ABSTRACT

• **Objective:** To present an overview of the human and economic burden of poststroke spasticity and muscle overactivity.

• **Methods:** Literature review.

• **Results:** The burden of stroke for patients, their caregivers, and society is enormous. Stroke can result in markedly diminished quality of life (QOL), loss of productivity, and considerable economic costs. The subset of stroke patients who have poststroke spasticity experience additional burdens. Patients with poststroke spasticity represent at least 20% of all stroke patients. Poststroke spasticity can manifest in multiple ways, with potentially profound and detrimental effects on patient function and QOL. The risk of falling is higher among stroke patients than in the general population, and even higher in poststroke spasticity patients, with the consequent risk of fractures. Estimated direct costs for poststroke spasticity patients are 4 times higher than those for stroke patients without residual spasticity.

• **Conclusion:** The burden of poststroke spasticity is high in terms of treatment costs, QOL consequences, caregiver burden, and the effects of comorbidities such as falls and fractures.

Stroke is a common event, with nearly 800,000 people in the United States affected by a stroke each year [1]. In 2006, the estimated prevalence was 6.4 million, representing 2.9% of the US adult population [1]. Stroke constitutes the third largest cause of death in the United States [1] and is the leading cause of disability in adults [2]. Stroke may affect a person’s cognitive, language, perceptual, sensory, and motor functions, and can be the cause of the upper motor neuron syndrome [2], characterized by spasticity, muscle overactivity, cocontraction, clonus, muscle weakness, and a variety of motor control abnormalities that impair the regulation of voluntary movement that can profoundly affect function [3–5]. For 2010, the overall direct and indirect economic costs related to stroke care in the United States are estimated to be $73.7 billion [1].

Spasticity is one of the positive signs of upper motor neuron syndrome. It is most frequently defined 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, as one component of the upper motor neuron syndrome [6]. If left untreated, spasticity and other consequences of upper motor neuron syndrome muscle overactivity can lead to numerous symptomatic and functional problems that can cause substantial disability and have damaging effects on patient quality of life (QOL) [8–10]. Spasticity subsequent to stroke is associated with an elevated risk for comorbidities, most obviously those resulting from falls and fractures [11–13], which can substantially add to the cost of care. Recent data have shown that direct costs for patients with poststroke spasticity are approximately 4 times those for stroke patients without spasticity [7].

Relatively few studies focus specifically on poststroke spasticity issues, and it is difficult to precisely quantify the burden of poststroke spasticity either in indirect economic costs or in human terms. This article will review available data to present an overview of the economic and human burdens directly related to spasticity and other aspects of upper motor neuron syndrome that are borne by the poststroke population.

Defining and Measuring Spasticity

Prevalence and incidence rates of poststroke spasticity are somewhat variable, since the definition and measurement of spasticity are themselves variable and individually affected by (a) the definition used; (b) the techniques used to measure any specific presenting phenomenon; and (c) the circumstances under which the information is collected. Despite these inconsistencies, it can be stated that excessive resistance of muscle to passive stretch at different velocities constitutes the defining clinical characteristic of spasticity, and this resistance increases proportionately with increases in the rate of stretch [5]. The prevalence of spasticity following stroke is generally estimated to be in the range of 17% to 46% [14–20].
However, in studies where broader definitions of spasticity have been utilized, the rate was as high as 58% [21]. The term “spastic” has been commonly yet imprecisely applied to a range of clinical attributes, including spastic hemiparesis, spastic gait, and spastic hand. However, the use of “spastic” as a descriptive term can be deceiving because the term itself may not be associated with the cause of the condition or a specific presentation. For example, “spastic gait” may not be caused by spasticity, so attempting to treat spastic gait as one would treat spasticity may not result in improvement of gait function [5]. Furthermore, because clinical management of spasticity and muscle overactivity has a wide variety of treatment goals—for example, those associated with symptomatic problems, issues related to passive function, and problems of active function—no consensus exists regarding the actual incidence of clinically meaningful poststroke spasticity [14].

There are several scales used to measure muscle tone and spasticity, each with advantages and disadvantages. The Ashworth Scale allows for assessment of muscle tone using a 5-point scale, while the Modified Ashworth scale uses a 6-point scale [22,23]. The Ashworth scale was originally validated for the elbow joint, although it is frequently used to assess other joints in both the upper and lower limbs. Ideally, the test is always conducted with the patient in the same position and under similar conditions. A disadvantage of both Ashworth scales is that they do not take into consideration the presence of contracture or other soft tissue factors that may limit joint motion. They also do not allow for the application of a variable-speed during passive motion.

The Tardieu Scale, developed for the pediatric population in the mid-1960s, attempts to assess spasticity by varying the speed of joint motion available, from “very slow” (V1) to “as fast as possible” (V3). The difference between these 2 variables permits an estimation of the effect of spasticity [24]. Although it is more difficult to administer and rate than either of the Ashworth scales, the Tardieu Scale appears to be a more accurate indicator of spasticity because its assessment more closely follows Lance’s definition of spasticity, as it evaluates resistance to passive movement at both slow and fast speeds [25]. However, this scale has not yet been validated in a systematic way and must be administered with caution, as sensitivity and reliability have not been fully determined [26].

Unfortunately, scales that assess only muscle tone and spasticity provide no information on the presence of spasticity and muscle overactivity as well [27]. Other functional scales, such as those for gait or motor assessment, may also provide useful measurements of functional deficit [13].

Despite this lack of a universal standard for defining or measuring poststroke spasticity, a review of data from a variety of studies provides some insight into the nature of the condition. For example, a study by Sommerfeld et al of 95 first-time stroke patients in Sweden (mean age, 78 years) found that between a short-term analysis (5.4 days’ poststroke) and a medium-term analysis (3 months’ poststroke), the incidence of poststroke spasticity was little changed (21% and 19%, based on Modified Ashworth scale measurements) [14]. A long-term follow-up study of spasticity rates after 18 months (by which time 66 subjects were available for evaluation) found that almost exactly the same proportion of subjects still had spasticity (20%) [17]. These data are consistent with a 2008 study that examined the 1-year spasticity rate in 140 Swedish patients after first-ever stroke, with 17% of subjects having any spasticity and 4% having disabling spasticity as rated on the Modified Ashworth scale 1 year after stroke [19].

In Germany, a recent prevalence study of poststroke spasticity that focused on 211 patients who exhibited limb paresis within 5 days poststroke found that the rate of spasticity 6 months’ poststroke was 42.6% using the Modified Ashworth scale. The likely explanation for this higher spasticity prevalence is that initial limb paresis confers a higher spasticity risk [28]. Two south Asian studies reported rates of spasticity that were similar to the rate reported in the German study. A 2009 poststroke spasticity study in Thailand observed increased muscle tone on the Modified Ashworth scale in 41.6% of 327 patients [29]. A 2005 study from rural eastern India on the occurrence of spasticity 1 year after stroke found that 46% of patients were identified as having spasticity, based on the Modified Ashworth scale [20].

In the United States, the National Stroke Association conducted a national survey of stroke survivors who primarily had problems associated with movement and mobility (n = 504) [21]. The survey defined spasticity as “muscle tightness that makes arm and leg movement difficult.” They found that 58% of patients reported experiencing spasticity by this definition [21]. Half of those patients had never received treatment. It is not known if this rate of spasticity, which is higher than what was found in the European and Asian studies, reflects a lower threshold for defining spasticity, is truly representative of the populations examined, or is the result of a possible sampling bias.

Clinical and Quality of Life Impact

Morbidity

Poststroke upper motor neuron syndrome typically involves the presence of spasticity and muscle overactivity as well
as muscle weakness in addition to abnormalities of motor control that interfere with the regulation of voluntary movement [4,9]. Clinically significant functional disturbances arising from upper motor neuron syndrome can occur if muscle overactivity and spasticity remain untreated. Problems can be classified as symptomatic or relate to passive or active function. Symptomatic issues include pain, flexor spasms, extensor spasms, and clonus. Passive function refers to passive manipulation of limbs for the purpose of achieving functional ends that is typically performed by caregivers, although patients may also passively manipulate their limbs with the noninvolved limb. Active function, in contrast, refers to a patient directly using a limb to perform a functional activity [3].

Poststroke spasticity can manifest in multiple ways, with potentially profound and detrimental effects on patient function and QOL. The most common patterns of dysfunction resulting from poststroke spasticity are equinovarus foot and flexed elbow; other dysfunctions include flexed wrist, clench fist, stiff knee, adducted/internally rotated shoulder, thumb-in-palm deformity, excessive hip flexion, and adducted thighs [4,30]. Other patterns may manifest, and most cases do not present as a single entity but instead affect function in combination. The dysfunction and interference with activities of daily living that result from poststroke spasticity can promote a broad spectrum of clinical morbidities, not the least of which are the results of falls and fractures [11].

Falls experienced by stroke survivors are, to a large extent, a function of decreased mobility and compromised balance. However, the fear of falling may be an even greater factor in conferring increased risk of falls and immobility in stroke patients in that the fear may lead to avoidance of physical activity, resulting in physical deconditioning and thus increased impairment of balance and mobility [31]. Within the poststroke patient population, Soyuer and Oztürk found that spasticity is associated with a considerable increase in the risk of falling [13]. Concurrent with a greater number of falls is an increase in the rate of fractures. Hip fractures occur 2 to 4 times more often in stroke patients than in controls [32]. Exacerbating the risk of fractures in the overall poststroke population is the increased probability of low bone mineral density. Stroke patients experience increasing bone mineral density loss up to 1 year poststroke and are at higher risk of experiencing bone mineral density loss on the paretic side resulting from disuse, with consequent increased likelihood of hemiosteoporosis (Figure 1) [33]. Intriguingly, the bone mineral density of the unaffected arm of stroke patients has been seen to increase significantly, possibly as a result of increased physical demands on the unaffected side and/or redistribution of bone mass [33]. Based on the Soyuer data cited above, poststroke falls are

*Figure 1.* Percentage change in bone mineral density (BMD) on the paretic side of stroke patients (*n* = 19) from 1 month to 1 year poststroke. Bivariate differences between baseline (1 month poststroke) and 12 months poststroke: total arm, *P* < 0.001; humerus, *P* < 0.001; ulradistal (UD) radius, *P* < 0.01; total femur, *P* < 0.001; proximal femur, *P* < 0.001. Reprinted from reference 31.
likely to occur even more frequently in stroke patients with poststroke spasticity.

The financial burden resulting from stroke-related falls and fractures is substantial, with the lifetime cost for a person with hip fracture estimated at $81,300 (2001 dollars). Total costs for hip fractures in the United States are projected to exceed $45 billion by the year 2040 [34]. Although no data are currently available on the rate of falls and fractures in poststroke spasticity patients as a specific subgroup, it is likely that a higher risk of falls and fractures in this patient population would account for larger associated costs.

**Quality of Life and Caregiver Burden**

Studies of QOL in stroke and spasticity focus either on stroke as the diagnostic entity and do not isolate spasticity or other upper motor neuron syndrome phenomena, or on spasticity as a whole and do not isolate spasticity and muscle overactivity resulting from stroke. One of the few exceptions to this is a 2009 online survey conducted by the organization We Move, which provides education and research on movement disorders [35]. The We Move survey of 589 people with a diagnosis of spasticity examined patient self-reports on the effects of spasticity, 441 (75%) of whom had a diagnosis of stroke [35]. When asked which single aspect of spasticity had the greatest impact on their QOL, the most common answer (36%) was limited range of motion in the affected limbs. The second and third most common responses were stiffness or contractures of muscles (23%) and limitations in activities of daily living (22%) [35]. Almost 60% of respondents stated that they were unemployable as a result of their poststroke spasticity, and 29% reported that their work was limited to some extent by their condition [35]. Of those surveyed, 78% said that spasticity had a negative impact on their ability to perform activities of daily living, and 62% reported that spasticity limited their ability to engage in activities outside the home or that they required assistance to do so [35].

Since there is a paucity of data from studies aimed at evaluating QOL in poststroke spasticity patients, it is useful to examine baseline study data to determine the status of untreated patients and gain insight regarding their pretreatment QOL. A study by McCrory et al (2009) involved 96 poststroke spasticity patients with upper limb spasticity randomized to receive botulinum toxin type A or placebo injection [36]. The variables recorded during this study included patient scores for QOL, pain, mood, disability, and caregiver burden [36]. The mean elapsed time since stroke of the study population was nearly 6 years, and the mean composite Modified Ashworth scale score was 7.0. Using the Assessment of Quality of Life (AQoL) instrument, where 1.00 indicates full QOL and 0.00 indicates death-equivalent QOL, the investigators found a mean baseline score of 0.35, signifying a rather poor QOL [36,37]. The mean baseline pain score using the 100-mm visual analogue scale was 40.7, indicating moderate levels of pain. Depression was measured by the Hospital Anxiety and Depression Scale [38], and the mean baseline score was 5.5, indicating a fairly high level of depression. Disability in these patients was found to be moderate, with mean scores totaling 1.8 based on the Patient Disability Scale, where 0 = no disability and 4 = maximum disability. Finally, caregiver burden was measured with the Carer Burden Scale in which 0 = no burden and 4 = maximum burden; the mean score was 1.7 [36].

A number of clinical studies assessing botulinum toxin type A for the treatment of spasticity have evaluated QOL and impact on activities of daily living. A recent open-label study by Elovic et al evaluated health-related QOL using the Stroke Adapted Sickness Impact Profile and EuroQoL-5D and impact on ADL using the Disability Assessment Scale [39]. Baseline scores (41.5 ± 199 on a scale of 0 [no dysfunction] to 100 [maximal dysfunction] for the Stroke Adapted Sickness Impact Profile, and 66.4 ± 21.3 on a scale of 0 [worst imaginable health] to 100 [best imaginable health] for the EuroQoL-5D) indicated a relatively low health-related QOL. Additionally, patients were asked to identify a principal area of treatment at baseline based on the domains of the Disability Assessment Scale (limb posture, pain, dressing and hygiene); the majority of patients chose limb posture (49%) and dressing (29%) as domains that were most affected by their spasticity [39]. Similar findings were seen in a study by Brashear et al [40]. An additional post-hoc analysis of the Elovic et al study revealed a correlation between severity of disability as evaluated by the Disability Assessment Scale and caregiver burden: at baseline, more severe levels of disability were associated with a greater number of hours of required caregiver assistance [41].

Similar levels of disability and caregiver burden were observed in a clinical trial from the United Kingdom in which 40 patients with poststroke spasticity, all having in common the disability of a nonfunctional arm, were evaluated using the Patient Disability Scale and Carer Burden Scale [8]. At baseline, the mean Patient Disability Scale score was 2.5 (out of 4), and the mean caregiver burden was 1.7 (out of 4) [8]. This is consistent with a 2001 study of botulinum toxin type A versus placebo, which included 59 poststroke patients with upper limb spasticity. Baseline measurement of activities of daily living using the Barthel Index (0–20 point scale, where 20 indicates a person is continent, able to wash, feed and dress himself, and independently mobile) showed a mean score of 12.9 [42,43]. Both studies demonstrate that spasticity can be severely disabling and can significantly impact caregivers.

A recent study assessing caregiver burden for patients with spasticity due to either stroke or multiple sclerosis
found that caregivers for stroke survivors experienced a significantly higher burden than caregivers for multiple sclerosis patients, based on the Oberst Caregiving Burden Scale time subscale ($P < 0.05$) [44]. A detrimental impact on caregivers’ employment was also observed, with 7% reporting work absenteeism and 24.2% reporting loss in work productivity. Because this study did not include nonspastic patients or all impairments associated with stroke, it is difficult to say whether stroke recovery as a whole results in greater caregiver burden than that posed by patients with multiple sclerosis–related needs, but it is clear that caring for patients with poststroke spasticity is complicated and time-demanding [44].

It should be noted that not all patients with spasticity are severely disabled and require caregiver assistance. A study that evaluated prevalence of spasticity among stroke survivors determined prevalence of both total spasticity and disabling spasticity. Disabling spasticity was defined as spasticity severe enough to warrant intervention or treatment, the implication being that some patients have spasticity that is mild and does not severely impact function [19].

**Cost Burden**

A recent study from Sweden comparing 1-year direct costs for poststroke patients with and without spasticity is the only published cost study specific to this patient population. Lundström et al studied 140 patients hospitalized with first-ever stroke (median age 73 years, 48% female), 115 of whom did not suffer from spasticity and 25 of whom did [10]. Direct costs for the year following stroke were 4 times greater for patients with spasticity than for those without [7]. Most of the costs (78%) were associated with hospitalization; 20% were attributable to municipal services. Medications and primary care each constituted 1% of total direct costs. Based on a conversion of the Swedish krona to purchasing power parity with the 2003 US dollar, the mean direct costs for patients with spasticity were $84,195. For patients without out spasticity, the costs dropped to $21,842. A significant correlation was found to exist between increased spasticity (based on the Modified Ashworth Scale) and higher costs ($P < 0.001$) (Figure 2) [7].

Radensky et al evaluated treatment costs for poststroke patients with either upper or lower extremity spasticity [49]. Thirty physicians from across the United States were asked to describe management strategies for 4 hypothetical case studies of poststroke spasticity patients. Potential costs for each examined case were calculated, including all therapeutic interventions, such as medications, surgery, and physical therapy as well as counseling, in-office and telephone consultations, home nurse visits, orthopedic apparatus, wheel chairs, and laboratory services [49]. Although 60% of patients were covered by Medicare, patients’ insurance coverage overall was diverse [49]. The mean cost per patient with upper extremity spasticity symptoms was $5131 (range, $1314–$15,776), while for those with lower extremity symptoms the mean treatment cost was slightly higher at $5384 (range, $1331–$21,961). A comparison of the relative costs of different spasticity therapies within the study found that treatment with either oral baclofen or diazepam was associated with increased overall management costs, while treatment with dantrolene, botulinum toxin A, or phenol was associated with lower overall management costs. A comparison of treatment strategies that included strategies with and without botulinum toxin A in upper extremity poststroke spasticity demonstrated a reduction in costs that reached statistical significance ($p < 0.05$) when botulinum toxin A was included [49]. The figures reflect treatment costs for the years 1994–1999.

**Cost-Effectiveness Studies**

Because poststroke spasticity patients are commonly treated with a combination of physical and pharmacologic therapies, it is of some interest to evaluate the relative cost-effectiveness of physical therapy alone compared with a combination of therapy regimens. Wallesch et al did just that in comparing physiotherapy alone to physiotherapy combined with either botulinum toxin A or oral baclofen over 1 year [50]. Cost-effectiveness was determined based on the surveyed opinion of neurologists who were asked to evaluate the degree of improvement, if any, resulting from each of the 3 treatments alone or in combination. Cost-effectiveness was measured using the ratio cost/unit of improvement on the Ashworth Scale. Botulinum toxin A plus physiotherapy was by far the most cost-effective therapy, costing 10 times less than physiotherapy alone and nearly 3 times less than baclofen plus physiotherapy (Table) [50].

In 2005, Ward et al compared the cost-effectiveness and outcomes of oral spasticity therapy (benzhexol, baclofen, or tizanidine) with treatment with first-line botulinum toxin A or second-line botulinum toxin A (in patients who had failed oral therapy) in patients with flexed wrist/clenched fist deformity resulting from poststroke spasticity [51]. Results were expressed as cost in British pounds (£) per successfully treated month (STM) as well as in number of STMs per year, the combination of these criteria allowed for a cost-eficacy analysis. At the time the article was written, £1 was worth approximately US $1.6. The investigators observed that first-line botulinum toxin A treatment cost £942 (£1507) per STM, second-line botulinum toxin A cost £1387 (£2219) per STM, and oral therapy cost £1697 (£2715) per STM. Thus, in order to achieve STM, oral therapy cost approximately 80% more than first-line botulinum toxin A and approximately 22% more than second-line botulinum toxin A. Additional context may be provided by a comparison of the percentage...
**Figure 2.** Direct costs of stroke by Modified Ashworth Scale (MAS). MAS maximum 1 refers to a maximum score of 1 in any tested joint. A round circle (°) denotes an outlier, (ie, between 1.5 and 3 box lengths from the end of the box). An asterisk (*) denotes an extreme (ie, 3 box lengths from the end of the box). Reprinted from reference 10.

**Table 1.** Cost-Effectiveness Analysis: Averages for Upper Limb, Lower Limb, Hemispasticity, and All Degrees of Spasticity

<table>
<thead>
<tr>
<th></th>
<th>Physiotherapy Alone</th>
<th>Physiotherapy plus Baclofen</th>
<th>Physiotherapy plus BTX-A</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total expected costs, DM</strong></td>
<td>25,596</td>
<td>24,378</td>
<td>27,097</td>
</tr>
<tr>
<td><strong>Average expected effects, UOI</strong></td>
<td>0.52</td>
<td>0.179</td>
<td>0.538</td>
</tr>
<tr>
<td><strong>Mean cost-effectiveness ratio</strong></td>
<td>495,267</td>
<td>136,067</td>
<td>50,344</td>
</tr>
<tr>
<td><strong>Incremental costs, DM</strong></td>
<td>—</td>
<td>-1,218</td>
<td>1,501</td>
</tr>
<tr>
<td><strong>Incremental effects, UOI</strong></td>
<td>—</td>
<td>0.127</td>
<td>0.486</td>
</tr>
<tr>
<td><strong>Incremental cost-effectiveness ratio</strong></td>
<td>—</td>
<td>-9,591</td>
<td>3,088</td>
</tr>
</tbody>
</table>

Note: Total expected costs are direct medical costs relating to spasticity for the first year for one patient (e.g., costs of treatment, hospitalization, nursing home and rehabilitation). The mean cost-effectiveness ratio represents the costs in DM per unit of improvement on the Ashworth scale. The incremental cost-effectiveness ratio represents the difference in costs divided by the difference in effects. All incremental factors represent increases over physiotherapy alone. BTX-A = botulinum toxin A; DM = deutsche mark; UOI = units of improvement on the Ashworth scale. (Adapted with permission from Wallesch 1997.)
of STMs achieved per year for each treatment: for first-line botulinum toxin A this result was 73% (266 of 365 days) versus 68% for second-line botulinum toxin A and 35% for oral therapy [51].

Conclusion

The burden of poststroke spasticity is high in terms of treatment costs, QOL consequences, caregiver burden, and the effects of comorbidities such as falls and fractures. Stroke is already notable for the heavy burdens related to the physical impairment it produces as well as associated economic costs; stroke patients with spasticity represent a particularly overburdened subpopulation within the larger stroke patient group. Poststroke spasticity management requires a multidisciplinary team approach and is complicated by lack of clarity of the true definition of spasticity among medical professionals and the implications of this on treatment selection. There are several effective treatments, including physical rehabilitation, oral medications, surgery, and focal medication, such as phenol or botulinum toxin A. In addition to having demonstrated efficacy and safety, studies have shown that botulinum toxin A may also be more cost-effective for the treatment of patients with poststroke spasticity [50,51,54,55]. Cost-effective treatment represents one way in which the burden of poststroke spasticity may be reduced in both human and economic terms.

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POSTSTROKE SPASTICITY


