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Version: Accepted Version

Article:
Collins, A.F., Brown, S.T.R. and Baker, M.R. (2020) Minimum EMG burst duration in healthy controls : implications for electrodiagnosis in movement disorders. Movement Disorders Clinical Practice, 7 (7). pp. 827-833. ISSN: 2330-1619

https://doi.org/10.1002/mdc3.13044

This is the peer reviewed version of the following article: Collins, A.F., Brown, S.T. and Baker, M.R. (2020), Minimum EMG Burst Duration in Healthy Controls: Implications for Electrodiagnosis in Movement Disorders. Mov Disord Clin Pract. , which has been published in final form at https://doi.org/10.1002/mdc3.13044. This article may be used for non-commercial purposes in accordance with Wiley Terms and Conditions for Use of Self-Archived Versions.

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Minimum EMG Burst Duration in Healthy Controls: Implications for Electrodiagnosis in Movement Disorders

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Word Count (Abstract: 154/250 Body: 2842/3700; 2 Figures; 1 Table; References 26/80)

Key words: (up to 5) EMG; functional movement disorders; myoclonus Running title:

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Accepted for publication in *Movement Disorders Clinical Practice*

Abstract

Background: EMG burst duration can provide additional diagnostic information when investigating hyperkinetic movement disorders, particularly when a functional movement disorder is suspected. It is generally accepted that EMG bursts <50ms are pathological.

Objective: To re-assess minimum physiological EMG burst duration.

Methods: Surface EMG was recorded from face, trunk and limb muscles in controls (n=60; age 19-85). Subjects were instructed to generate the briefest possible ballistic movements involving each muscle (40 repetitions) or, in muscles spanning joints, to generate rapid rhythmic alternating movements (20-30s), or both.

Results: We found no effect of age on EMG burst duration. However, EMG burst duration varied significantly between body regions. Rhythmic EMG bursts were shorter than ballistic bursts but only significantly so for lower limbs ($p<0.001$). EMG bursts of duration <50ms were frequently observed, particularly in appendicular muscles.

Conclusion: We present normal reference data for minimum EMG burst duration, which may assist clinical interpretation when investigating hyperkinetic movement disorders.

Introduction

Electrophysiological investigations are widely used in the diagnosis of hyperkinetic movement disorders, most notably tremor and myoclonus (1,2), where they may be helpful in identifying patients with functional hyperkinetic movement disorders. Electromyography (EMG), which is a commonly available modality, is perhaps the least challenging of the electrophysiological techniques in terms of data acquisition. Whilst there is increasing interest in the application of complex signal processing techniques to such data (e.g. (3,4)), analysis limited to the simple parameter of EMG burst duration is widely used to guide diagnosis (and localisation) of myoclonus (5), and has the additional advantage of being very simple to measure. Typically EMG is recorded simultaneously with video-EEG and only a limited number of channels are available for EMG (sometimes a single channel). The placement of EMG electrodes is then determined by the number available and the clinician's assessment as to which muscles are most involved. Surprisingly, despite the widespread application of EMG burst duration in clinical practice, normal values are only available from small studies and only for a limited selection of appendicular muscles.

Sampling EMG burst duration for a ballistic movement generated by an individual muscle in a large population of healthy controls to determine normal limits for burst duration based on mean and standard deviation would be one approach. However, because the individual discrete EMG bursts contributing to the triphasic agonist-antagonist-agonist EMG pattern of ballistic limb movements cannot always be distinguished visually (6), ballistic EMG bursts appear artificially prolonged (i.e. composites of the first and second agonist burst), thus skewing the distribution of data. To address this, where possible, it is recommended that EMG is recorded from the antagonist muscle acting at the same articulation in order to exclude

contaminated agonist bursts from analysis. However, this is not always possible, particularly where there is no antagonist muscle (e.g. facial muscles) or where clean EMG recording from an antagonist muscle is difficult to achieve non-invasively (e.g. abdominal muscles). To circumvent this problem, the approach adopted has therefore been to simply measure the shortest EMG burst duration, rather than the mean, and interpret this with reference to a lower limit for the duration of normal physiological EMG bursts.

Whilst there is general agreement in the literature that short duration EMG bursts are pathological, there is debate as to the upper limit, with some accepting a burst duration of <70ms (1,7) and others advocating a cut-off of <50ms (5,8,9). By contrast, in functional movement disorders (FMDs), which are thought to arise as a consequence of abnormal predictive coding by the brain (10) and thus by definition remain constrained by physiological mechanisms of movement control, the duration of EMG bursts observed, particularly in functional myoclonus, should be comparable to those generated by voluntary ballistic movements; based on data collated from a number of small studies, this appears to be consistently longer than 50ms in duration (e.g. neck (11); upper limb (6,12–14); lower limb (15)).

Here we have addressed the need for a more comprehensive dataset of normal values for minimum EMG burst duration by measuring the minimum voluntary EMG burst duration in cranial, axial and appendicular muscles in healthy controls instructed to produce brief ballistic voluntary contractions (to mimic myoclonus) or rhythmic contractions (mimicking tremor) in these muscles. As a reference resource, these data should assist clinicians in the electrodiagnostic investigation of complex hyperkinetic movement disorders.

Methods

Participants: Experiments were conducted in 60 healthy volunteers (age range 19-85; mean age 34; 30 female). Participants were excluded if they had a neurological disorder or an implanted device (e.g. cardiac pacemaker). Experiments were approved by the Newcastle University Ethics Committee and conformed to the Declaration of Helsinki. All participants provided written informed consent. Analysis also included anonymised EMG data acquired from patients with movement disorders (n=3) referred for neurophysiologic investigation, as part of routine diagnostic work-up, and identified by a retrospective case note review.

Recordings: Referential surface electromyogram (EMG) recordings were made using adhesive (Ag/AgCl) electrodes attached to the skin overlying cranial (*temporalis; orbicularis oculi; risorius*), trunk (*trapezius; infraspinatus; rhomboids; latissimus dorsi; pectoralis major; rectus abdominis superior; rectus abdominis inferior*), upper limb (*deltoid; triceps brachii; biceps brachii; extensor digitorum communis; flexor carpi ulnaris; abductor pollicis brevis; first dorsal interosseous*) and lower limb (*vastus lateralis; biceps femoris; tibialis anterior; medial gastrocnemius; extensor digitorum brevis; abductor hallucis*) muscles. The anode (reference electrode) was placed on the bony prominence or tendon and the cathode (active electrode) on the muscle belly (approximate inter-electrode distance of 3cm). Consistent EMG electrode placement is important when comparing EMG burst durations across individuals; if the electrode spacing is too large it becomes in essence a monopolar recording (with the reference electrode behaving as an indifferent), which could reduce the duration of the recorded EMG burst. Surface EMG signals were amplified (5K) and filtered (3Hz-2KHz) using an 8-channel Digitimer D360 amplifier (Digitimer Ltd, Welwyn Garden City, UK), controlled via a

dedicated laptop (NB. The sampling frequency should ideally be set at four times the low pass filter frequency). The output from the D360 was connected to an analogue to digital converter (CED micro 1401; Cambridge Electronic Design Ltd, UK) and signals digitized at a sampling frequency of 5KHz. EMG signals could be viewed on the data acquisition laptop using dedicated software (Spike 2; CED Ltd).

Behavioural tasks: Once all surface EMG electrodes were in position, subjects were asked to contract the individual muscles separately. Participants were provided with guidance as to the optimum method for activating individual muscles for each task.

1. Mimicking brief myoclonic jerks. For this task, subjects made ballistic movements (brief muscle contractions generated with maximum velocity and acceleration) such that the EMG exhibited very high motor unit firing rates. The instruction given to subjects for each muscular contraction was that it should be as brief and rapid as possible (thus mimicking myoclonic movements). Where necessary, the investigator also provided a demonstration to the participants of what was required. For proximal and axial/trunk muscles, participants were shown an anonymised video of the movement required.

2. Mimicking repetitive myoclonic/tremulous movements. For articular muscles (i.e. muscles spanning joints in the limbs), subjects were also asked to make self-paced (i.e. without external feedback) rhythmic alternating (oscillating) movements as rapidly as possible, thus mimicking tremor.

In the interests of time, participants were separated into three experimental sub-groups, each sub-group testing a set of different muscles. Recordings from the upper body were made from a total of 18 volunteers, cranial, facial, arm and foot muscles were tested in 20 subjects, and lower limb muscle contractions were recorded from

36 participants. There was some overlap of participants for the three experiments. Each muscle was recorded for a duration of 20-30 seconds (~ 40 muscle contractions) while the volunteers executed the movements. Each contraction task was separated by 1-2 minutes of rest.

Data analysis: EMG data were first inspected visually and any data contaminated by noise excluded from the analysis. Unrectified EMG was then reviewed and a preliminary analysis of burst duration completed in Spike2 by assigning onset and offset markers by eye (as illustrated in Figure 1). This approach to measurement will inevitably introduce human error into the analysis, affecting accuracy (by increasing bias), precision and stability. Measurement of burst duration was therefore automated using custom scripts compiled in Matlab (Mathworks, Natick, USA) as follows: EMG was full-wave rectified; EMG burst onset/offset markers were assigned when the EMG amplitude increased by two standard deviations above baseline for both onset and offset; burst duration was then measured between burst onset and burst offset markers and an average burst duration determined for each muscle in each subject. The outputs from this process were again reviewed by eye and where bursts were detected erroneously, or where co-contraction was observed by reviewing the antagonist EMG recording simultaneously, outputs were rejected. The Shapiro-Wilk test was used to check for normality. The Wilcoxon signed-rank test was used for non-parametric data (null hypothesis rejected if $p<0.05$). Where correlations between data were explored, Spearman's rank correlation co-efficient was used.

Results

EMG recordings were obtained from 60 subjects (age range 19-85; mean age 34; 30 female), across a total of 23 muscles. Examples of typical unrectified EMG burst recordings are shown in Figure 1A, illustrating typical EMG bursts acquired from cranial (*risorius*), shoulder (*trapezius*, PM, *deltoid*), upper limb (FCU) and lower limb (VL, MG) muscles on the right. The width of the grey box in Figure 1A shows the duration of the longest EMG burst (*risorius*) for ease of comparison with other EMG bursts.

Inspection of Figure 1A would suggest that there are regional differences in the duration of EMG bursts. For example, the forearm flexor muscle illustrated appears to generate the shortest EMG burst and the facial muscle the longest, whereas proximal upper limb and lower limb muscles generate EMG bursts of comparable but intermediate durations.

Figure 1B plots the mean EMG burst duration (and standard error) for representative regional muscles averaged across subjects (cranial, trunk and upper limb muscles (n=20); PM (n=18); lower limb muscles (n=36)), showing a different pattern of EMG burst durations from that illustrated in Figure 1A.

Figure 1C illustrates typical unrectified EMG recordings (from the same subject as in Figure 1A) of lower limb muscles whilst making either brief voluntary ballistic muscle contractions or rapid alternating rhythmic movements. Note that EMG burst duration in rhythmic movements was consistently reduced (vertical dashed lines in Figure 1C indicate onset and offset of EMG bursts). These data are presented in more detail in Table 1, where mean burst EMG durations for rapid ballistic and rhythmic movements are listed for each muscle averaged across subjects. Whilst on visual inspection most of our data appeared normally distributed, only ballistic EMG burst

durations recorded from cranial muscles and rhythmic EMG burst durations recorded from trunk muscles satisfied the Shapiro-Wilk test. Non-parametric statistical tests were therefore applied to the data. Analysis of regional data showed that despite rhythmic EMG bursts appearing shorter than ballistic EMG bursts (see Fig. 1C), it was only in the lower limbs that rhythmic EMG bursts were significantly shorter than ballistic EMG bursts ($p<0.0001$, Wilcoxon signed-rank test); there was no difference between ballistic and rhythmic trunk or upper limb muscle movements ($p=0.88$ and $p=0.51$ respectively, Wilcoxon signed-rank test). For ballistic movements, EMG bursts in cranial muscles were significantly longer than those recorded from trunk muscles ($p=0.04$, Wilcoxon signed-rank test) and distal arm muscles ($p<0.001$; Wilcoxon signed-rank test), but not compared to lower limb muscles.

Further analysis of appendicular muscles showed that ballistic EMG bursts recorded from proximal muscles compared to distal muscles have a significantly longer duration in both upper limbs and lower limbs ($p<0.0001$ and $p= 0.037$ respectively, Wilcoxon signed-rank test). For rhythmic muscle contractions, EMG bursts recorded from upper limb muscles were significantly longer than those recorded from lower limb muscles ($p<0.0001$, Wilcoxon signed-rank test). Whilst there was no significant difference between the durations of rhythmic EMG bursts recorded from proximal and distal upper limb muscles ($p=0.142$, Wilcoxon signed-rank test), bursts recorded from distal lower limb muscles were significantly shorter than those recorded from proximal lower limb muscles ($p=0.003$, Wilcoxon signed-rank test).

There was no significant correlation between age and EMG burst duration for ballistic movements, rhythmic movements or all movements combined ($p=0.93$, $p=0.44$ and $p=0.40$ respectively; Spearman's rank correlation coefficient).

Minimum EMG burst durations for all muscles tested are summarised in Table 1. We observed very short duration physiological EMG burst durations in a large proportion of our healthy control population. The highest percentage of short EMG bursts was seen in the rhythmic EMG lower limb muscle group with 57% of participants averaging <70ms. Somewhat unexpectedly, we also observed that 18% of healthy subjects could generate average voluntary EMG bursts of less than 50ms in duration when contracting muscles rhythmically (see Fig. 1D). As can be seen from Table 1, short duration EMG bursts were most frequently observed in forearm, hand and lower limb muscles, and occasionally in shoulder muscles (*pectoralis major* and *deltoid*). EMG bursts <50ms were never observed in *orbicularis oculi, risorius, trapezius, infraspinatus, rhomboid, latissimus dorsi, rectus abdominis superior, triceps brachii, biceps brachii, abductor pollicis brevis* or *extensor digitorum brevis.* As an initial test of the diagnostic utility of the minimum EMG burst duration data collected during this study and presented in Table 1, we reviewed EMG data from a sample of patients with a diagnosis of myoclonus. These results are shown in Figure 2. In a 48 year old female with a 5 year history of action myoclonus after recovering from hypoxic-ischaemic encephalopathy (the result of an out of hospital respiratory arrest requiring intubation and prolonged admission to the intensive care unit i.e. Lance-Adams Syndrome; Fig. 2A), the duration of EMG bursts from each muscle was less than the minimum voluntary EMG burst duration for the same muscles recorded from healthy controls (see Table 1). In contrast, in the two patients with a diagnosis of functional movement disorder, one a 60 year old male with a history of recurrent psychotic episodes since his teenage, each necessitating prolonged admission to a psychiatric facility, and a 2 year history of relapsing-remitting generalised myoclonic jerks (Fig. 2B), and the other, a 64 year old male (Fig. 2C)

with a background of chronic right leg pain, who developed abdominal myoclonus following two significant and simultaneous life events (the death of his mother and the arrest and imprisonment of his son), EMG bursts in the affected muscles were clearly longer than minimum EMG burst durations, as summarised in Table 1.

Discussion

The primary motivation for this study was to define the physiological limits of EMG burst durations during rapid voluntary movements for a range of muscles, thus providing a comprehensive reference for the neurologist or neurophysiologist investigating complex hyperkinetic movement disorders, particularly functional movement disorders. Our preliminary analysis did not appear to show a significant change in EMG burst duration with age. However, we acknowledge that older age groups were under-represented in our sample. Future studies of the physiology of EMG burst duration should include a more systematic investigation of the effects of age.

Whilst we have focused on the utility of minimum EMG burst duration in the clinic, the variability of EMG burst duration might also be a useful clinical measure. Voluntary EMG bursts are generated by engaging a number of different neural pathways, from movement to movement, and are thus highly variable (see Table 1). By contrast, in pathological involuntary ballistic movements, such as myoclonus, which are more stereotyped and generated by a much more limited repertoire of neural pathways, the associated EMG activity should be less variable. However, to our knowledge, this hypothesis has not been investigated formally. The standard deviation of pathological EMG burst duration might therefore be another measurable electrodiagnostic parameter in the investigation of movement disorders and merits further investigation.

Although the prevalence of FMDs is unknown, they are thought to be relatively common; it has been estimated that up to 40% of patients seen in movement disorder clinics are diagnosed with FMD (7,8). Whilst the aetiology is not entirely understood, recent accounts have suggested that FMDs arise because of abnormal

predictive coding by the brain, specifically miscalibration of internal predictions of the sensory consequences of movement (10). Within this conceptual framework, motor manifestations of FMDs remain constrained by physiological limits imposed by motor control circuitry.

Making the correct diagnosis in certain hyperkinetic movement disorders can be challenging, particularly when FMD is within the differential (e.g. dystonia; (16); myoclonus; (17)). When FMDs are not recognized, unnecessary and costly investigation ensues (18), appropriate early therapeutic interventions may not be provided (19,20) and of more concern, inappropriate (and occasionally high risk) therapeutic interventions are considered (21).

EMG burst duration is a simple, widely used guide to diagnosis (and localisation) of myoclonus (4) and has the advantage of requiring very little expertise to measure. However, our data suggest that EMG burst duration <50ms is not a reliable criterion for deciding whether a movement is pathological, particularly when applied to certain muscle groups (as is evident from one previous study (22)).

Our results also provide potential insights into the physiology of motor control. For ballistic contractions in the limbs (see Table 1), minimum EMG burst durations were longer in what are traditionally considered ‘pyramidal’ muscles (EDB, TA and EDC) compared to their antagonists (AH, MG and FCU respectively). Whilst voluntary control of the latter group of muscles is mediated to a greater extent by polysynaptic descending pathways which exert both excitatory and inhibitory effects on motoneurons (e.g. cortico-reticulospinal inputs; (23)), that of the former is mediated mainly by direct, monosynaptic corticospinal connections (24). This would suggest that rapid and brief movements are better generated by these polysynaptic descending pathways. Intriguingly, motoneurons controlling PM, which has an

exceptionally short minimum EMG burst duration for a trunk/proximal arm muscle (27.6ms; see Table 1), receive particularly strong cortico-reticular input (25,26). Finally, as noted by others (1,4), in the context of FMD it is important that EMG burst duration is not interpreted in isolation and where possible the presence or absence of co-contraction of agonist-antagonist muscles should also be assessed.

Author Roles

Author AFC: 1C, 2B, 3A

Author STRB: 1C

Author MRB: 1A, 1B, 2A, 2C, 3B

Funding Sources and Conflicts of Interest

None of the authors has any conflict of interest to disclose and there was no specific research funding for this project.

Financial Disclosures of all authors

AFC is employed by the University of Sheffield and has an honorary contract with Sheffield Teaching Hospitals NHS Foundation Trust

STRB was funded by an undergraduate scholarship from INSPIRE

MRB is employed by Newcastle upon Tyne Hospitals NHS Foundation Trust and Newcastle University. His research has been funded by grants from the following organisations: MRC; the William Leech Charity; NIHR; Ataxia UK; Friedreich's Ataxia Research Alliance

Ethical Publication Statement

The Ethics Committee at Newcastle University approved this study and we conformed to the Declaration of Helsinki. Written informed consent was obtained from all participants in this study on the day of the data collection. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this work is consistent with those guidelines.

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Figure Legends

Figure 1. A. Raw EMG data. Examples of unrectified raw surface EMG illustrating typical EMG bursts acquired from cranial (*risorius*), shoulder (*trapezius*, PM, *deltoid*), upper limb (FCU) and lower limb (VL, MG) muscles on the right. The grey box shows the duration of the longest EMG burst (*risorius*) for comparison. **B. Summary bar graph.** Minimum EMG burst duration was averaged for each muscle across all subjects (cranial, back and upper limb muscles (n=20); PM (n=18); lower limb muscles (n=36)). Error bars show standard error. **C. Ballistic and rhythmic EMG bursts.** Examples of unrectified surface EMG recorded from lower limb muscles in the same subject (Subject 6) whilst making either brief voluntary ballistic muscle contractions or rapid alternating rhythmic movements. Note that EMG burst duration in rhythmic movements was consistently reduced. Vertical dashed lines indicate onset and offset of EMG bursts and grey boxes highlight the duration of the shorter rhythmic EMG bursts. **D. Example of a ballistic EMG burst <50ms recorded from the *tibialis anterior* muscle.** The grey box and dashed vertical lines here delimit a 50ms time window. [*flexor carpi ulnaris* (FCU); *medial gastrocnemius* (MG); *pectoralis major* (PM); *tibialis anterior* (TA); *vastus lateralis* (VL)].

Figure 2. Examples of unrectified surface EMG recorded from three patients with a diagnosis of myoclonus. **A.** A 48-year-old female with a 5-year history of action myoclonus after recovering from hypoxic-ischaemic encephalopathy. **B** and **C.** Two patients with a diagnosis of functional movement disorder: **B.** a 60 year old male with a psychiatric history of more than 40 years (multiple prolonged admissions with psychosis) and a 2 year history of relapsing-remitting generalised myoclonic jerks; and **C.** A 64 year old male with a background of chronic right leg pain who developed

abdominal myoclonus following two significant and simultaneous life events (the death of his mother and the arrest and imprisonment of his son). Dashed boxes indicate the limits of each EMG burst. Grey boxes demarcate the minimum EMG burst duration for each muscle (see Table 1). In **A** note that there are no dashed boxes because each EMG burst falls within the grey box. [*abductor hallucis* (AH); *abductor pollicis brevis* (APB); *biceps brachii* (BB); *extensor digitorum communis* (EDC); Deltoid (Delt); *rectus abdominis* (RA); *vastus lateralis* (VL)]

Table Legends

Table 1. Average (and median) ballistic EMG burst durations with standard deviations and number of participants with average EMG bursts of less than 50ms. Average (and median) rhythmic EMG burst durations with standard deviations and with number of participants with average EMG bursts of less than 50ms. [Abbreviations: UL – upper limb; LL – lower limb; Temp - *temporalis*; OO – *orbicularis oculi*; Ris - *risorius*; Trap - *trapezius*; IS - *infraspinatus*; RB - *rhomboid*; LD – *latissimus dorsi*; PM – *pectoralis major*; RAS – *rectus abdominis superior*; RAI – *rectus abdominis inferior*; Delt - *deltoid*; TB – *triceps brachii*; BB – *biceps brachii*; EDC – *extensor digitorum communis*; FCU – *flexor carpi ulnaris*; APB - *abductor pollicis brevis*; FDI – *first dorsal interosseous*; VL - *vastus lateralis*; BF – *biceps femoris*; TA – *tibialis anterior*; MG – *medial gastrocnemius*; EDB – *extensor digitorum brevis*; AH - *abductor hallucis*].