



Revista Ciencias de la Salud

ISSN: 1692-7273

ISSN: 2145-4507

Editorial Universidad del Rosario

Sepúlveda, Paulina; Rivas, Claudia; Rivas, Rodrigo; Saiz, José L; Doussoulín, Arlette
Prevalence, Onset, Evolution, and Prediction of Spasticity Poststroke: A Longitudinal Study

Revista Ciencias de la Salud, vol. 21, no. 2, 2023, pp. 1-19

Editorial Universidad del Rosario

DOI: <https://doi.org/10.12804/revistas.urosario.edu.co/revsalud/a.11318>

Available in: <https://www.redalyc.org/articulo.oa?id=56276342007>

- How to cite
- Complete issue
- More information about this article
- Journal's webpage in redalyc.org

UDEM [redalyc.org](https://www.redalyc.org)

Scientific Information System Redalyc

Network of Scientific Journals from Latin America and the Caribbean, Spain and Portugal

Project academic non-profit, developed under the open access initiative

Prevalence, Onset, Evolution, and Prediction of Spasticity Poststroke: A Longitudinal Study

Prevalencia, inicio, evolución y predicción de la espasticidad posterior a un ACV: un estudio longitudinal

Prevalência, início, evolução e previsão de espasticidade após AVC: um estudo longitudinal

Received: November 01, 2022 • **Approved:** March 02, 2023

Doi: <https://doi.org/10.12804/revistas.urosario.edu.co/revsalud/a.11318>

To cite this article: Sepúlveda P, Rivas C, Rivas R, Saiz JL, Doussoulin A. Prevalence, Onset, Evolution, and Prediction of Spasticity Poststroke: A Longitudinal Study. Rev Cienc Salud. 2023;21(2):1-19. <https://doi.org/10.12804/revistas.urosario.edu.co/revsalud/a.11318>

Paulina Sepúlveda, PhD¹

Claudia Rivas, Phys.²

Rodrigo Rivas, MD Neurologist³

José L. Saiz, Psychologist, PhD⁴

Arlette Doussoulin, Phys., PhD^{5*}

Abstract

Introduction: Because of the the complex physiopathology of spasticity, it is distinguished as one of the most significant positive clinical signs of upper motor neuron syndrome, constituting a clinical feature

1 Departamento de Ciencias Pre-clínicas, Universidad de La Frontera (Temuco, Chile).

2 MV Clinical (Temuco, Chile).

3 Departamento de Especialidades Médicas, Universidad de La Frontera (Temuco, Chile). Clínica Alemana de Temuco (Chile).

4 Departamento de Psicología, Universidad de La Frontera (Temuco, Chile).

5 Departamento de Pediatría y Cirugía Infantil, Universidad de La Frontera (Temuco, Chile)

Paulina Sepúlveda, ORCID: <https://orcid.org/0000-0002-6209-6592>

Claudia Rivas, ORCID: <https://orcid.org/0000-0002-1638-9036>

Rodrigo Rivas, ORCID: <https://orcid.org/0000-0001-51868262>

José L. Saiz, ORCID: <https://orcid.org/0000-0002-7137-4646>

Arlette Doussoulin, ORCID: <https://orcid.org/0000-0002-12715535>

* Corresponding author: arlette.doussoulin@ufrontera.cl

that has great impact in the neurorehabilitation setting. Thus, the current study aimed to determine the prevalence, onset, evolution, and prediction of spasticity after a stroke. *Materials and Methods:* A correlational, longitudinal design was used. A total of 136 patients were evaluated at the following times: 10 days (T1), 3 months (T2), and 12 months (T3) poststroke. The initial evaluation included sociodemographic and clinical data (T1). Muscle tone was measured (T1, T2, and T3) using the Modified Ashworth Scale. *Results:* The prevalence of poststroke spasticity in the elbow was 37.5% at T1 and 57.4% at T2 and T3. Among patients with motor damage, the onset of spasticity occurred at T1 in 44.7%, between T1 and T2 in 23.7%, and between T2 and T3 in 0.9%. Significant predictors of the alteration in muscular tone for at least two of the evaluation times were ethnic self-classification, type, area, extent of stroke, and number of sessions. *Conclusions:* Spasticity onset occurs during the first 10 days after a stroke. More clinical than sociodemographic variables predicted spasticity.

Keywords: Muscle spasticity; rehabilitation; risk factors; stroke; upper extremity.

Resumen

Introducción: la espasticidad se destaca como uno de los signos clínicos positivos más significativos del síndrome de motoneurona superior, por su compleja fisiopatología, y constituye una característica clínica de gran impacto en el ámbito de la neurorrehabilitación. Por lo tanto, el objetivo fue determinar la prevalencia, el inicio, la evolución y la predicción de la espasticidad después de un accidente cerebrovascular. *Materiales y métodos:* se utilizó un diseño longitudinal correlacional. Se evaluaron 136 pacientes: 10 días (T1), 3 meses (T2) y 12 meses (T3) pos-ACV. La evaluación incluyó datos sociodemográficos y clínicos (T1) y se midió el tono muscular (T1, T2 y T3) mediante la Escala de Ashworth Modificada. *Resultados:* la prevalencia en el codo fue del 37.5% en T1, y del 57.4% en T2 y T3. Entre los pacientes con daño motor, el inicio de la espasticidad ocurrió en T1 para el 44.7% de ellos, entre T1 y T2 para el 23.7% y entre T2 y T3 para el 0.9%. La autclasificación étnica, el tipo, el área, la extensión del ictus y el número de sesiones predijeron significativamente la alteración del tono muscular en al menos dos ocasiones. *Conclusiones:* el inicio de la espasticidad ocurre durante los 10 primeros días después de un ACV. Más variables clínicas que sociodemográficas predijeron espasticidad.

Palabras clave: espasticidad muscular; rehabilitación; factores de riesgo; accidente cerebrovascular; extremidad superior.

Resumo

Introdução: a espasticidade destaca-se como um dos sinais clínicos positivos mais significativos da síndrome do neurônio motor superior, devido à sua fisiopatologia complexa, e constitui uma característica clínica de grande impacto no campo da neurorreabilitação. Portanto, nosso objetivo foi determinar a prevalência, início, evolução e predição da espasticidade após o acidente vascular cerebral. *Materiais e métodos:* foi utilizado um desenho correlacional longitudinal. Foram avaliados 136 pacientes: 10 dias (T1), 3 meses (T2) e 12 meses (T3) pós-avc. A avaliação incluiu dados sociodemográficos e clínicos (T1) e o tônus muscular (T1, T2 e T3) foi medido por meio da Escala Modificada de Ashworth. *Resultados:* a prevalência no cotovelo foi de 37,5% em T1 e 57,4% em T2 e T3. Entre os pacientes com prejuízo motor, o início da espasticidade ocorreu em T1 em 44,7% deles, entre T1 e T2 em 23,7% e entre T2 e T3 em 0,9% dos pacientes. A autoclassificação étnica, o tipo, a área, a extensão do AVC e o número de sessões predisseram significativamente as anormalidades do tônus muscular em pelo menos duas ocasiões. *Conclusões:* o início da espasticidade ocorre durante os primeiros 10 dias após o acidente vascular cerebral. Mais variáveis clínicas do que sociodemográficas previram a espasticidade.

Palavras-chave: espasticidade muscular; reabilitação; fatores de risco; acidente vascular cerebral; extremidade superior.

Introduction

Because of the complex physiopathology of spasticity, it is distinguished as one of the most significant positive clinical signs of upper motor neuron syndrome, constituting a clinical feature with great impact in the neurorehabilitation setting (1,2). Although the most commonly cited definition of spasticity is the one offered by Lance, other studies have proposed more modern concepts, with that formulated by Pandyan et al. referring to “a disordered sensory-motor control, resulting from an upper motor neuron lesion, which presents with involuntary, intermittent, or sustained activation of the muscles” (3-5).

The evidence suggests that poststroke spasticity (PSS) is caused by the loss of equilibrium between the supraspinal excitatory and inhibitory mechanisms, particularly of the dorsal reticulospinal tract, and abnormal intraspinal processing of the stretch reflex due to changes in the intrinsic properties of the spinal motor neuron, which is associated with modifications in the biomechanical properties of the muscle and surrounding soft tissue (6-8).

There is great variability in the beginning and evolution of PSS (9). This variation is related to changes in neural plasticity and mediated by different intrinsic and extrinsic factors after a brain injury (10,11). Studies have projected a prevalence of 24 % in the first week, 19 % at 3 months, 22 % to 43 % at 4 and 6 months, and 17 % to 38 % at 12 months poststroke (12,13). This high variability can be explained by the various assessment methods and multiple definitions used, for which there is currently no consensus (14).

Depending on the location and extent of the brain damage, the presentation of PSS can differ (15); however, the lesion does not predict the intensity nor impact it will have, as this can change within the same day or over longer periods as well as fluctuate in different physiological and psychological situations (6,16). The degree of PSS can vary from the effects of some antigravity muscle groups to global manifestations. In this way, three types of involvement are clear: focal, segmental, and generalized. Likewise, the spastic condition is typically manifested in the upper and lower extremities, through characteristic patterns, each with specific muscles involved, mainly affecting the elbow (79 %), wrist (66 %), and ankle (66 %) (9,17-19). The typical pattern of the spastic upper extremity (UE) is described as shoulder adduction with internal rotation, flexed elbow, flexed wrist, and closed fist; however, this is not the only pattern of spasticity that can manifest (17).

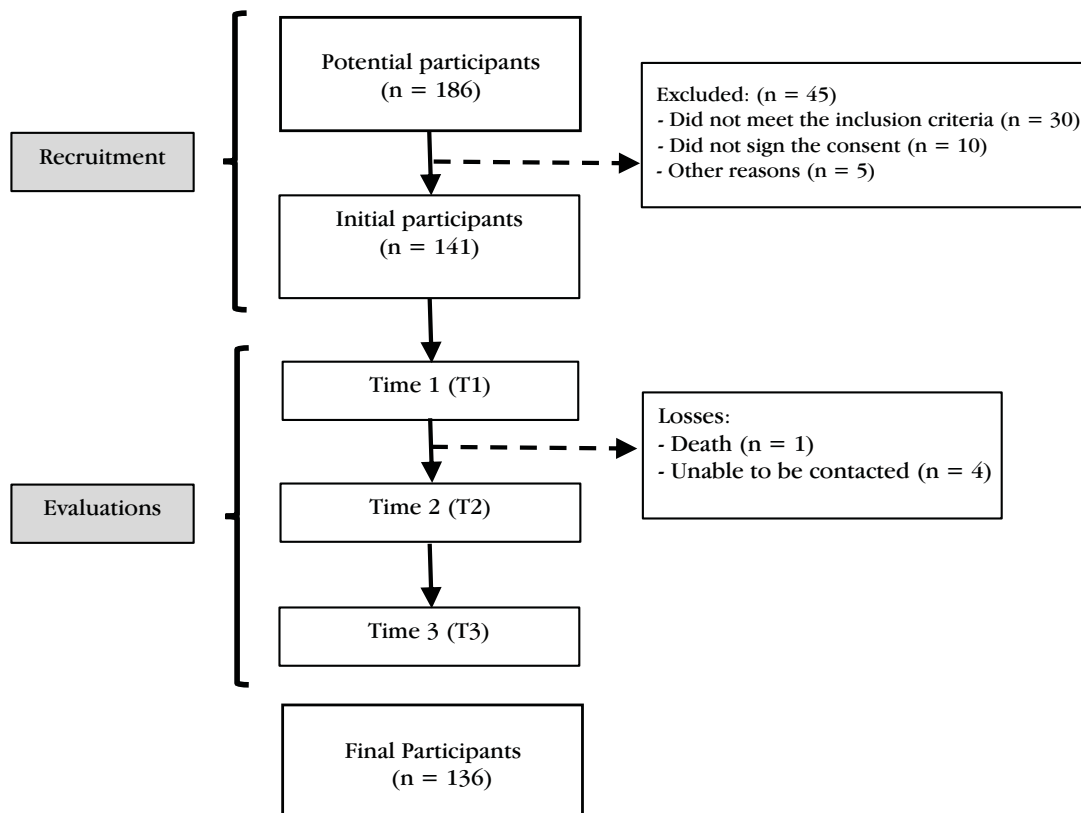
Although spasticity is a source of functional involvement, associated with reduced quality of life due to the generated disability, it is not always detrimental and does not always require treatment (20-22). In fact, some effects of spasticity may be beneficial, such as during the support phase in walking rehabilitation and when carrying weight through support on the UE (23). Lundström et al. concluded that intervention is required when spasticity causes disability (24), which makes it necessary to consider the final impact of PSS before seeking therapeutic strategies (25).

Despite the broad scientific evidence on this topic, there remains no consensus on the moment when spasticity appears, its location, or its predictors (4,6). Therefore, new evidence could guide professionals in the investigation and handling of spasticity, supporting intervention planning and reducing functional limitations and rehabilitation costs (26). Thus, the purposes of the current study were to examine (i) the prevalence, (ii) onset time, (iii) evolution, and (iv) prediction of elbow and wrist spasticity during the first year poststroke.

Materials and methods

We used a correlational, longitudinal design for this study, with prospective follow-up, for descriptive and predictive purposes. We obtained the population and samples as described in Figure 1. In total, we recruited 186 patients, among whom 45 were excluded for not fulfilling the eligibility criteria, leaving 141 to participate in the first assessment period (T1). The final sample, with all three assessments, was composed of 136 patients, as 5 were lost between T1 and T2.

Figure 1. Flow chart of the study



Participant selection was controlled by strict compliance with the inclusion and exclusion criteria. Inclusion criteria were age between 18 and 90 years, without prior history of motor disability, being in the hospital phase diagnosed with an ischemic or hemorrhagic stroke, without severe cognitive impairment (Mini-Mental Scale score ≥ 11), hemodynamically stable, presenting a stroke confirmed by computed tomography (CT) and/or magnetic resonance imaging, and signing the informed consent. Exclusion criteria were presenting with sensitive aphasia, having a medical contraindication, and having had more than one stroke.

Table 1 presents the sociodemographic and clinical characteristics of the final sample, showing that 62.5 % of the participants were men, 49.3 % were aged 65 years or older, 76.5 % lived in an urban area, and 32.4 % self-classified as belonging to the Mapuche ethnic minority group. With regard to the extent and severity of the damaged brain area, 58.1 % of the sample showed moderate to slight damage in the middle cerebral artery (MCA; as measured by the Alberta Stroke Program Early CT Score [ASPECTS]), and 56.6 % showed a partial anterior circulation infarct (PACI) according to the Oxfordshire Community Stroke Project (OCSP) classification.

Table 1. Sociodemographic characteristics of the sample

		Frequency	%
Sex	Female	51	37.5
	Male	85	62.5
Age range, years	25–44	15	11.0
	45–64	54	39.7
	≥ 65	67	49.3
Ethnic self-classification	Mapuche	44	32.4
	Non-Mapuche	92	67.6
Type of stroke	Ischemic	105	77.2
	Hemorrhagic	31	22.8
Location	Right	73	53.7
	Left	63	46.4
NIHSS	0: No stroke	12	8.8
	1–4: Mild stroke	75	55.1
	5–15: Moderate stroke	40	29.4
	16–20: Severe stroke	9	6.6
Hypertension	Yes	103	75.7
	No	33	24.3
Coronary disease	Yes	25	18.4
	No	111	81.6

		Frequency	%
Insulin dependent diabetes	Yes	27	19.9
	No	15	11.0
	No diabetes	94	69.1
ASPECTS	Severe damage MCA	35	25.7
	Mild damage MCA	44	32.4
	Without damage MCA	25	18.4
	Different damaged structures	32	23.5
OCSF	TACI	17	12.5
	PACI	77	56.6
	LACI	11	8.1
	POCI	31	22.8
Motor damage	Hemiplegia upper extremity	47	34.6
	Hemiparesis upper extremity	67	49.3
	Without alteration	22	16.2
Quantity of rehabilitation sessions	No	25	18.4
	1–10	21	15.4
	11–30	24	17.6
	>30	66	48.5

ASPECTS: Alberta Stroke Program Early CT Score; **LACI:** lacunar circulation infarcts; **MCA:** middle cerebral artery; **NIHSS:** National Institute Health Stroke Scale; **OCSF:** Oxfordshire Community Stroke Project Classification; **PACI:** partial anterior circulation infarcts; **POCI:** posterior circulation infarcts; **TACI:** total anterior circulation infarcts.

Initially, the potential participants were evaluated by two neurologists, who also confirmed compliance with the eligibility criteria, in each health center, and for the 10 hospitalization days. The sample was then recruited and identified. A previously trained physiotherapist belonging to each health center performed the evaluations of UE motor functions. Each participant was assessed on three occasions: during the first 10 hospitalization days (T1) and 3 months (T2) and 12 months (T3) after stroke. The length of the follow-up period was based on previous studies (27).

At T1, the neurologists registered sociodemographic data such as sex, age, residence, and ethnic self-classification (Mapuche, nonindigenous); clinical aspects referring to the type, extent, and severity of the stroke, assessment of the neurologic deficit (National Institute Health Stroke Scale), and motor damage; and comorbidities such as high blood pressure (HBP), cardiopathies, and diabetes. The physiotherapist assessed muscle tone at all time points.

At T3, the number of rehabilitation sessions of each patient was registered as one of four categories: none, 1 to 10, 11 to 30, and 31 sessions or more.

The extent and severity of the damage were recorded using the ASPECTS, a quantitative topographic scale (0 to 10 points) that assesses acute ischemic changes in patients with a recent stroke in the territory of the MCA at the level of the basal ganglia and above the basal ganglia through cranial CT (28). In the present study, we classified the scores derived from the CT according to four MCA damage categories: severe (0–4), moderate (5–7), slight (8–9), and no damage (10). (29).

The damaged area of the brain was identified using the four subtypes included in the OCSF classification: total anterior circulation infarct (TACI), PACI, lacunar circulation infarct (LACI), and posterior circulation infarct (POCI). This classification provides fast, simple, and prognostically valuable information, as well as predicts the size and location of the lesion in 75 % of patients (30,31).

The elbow and wrist muscle tone were evaluated as flaccid, normal, and spastic. We used the Modified Ashworth Scale (MAS) to differentiate the degree of spasticity, given its utility when applied to the UE and its high interobserver reliability (32–34). The MAS classifies the tone according to six categories, which, in this study, were regrouped into the following four categories: normal (value 0), slight (values 1 and +1), moderate (values 2 and 3), and severe spasticity (value 4). Other studies have also used this abbreviated classification (35). We added a fifth category, flaccidity, to the classification. Although flaccidity is not part of the MAS, its inclusion allows a more comprehensive view of the different alterations in muscle tone after stroke.

To evaluate the presence of motor damage in the affected UE, the patient was asked to place their affected hand on their head and was categorized as normal if they achieved the complete movement, hemiparesis if they began the movement but did not complete it or hemiplegia if they did not perform the movement.

We used the statistical package SPSS 25. The prevalence of spasticity was calculated as the percentage of patients with at least slight muscle tone according to the MAS, thus excluding those with normal tone and flaccidity. We used Cohen's kappa coefficient to evaluate the degree of agreement in muscle tone between the elbow and wrist at each evaluation time. The evolutionary pattern of the elbow and wrist tone was examined by comparing T1 and T2 and then T2 and T3, using the marginal homogeneity test and reporting its standard Mantel–Haenszel statistic. This statistical test evaluates changes in repeated measures of a variable composed of three or more categories. Finally, to predict muscle tone, we used Cramer's *V* coefficient when the predictor was nominal and Goodman and Kruskal's gamma (*G*) coefficient when the predictor was ordinal. Obtaining significant *V* values was followed by a post hoc analysis of the contingency table using the *z* test, with the Bonferroni correction for pairwise comparisons of the categories (percentages) of the predictor variable.

Results

Prevalence and onset of spasticity in the elbow and wrist

The prevalence of PSS in the elbow was 37.5 % at T1 and 57.4% at T2 and T3. We observed similar or identical percentages in the wrist: 36.8% at T1 and 57.4% at T2 and T3. These results indicate that, for both joints, the prevalence of spasticity increased from T1 to T2 but remained the same from T2 to T3. Among the 136 patients with motor damage, elbow spasticity set in at T1 (i.e., during the first 10 days), between T1 and T2, and between T2 and T3 in 44.7 %, 23.7 %, and 0.9 % of the cases, respectively. Almost one-third (30.7 %) of these patients lacked spasticity in the elbow at all three evaluation times. Similar results occurred with wrist spasticity: for 43.9 % of patients, it began at T1; for 24.6 %, between T1 and T2; and for 0.9 %, between T2 and T3. In addition, 30.7 % of these patients did not present spasticity in the wrist.

Evolution of muscle tone

The kappa coefficients were 0.97 at T1 and 0.84 at T2 and T3, with $p < 0.001$ for all, which indicates a high degree of agreement between the muscle tone of the elbow and wrist at every measurement time. Taking into consideration this high convergence and the high similarity between these two joints in terms of prevalence and onset of spasticity, a decision was made to continue examining only the muscle tone of the elbow in the subsequent analysis. In fact, the evolution pattern of muscle tone in the elbow was nearly identical to that in the wrist. Equally, we observed high similarity between these two joints in terms of the muscular tone prediction patterns. Another reason for this decision was that the literature reveals a higher prevalence of spasticity in the elbow (79 %) compared with the wrist (66 %) (19).

Table 2 presents the crossed distributions of muscle tone categories in the elbow, comparing T1 and T2 and comparing T2 and T3. For correct interpretation, it must be considered that although the cells of the downward diagonal (indicated with a border) include the patients who demonstrated maintained tone between assessment times, the cells above and below the diagonal indicate the patients who presented increased or reduced tone, respectively.

Table 2. Evolution of muscle tone in the elbow: T1 × T2 and T2 × T3

T2					
Spasticity					
T1	Flaccidity	Normal	Mild	Moderate	Severe
Flaccidity	4	13	21	4	0
Normal	0	41	1	1	0
Mild	0	0	32	14	1
Moderate	0	0	0	1	3
Severe	0	0	0	0	0
T3					
Spasticity					
T2	Flaccidity	Normal	Mild	Moderate	Severe
Flaccidity	1	2	1	0	0
Normal	0	54	0	0	0
Mild	0	1	53	0	0
Moderate	0	0	0	20	0
Severe	0	0	0	0	4

Significant changes in elbow tone between T1 and T2 were shown in the test of marginal homogeneity, $\text{HM} = -6.98, p < 0.001$. As noted in Table 2 (section T1 × T2), no patients presented reduced tone, whereas 58 patients (42.6%) presented increased tone. Of the 42 patients with flaccidity at T1, 13 changed to normal tone, 21 to slight spasticity, and 4 to moderate spasticity. Of the 43 patients with normal tone at T1, 1 changed to slight spasticity and 1 to moderate spasticity. Of the 47 who exhibited slight spasticity at T1, 14 moved to moderate and 1 to severe spasticity. Finally, of the 4 patients with moderate spasticity at T1, 3 changed to severe spasticity. It is relevant to note that, of the 41 patients who maintained normal tone between T1 and T2, 21 presented no motor damage. We noted no significant changes in elbow tone between T2 and T3 ($\text{HM} = -1.41; p = 0.157$). Indeed, at T3, 132 patients (97.1%) showed conservation of the tone they presented at T2 (see Table 2, section T2 × T3).

Prediction of muscle tone in the elbow

Table 3 presents the results of three sociodemographic and five clinical variables as predictors of muscle tone in the elbow at each evaluation time. Sex, age, type of stroke, and hypertension did not significantly predict elbow muscle tone at any of the measurement times.

Table 3. Prediction of muscle tone in the elbow from sociodemographic and clinical variables

Predictor	Test	T1	T2	T3
Sex	V	0.14	0.15	0.12
Age range	G	0.16	0.13	0.12
Ethnic self-classification	V	0.19	0.31*	0.28*
Type of stroke	V	0.31**	0.27*	0.26
Hypertension	V	0.21	0.18	0.17
Brain damaged area (OCSP)	V	0.34***	0.37***	0.39***
Extent of damage MCA (ASPECTS)	G	0.17	0.52***	0.50***
Quantity of rehabilitation sessions	G	0.16	0.69***	0.70***

ASPECTS: Alberta Stroke Program Early CT Score; G: Goodman and Kruskal's gamma coefficient; MCA: middle cerebral artery; OCSP: Oxfordshire Community Stroke Project Classification; V: Cramer's V coefficient.

* $p > 0.05$. ** $p > 0.01$. *** $p > 0.001$.

Ethnic self-classification was the only sociodemographic variable that significantly predicted tone, although only at T2 and T3. As shown in Table 4, post hoc analyses at T2 revealed significant differences in the categories of normal tone (46.7% in nonindigenous people vs. 25.0% in Mapuches) and slight spasticity (33.7% in nonindigenous people vs. 52.3% in Mapuches). The significant difference observed in the normal category at T2 persisted at T3 (48.9% in nonindigenous people vs. 27.3% in Mapuches). These results suggest that, comparatively, the nonindigenous patients tended to present normal tone and the Mapuche patients tended to present slight spasticity.

Table 4. Prediction of muscular tone in the elbow according to ethnic self classification at T2 and T3 (%)

T2					
Spasticity					
Ethnic self-classification	Flaccidity	Normal	Mild	Moderate	Severe
Mapuche	0.0	25.0 ^a	52.3 ^a	22.7	0.0
Non-Mapuche	4.3	46.7 ^b	33.7 ^b	10.9	4.3
T3					
Mapuche	0.0	27.3 ^a	50.0	22.7	0.0
Non-Mapuche	1.1	48.9 ^b	34.8	10.9	4.3

Note: At each time, percentages in a column with different superscripts differ significantly ($p < 0.05$) according to paired comparisons using the Bonferroni correction z test.

With the exception of HBP, all clinical variables significantly predicted elbow tone for at least two evaluation times (see Table 3). In addition, the damaged brain area (according to the OCSP) significantly predicted the muscle tone of the elbow (see Table 3). We also observed

greater ischemic damage (according to ASPECT) at T2 and T3. Finally, a higher number of rehabilitation sessions predicted increased elbow tone (see Table 3).

Discussion

This study is the first in Chile to longitudinally examine PSS and its predictors. Currently, the evidence concerning the onset and predictions of spasticity is controversial (36). It would be advantageous for professionals in the area of rehabilitation to have guiding scientific evidence during the acute period after a stroke that identifies which patients are at risk of developing spasticity and which motor functions could be affected, as well as evidence that provides details of the prevalence and onset of spasticity in the elbow and wrist.

There is disparate scientific evidence regarding the evolution time of spasticity, stimulating the search for new knowledge. In addition, although not all patients with spasticity require therapeutic intervention, early attention could reduce disability levels in those who need it (38), making early investigation fundamental (37). The reported prevalence of spasticity varies from 4% to 27% during the first 6 weeks after stroke, with the peak between 1 and 3 months after the damage, results that are similar to those found in the present study (9,13,39).

Among all patients assessed, the prevalence of spasticity in the elbow was 57.4% at 3 months, which remained steady at 12 months poststroke. These findings are comparable with other studies in which the prevalence varied between 16% and 42.6% in at least one joint in cohorts of 83 to 328 patients (19,24,40). The increase between T1 and T2 can be explained through studies with electromyography, which showed that muscle tone reached its maximum at 3 months poststroke, implying that spasticity may be caused not only by damage in the neural structures but also by adaptive characteristics, such as intrinsic changes in the muscle (41-43). The UE adopts specific patterns causing immobilization (especially in short lengths), which triggers muscle contracture (8,17,44). The contracture and subsequent muscle fibrosis could even increase spasticity via the hyperactivation of the afferent pathways of the spindle during muscle lengthening (8). At the same time, muscle immobilization reduces postactivation depression, which is a fundamental mechanism in spasticity development (45).

Of the patients who had motor damage at T1, 47 presented with hemiparesis and 67 with hemiplegia in the UE, and for almost half of these individuals, spasticity set in during the first 10 days of evolution, results similar to those presented by Dorňák (14). With regard to the evolution of muscle tone, between T1 and T2, no patients presented reduced tone, whereas 58 patients demonstrated increased tone. This finding is in line with the results of Wissel et al., who suggested that patients with no initial signs of increase in tone can develop spasticity during a longer follow-up (19). The accumulated evidence suggests that the identification

of predictors of spasticity is limited, and the present study contributes to expanding this knowledge (9,38,46).

Ethnic self-classification (Mapuche or nonindigenous people) was the only sociodemographic variable that significantly predicted elbow tone at T2 and T3, indicating that, comparatively, nonindigenous patients tended to exhibit normal tone and Mapuche patients increased tone—specifically, slight spasticity. Although this finding is highly relevant, because it contributes new information in this area of study, possible explanations should be considered.

Some evidence has revealed a greater incidence of stroke and worse outcomes in native populations and ethnic minorities (47,48). The largest Mapuche indigenous population in the country is concentrated in the Chilean region of La Araucanía, where this study was conducted (49). In this region, the Mapuches have a mortality rate from stroke that is twice the rate observed in the rest of the country, as they also have a high prevalence of poverty, HBP, diabetes, sedentary lifestyle, and overweight, all risk factors for stroke (50-53). However, there is no evidence that specifically associates this ethnic group with the occurrence of stroke (54).

It is possible that the increased tone in the Mapuche population, in contrast to nonindigenous people, is due to the stroke being more serious in the indigenous group, generating greater spasticity. Two additional findings in the present study seem to support this possible explanation. On one hand, there was a marginally significant association between ethnic group and motor damage ($p = 0.08$), indicating that 52.6 % of Mapuches presented with hemiplegia (vs. 35.5 % of nonindigenous people) and 64.5 % of nonindigenous people presented with hemiparesis (vs. 47.4 % of Mapuches). These results suggest a tendency for Mapuches to present with hemiplegia and nonindigenous people to present with hemiparesis. From the point of view of motor function, hemiplegia is known to involve greater disability than hemiparesis does. On the other hand, when examining the relation between ethnic group and number of rehabilitation sessions, we found that a significantly higher percentage of Mapuches required more than 30 sessions ($p = 0.04$). If strokes were more serious in Mapuches, their spasticity would also be greater, and they would consequently require more rehabilitation sessions.

From the clinical point of view, three variables significantly predicted elbow tone for at least two evaluation times.

1. The type of stroke predicted muscle tone at T1 and T2; in relative terms, patients with ischemic stroke tend to have normal tone, and patients with hemorrhagic stroke have slight spasticity. These results agree with studies indicating that after a 3-month follow-up period, patients who developed spasticity had a previous diagnosis of hemorrhagic stroke (55). In addition, Lundström et al. found that hemorrhagic stroke was a predictor of spasticity at both the first month and 6 months after the damage, with a similar statistical significance to this study (13). Katoozian et al.

suggested that the risk of presenting spasticity due to hemorrhagic stroke is 2.5 times, with no differences regarding anterior or posterior circulation (56).

2. The damaged brain area (according to the OCSF) significantly predicted muscle tone in the elbow at the three assessment times, identifying a significantly greater presence of patients with LACI and POCI in the normal tone category and more patients with TACI and PACI in the slight spasticity category. Previous research remains controversial regarding this issue. In their study, Kong et al. found that the lesion site, evaluated through the OCSF, was not a prognostic factor for spasticity (35). However, in the SALGOT study, in which several predictive models were evaluated, the OCSF was an important predictor of severe spasticity (57). However, these disparities could be due to the distribution by location; for example, in the two cited studies, patients with TACI or PACI comprised fewer than 50% of the sample, whereas in the present study, they represent 69.1% indicating that, clinically, patients with greater damage have higher chances of presenting with PSS.

3. The association between PSS and the extent of the lesion is not well known (58). The scientific evidence presents some software-support imaging studies. In our study, the extent of the damage assessed through ASPECTS significantly predicted elbow tone at T2 and T3. In line with this result, Picelli et al. conducted a retrospective analysis of patients with chronic stroke who had lesions in the insula, basal ganglia, and thalamus as well as in the internal and external capsule, corona radiata, and superior longitudinal bundle, concluding that these patients can suffer severe PSS in both the UE and the lower extremity (59). Cheung et al. observed a correlation between spasticity and lesions occurring in the putamen (60). This association was not unexpected, as these structures are irrigated by the MCA and its lenticulostriate branches, encompassing broad areas of the brain that receive primary sensory input, the motor and premotor cortices, and the extrapyramidal cortex.

Among its strengths, our study is the first conducted in Latin America to include a longitudinal evaluation of the prevalence, onset, evolution, and prediction of elbow PSS. This study also included ethnic self-identification, a variable that seems to be a relevant predictor of muscle tone and a finding that should be considered when studying ethnically heterogeneous populations. Another aspect to emphasize is the low percentage of follow-up loss over the three measurement times. In addition, we assessed all patients clinically and through neuroimaging, using scales widely validated in vascular neurology.

The limitations of this study include the recruitment of the sample, as only two health centers in the city were included. Although these centers attend 80% of the stroke admissions in the geographic sector of reference, they do not necessarily represent the reality of other centers in the geographical study area. Another limitation is the inclusion of a low percentage of patients with severe stroke, which could lead to underestimation of spasticity prevalence; however, this was a characteristic of the subjects who comprised the study sample.

Acknowledgments

We would like to thank the patients and professionals who participated during the study period.

Funding

Project DIUFRO DI18-0034 y DIUFRO DIE18-0010, Universidad de La Frontera (Chile).

Ethical considerations

This study was conducted in 2019 and 2020, with prior approval from the Ethics Committee of the South Araucanía Health Service (SSAS) and recorded at ClinicalTrials.gov (NCT03588832).

Authors' contribution

(1) Conceptualization; (2) Data curation; (3) Formal analysis-statistical techniques, data analysis; (4) Fund acquisition; (5) Research-research process; (6) Methodology-design and development; (7) Project administration-management and coordination; (8) Resources-supply of study materials; (9) Software-programming, design, and implementation; (10) Planning supervision and leadership; (11) Validation-verification; (12) Data visualization-presentation; (13) Writing-original document; (14) Writing-review and editing.

Author	Codes authorship
Paulina Sepúlveda	1, 5, 6, 7, 8, 9, 10, 12, 13, 14
Arlette Doussoulin	1, 5, 6, 7, 8, 9, 10, 13, 14
Claudia Rivas	1, 5, 6, 7, 13, 14
Rodrigo Rivas	1, 5, 6, 7, 13, 14
José Luis Saiz	1, 2, 3, 6, 11, 12, 13

Conflict of interest

All authors declare that they have no conflict of interest concerning the research related to the manuscript.

References

1. Mayer N. Spasticity and other signs of the upper motor neuron syndrome. In: Brashear A, editor. Spasticity, diagnosis and management. New York: Demos Medical Publishing; 2016. p. 17-31.
2. Li S, Francisco GE. New insights into the pathophysiology of post-stroke spasticity. *Frontiers in human neuroscience*. 2015;9:192. <https://doi.org/10.3389/fnhum.2015.00192>
3. Lance JW. The control of muscle tone, reflexes, and movement: Robert Wartenberg Lecture. *Neurology*. 1980;30(12):1303-13.
4. Dan B. The end of spasticity? *Dev Med Child Neurol*. 2017;59(9):882. <https://doi.org/10.1111/dmcn.13496>
5. Pandyan AD, Gregoric M, Barnes MP, Wood D, Van Wijck F, Burridge J, et al. Spasticity: clinical perceptions, neurological realities and meaningful measurement. *Disabil Rehabil*. 2005;27(1-2):2-6. <https://doi.org/10.1080/09638280400014576>
6. Li S. Spasticity, motor recovery, and neural plasticity after stroke. *Front Neurol*. 2017;8(120). <https://doi.org/10.3389/fneur.2017.00120>
7. Sepúlveda P, Bacco J, Cubillos C, Doussoulín A. Espasticidad como signo positivo de daño de motoneurona superior y su importancia en rehabilitación. *CES Med*. 2018;32(3):259-69. <https://doi.org/10.21615/cesmedicina.32.3.7>
8. Gracies JM. Pathophysiology of spastic paresis. I: Paresis and soft tissue changes. *Muscle Nerve*. 2005;31(5):535-51. <https://doi.org/10.1002/mus.20284>
9. Wissel J, Verrier M, Simpson DM, Charles D, Guinto P, Papapetropoulos S, et al. Post-stroke spasticity: predictors of early development and considerations for therapeutic intervention. *PM & R: Journal of Injury, Function, and Rehabilitation*. 2015;7(1):60-7. <https://doi.org/10.1016/j.pmrj.2014.08.946>
10. Burke D, Wissel J, Donnan GA. Pathophysiology of spasticity in stroke. *Neurology*. 2013;80(3 Suppl 2):S20-6. <https://doi.org/10.1212/WNL.0b013e31827624a7>
11. Sunnerhagen K. Predictors of spasticity after stroke. *Curr Phys Med Rehabil Rep*. 2016;4:182-85. <https://doi.org/10.1007/s40141-016-0128-3>
12. Wissel J, Manack A, Brainin M. Toward an epidemiology of poststroke spasticity. *Neurology*. 2013;80(3 Suppl 2):S13-9. <https://doi.org/10.1212/WNL.0b013e3182762448>
13. Lundström E, Smits A, Terent A, Borg J. Time-course and determinants of spasticity during the first six months following first-ever stroke. *J Rehabil Med*. 2010;42(4):296-301. <https://doi.org/10.2340/16501977-0509>

14. Dornák T JM, Konvalinková R, Říha M, Mužík J, Hoskovcová M. Prevalence and evolution of spasticity in patients suffering from first-ever stroke with carotid origin: a prospective, longitudinal study. *European journal of neurology*. 2019;26(6):880-6. <https://doi.org/10.1111/ene.13902>
15. Mirbagheri MM, Alibiglou L, Thajchayapong M, Rymer WZ. Muscle and reflex changes with varying joint angle in hemiparetic stroke. *J Neuroeng Rehab*. 2008;5:6. <https://doi.org/10.1186/1743-0003-5-6>
16. Doussoulín A, Rivas C, Bacco J, Rivas R, Sepúlveda P. Effects of spasticity on motor recovery after a stroke. *Rev Chil Neuro Psiquiat*. 2019;57(4):377-86.
17. Hefter H, Jost WH, Reissig A, Zakine B, Bakheit AM, Wissel J. Classification of posture in poststroke upper limb spasticity: a potential decision tool for botulinum toxin A treatment? *Int J Rehabil Res*. 2012;35(3):227-33. <https://doi.org/10.1097/MRR.0b013e328353e3d4>
18. Friden J, Lieber RL. Spastic muscle cells are shorter and stiffer than normal cells. *Muscle Nerve*. 2003;27(2):157-64. <https://doi.org/10.1002/mus.10247>
19. Wissel J, Schelosky LD, Scott J, Christe W, Faiss JH, Mueller J. Early development of spasticity following stroke: a prospective, observational trial. *J Neurol*. 2010;257(7):1067-72. <https://doi.org/10.1007/s00415-010-5463-1>
20. Wei TS, Liu PT, Chang LW, Liu SY. Gait asymmetry, ankle spasticity, and depression as independent predictors of falls in ambulatory stroke patients. *Plos One*. 2017;12(5):e0177136. <https://doi.org/10.1371/journal.pone.0177136>
21. Tedesco Triccas L, Kennedy N, Smith T, Pomeroy V. Predictors of upper limb spasticity after stroke? A systematic review and meta-analysis. *Physiotherapy*. 2019;105(2):163-73. <https://doi.org/10.1016/j.physio.2019.01.004>
22. Gomez-Soriano J C-d-l-CR, Munoz-Hellin E, Ortiz-Gutierrez R, Taylor J. Evaluation and quantification of spasticity: a review of the clinical, biomechanical and neurophysiological methods. *Rev Neurol*. 2012;55(4):217-26.
23. Bernhardt J, group AC. Response to letter: and yet it moves - AVERT enlightens translations stroke research. *Int J Stroke*. 2017;12(9):NP14-NP5. <https://doi.org/10.1177/1747493015621453>
24. Lundström E, Terent A, Borg J. Prevalence of disabling spasticity 1 year after first-ever stroke. *Eur J Neurol*. 2008;15(6):533-39. <https://doi.org/10.1111/j.1468-1331.2008.02114.x>
25. Lieber RL, Runesson E, Einarsson F, Friden J. Inferior mechanical properties of spastic muscle bundles due to hypertrophic but compromised extracellular matrix material. *Muscle Nerve*. 2003;28(4):464-71. <https://doi.org/10.1002/mus.10446>
26. Sunnerhagen K. OAMM. Onset, time course and prediction of spasticity after stroke or traumatic brain injury. *Ann Phys Rehabil Med*. 2019;62(6):431-4. <https://doi.org/10.1016/j.rehab.2018.04.004>
27. Thilmann AF, Fellows SJ, Garms E. The mechanism of spastic muscle hypertonus. Variation in reflex gain over the time course of spasticity. *Brain*. 1991;114(Pt 1A):233-44. <https://doi.org/10.1093/oxfordjournals.brain.a101859>
28. Barber PA, Demchuk AM, Zhang J, Buchan AM. Validity and reliability of a quantitative computed tomography score in predicting outcome of hyperacute stroke before throm-

- bolytic therapy. ASPECTS Study Group. Alberta Stroke Programme Early CT Score. *Lancet*. 2000;355(9216):1670-4. [https://doi.org/10.1016/S0140-6736\(00\)02237-6](https://doi.org/10.1016/S0140-6736(00)02237-6)
29. Nagel S, Sinha D, Day D, Reith W, Chapot R, Papanagiotou P, et al. e-ASPECTS software is non-inferior to neuroradiologists in applying the ASPECT score to computed tomography scans of acute ischemic stroke patients. *Int J Stroke*. 2017;12(6):615-22. <https://doi.org/10.1177/1747493016681020>
 30. Bamford J, Sandercock P, Dennis M, Burn J, Warlow C. Classification and natural history of clinically identifiable subtypes of cerebral infarction. *Lancet*. 1991;337(8756):1521-6. [https://doi.org/10.1016/0140-6736\(91\)93206-O](https://doi.org/10.1016/0140-6736(91)93206-O)
 31. Mead GE, Lewis SC, Wardlaw JM, Dennis MS, Warlow CP. How well does the Oxfordshire community stroke project classification predict the site and size of the infarct on brain imaging? *J Neurol Neurosurg Psychiatry*. 2000;68(5):558-62. <https://doi.org/10.1136/jnnp.68.5.558>
 32. Arturo Agredo C, Bedoya JM. Validación Escala Modificada de Ashworth [Internet]. *Fisioterapia*. 2005 Jun 6. Available from: <https://www.efisioterapia.net/articulos/validacion-escala-ashworth-modificada>
 33. Platz T, Eickhof C, Nuyens G, Vuadens P. Clinical scales for the assessment of spasticity, associated phenomena, and function: a systematic review of the literature. *Disabil Rehabil*. 2005;27(1-2):7-18. <https://doi.org/10.1080/09638280400014634>
 34. Charalambous C. Interrater Reliability of a Modified Ashworth Scale of Muscle Spasticity. In: Banaszkiewicz P. KD, editor. *Classic Papers in Orthopaedics*. London: Springer; 2014.
 35. Kong KH, Lee J, Chua KS. Occurrence and temporal evolution of upper limb spasticity in stroke patients admitted to a rehabilitation unit. *Arch Phys Med Rehabil*. 2012;93(1):143-8. <https://doi.org/10.1016/j.apmr.2011.06.027>
 36. Brainin M, Norrving B, Sunnerhagen KS, Goldstein LB, Cramer SC, Donnan GA, et al. Poststroke chronic disease management: towards improved identification and interventions for poststroke spasticity-related complications. *Int J Stroke*. 2011;6(1):42-6. <https://doi.org/10.1111/j.1747-4949.2010.00539.x>
 37. Yelnik AP, Simon O, Parratte B, Gracies JM. How to clinically assess and treat muscle overactivity in spastic paresis. *J Rehabil Med*. 2010;42(9):801-7. <https://doi.org/10.2340/16501977-0613>
 38. Leathley MJ, Gregson JM, Moore AP, Smith TL, Sharma AK, Watkins CL. Predicting spasticity after stroke in those surviving to 12 months. *Clin Rehabil*. 2004;18(4):438-43. <https://doi.org/10.1191/0269215504cr727oa>
 39. Kong KH, Lee J, Chua KS. Occurrence and temporal evolution of upper limb spasticity in stroke patients admitted to a rehabilitation unit. *Arch Phys Med Rehabil*. 2012;93(1):143-8. <https://doi.org/10.1016/j.apmr.2011.06.027>
 40. Dornák T, Justanová M, Konvalinková R, Říha M, Mužík J, Hoskovicová M, et al. Prevalence and evolution of spasticity in patients suffering from first-ever stroke with carotid origin: a prospective, longitudinal study. *Eur J Neurol*. 2019;26(6):880-6. <https://doi.org/10.1111/ene.13902>
 41. Bhakta BB. Management of spasticity in stroke. *Br Med Bull*. 2000;56(2):476-85. <https://doi.org/10.1258/0007142001903111>

42. Welmer AK, von Arbin M, Widen Holmqvist L, Sommerfeld DK. Spasticity and its association with functioning and health-related quality of life 18 months after stroke. *Cerebrovasc Dis.* 2006;21(4):247-53. <https://doi.org/10.1159/000091222>
43. Galiana L, Fung J, Kearney R. Identification of intrinsic and reflex ankle stiffness components in stroke patients. *Exp Brain Res.* 2005;165(4):422-34. <https://doi.org/10.1007/s00221-005-2320-z>
44. Trompetto C, Marinelli L, Mori L, Pelosin E, Curra A, Molfetta L, et al. Pathophysiology of spasticity: implications for neurorehabilitation. *BioMed Res Int.* 2014;2014:354906. <https://doi.org/10.1155/2014/354906>
45. Schinwelski MJ, Sitek EJ, Waz P, Slawek JW. Prevalence and predictors of post-stroke spasticity and its impact on daily living and quality of life. *Neurol Neuroch Polska.* 2019;53(6):449-57. <https://doi.org/10.5603/PJNNS.a2019.0067>
46. Trimble B, Morgenstern LB. Stroke in minorities. *Neurol Clin.* 2008;26(4):1177-90. <https://doi.org/10.1016/j.ncl.2008.05.010>
47. Pathak EB, Sloan MA. Recent racial/ethnic disparities in stroke hospitalizations and outcomes for young adults in Florida, 2001-2006. *Neuroepidemiology.* 2009;32(4):302-11. <https://doi.org/10.1159/000208795>
48. Estadísticas. INd. Resultados Censo 2017 [Internet]. Chile: Gobierno de Chile; 2017. Available from: http://www.censo2017.cl/wpcontent/uploads/2018/05/presentacion_de_la_segunda_entrega_de_resultados_censo2017.pdf
49. Doussoulín A, Rivas R, Sabelle C. [Hospital discharges due to stroke in the period 2001-2010 in a southern Chilean region]. *Rev Med Chil.* 2016;144(5):571-6.
50. Navarrete CC, Cartes-Velásquez R. Prevalencia de hipertensión arterial en comunidades Pehuenches del Alto Bio Bio. *Rev Chil Cardiol.* 2012;31(2):102-07. <http://dx.doi.org/10.4067/S0718-85602012000200004>
51. Lavados PM, Díaz V, Jadue L, Olavarria VV, Carcamo DA, Delgado I. Socioeconomic and cardiovascular variables explaining regional variations in stroke mortality in Chile: an ecological study. *Neuroepidemiology.* 2011;37(1):45-51. <https://doi.org/10.1159/000328872>
52. Perez F, Carrasco E, Santos JL, Calvillan M, Albala C. Prevalence of obesity, hypertension and dyslipidemia in rural aboriginal groups in Chile. *Rev Med Chil.* 1999;127(10):1169-75.
53. Soto A, Morales G, Provoste R, Lanas F, Aliaga I, Pacheco D, et al. Association between Mapuche Ethnicity and Stroke: A Case-Control Study. *J Stroke Cerebrovasc Dis.* 2019;28(5):1311-6. <https://doi.org/10.1016/j.jstrokecerebrovasdis.2019.01.029>
54. Cha KS, Lee H, Kim D, Lee J, Sohn M, Lee S, et al. Prevalence rate of spasticity at 3 months after stroke in Korea: The Korean Stroke Cohort for Functioning and Rehabilitation (kosco) study. *Brain Neurorehabil.* 2016;9(2):e6.
55. Katoozian L, Tahan N, Zoghi M, Bakhshayesh B. The Onset and Frequency of Spasticity After First Ever Stroke. *J Nat Med Assoc.* 2018;110(6):547-52. <https://doi.org/10.3389/fneur.2020.616097>
56. Opheim A, Danielsson A, Murphy M, Persson HC, Sunnerhagen KS. Early prediction of long-term upper limb spasticity after stroke: Part of the SALGOT study. *Neurology.* 2015;85(10):873-80. <https://doi.org/10.1212/WNL.0000000000001908>

57. Lee KB, Hong BY, Kim JS, Sul B, Yoon SC, Ji EK, et al. Which brain lesions produce spasticity? An observational study on 45 stroke patients. Plos One. 2019;14(1):e0210038. <https://doi.org/10.1371/journal.pone.0210038>
58. Picelli A, Tamburin S, Gajofatto F, Zanette G, Praitano M, Saltuari L, et al. Association between severe upper limb spasticity and brain lesion location in stroke patients. BioMed research international. 2014;2014:162754. <https://doi.org/10.1155/2014/162754>
59. Cheung DK, Climans SA, Black SE, Gao F, Szilagyi GM, Mochizuki G. Lesion Characteristics of Individuals With Upper Limb Spasticity After Stroke. Neurorehabil Neural Repair. 2016;30(1):63-70. <https://doi.org/10.1177/154596831558>