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Frequency analysis of lower extremity electromyography signals for the quantitative diagnosis of dystonia

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ABSTRACT

The purpose of this study was to develop an objective, quantitative tool for the diagnosis of lower extremity dystonia. Frequency domain analysis was performed on surface and fine-wire electromyography (EMG) signals collected from the lower extremity musculature of ten patients with suspected dystonia while performing walking trials at self-selected speeds. The median power frequency (MdPF) and percentage of total power contained in the low frequency range (%AUC_{Total}) were determined for each muscle studied. Muscles exhibiting clinical signs of dystonia were found to have a shift of the MdPF to lower frequencies and a simultaneous increase in the %AUC_{Total}. A threshold frequency of 70 Hz identified dystonic muscles with 73% sensitivity and 63% specificity. These results indicate that frequency analysis can accurately distinguish dystonic from non-dystonic muscles.

1. Introduction

Dystonia is a neurologic movement disorder typified by repetitive gestures or abnormal positioning of involved body parts due to sustained involuntary muscle contractions (Berardelli et al., 1998; Pont-Sunyer et al., 2010). The onset of symptoms is usually associated with or exacerbated by voluntary actions, and prolonged dystonic postures may constrain normal motions; affected individuals may even slow their movements in an attempt to suppress the dystonic drive (Sanger et al., 2010, 2003).

Given the debilitating impact that dystonia may have on an affected individual's quality of life, accurate diagnosis and identification of involved muscles is paramount for effective management and treatment. However, no current diagnostic tools allow for the objective and definitive diagnosis of the disease. Although studies utilizing positron emission tomography (PET) and magnetic resonance imaging (MRI) (Albanese et al., 2006) suggest a correlation between dystonia and the presence of lesions in the thalamus or the basal ganglia, the lack of consistent findings across all forms of dystonia has limited their utility (Albanese et al., 2006; Brin et al., 2004). As such, the diagnosis of dystonia is made purely on clinical assessment and is subject to the examining clinician's experience and ability to differentiate the symptoms of dystonia from other movement disorders (Lalli and Albanese, 2010).

Electromyography (EMG) is a technique used to measure the electrical activity produced by skeletal muscles that results in contraction. Dystonia is thought to be associated with distinct EMG patterns in the time-domain, including co-contraction of antagonist muscles, continuous EMG activity, multi-phasic (oscillatory) activation patterns, and antiphasic activity due to overflow of electrical activity to surrounding or uninvolved muscles (Berardelli et al., 1998; Marsden, 1984). Recent efforts to develop diagnostic tools for dystonia have shifted the focus from time-domain analyses to frequency-domain analyses of EMG signals. Frequency analysis of an EMG signal allows for the decomposition of the signal into its frequency components. Previous studies describe a low-frequency (<30 Hz) drive to co-contraction in muscle pairs in primary forms of cervical (Tijssen et al., 2000) and limb dystonia (Farmer et al., 1998; Grosse et al., 2004). However, the presence of co-contraction is not a specific marker for dystonia (Carolan and Cafarelli, 1992; Malfait and Sanger, 2007) and hinges on proper identification of involved muscle pairs; thus, these results, although promising, must be interpreted with caution.

Due to the variability in the clinical and electrophysiological presentation of individuals affected by dystonia, we believe that diagnosis may not be made purely on the presence of co-activation. Instead, we propose that analysis of muscle activity *independent* of the activity of surrounding or antagonist muscles will provide significantly more valuable information that, when used in conjunction with overall clinical assessment, would allow for definitive diagnosis of dystonia. Thus, the purpose of this study was to develop an objective frequency domain-based method that would allow for the identification and diagnosis of dystonia in lower

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extremity muscles. We hypothesized that the previously described low-frequency drive would manifest in lower limb dystonia and that frequency domain analysis of EMG signals collected from affected muscles would demonstrate a spectral shift to lower frequencies.

2. Methods

Ten patients (mean ± SD: 24 ± 18 years, 59 ± 25 kg, 1.61 ± 0.16 m, 22 ± 8 kg/m^2, 1 M/9 F) with suspected primary lower extremity dystonia were selected for this study. A retrospective analysis was performed on EMG recordings collected from the lower extremity musculature during clinical evaluation of their gait (Motion Analysis Laboratory, Mayo Clinic, Rochester, MN).

2.1. Lower extremity EMG recordings

Electromyography signals from 121 lower extremity muscles (3–10 muscles per leg) were collected while subjects performed three walking trials at self-selected speeds, with the option to rest between each trial. Each trial was completed prior to the onset of subjective fatigue. Fatigue was not reported by the patient or assessed by the physical therapist conducting the gait study. Furthermore, each walking trial involved submaximal contractions of the lower extremity musculature and was completed within 5 ± 2 s, which is well below the reported endurance time for the ankle and knee at all intensity levels (Frey Law and Avin, 2010). Muscles studied included the extensor digitorum longus (EDL), gastrocnemius (GC), gluteus maximus (GX) and medius (GD), hamstrings (HS), medial and lateral hamstrings (MHS and LHS), iliopsoas (IL), lumbar paraspinals (LP), peroneus longus (PL), rectus femoris (RF), tibialis anterior (TA), and tibialis posterior (TP).

Bipolar stainless steel surface electrodes (12 mm disks; Motion Lab Systems, Baton Rouge, LA) were used to measure activity from the GC, GX, GD, HS, MHS, LHS, LP, RF, and TA muscles. Inter-electrode distance was 18 mm with a 12 × 3 mm reference electrode bar between the sensors. All surface electrodes were connected to a differential input preamplifier with a high common mode rejection ratio (>100 dB), input impedance (>100 MΩ) and a base gain of 20 (Motion Lab Systems). Skin in the area where the electrode was placed was prepared by shaving if necessary and mildly abraded in accordance with the International Society of Electrophysiology and Kinesiology (ISEK) and SENIAM standards (Hermens et al., 2000). Electrodes were placed over the target muscle belly, parallel with the muscles fibers, and secured with Tegaderm (3M Health Care, Neuss, Germany). Target muscle electrode placement was confirmed by having each subject perform a voluntary contraction of the target muscle while verifying that EMG signal was present in that channel. Each subject also performed voluntary contractions of muscles which could contribute to crosstalk in the target channel (adjacent, antagonist, or agonist muscles) while confirming that EMG signal was not present in the channel for the target muscle.

Paired, fine-wire indwelling electrodes (nylon insulated stainless steel, 2 mm exposed tip, 25 gauge hypodermic needle; Motion Lab Systems) were used to measure activity from the EDL, IL, PL, and TP muscles. All fine-wire electrodes were connected to a differential input preamplifier with a high common mode rejection ratio (>100 dB), input impedance (>100 MΩ) and a base gain of 20 (Motion Lab Systems). Skin in the area where the electrode was inserted was sterilized with alcohol in accordance with ISEK standards. Electrodes were inserted under ultrasound guidance with respect to anatomical landmarks as described in literature (Perotto et al., 2005) and secured with Tegaderm (3M Health Care). Target muscle electrode placement was confirmed by electrical stimulation with a peripheral nerve stimulator (Neuro Technology, Kerrville, TX). The current (200 μs, 1 Hz, square wave monophasic pulses) was increased to a level which created a minimal muscle contraction in the target muscle, and it was visually verified that the twitch only occurred in the target muscle.

Surface and fine-wire EMG signals were acquired at 2400 Hz per channel with an MA300 electromyography system (Motion Lab Systems) and digitized at 2400 Hz (PCI-6071e A/D card; National Instruments, Austin, TX). It has been reported in literature that the bandwidth of usable energy for surface and fine-wire EMG signals is between 20–500 Hz and 20–1000 Hz, respectively (Basmajian and De Luca, 1985). As such, the sampling rate is in accordance with the Nyquist theorem, which states that the sampling rate must be greater than twice the highest frequency component of the analog signal.

2.2. Frequency domain analysis

Frequency analysis was performed on raw EMG signals collected with both surface and fine-wire electrodes with a custom Matlab software program (MathWorks, Natick, MA). Raw EMG signals from individual muscles were detrended and filtered with a 10th order infinite impulse response notching comb filter (1.5 Hz bandwidth) to remove background instrumentation noise (60 Hz) and its harmonics. The signals were then transformed into the frequency domain using an n-point discrete fast Fourier transform (DFT). The number of points, n, used to calculate the DFT was determined by the next power-of-two from the number of data points in the raw EMG signal. The power spectral density (PSD) for each muscle was estimated using Welch's periodogram method with 50% overlap between segments and 1 Hz frequency resolution; segments were windowed with a rectangular window. Each PSD was then normalized to the maximum power value in each spectrum.

2.3. Quantification of spectral shift: MdPF and %AUC$_{Total}$ calculations

Spectral shift was quantified by two parameters: the median power frequency (MdPF) and the percentage of the total power contained in the low frequency range (%AUC$_{Total}$). The MdPF describes the relative proportion of low and high frequencies in the spectrum. Thus, a left-shift of the MdPF to a lower frequency indicates a greater proportion of muscle fibers firing at a low frequency. Mathematically, the MdPF is the point that divides the spectrum into regions containing equivalent power, and was calculated for each trial using Eq. (1) (Stulen and DeLuca, 1981) and averaged across the three trials for each muscle:

$$\sum_{0}^{MdPF} PSD(f) = \sum_{MdPF}^{\infty} PSD(f) = \frac{1}{2}\sum_{0}^{\infty} PSD(f) \quad (1)$$

The total power contained within each spectrum was found by calculating the area under the PSD curve (AUC$_{Total}$) from 0 to 500 Hz. Although it has been reported that the usable energy within an EMG signal collected with fine-wire electrodes is in the 20–1000 Hz range (Basmajian and De Luca, 1985), visual inspection of the power spectra constructed from both surface and fine-wire EMG data showed minimal power above 500 Hz. The %AUC$_{Total}$ was calculated according to Eq. (2):

$$\%AUC_{total} = \frac{AUC_{Low}}{AUC_{Total}} = \frac{\sum_{10}^{t} PSD(f)}{\sum_{0}^{500} PSD(f)} \quad (2)$$

where AUC$_{Low}$ is the power contained in the range from 10 Hz to the upper threshold, t. The upper threshold was selected after two iterations. Initially, a value of 50 Hz was arbitrarily selected for preliminary analysis; subsequently, analysis of the receiver operating