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Original Article

Torque variability of the plantar flexors in people with Huntington's disease

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Abstract: Background: Torque steadiness can be impaired in people with Huntington's disease (HD) and worsen with disease advancement. However, existing studies have several methodological oversights. Studies have used absolute torque targets, which do not account for differences in maximal torque capacity between people. Furthermore, despite its known influence on torque steadiness, previous studies in HD have not controlled for visual feedback. This study evaluated torque variability at relative intensities with and without visual feedback between people with prodromal HD and healthy controls. Methods: Twenty-four people with prodromal HD and twenty-seven age- and sex-matched healthy controls were recruited for this study. Torque variability was evaluated, with and without visual feedback, in the right plantar flexors at 10% and 30% of each participant's maximum voluntary isometric contraction (MVIC). Measures of disease burden included the CAG age product, diagnostic confidence level and Unified Huntington's Disease Rating Scale - Total Motor Score. Results: Significant differences in torque variability were observed, though not in overall MVIC, between people with prodromal HD and healthy controls. Significantly higher torque fluctuations were observed for both groups when visual feedback was removed. No associations were observed between torque variability and disease burden in people with prodromal HD. Torque variability measurements showed higher reliability in healthy controls. Conclusions: People with prodromal HD exhibited greater torque variability than healthy controls. Torque variability worsened for both groups when visual feedback was removed. These findings support further investigation into the utilisation of torque variability measurements as markers of disease progression in people with prodromal HD.

Keywords: Force steadiness, isometric contraction, Huntington's disease, visual feedback

Introduction

The inability to regulate force production, often referred to as force variability, is a common and disabling aspect of Huntington's disease (HD) [1, 2]. In particular, studies have reported greater force variability in the hands, tongue and feet of people with prodromal and manifest HD [3-6]. Measurement of alterations in force regulation is therefore of relevance as deterioration can negatively impact activities of daily living, including the ability to hold objects, eat with utensils, brush teeth, and drive a motor vehicle [3, 4, 6-8].

Several methodological approaches have been used to examine force control in people with HD. The most common ones have involved the utilisation of portable force transducers to characterise alterations in force control [3, 5, 6]. While informative, existing methodologies have several notable shortcomings. First, existing studies have not controlled joint position during force control tasks [5]. Changes in joint position enables engagement of non-specific muscle groups, which alters force characteristics and proprioceptive feedback to the motor system, subsequently making it difficult to reliably determine force variability [9-11]. Second,

available studies have used predefined absolute force targets (e.g., 0.5 N, 1 N, 10 N), which do not account for the innate differences in muscle strength between participants [5, 6]. This is noteworthy given the interindividual differences in muscle strength, thus requiring different muscular efforts between people during force control tasks [12]. It is therefore not possible to ascertain whether observed force variability is reflective of more pronounced muscle weakness or changes in neuromuscular function (i.e., motor unit recruitment or discharge frequency). For these reasons, studies in other healthy and clinical populations have used force targets that represent the percentage of an individual's maximum voluntary contraction (MVIC) [12, 13]. This methodological approach removes the potential strength bias that may influence the force variability outcomes. Third, the vast majority of studies have examined torque variability at low force targets (e.g., 1, 5 and 10 N) [5, 6], with little emphasis given to the examination of torque variability at higher force targets. Finally, no studies in people with HD have investigated torque variability of the plantar flexors, which are involved in activities such as driving and walking and are notably impaired in HD over time [14-16].

Beyond the aforementioned methodological limitations, no study has evaluated the influence of the visuomotor system on the force variability in people with HD. This is of relevance as previous studies in healthy and clinical populations, including Parkinson's disease, have noted the contribution of the visuomotor system in force control [13, 17, 18]. Specifically, force quantity and rate declined from target forces (5, 10, 15 and 25% MVIC) when visual feedback was removed in people with PD, compared to healthy controls [17, 18]. Furthermore, greater force variability was observed with the removal of visual feedback, particularly at the higher MVIC outputs (20 and 30%) in a healthy older population. Given the finding that visuomotor system is impaired in people with HD it is critical to investigate its role in force variability.

The aim of this investigation was threefold: (1) to evaluate torque (rotational force) variability at 10% and 30% maximal voluntary isometric contraction (MVIC) values with and without visual feedback between people with prodromal HD and healthy controls; (2) to evaluate the reliability of torque steadiness tasks in people with prodromal HD and healthy controls; and (3) to evaluate associations between clinical measures of disease burden and torque variability values. We hypothesised that torque variability would be greater in people with prodromal HD and that this would be greater during no visual feedback conditions. In addition, we hypothesised that torque steadiness tasks would be reliable in people with prodromal HD and healthy controls and would be associated with clinical measures of disease burden.

Methods

Ethical approval and participant consent

The study was conducted in accordance with the Declaration of Helsinki and approved by Edith Cowan University (approval number: 13-145), North Metropolitan Area Mental Health Service (approval number: 2009_16) and Deakin University (approval number: 2015-052) Human Research Ethics Committees. Researchers ensured that all participants understood the requirements of the study and provided written and informed consent prior to engagement in study procedures.

Study design

The present study examines cross-sectional data collected as part of a recently completed clinical trial (ACTRN12618001717246) and observational data collected in healthy controls.

Participants

Twenty-four people with prodromal HD and twenty-seven age- and sex-matched healthy controls were recruited for this study (**Table 1**). Given previous findings by Medzech, et al. [5], authors were interested in evaluating whether similar, albeit more subtle, torque steadiness impairments could be detected in the plantar flexors of people with prodromal HD. People with prodromal HD as well as healthy controls were recruited through existing databases. Inclusion criteria for the prodromal HD group were as follows: (1) a cytosine-adenine-guanine (CAG) repeat length greater than 39; (2) a total

Table 1. Comparison of baseline characteristics between the prodromal HD group and healthy control group

Variable	Control (n=27)	Prodromal HD (n=24)	*P*-value
Age (years)	43.84 (2.14) [21; 58]	42.50 (2.52) [22; 59]	0.343[a]
Sex (male/female)	6/16	9/13	0.215[b]
MVIC	85.70 (49.16) [24.37; 202.50]	105.87 (62.37) [16.63; 260.49]	0.101[a]
CAGn	-	43.23 (3.23) [39; 51]	-
CAP Score	-	0.88 (0.21) [0.54; 1.31]	-
DBS	-	302.22 (87.79) [166.5; 467.5]	-
UHDRS-TMS	-	8.27 (10.14) [0; 36]	-
DCL	-	0.77 (1.06) [0; 4]	-
TFC	-	13.00 (13.00) [13; 13]	-

MVIC: maximal voluntary isometric contraction; CAGn: cytosine-adenine-guanine repeat number; CAP: CAG-age product; DBS: disease burden score; CAP score: CAG-age product scaled score; UHDRS-TMS: Unified Huntington's Disease Rating Scale; DCL: diagnostic confidence level; TFC: total functional capacity. Data are presented as mean (standard deviation) [range]. [a], Independent t-test was used for comparison between groups. [b], Chi-square test was used for comparison between groups.

functional capacity (TFC) score of 13; (3) diagnostic confidence score equal to or less than 2 on the Unified Huntington's Disease Rating Scale-Total Motor Score (UHDRS-TMS); and (4) the ability to follow instructions and provide informed written consent. Exclusion criteria for both people with prodromal HD and healthy controls were the presence of musculoskeletal, metabolic, endocrine or cardiovascular disorders or recent or longstanding substance abuse. Eligibility was assessed over the phone and in person.

Disease burden scores were calculated using the methods suggested by Penney Jr, et al. [19]. To determine the proximity to diagnosis at the start of the study, an index was calculated using the CAG-age product (CAP) score, according to previously established methods [20].

Study procedures

Warm up: Prior to the commencement of torque steadiness procedures, participants were asked to undertake a 5-minute physical warm-up on a Monark cycle ergometer at 60 Watts. Following the completion of this warm-up, participants were seated on an isokinetic dynamometer (Biodex System 4), with the hip joint slightly flexed (35°), the knee joint fully extended (0°), and the ankle joint of the right foot in a neutral position (0°) such that the sole of the foot was perpendicular to the shank and the lateral malleolus of the fibula aligned to the centre of rotation of the dynamometer. Once seated correctly, participants were asked to perform 5 submaximal contractions of increasing intensity ranging from 20 to 100% of perceived exertion using the plantar flexors as part of a task-specific warmup, with 30 s rest between contractions. Upon conclusion of these warmup and familiarisation procedures, participants were instructed on the MVIC procedures.

MVICs: Participants were asked to perform 3-5 MVICs of the right plantar flexors for a duration of 3 to 5 s each, and a rest period of one minute was given between contractions. Participants received encouragement along with visual feedback throughout the MVIC contractions. The MVIC contraction was performed until the maximal torque had a difference of less than 5%. Torque was sampled at 2000 Hz at the ankle joint using LabChart Software (ADInstruments, NSW, Australia) and a 16-bit analogue-to-digital converter (PowerLab 16/35, ADInstruments, NSW, Australia).

Torque steadiness: Torque steadiness was evaluated in the right plantar flexors at 10% and 30% of each participant's maximum voluntary contraction torque. Two conditions were used for torque steadiness measurements: with and without visual feedback. During the visual feedback condition, participants were asked to apply force through the plantar flexors to match and maintain a real-time torque generation line with a horizontal target torque line at 10% and then 30% of the participant's MVIC for 20 seconds. Real-time and horizontal target torque lines were presented on a television located directly in front of participants. For the

no visual feedback condition, participants were similarly asked to match their real-time torque generation line with the horizontal target torque line, however visual feedback was removed once participants' real-time torque generation line met the horizontal target torque line. Participants were asked to maintain the expected target torque (10% and then 30% MVC) for 20 seconds following the removal of visual feedback (TV turned off), with the entirety of the 20 second trial analyzed. The amplitude of torque fluctuations above and below the horizontal target torque lines (torque control) were quantified and used for analysis. Greater coefficient of variation (CV) values was indicative of worse torque steadiness. Participants performed one familiarisation trial, followed by three trials for each condition, with the average of trials used for analysis. The order of study assessments was as follows: 10% MVC (visual feedback provided), 30% MVC (visual feedback provided), 10% MVC (visual feedback not provided) and 30% MVC (visual feedback not provided).

The 10% MVIC was selected as it was the lowest relative value that participants could perform plantar flexion of the ankle without the need to engage extensor muscles (e.g., tibialis anterior). It is noteworthy that torque levels below 10% MVIC were not undertaken as they are associated with a lower signal to noise ratio [21]. The 30% MVIC was selected based on previous research which indicated that 30-40% MVIC is the optimal range for reducing force variability, within this range both the number of active motor units and their discharge frequency can be adjusted [21]. The plantar flexors were specifically selected given their role in postural stability, gait and driving [22-24]. This muscle group was also selected as it has been previously examined and shown to be impaired in people with manifest HD [5].

Statistical analysis

Sample size was calculated based on the results reported by Reilmann, et al. [6] on isometric torque steadiness in people with prodromal HD and controls performing three level tasks. From the results of this study, it suffices that the minimum detectable effect size (Cohen's f^2) is set at 0.45 (large). For a two-way analysis of variance (ANOVA) to examine the effect of group, visual feedback and interactions and using an alpha level of 0.05 and a statistical power of 0.8, it was estimated that a sample size of at least 20 participants per group would be sufficient to detect a difference in isometric torque steadiness between people with prodromal HD and healthy controls.

Data are presented as mean and standard deviation. Normality assumptions were evaluated with the Shapiro-Wilk test. Age differences between groups were examined with an independent t-test. Sex differences were evaluated using a Chi-square test. The intraclass correlation coefficient (ICC), estimated with a two-way mixed model, was used to determine the reliability of the torque steadiness variables for each group. A nested model analysis of variance (ANOVA) was used to examine the effects of torque steadiness level (10% vs. 30%), group (prodromal HD vs. control), visual feedback (present vs. absent) and two-way interactions on torque steadiness. Contrasts were used to compare the prodromal HD group to healthy controls. Spearman's correlation coefficients were considered to assess associations between torque variability and measures of disease progression. Statistical significance was set at $P \leq 0.05$. Statistical analyses were undertaken with STATA version 15.1.

Results

Demographic characteristics

Participants completed all study procedures. Demographic and clinical data are provided in **Table 1**.

Test-retest reliability

Test-retest reliability values of torque steadiness data are presented in Table S1. Torque steadiness outcomes showed higher reliability in controls (range: 0.40-0.99) than in people with prodromal HD (range: 0.36-0.99) overall and after controlling for the target torque and presence or absence of visual feedback.

Torque steadiness

Group differences in torque steadiness levels with and without visual feedback are presented in **Figure 1**. When comparing group effects across all torque intensity and visual feedback

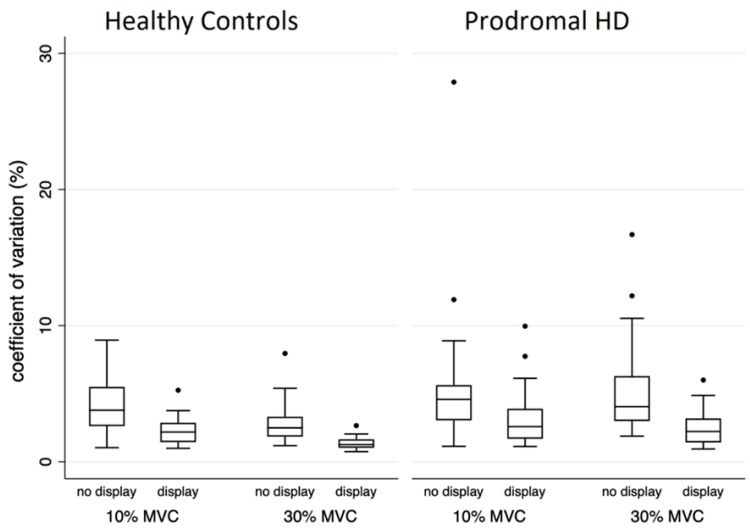

Figure 1. The box and whisker plots show the amount of torque variability, as indicated by a greater coefficient of variation, between the levels of torque steadiness and visual feedback conditions in people with prodromal HD and healthy controls. The solid dots represent outliers in the data.

trials the people with prodromal HD had significantly higher torque variability (P=0.018), as indicated by the coefficient of variation of torque, in comparison with healthy controls. People with prodromal HD also had significantly higher torque variability, compared to healthy controls, in the 10% MVIC with visual feedback (*P*=0.020), 30% MVIC with visual feedback (*P*<0.001) and 30% MVIC without visual feedback (*P*=0.001) conditions but not in the 10% MVIC without visual feedback (*P*=0.061) condition. Significantly higher torque fluctuations (coefficient of variation of torque) were observed for people with prodromal HD (10% MVIC [*P*=0.019], 30% MVIC [*P*<0.001]) and healthy controls (10% MVIC [*P*<0.001], 30% MVIC [*P*<0.001]) when visual feedback was removed. No significant torque fluctuations were observed in people with prodromal HD (with visual feedback [*P*=0.089], without visual feedback [*P*=0.372]) for the 10% compared to 30% MVIC conditions. However, significantly greater torque fluctuations were observed in the 10%, compared to 30%, MVIC condition in healthy controls (with visual feedback [*P*< 0.001], without visual feedback [*P*=0.011]).

Associations

A moderate association was found between the UHDRS-TMS and torque variability (CV) at 10% MVIC when visual feedback was provided (Spearman rho =0.44; *P*= 0.049, not significant after correction). However, no associations were found between the UHDRS-TMS and torque variability at 10% without visual feedback, nor 30% MVIC, with or without visual feedback (see **Figure 2**). Additionally, no associations were found for CAP or DCL score and torque variability regardless of the MVIC condition and visual feedback (see **Figure 3**).

Discussion

Reduced force control has previously been documented in people with HD and adversely impacts activities of daily living, including driving, holding a cup, and brushing teeth. Here, we investigated torque variability profiles with and without visual feedback in people with prodromal HD and healthy controls, while controlling for joint position and maximal isometric strength. In addition, we evaluated the reliability of torque variability measurements and potential associations between torque variability values and measures of disease burden. We observed significant differences in torque variability profiles between people with prodromal HD and healthy controls. Furthermore, we found that torque variability measurements were reliable, but were not associated with motor symptom severity in people with prodromal HD.

Consistent with previous findings, we found significant differences in torque variability profiles between people with prodromal HD and healthy controls. In particular, people with prodromal HD demonstrated significantly greater torque variability at 10% and 30% MVIC values compared to healthy controls. These findings are relevant as the present study controlled for limb position and interindividual differences in maximal isometric strength of participants, which have been shown to influence torque control [9-11, 13]. Together, these findings suggest that torque variability may be a useful measure of disease onset, however this needs to be explored further in longitudinal studies.

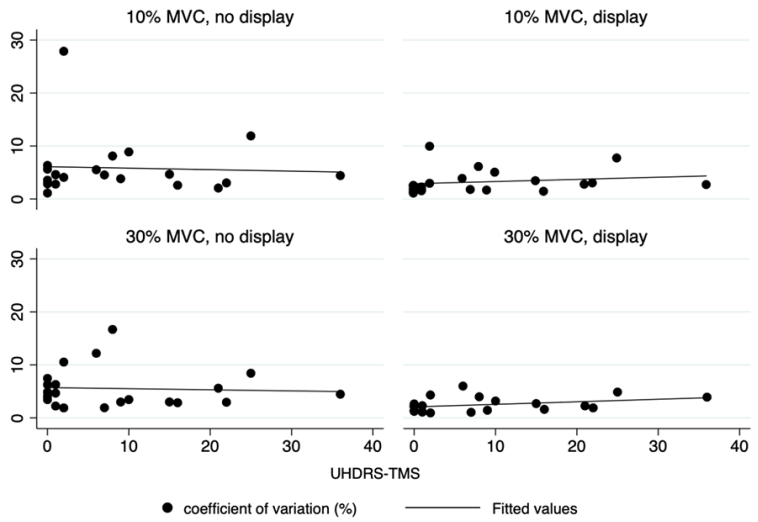

Figure 2. Scatterplots of the relationship between the UHDRS-TMS and the coefficient of variation at the various levels of torque steadiness and visual feedback conditions in people with prodromal HD.

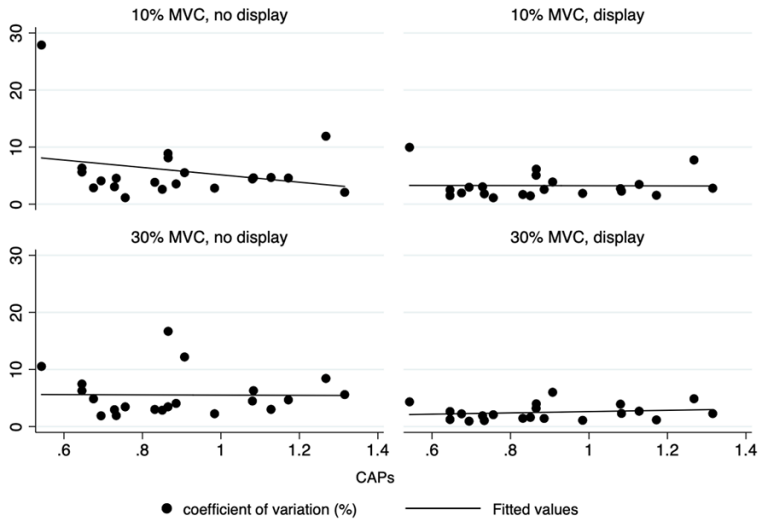

Figure 3. Scatterplots of the relationship between the CAPs and the coefficient of variation at the various levels of torque steadiness and visual feedback conditions in people with prodromal HD.

A noteworthy finding of this study was that torque variability worsened for prodromal HD and healthy control groups when visual feedback was removed. To our knowledge, this is the first study to report this observation in people with prodromal HD. This finding contrasts previous work in subacute stroke [25] and healthy older adults [13], where removal of visual feedback was associated with reduced force (torque) variability. This discrepancy in findings may be explained by differences in the force/torque variability protocols used between studies, with previous studies in healthy populations evaluating force profiles in the index finger of dominant and non-dominant hands, which have greater sensitivity to sensorymotor stimuli, particularly proprioceptive feedback [26]. However, this explanation requires further investigation in HD. Additionally, while complete removal of visual feedback was necessary to ascertain the influence of the visuomotor system, future studies should investigate partial reduction in visual information as this may better reflect events in daily life.

Contrary to previous studies, our results show that torque variability of the plantar flexors did not significantly differ between 10% and 30% MVIC torque outputs for the prodromal HD group, despite being significantly different in the healthy control group. This finding was unexpected given the recent findings by Medzech, et al. [5], who reported greater force variability at lower force outputs in people with manifest HD. Additionally, Reilmann, et al. [6] found that lower force targets were more sensitive at detecting motor deficits in people with prodromal HD. The exact reason for this discrepancy in finding is not known. Methodological differences, including equipment utilized and the sampled population between the present and previous studies may explain this discrepancy in findings, however this needs to be explored by future studies.

It is noteworthy that we did not observe a significant association between greater torque variability and disease burden measures, despite previous literature reporting such associations [3-6]. This is presumably due to the low

total motor score ranges, which are typical during the prodromal stage of the disease.

Several limitations need to be considered when interpreting our study findings. First, this was a cross-sectional investigation, which does not provide information of the sensitivity of measures over time. Second, while our study was appropriately powered, only people with prodromal HD were included, limiting the generalisability of our findings to people earlier or later in the disease course. Finally, this study only examined associations between torque variability and clinical measures of disease burden, therefore our findings do not represent a causal link between torque variability and motor severity and cognitive function.

Our findings show that torque variability measurements are reliable and discriminate between people with prodromal HD and healthy controls. In addition, our findings show that torque variability increases when visual feedback is removed in people with prodromal HD and healthy controls. Finally, our findings show that torque variability did not differ between torque intensities, nor was it associated with disease burden in people with prodromal HD. Although there is still a fundamental need to determine the sensitivity of torque variability measurements over time, our findings provide preliminary support for the utilisation of these measurements as markers of disease progression.

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Disclosure of conflict of interest

None.

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Table S1. Reliability of force steadiness outcomes (intraclass correlation coefficients) and 95% intervals confidence for both groups

Variable	Force Steadiness	Display	Control			HD		
			ICC	95% Confidence Interval		ICC	95% Confidence Interval	
Max Corrected Torque	10%	Absent	0.989	0.980	0.995	0.823	0.684	0.914
		Present	0.997	0.994	0.999	0.998	0.997	0.999
	30%	Absent	0.995	0.991	0.998	0.935	0.876	0.970
		Present	0.999	0.999	0.999	0.997	0.993	0.998
Mean Corrected Torque	10%	Absent	0.991	0.983	0.995	0.810	0.662	0.907
		Present	0.999	0.997	0.999	0.999	0.999	0.999
	30%	Absent	0.992	0.984	0.996	0.930	0.865	0.967
		Present	0.999	0.999	0.999	0.999	0.999	0.999
Standard Deviation Corrected Torque	10%	Absent	0.453	0.218	0.671	0.601	0.370	0.787
		Present	0.580	0.363	0.760	0.463	0.209	0.696
	30%	Absent	0.402	0.161	0.638	0.657	0.442	0.822
		Present	0.802	0.662	0.898	0.730	0.542	0.863
Standard Error Corrected Torque	10%	Absent	0.453	0.219	0.672	0.595	0.362	0.784
		Present	0.580	0.363	0.760	0.453	0.198	0.689
	30%	Absent	0.402	0.161	0.637	0.657	0.441	0.821
		Present	0.802	0.662	0.898	0.728	0.539	0.862
EMG SOL rms mV	10%	Absent	0.979	0.961	0.990	0.891	0.797	0.948
		Present	0.993	0.986	0.996	0.988	0.977	0.995
	30%	Absent	0.989	0.979	0.995	0.943	0.890	0.973
		Present	0.993	0.986	0.996	0.984	0.968	0.993
EMG MG rms mV	10%	Absent	0.966	0.937	0.983	0.740	0.557	0.869
		Present	0.977	0.957	0.989	0.366	0.109	0.625
	30%	Absent	0.888	0.798	0.944	0.909	0.828	0.957
		Present	0.906	0.829	0.953	0.817	0.674	0.911
EMG LG rms mV	10%	Absent	0.999	0.999	0.999	0.988	0.976	0.995
		Present	0.999	0.999	0.999	0.927	0.861	0.966
	30%	Absent	0.993	0.986	0.997	0.632	0.409	0.807
		Present	0.999	0.998	0.999	0.884	0.784	0.945
EMG TA rms mV	10%	Absent	0.986	0.973	0.993	0.733	0.547	0.865
		Present	0.999	0.998	0.999	0.500	0.250	0.722
	30%	Absent	0.998	0.997	0.999	0.765	0.593	0.883
		Present	0.994	0.988	0.997	0.414	0.157	0.661
Coefficient of Variation (%) Corrected Torque	10%	Absent	0.513	0.285	0.715	0.689	0.485	0.840
		Present	0.606	0.395	0.777	0.784	0.622	0.893
	30%	Absent	0.429	0.188	0.658	0.397	0.139	0.648
		Present	0.611	0.397	0.784	0.670	0.459	0.829