Improved Biomedical Device for Spasticity Quantification

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***Abstract*—Spasticity is a disabling motor disorder, resulting from neurological impairments. This disorder has now a remarkable social and economic impact, affecting approximately 1.9% of the world population. The methods for spasticity quantification play an important role in the medical treatments' success and may reduce the associated costs. The Modified Asworth Scale is the most commonly method used for the evaluation of this disorder, but experts agree that it is not a precise and reproducible method. Despite the literature presents several innovative methods, there is still a need for improvements. This paper presents the development of a biomedical device for spasticity quantification, based on the velocity dependent increase of the stretch reflexes threshold. The proposed approach was tested in clinical environment, with the collaboration of patients with spasticity. The experimental trials allow confirming the sensibility, reproducibility and reliability of the proposed approach.**

Keywords—Spasticity quantification, Tonic Stretch Reflex Threshold.

I. INTRODUCTION

Spasticity is a common pathological phenomenon, in clinical practices, that interferes in the motor control function causing difficulties in the daily activities [1]. This phenomenon is typically associated with lesions in the central nervous system, frequently expressed in diseases such as stroke, multiple sclerosis or cerebral palsy [2], [3]. Spasticity has now an remarkable social and economic impact, affecting approximately 1,9% of the world population [4], [5], [6].

This pathology is integrated in the syndrome of the upper motor neuron and it occurs with lesions in the first motoneuron of the pyramidal tract. In spasticity, the principal mechanism that determines the appearance of the clinical manifestations is the deregulation/hyper-excitability of the stretch reflex (SR), conducing to muscle hyperton, hyperactivity reflexive and spasms [2], [7], [8].

The mechanism of spasticity is commonly thought as an exaggerated SR excitability, due to the reduction of the SR threshold (SRT) which is velocity-dependent. This reduction in the SRT is correlated with the increase in reflex joint torque [7], [8].

Several treatments and therapies have emerged for the treatment of spasticity [9]. The treatment must be appropriate to the symptoms presented by each patient [10]. The existence of a sensitive, consistent and reproducible method for clinical evaluation of the level of spasticity plays an important role in the selection of the appropriate treatment. This clinical evaluation enables the monitoring of the evolution of spasticity over time, in response to the applied therapy [9], [11].

The quantification of spasticity is a hard and complex task and has been under an extensive study by the scientific community. Despite the constant progresses, there is not yet available a well-accepted standard method. The literature presents several methods for this propose: the Ashworth Scale (AS) and the modified AS version (MAS); isokinetic device; Pendulum Test [4], [11], [12]. These methods are typically based on the phase and magnitude of the SR and the resistance to passive stretch. Nevertheless, this measure is not correlated with the clinical impression of spasticity and cannot differentiate the mechanical stiffness from the reflective stiffness. On the other hand, the implementation of the device is still complex. Hence, there is still a need for a device that meets these requirements [13], [11], [12].

The key issue for spasticity quantification is to determine which variables can correctly quantify this disorder. A correct measure must follow the physiological mechanisms related to the motor control system in healthy individuals allowing detecting the deregulation in these mechanisms that lead to motor disorders. For this purpose the applied approach must be in accordance to a standard definition of spasticity [11], [12], [14].

Lance [15] defined spasticity as: "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes ("muscle tonus") with exaggerated tendon jerk, resulting in hyper excitability of the stretch reflexes; it is one of the components of the upper motor neuron syndrome". This definition is still accepted nowadays and includes some important aspects: refers to spasticity as a disorder in the somatic mobility, related to the high tonic component of the SR; it is due to the spinal reflex disorder; it is one of the upper motoneuron syndrome, reflects the physical component of the SR; the SRT is the basis of the tonus; it is referred that the excess of the reflex depends on the stretch velocity. This last statement can be the key issue for spasticity quantification [4], [5].

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This paper presents the study, design and development of a mechatronic device for the quantification of spasticity, in joints of ankle, elbow or knees. The proposed approach is based on the velocity dependent increase in the tonic SRT.

II. PROPOSED APPROACH

Based on the proposed approach it was developed a system, including hardware and software that can be used in the joint of elbow, ankle and knee. The system was tested in the Biceps Braqualis of patients with spasticity. For this study it is considered 30º as the maximum flexion of the elbow and 180º as full extension of the elbow [16].

This approach is focused on the velocity-dependent increase of the SRT. The variable to be quantified is the Tonic SRT (TSRT) that represents the angle joint position at which a SR is evoked at velocity equals to zero (at rest). The value of TSRT (parameter λ in Figure 1) represents the muscle length from which the muscle is activated, in response to any stretch, registering an increase of torque due to the increase of muscle length and of the velocity of stretching. In healthy subjects λ is out of the range of motion of the limb, presenting full freedom of movements. In subjects with spasticity the deregulation of the SR cause λ to lie into the range of motion of the limb, presenting limitation in movements [17], [18].

In order to achieve the desired goals, it was developed a system to record the angle joint position and the angular velocity of a passive muscle stretch and the EMG signal. Both angle joint position and angular velocity was computed based on the signal recorded by the same sensor (electro-goniometer, Biometrics SG 110).

The EMG signal is recorded by surface electrodes (Ag/AgCL), using bipolar configuration. Both signals were filtered, amplified and sampled with an analog-to-digital converter (ADC) and sent to a computer for signal and data acquisition and processing.

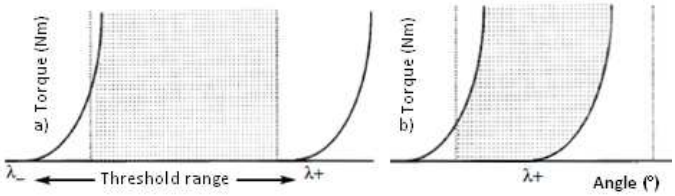

Figure 1 Relation between range of regulation of TSRT (λ) and biomechanical range of joint in healthy individuals, a), and individuals with spasticity, b) Limit of biomechanical range are shown by vertical dotted line [17].

The process consists in manually promoting a passive muscle stretch at different velocities, in order to determine the dynamic SRT (DSRT). The DSRT is defined pairing the angle joint position and the velocity of stretch at which it was evoked a SR. SR is an involuntary muscle contraction, identified by an increase in the amplitude of the EMG signal (Figure 2).

The precise time activation of the SR is identified by an algorithm developed for this propose. This algorithm was developed considering the disturbances, in the EMG signal, induced by the deregulation of the SRT, enabled to detect muscle activations in signals with low and high signal to noise ratio [19]. Each DSRT is plotted on an orthogonal plan, angular velocity versus angle. In this analysis velocity is considered as an independent variable and the angle as a dependent variable. The TSRT value is estimated using a regressing analysis, interpolating a regression line between each DSRT. The TSRT is estimated when the regression line cross the horizontal axis, corresponding to velocity zero.

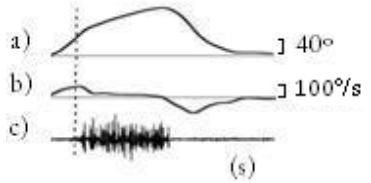

Figure 2 Identification of DSRT. a) Angle joint position, b) velocity of the movement c) and EMG signal [4].

Figure 3 shows the freedom of movements available in this limb, at the left side of the regression line. The area at the right side of the regression line, which originates a TSRT, corresponds to the area in which any muscular stretch would originate a spastic reaction to the movement. In this case, even with slower velocities of stretching, the range of movement is limited to this amplitude, showing a wide range of movements compromised by spasticity. Under this classification a low value of TSRT, near the beginning of the joint range of motion, indicates a high level of spasticity, showing a large range of movements compromised by spasticity; a high value of TSRT, near or above the end of the range of joint movement, indicates a low degree of spasticity, showing a small range of movement compromised by spasticity [4], [17].

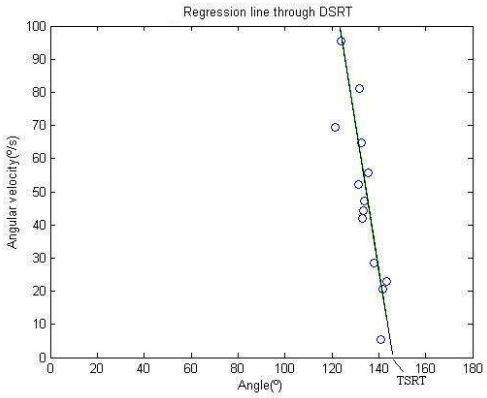

Figure 3 Example of the TSRT estimation, regression line and DSRTs presented as small circles.

The degree of approximation of each regression line to the experimental data was evaluated considering the coefficient of determination (r^2) and it was considered a prediction interval of 95% (Figure 4). The DSRT that does not belong to the considered prediction interval are considered "false detections" and were classified as technical errors. These technical errors can be associated with artifacts originated by the system or by the evaluator during the experimental tests.

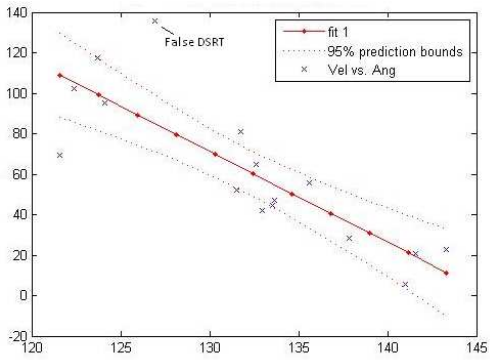

Figure 4 Prediction interval of 95%

A. Patients

To participate in this study 11 patients were selected, aged between 37 and 86 years old. The selected patients present spasticity caused by stroke or cerebral palsy.

The patients' selection was based on the occurrence of spasticity symptoms in the flexor muscles of the elbow. It was assured, by the clinical assistant that each patient was not under the influence of drugs; this situation could interfere in the neuromuscular functions. All the patients received the necessary information regarding the experimental procedure and research objective. After reflecting on their participation in the study, each patient signed the Informed Consent [20].

B. Experimental procedure

Each patient was evaluated, in clinical environment, in three consecutive sessions separated by two days, in order to verify the reproducibility of the results. In each session each patient performed three evaluations, taking into account that the evaluation may be affected by artifacts or failures. In each session the instrumentation was placed in accordance to the experimental protocol. The electro-goniometer was placed in the arm of the patient with the axis of rotation in concordance to the axis of rotation of the elbow joint. Then, the surface EMG electrodes were placed close to the motor point of the target muscle [19]. Each evaluation consists in promoting passive muscle stretch at different velocities, to identify the SRT related to each velocity. It was promoted thirty muscle stretch divided by three range of velocity: low (2º/s to 150º/s), moderated (151º/s to 300º/s) and fast (higher than 300º/s). It was considered 500º/s as the maximum velocity, because at this velocity it is possible to evoke a SR in healthy subjects [21]. For each muscle stretch, the arm of the patient was extended, by the evaluator, from the initial position to the final position. The initial position corresponds to the full flexion of the limb and the final position to the maximum extension of the limb.

After checking all the initial conditions, the evaluator performed the passive muscle stretch recording the EMG, angle and angular velocity signals. These signals were sent and stored in the computer for further analysis.

III. RESULTS

As expected, the SR responds proportionally to the velocity of stretch. In figure 3, it can be seen the distribution of DSRTs, in the orthogonal plan. This example shows that a higher velocity the SR responds faster than at lower velocity of stretch.

It can be stated that the regression line intersects the axis corresponding to angle joint position, clearly between the amplitude of movement of the limb. In fact, this patient present limitations of movements from this angle joint. As expected, the freedom of movements of the limb decreases when the velocity of the movements increases.

As mentioned above, the TSRT was estimated in three different tests in order to test the reproducibility between assessments. This reproducibility was tested with the analysis of variance ANOVA. Despite the variability observed in some patients, the analysis shows a good correlation between the TSRT values estimated (0.362).

Table 1 Results of the analysis of variance ANOVA.

	Sum of squares	df	Average of squares	F	Significance
Between groups	1523,292	2	761,646	1,039	0,362
Into groups	32999,375	45	733,319		
Total	3 4522,667	47			

The variability observed between assessments may be explained by the difference in the coefficients of determination, alterations in the excitability of the UMN and technical errors. The presence of artifacts, technical errors and/or muscle activation originated stretching shortened muscles can originate false DSRTs that conduce to lower coefficients of determination [22], [23]. An indicator of alterations in the excitability of the Upper Motor Neuron is the discrepancy in the number of DSRT observed [4]. A lower excitability of the UMN justifies a higher value of TSRT. Several factors may contribute to changes in the excitability of the UMN, including: emotional stress, discomfort or anxiety. associated to the participation in the trials; consume of stimulants, for example caffeine or tobacco; the etiology of spasticity [4], [24]. Beyond these factors, previous studies demonstrated that promoting repetitive passive muscle stretch can leave to situation of "muscle accommodation" conducing to a decrease in the response of the SR, essentially in individuals with lower level of spasticity [22].

IV. CONCLUSIONS

Spasticity is a disabling motor disorder and has now a remarkable social and economic impact. This article presented a device for spasticity quantification. The analysis of the results obtained in the experimental tests revealed reproducibility of the estimated value of the TSRT, assessed in different days. The obtained results demonstrated the reliability of the quantification of spasticity based on the velocity-dependent increase of the SRT. This work demonstrates that is possible to quantify the range of motion affected by spasticity, presenting a quantitative value for the muscle length from which the spastic resistance to muscle stretch begins. However, it would be important to repeat the tests in order to increase sample dimension.

Table 2 Obtained results, in 3 sessions. "nd" indicates evaluation at which results could not be obtained

Patients	Session 1					Session 2					Session 3				
	Nº DSRT	TSRT	Slope	r^2	>95 %	Nº DSRT	TSRT	Slope	r^2	>95 %	Nº DSRT	TSRT	Slope	r^2	>95 %
M1	12	158	-6.14	0.28	2	14	180	-1.34	0.16	2	12	157	-7.85	0.47	3
M2	3	nd	nd	nd	nd	6	193	-1.73	0.83	1	11	200	-1.96	0.23	2
M3	14	155	-3.61	0.47	3	15	178	-3.92	0.33	6	14	167	-6.75	0.46	3
M4	15	178	-1.2	0.79	3	7	168	284	0.69	6	19	173	-7.68	0.59	10
M5	21	142	-8.08	0.63	8	29	155	-8.16	0.58	11	7	199	-2.5	0.73	1
M6	27	147	-3.98	0.73	11	6	154	-1.57	0.56	0	14	171	-4.13	0.5	3
M7	9	167	-7.69	0.72	1	0	nd	nd	nd	nd	9	240	-1.47	0.69	3
M8	20	164	-0.081	0.8	8	22	169	-0.06	0.75	6	10	208	-1.38	0.57	3
M9	13	162	-3.69	0.7	2	7	169	-10.74	0.71	1	22	168	-3.66	0.35	10
M10	8	207	-2.19	0.15	2	9	177	-3.4	0.6	1	7	199	-3.01	0.13	1
M11	14	204	-1.73	0.65	3	11	205	-1.55	0.047	2	7	201	-2.06	0.51	1
M12	7	252	-8.39	0.19	6	7	142	-7.44	0.46	1	23	259	0.65	0.07	4
M13	23	151	-7.36	0.57	6	11	175	-3.84	0.71	3	17	175	-4.93	0.27	4
M14	14	192	-2.1	0.54	3	12	195	-1.6	0.19	4	8	188	-7.41	0.47	0
M15	15	152	-5.61	0.68	4	30	161	-6.37	0.54	1	16	211	-2.61	0.11	4
M16	23	183	-3.21	0.65	12	29	182	-5.78	0.77	16	33	192	-4.44	0.54	20
M17	7	136	-11.02	0.64	1	41	151	-3.91	0.52	20	28	124	-13,6	0.91	7
M18	9	182	-8.59	0.5	3	7	222	-3.76	0.82	1	7	173	-3.81	0.42	1

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