Using sEMG Signal Frequency to Evaluate Post-Stroke Elbow Spasticity

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***Abstract*— Spasticity is a motor disorder with high prevalence and critical consequences following a stroke. Reliable and sensitive measurements are important to guide the selection and evaluation of treatment strategies. Technology-assisted methods, such as the surface electromyography (sEMG) technique, have been developed to measure spasticity as sensitive and accurate alternatives to commonly used clinical scales. However, sEMG amplitude based measures may confound spasticity-induced muscle activities with other types of muscle contractions. This study thus introduces the idea of using sEMG frequency information to detect spasticity as a potential solution to overcome the limitations of existing sEMG based measures. The preliminary results of three patients demonstrate the possibility and future research directions for this approach.**

I. Introduction

Spasticity is a common sequela in people suffering a neurological injury. It manifests as involuntary muscle contractions secondary to an upper neuron motor disorder. Over the last four decades, the most commonly cited definition of spasticity is "a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex" [1]. Spasticity is highly prevalent following a stroke with a prevalence rate estimated from 30% to 80% [2]. Upper-limb spasticity is strongly correlated with post-stroke pain [3], and it also limits patient engagement in rehabilitation [4]. In addition, spasticity could lead to an increase of socioeconomic burden of stroke by 400% [5]. Therefore, effective management of spasticity is a critical issue in the neuro-rehabilitation field [6], and accurate measures of spasticity are important to evaluate the effectiveness of the treatment strategies [7].

Clinical scales — Modified Ashworth Scale (MAS) and Modified Tardieu Scale (MTS) — are the main widely used conventional solutions by clinicians to measure spasticity [8]. However, both MAS and MTS rely on the perceptive resistance of practitioners to assess the reaction of affected muscles during passive stretching [9], [10]. Not surprisingly, the reliability of these measures is questionable given their dependence on the experience of clinicians [9]. These clinical measures are also limited by their ability to distinguish between spasticity (neural origin) and muscle stiffness (non-neural origin).

To address the aforementioned limitations of the clinical scales, measurements from surface electromyography (sEMG) have been used to quantify the spastic muscle response to stretching, and this is specific to the stretch reflex as per definition in [1]. The sEMG-based muscle activity measures of spasticity are promising relevance and feasibility and thus have the potential to be widely adopted in clinical practice [11]. However, spastic muscle reactions might be confounded by other types of muscle activities when monitored with sEMG signal amplitudes. For example, muscle firing was also observed in the healthy subjects who were not expected to present any spastic response during passive stretching [12], so it is suspected that voluntary muscle contractions may happen. This phenomenon thus leads to the complications of the sEMG amplitude based outcome measures in detecting spasticity-induced muscle activities.

A previous study [13] analysed both the time and frequency domain features of sEMG measurements for spasticity evaluation when manual elbow stretching was performed on post-stroke patients. The results showed the MAS scores of patients had a positive correlation with root-mean-square value and a negative correlation with mean power frequency of the antagonist's sEMG signals. In healthy individuals, it has been shown that higher force production usually corresponds to an increased median frequency [14]. For individuals post-stroke, more severe spasticity — measured as a higher MAS in clinical practice — and higher stretching velocities correspond to higher force resistance production. We thus seem to observe opposite behaviours in healthy individuals voluntary contractions and patients spastic reactions. This therefore suggests a specific sEMG frequency composition of spastic reactions, which could be leveraged for spasticity measurement.

This study thus aims to explore the use of sEMG signal frequency to assess spasticity. Specifically, we report the study protocol for inducing and measuring spastic reactions, and to analyse their frequency composition. Frequency pattern changes of three subjects with elbow spasticity are evaluated at different movement velocities, aimed to induce different levels of spastic reactions [15].

II. Methods

A. Subjects

Patients with spasticity were recruited at Royal Melbourne Hospital (Melbourne, Australia) and Ruijin Hospital, School

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of Medicine, Shanghai Jiao Tong University (Shanghai, China). The following inclusion criteria were applied in recruitment: from 18 to 75 years old; in the chronic phase (at least 3 months post onset) of stroke recovery; upper limb hemiparesis due to a unilateral single stroke; spasticity in the elbow flexors as identified by treating clinicians; and adequate cognition to provide informed consent. Individuals with any following conditions were excluded from the study: co-morbid neurological conditions; a painful shoulder or elbow; significant non-neurological upper limb pathology; or contractures in the affected upper limb. The ethical approvals were obtained from the Melbourne Health HREC (#HREC/62637/MH-2021) and the Ruijin Hospital CTEC (#2021.356). Written informed consent was collected from all participants.

B. Experimental Setup

To measure muscle activities from elbow flexors and extensors, four sEMG sensors (Delsys Inc., Natick, MA, USA) with a sampling rate of $1.11kHz$ were placed on the biceps brachii short head (BSH), biceps brachii long head (BLH), brachioradialis (BRA), and triceps lateral head (TRI) of the subjects' impaired arm (see Fig.1). An upper limb rehabilitation robot ArmMotus-M2 (Fourier Intelligence Co., Ltd, Shanghai, China) was used to perform passive elbow stretching while recording the hand kinematic and interaction force at $500Hz$ (see Fig.1). Both sites had the same experimental setup.

C. Procedure

In the experiment, the subjects were seated in front of the ArmMotus-M2 with their impaired forearm attached to the end-effector. After that, the subjects were asked to fully relax their upper limb for the entire procedure. The clinician first helped the subjects to achieve an initial steady posture of approximately 90° shoulder elevation in the sagittal plane and 90° elbow flexion in the transverse plane as illustrated in Fig.2. Then, the robot performed elbow extension movements in an arc circle trajectory for a range of motion of 80° (see Fig.2) at nine different angular velocities (from $10° \cdot s^{-1}$ to $90° \cdot s^{-1}$) in a randomised order. This design of elbow

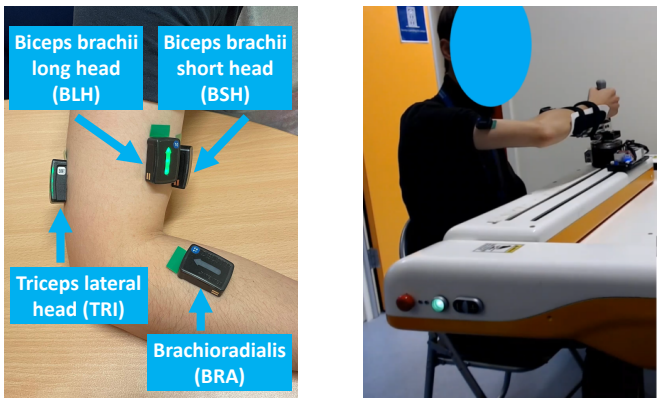

Fig. 1. The sEMG sensors (left) and ArmMotus-M2 robot (right) used in this experiment.

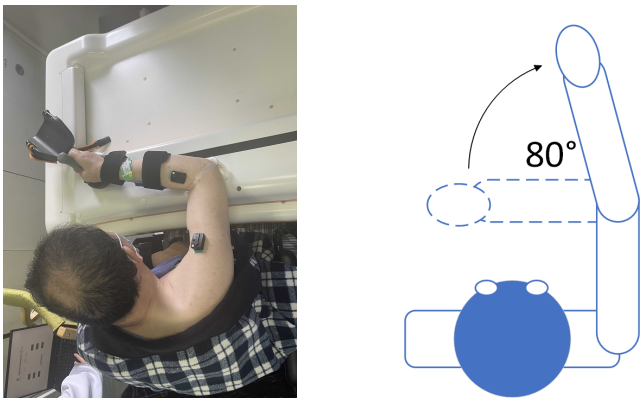

Fig. 2. The posture used and the elbow extension movements performed in the experiment.

stretching movement is aligned with [16] where spastic reactions could be observed. The entire procedure was repeated after a 10-minute break. The subjects also undertook a MAS and an MTS assessment either before or after the procedure.

D. Data Analysis

In accordance with [17], the raw sEMG signal was filtered by a 6^{th} order Butterworth band-pass filter with the effective frequency range of $10Hz$ to $450Hz$. The filtered signal was then resampled at $1kHz$ to synchronise with the force measurements. Next, the outliers and power line interference of the resampled signal were removed [18]. Finally, the signal was rectified and filtered by a 6^{th} order Butterworth low pass filter with a cutoff frequency of $30Hz$ to obtain the sEMG envelope [19].

A baseline of the sEMG was acquired by averaging the signal envelope during the initial steady posture of $3s$ prior each passive stretching movement. The stretch reflex onset was then defined as any sEMG envelope magnitude over three standard deviations from the baseline [20]. When an onset was detected during the constant velocity phase of each movement, the following $200ms$ period ("onset period") was considered for frequency analysis.

Inspired by [13], the mean frequency (MF) of the resampled sEMG signal during the onset period was calculated. The spectral composition of the period was also analysed. Only the antagonist muscles during passive elbow extension movements (BSH, BLH, and BRA) were investigated. Additionally, in order to describe the intensity of spastic response, the elbow stretch reflex torque of each movement was obtained from the interaction force measurements based on the method introduced in [16]. The sEMG signal processing and spectrum analysis were performed with MATLAB-R2022a.

Two-way repeated ANOVA was performed to analyse the trend of MF with increasing velocities. The within-subject factors were defined as experiment trials and movement velocities. The statistical analysis was performed with Python 3.9.

III. Results

Three subjects with upper-limb impairment following a stroke were recruited. The demographics and the clinical

TABLE I

SUBJECTS' DEMOGRAPHICS AND CLINICAL ASSESSMENT RESULTS.

No.	Age	Gender	Affected arm	Post injury (month)	MAS	MTS		
						X	R1(°)	R2(°)
#1	61	Male	Left	28	2	2	90	180
#2	62	Male	Right	163	3	2	80	110
#3	65	Male	Right	3	1+	2	90	110

assessment (MAS and MTS) results of the patients are reported in Table I. The MAS is presented as a single score, and MTS is presented as the quality of muscle reaction (X), angle of catch (R1) and full range of motion (R2).

The sEMG signal onset was detected in 52 of 54 passive stretching for BSH, 51 out of 54 for BLH, and 43 out of 54 for BRA. Only the sEMG measurements during the onset period were used for frequency analysis.

Repeated measures ANOVA did not show a statistically significant term effect on sEMG mean frequencies for movement velocities (within-subject factor) at BSH [$F(6,12) = 0.89$, $p = 0.53$], at BLH [$F(5,10) = 2.25$, $p = 0.13$], or at BRA [$F(1,2) = 0.11$, $p = 0.77$].

Along with a clear increase of reflex torque produced by the patients at higher velocities, it could only be observed a slight velocity-dependent decreasing trend of the sEMG mean frequency (MF) (see Fig.3). Among the three muscles, BLH showed a more monotonic trend, where the median of MF at the highest speed (*e.g.* $62.9Hz$ at $90° \cdot s^{-1}$) was roughly a quarter lower than that at the lowest speed (*e.g.* $83.8Hz$ at $10° \cdot s^{-1}$).

To understand the observed MF trend against movement velocities, such as at BLH, the sEMG frequency spectrum (presented as a percentage of the normalised power in an onset period) at a low, a medium, and a high speed was obtained for each individual (see Fig.4). Except for the first trial of Subject #3, the sEMG frequency spectrum demonstrated increased power at a lower frequency (mostly below $50Hz$) with an increasing velocity. This explains the slightly decreasing trend of MF against movement speed showed in Fig.3.

IV. DISCUSSION

The patients' sEMG mean frequency (MF), obtained from the simultaneously recorded onset signals during passive stretching, was observed to have an overall slightly decreasing trend with increasing speeds. This result thus presents a similar phenomenon with the previous study by Wang et al. [13]: a decreasing sEMG frequency corresponds to a more intense spastic response — represented by a higher MAS in [13] but a higher stretching velocity in our case. More interestingly, while sEMG frequency tends to increase with force production in healthy individuals (as summarised in [14]), an opposite trend seems to exist for spastic reactions: a larger reaction corresponding to lower frequencies. This difference may potentially be exploited to detect spasticity.

Looking into the pathophysiological mechanism of spasticity, the lesion of upper motor neurons (UMN) following a neurological injury (such as stroke) may interrupt the supraspinal descending pathways of lower motor neurons (LMN) at the spinal cord level. It is to note that the imbalance of the descending inhibitory and facilitatory influences, especially the loss of inhibitory control, will lead the LMN to tip in favour of excitation [21]. This may subsequently alter the balance between the innervations of intrafusal and extrafusal muscle fibres [21] and so potentially affect the sEMG frequency. However, due to the lack of research on the signal characteristics (*e.g.* time-frequency analysis) of the efferent motor fibres, it is still not clear how the changes in LMN could affect the frequency spectrum of the neuromuscular signals (*e.g.* sEMG).

In addition, different muscles showed noticeable variations regarding frequency distribution and velocity dependency: a more pronounced and monotonic decreasing trend for BLH compared to BSH and BRA. Even though all these muscles are elbow flexors, they may not be equally affected by spasticity and have different muscle fibres composition. This may not allow a straightforward translation of the frequency feature from one muscle to another.

Although the sEMG frequency can be observed to have

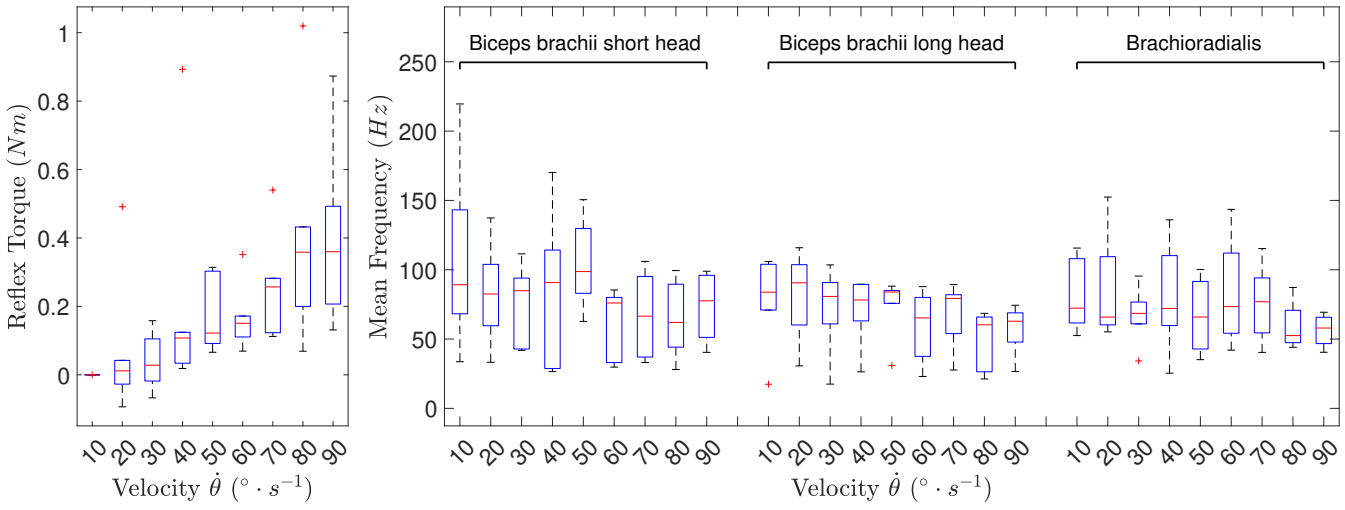

Fig. 3. Box plots of stretch reflex torque and sEMG mean frequency at different movement velocities.

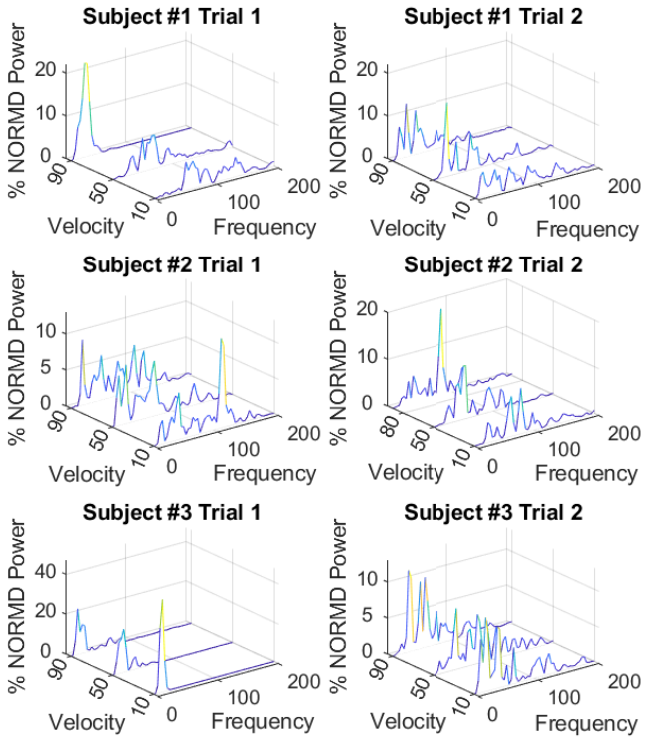

Fig. 4. sEMG normalised power spectrum for low, medium, and high speeds of each subject's BLH. Frequency is reported in Hz with a range of 0–200 where the spectrum changes can be observed. Velocity is reported in $^{\circ} \cdot s^{-1}$.

an overall decreasing trend against stretching velocities, this only constitutes preliminary results given that only three subjects were recruited so far. This study plans to recruit a total of 25 subjects to reduce the effect of individual variations introduced by the currently limited sample size. Moreover, the sEMG frequency was only measured during passive stretching and not during active, voluntary, movements. It would still be relevant to compare patients' voluntary contractions and passive stretching.

V. Conclusion

This study explored the possibility of using the sEMG frequency information in spasticity detection with the aim to optimise the existing sEMG based measures. The preliminary results showed an overall movement velocity dependent decreasing trend of the sEMG mean frequency with patients, which is in opposition with an expected increase observed in healthy individuals. Although this does not provide a definitive answer, it suggests that frequency analysis of sEMG measurements might be a useful tool to investigate spastic reactions.

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