group of younger patients, who had had stroke in the past and with more extensive lesions on CT or with haemorrhagic stroke. Additionally, Moura *et al.* [21] have found very interesting relationships between occurrence of spasticity and analysed demographic data. Patients who had been doing manual activities (more than 4 hours a day) before their strokes had a relative risk for spasticity of 3. Therefore, the way in which patients use their muscles before stroke seems to have an influence on the development of spasticity. This difference may have been caused by existing modifications and adaptations in the muscle cells, in relation to the size, distribution and contractile properties of the muscle fibres [29]. This may also explain the above-mentioned upper extremity and antigravity muscles as more commonly affected by spasticity.

Leathley *et al.* [15] have shown that it may be possible to develop models that can be used to predict the presence or absence of spasticity 12 months after a stroke. The predictor variables in the model depend on whether one is trying to predict either the presence of any spasticity or the presence of more severe spasticity, although day 7 BI is significant in both models and ever-smoking is significant in the second one. The variables used in developing these models are based on data that should be recorded routinely in hospital following admission with an acute stroke and that do not depend on complicated assessments or technology.

**Table 3.** Methods used to assess motor impairment and activities of daily living and health-related quality of life in cited studies

Study	Methods
Watkins <i>et al.</i> , 2002 [14]	BI
Leathley <i>et al.</i> , 2004 [15]	BI, mRS
Sommerfeld <i>et al.</i> , 2004 [17]	BI, BL, SSS, NHPT, RMI, GUG
Welmer <i>et al.</i> , 2006 [18]	BI, BL, NHPT, RMI, SF-36
Welmer <i>et al.</i> , 2009 [19]	BL
Lundström <i>et al.</i> , 2008 [20]	BI, NIHSS, mRS

BI – Barthel Index, mRS – Modified Rankin Scale, BL – Birgitta Lindmark Motor Assessment, SSS – Scandinavian Stroke Scale, NHPT – Nine Hole Peg Test, RMI – Rivermead Mobility Index, GUG – Get-up and Go Test, NIHSS – National Institutes of Health Stroke Scale, SF-36 – Short Form 36 Health Survey Questionnaire

Data on the impact of post-stroke spasticity on disability in ADL are more consistent. ADL was assessed with various methods (see Table 3) but modified BI was the most frequently used outcome measure. It is considered reliable, valid, and sensitive [30]. All of the reviewed studies found a relationship between appearance of spasticity and worse functional outcome. In only one study [18], where the association between spasticity and HRQL was analysed, did spasticity contribute to impairment of movement function and to limitation of activity, but it seemed to have a less pronounced effect on the other aspects of HRQL.

There were some differences between the impact of spasticity on ADL as related to the time of assessing increased muscle tone after stroke. Welmer *et al.* [18] compared functional outcome in the same group of patients 3 months and 18 months after stroke. Predominantly moderate to high correlations between spasticity and functional outcome 18 months post-stroke were found, contrary to rather low correlations between spasticity and functional loss 3 months post-stroke reported by Sommerfeld *et al.* [17]. Since reflex-mediated spasticity has been shown to reach a peak 1-3 months after stroke [31,32], the higher correlations at 18 months may merely be due to immobilization of affected limbs in patients with low scores on the functioning scales. That is why treatment of spasticity should be introduced as early as possible.

About 60 000 patients are hospitalized because of stroke every year in Poland [3] and 48 000 survive for more than one year. According to prevalence of spasticity from studies analysed in this review (range: 18-38%)

from 8600 to 18 000 of post-stroke survivors may develop spasticity. Alternatively, about 10 000 patients with severe spasticity (with its frequency of 21%, as in the UK study) and/or 2800 patients with disabling spasticity (with its frequency of 6% as in the Swedish study) can be expected every year. These latter figures indicate a clinical problem that deserves further attention and BoNT treatment, among others. Obviously, studies on Polish cohorts are needed to estimate the real prevalence, which may be different.

## Disclosure

Authors report no conflict of interest.

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