QUANTITATIVE CLINICAL ASSESSMENT OF MUSCLE SPASTICITY USING WEARABLE SENSORS

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INTRODUCTION

Muscle spasticity is a major contributor to chronic mobility impairment in people with neurological injury or disease [1], and is characterized by involuntary tonic (velocity dependent) stretch reflex that causes the muscle to activate inappropriately during functional movements [2]. Spasticity is also associated with abnormal tone [3] in opposing muscles that if not managed appropriately can lead to muscle contractures, chronic pain, and difficulties with basic motor tasks required for self-care and independence. Regardless of the management approach, instruments are needed to reliably and accurately quantify spasticity.

Although a number of laboratory-based technologies [4,5] are available for assessing muscle spasticity, none of these technologies are feasible for clinical use. The instrument used clinically, and for the vast majority of published studies related to muscle spasticity, is the Modified Ashworth scale [6]. Although the test is easy to administer, it is based on a limited ordinal scale (0-4) determined by subjective assessment. The test has been criticised due to lack of reliability and sensitivity to detect change [7], and inability to distinguish spastic reflex from muscle hypertonicity (high tone) [8].

Wearable technologies for sensing kinematics, force and muscle electromyography have the potential to deliver quantitative objective information that is of value to the treating therapist [9]. Wearable technologies are small, lightweight and generally unobtrusive, and could allow the clinician or researcher to perform unencumbered routine physical examinations while monitoring and collecting important clinical variables [10]. The sensing technology required to accomplish this aim already exist.

This paper describes the development and testing of a wearable sensor system called the "BioTone", developed at the Institute of Biomedical Engineering at UNB, to enable clinicians to acquire quantitative objective information during neurological examination of spasticity in their clients.

METHODOLOGY

BioTone hardware consists of a fibre-optic goniometer (ShapeSensor™, Measurand Inc., Fredericton, NB, Figure 1a), and a 2-channel EMG system (custom designed at UNB, uses DuoTrode Ag-AgCl electrodes, Figure 1b), that connect to an analog interface (BioSI™, also custom designed at UNB) that controls sampling and sends data to the laptop computer for storage, processing and real-time graphic display. The BioTone software guides the clinician through the testing protocol (including order of trials) and records all data during the clinician's examination of clients and provides real-time display of test results.

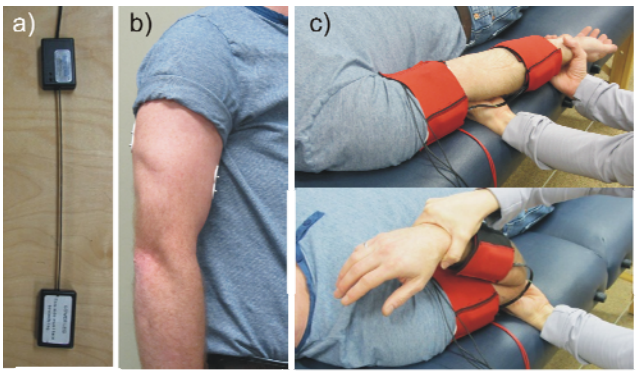

Figure 1. BioTone hardware: a) Fibre-optic goniometer; b) 2-ch EMG; c) stretch-reflex test.

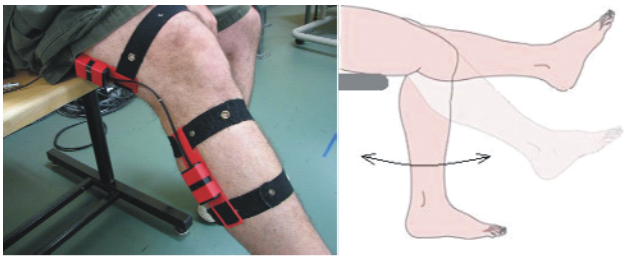

Figure 2. Pendulum test

At the time of writing this paper we have partially completed a pilot study at the Stan Cassidy Centre for Rehabilitation (SCCR, Fredericton, NB), and present here the study protocol and preliminary findings.

Participants

At the time of this preliminary analysis, 25 patients were recruited from the SCCR. Included were adult patients with acquired brain injury (ABI, including strokes), cerebral palsy (CP), multiple sclerosis (MS) and spinal cord injury (SCI). The research was reviewed and approved by both University and Regional Health Authority Research Ethics Boards, and all participants provided informed signed consent prior to data collection.

Testing Protocol

We focus here on assessment of spasticity and hypertonicity of the elbow and/or knee(s) using two different tests. For the elbow, spasticity was assessed using a "stretch-reflex test", and for the knee a "pendulum test" was used. For both protocols the client wore the BioTone system on the joint being tested. Sensors were mounted using a custom designed cuff system that allowed quick donning and doffing by the therapist, as illustrated in Figures 1 and 2.

Clinical assessment: Prior to BioTone assessment, a clinician recorded a Modified Ashworth Score (MAS) for elbow and knee flexion and extension based on subjective rating (ie. without the benefit of interpreting the instrumented results). Because the MAS scale (0-4) includes the inconvenient category of 1+, scores were re-assigned to a 0-5 scale (where 0=0; 1=1; 1+=2; 2=3; etc.).

Stretch-reflex test: The stretch-reflex test was conducted by first performing a single slow (~10-20^{o}/s) flexion and extension trial (throughout the passive range), followed by a series of fast (~120-140^{o}/s) flexion and extension trials (throughout the same passive range) (see Figure 1c). A short rest period was used between tests to allow muscles to relax, which could be monitored from the real-time display on the laptop computer. Fast flexion and extension trials were each performed three times. Nine clients being treated for elbow spasticity were included. The most affected elbow was tested in extension and flexion.

Pendulum test: The pendulum test was conducted by having the client seated on a reclining wheelchair with torso at approximately 50^{o} with thighs horizontal. The clinician raised the client's lower leg (shank and foot) to a horizontal position, waited until the muscle EMG signal trace indicated relaxation, then dropped the limb (see Figure 2). Clients were instructed not to voluntarily resist or aid in leg motion and to let the limb oscillate naturally until motion ceased. At least three pendulum tests were conducted. All clients were tested for one or both knees, and thus the sample consisted of forty-one knees tested.

BioTone data collection: Joint angle data from the fibre-optic goniometer and EMG data from electrodes placed on joint flexor and extensor muscles were collected at 1000Hz during the trials. Processing was done off-line. Kinematic data were filtered at 6 Hz and EMG data were band-pass filtered at 20-400 Hz (at the amplifiers), rectified and low-pass filtered at 6 Hz (4th order zero-lag Butterworth).

Data Analysis

For stretch-reflex test data, the clinician's intended passive motion curve was first estimated using a constant-jerk kinematic model. Departures from the intended passive motion profile by the actual motion profile were assessed by examining peak velocity departures at muscle onset and density (area under curve) departures over the length of the trial. Peak EMG intensity at onset and density of EMG signal over the trial, were similarly computed for both antagonist (the muscle being tested) and agonist muscles [11] (see Figure 3).

For pendulum test data, we used a previously published algorithm [12] for assessing the joint stiffness, viscosity and

relaxation index from the lower limb's pendulum motion profile, and we also computed the muscle EMG response as described above.

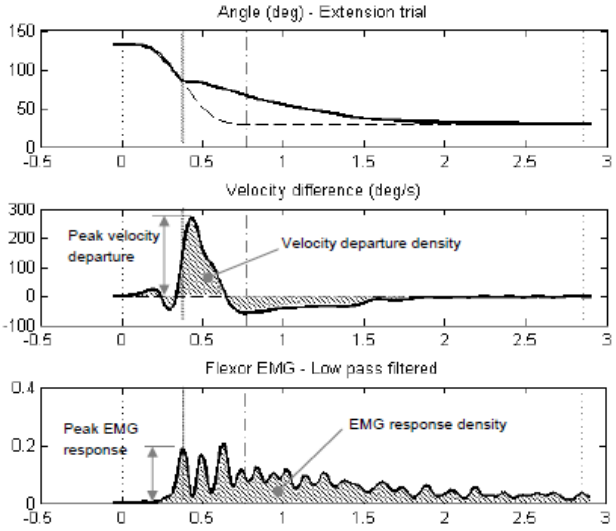

Figure 3. Stretch-reflex test data (elbow extension) for client with moderate spasticity.

Relationships were explored between the biomechanical (kinematics-based) and neuro-muscular (EMG-based) responses during the stretch-reflex and pendulum tests, as well as between these quantitative measures and subjective clinical MAS score. Spearman correlation analysis was used with α=.05. All statistical analyses were performed with SPSS (v19, SPSS Inc).

RESULTS

Stretch-Reflex Results

Biomechanical vs neuromuscular responses: There was a significant correlation between magnitude of peak angular velocity departure and peak antagonist EMG for both elbow extension (r=.683, p=.021) and flexion (r=.583, p=.050). There were also significant correlations between velocity departure density and density of EMG response over the trial for elbow extension (r=.767, p=.008) and flexion (r=.867, p=.001) trials.

Neuro-biomechanical responses vs clinical MAS score: MAS score for elbow flexor testing did not correlate significantly with peak velocity departure or EMG responses during elbow extension tests, but elbow extensor MAS score correlated with peak velocity (r=.843, p=.002) and peak EMG response (r=.738, p=.012) during elbow flexion tests. MAS score also correlated positively with velocity departure density (r=.687, p=.020) and EMG density (r=.721, p=.024) for extension testing, and with velocity departure density (r=.896, p=.001) and EMG density (r=.896, p=.001) for elbow flexion testing (see Figure 4).

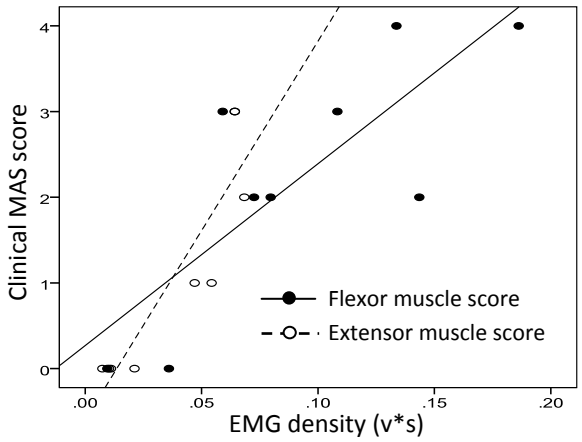

Figure 4. EMG density versus clinical MAS score during stretch reflex test.

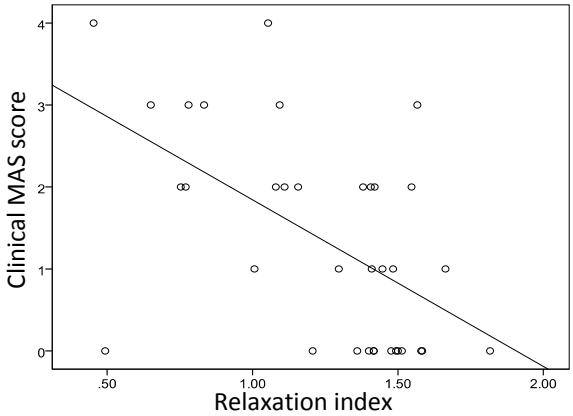

Figure 5. Relaxation index vs clinical MAs score during pendulum test.

Pendulum Test Results

Biomechanical vs neuromuscular responses: Muscle EMG peak responses correlated significantly with stiffness (extensor: r=.284, p=.036; flexor: r=.400, p=.005), viscosity (extensor: r=.375, p=.008), and relaxation index (extensor: r=-.302, p=.027; flexor: r=-.283, p=.036). Muscle EMG density also correlated with stiffness (extensor: r=.421, p=.003; flexor: r=.401, p=.005) and relaxation

index (extensor: r=-.289, p=.033; flexor: r=-.350, p =.013).

Neuro-biomechanical responses vs clinical MAS score: MAS score correlated with peak EMG intensity (r=.463, p=.004), EMG density (r=.418, p=.010), stiffness (r=.401, p=.007) and relaxation index (r=-.514, p=.001, see Figure 5).

DISCUSSION

Ongoing management of problematic spasticity is commonly required in patient populations with upper motor neuron syndrome [1]. Clinical assessment of spasticity is often complicated by concurrent conditions such as contractures and high tone. Clinical rating scales, such as the MAS [6] are used almost exclusively in both practice and in clinical trials, but the literature is unclear regarding their validity [7,4,12]. The current lack of reliable and valid objective measurement tools for spasticity, negatively impacts on research and management.

We have developed and pilot tested a wearable sensor system that measures biomechanical and neuromuscular responses during clinical testing for elbow and knee spasticity. Although the stretch-reflex and pendulum tests are quite different in nature, and were applied to different joints (elbow and knee, respectively), there was good agreement between the biomechanical response and neuromuscular response during both tests, and many of these variables had a significant correlation with the clinical MAS score.

For elbow testing with stretch-reflex, clinical MAS was correlated most strongly with density of antagonist muscle EMG response but also correlated with EMG peak intensity (spastic reflex). For knee pendulum tests, MAS was most strongly related to joint stiffness and relaxation index. These results suggest clinical MAS scoring is influenced by both muscle spastic reflex, and muscle hypertonicity.

Future work will concentrate on using BioTone data collected in the clinic for developing better models to separate spastic reflex and hypertonicity during clinical assessments of spasticity due to upper motor neuron syndrome.

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