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Research Article

Sustained Muscle EMG Activity to Contractile Failure During Incremental Exercise and Intense Constant Load Cycling: No Evidence of a Central Governor

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Since 1997, debate has continued over the presence of a central governor that constrains neuromuscular activity during severe, intense exercise. This study aimed to challenge the central governor model (CGM) by acquiring surface electromyography (sEMG) data from the vastus lateralis (VL) and gluteus maximus (Gmax) muscles of 14 healthy participants during 4 different bouts of constant load, non-steady state cycling exercise (110, 125, 140, 160 %watts at the ventilation threshold), and 1 incremental bout to volitional exhaustion. sEMG activity was processed to isolate and capture each contraction of the VL and Gmax during all bouts of exercise. sEMG data were then graphed to profile sEMG root mean square (rms) activity over time, with linear curve fitting used to quantify this relationship for data preceding (segment 1) and during the final 30s of each test (segment 2). A two-way repeated measures ANOVA was used to test for differences between the slopes of the two linear segments of the sEMG rms response of the VL for each bout. Results during the VO_2max trial revealed a significant main effect for SEGMENT, where segment 2 was significantly greater than segment 1 ($F=6.741$, $p=0.023$). During the critical power trials, there were significant differences in sEMG rms for each of INTENSITY ($F=9.349$, $p<0.001$), SEGMENT ($F=5.443$, $p=0.036$), and the interaction effect ($F=2.837$, $p=0.005$). Muscle sEMG rms data revealed sustained increases in muscle activity in all bouts of intense exercise to volitional exhaustion in both the VL and Gmax, which is inconsistent with the predictions made from the CGM.

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Introduction

In 1924, Hill, Long, and Lupton[1] proposed that a "governor mechanism" could exist to constrain heart and peripheral vascular function during intense exercise, with the net benefit of this speculated to be the prevention of catastrophic organ damage. In 1997, Tim Noakes, a South African cardiologist, further refined this proposition and named it The Central Governor Model (CGM).[2] Noakes explained the CGM to involve regulated behavior from complex systems within the central nervous system (CNS) which are designed to protect and maintain homeostasis of the body. Whilst the CGM has been continually developed by Noakes from 1997 to the present time, considerable debate continues over research data that are anomalous to expectations based on the CGM, in addition to how the CGM may violate core principles of science. [2][3][4][5]

Core evidence from prior research and scholarship that has been interpreted to support the CGM has been the inability of numerous subjects who undertake incremental exercise to volitional fatigue to demonstrate a VO_2 plateau near the end of the testing protocol. Noakes viewed this to be supportive of the CGM given that an absence of a VO_2 plateau could be interpreted as a premature termination of exercise. Nevertheless, added causes of the absence of a VO_2 plateau could be poor protocol design, insensitive or invalid equipment, poor exercise tolerance of the subject, and an increasing VO_2 cost of ventilation.[6]

A core issue within the CGM is that the CNS must constrain further increases in motor unit recruitment during intense exercise to prevent structural damage to one or more physiological systems. As stated by Noakes, "*... the central nervous system can ensure that homeostasis is maintained in all bodily systems, not just the heart, by regulating the number of motor units recruited in the exercising muscles by the brain .....*".[3] (see page 26) If this is true, then it is logical to assume that muscle EMG activity should not continue to increase, or be sustained, during the final minutes of exercise to volitional exhaustion.

Surface electromyography (sEMG) is a tool used to collect electrical signals by contracting motor units through electrodes placed over the skin directly above the muscles of interest. Whilst sEMG data may not be able to distinguish the difference between increased motor unit recruitment, firing rate, or muscular failure, it is understood to be the closest measurement for quantifying altered electrical activity of contracting skeletal muscle during increases in exercise intensity during complex movement in human subjects.[7] Research has demonstrated changes in root mean squared (rms) data during ramp

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incremental exercise protocols, along with the rms/work rate ratio at exhaustion.[8] Scheuermann et al.[9] demonstrated increases in both mentioned variables tested in a slow and fast ramp protocol, which revealed sustained increases in muscle rms sEMG activity. sEMG rms/force ratio data has also been shown to increase during a repeated cycling bout protocol[10] thereby supporting the involvement of peripheral factors contributing to the development of muscle contractile failure at exhaustion. Furthermore, motor unit recruitment strategies have been shown to produce similar results during different protocols (cycling 10% above and below critical power) and suggest the central nervous system has little influence over the development of the contractile failure of skeletal muscle.[11] This data is further supported by various other studies that have investigated sEMG data and found muscle sEMG activity does not dampen at volitional fatigue.[9][12][13]

Interestingly, there is also compelling evidence to suggest that in a subset of subjects, a disturbance in homeostasis occurs preceding VO_2max and eventual exhaustion. This involves a plateau or drop in cardiac output and muscle blood flow, and meaningful drops in oxy-hemoglobin saturation.[14][15] If there were a CGM, one would expect these features to be prevented.

Fatigue mechanisms can also be attributed to metabolic reactions and the accumulation of metabolites within the peripheral systems. Such metabolites include inorganic phosphate (P_i), potassium (K^+), ammonia, lactate ($[La^-]$), and hydrogen ions (H^+), all of which to some degree have been attributed to the reduction in muscle force during intense cycling exercise to failure.[16][17] The primary cellular mechanisms that control muscle force production include the calcium (Ca^{2+}) concentration and sensitivity surrounding myofilaments, and the total activation of Ca^{2+}.[16] The production of La^- and H^+ through glycolysis is thought to influence these mechanisms, and research has often shown correlations between acidosis and declining muscle force production.[18] However, this relationship is not always present, as muscle force recovers quicker than pH and as such gives rise to another metabolite that influences contractile performance.[18] Increased concentrations of muscular inorganic phosphate (Pi = HPO_3^{-2}) are considered to be more detrimental to contracting muscle when sustaining power outputs above critical power.[16][18] The accumulation of intramuscular Pi (due to dephosphorylation of ATP to ADP +Pi during intense muscle contraction in excess of rates of mitochondrial respiration) ionically interacts with Ca^{2+} and lowers the availability of Ca^{2+} for muscle contraction.[16][18] K^+ and ammonia are further by-products that may also influence muscle fiber excitability. Consequently, the data suggest that contractile failure is likely to be a combination of a variety of factors[19][20] independent of CNS involvement in dampened neural activation.

Consequently, the problem addressed by this research concerns the uncertainty over evidence that would support the CGM during intense exercise to volitional failure, as well as the responses of muscle sEMG activity to different bouts of incremental vs. constant load intense exercise, including contractile failure after different exercise durations. As such, the purpose of this research is to quantify the muscle sEMG activity during and near the end of intense exercise of differing durations to establish the slope of the sEMG activity as evidence of altered or unaltered muscle activation prior to volitional exhaustion.

Material and Methods

Recruitment

Selection and recruitment of participants were based on self-reporting of physical fitness, with included participants being moderately to highly trained in cycling as reflected by at least 45 minutes of cycling training, three times a week. Additional inclusion criteria for participants included either males aged 18 - 45 years or females aged 18 - 55 years and ensured participants did not have any musculoskeletal, cardio-pulmonary, or metabolic diseases. Participants were excluded if any of the following conditions were met: 1) current and/or history of smoking, 2) current or recent musculoskeletal injury within the past 3 months, and 3) any surgical procedures within the past 3 months that may prevent exercise participation.

A total of 14 healthy, well-trained participants (12 male and 2 female) were recruited to complete multiple exercise trials of cycle ergometry. The participants of this study were local cyclists in the Brisbane community. These participants were recruited through social media platforms targeting trained cyclists, along with recruitment flyers and emails sent out within the university. Participants were also required to complete an Exercise and Sports Science Australia: Adult Pre-Screening System tool to determine if they met the inclusion and exclusion criteria to participate in the study.

Sample Size

Apriori sample size was estimated using the free software from the University of Dusseldorf; GPower (version 3.1.9.7). Based on an effect size = 0.5, p-value = 0.05, statistical power = 0.8, groups = 4, measurements = 4, correlation among measures = 0.2, and a non-sphericity correction = 1, the estimated total sample size was calculated to be 12 subjects with an actual statistical power = 0.86. To ensure sustained adequate statistical power for the study, 14 subjects were recruited to allow for missing data or participant dropout.

Informed Consent

An informed consent form was completed prior to attending the familiarization session. This was completed via an online meeting to ensure the participant understood the testing protocols and consent form. The form was then required to be signed during the familiarization session. All research procedures were approved by the QUT University Human Research Ethics Committee (Ethics approval number 4252).

Data Collection Methods

Each participant completed a total of 5 trials over a 4-day period within the exercise physiology lab of the Institute of Health and Biomedical Innovation. The protocol included a familiarization session and a VO_2max test completed on the first day, with 4 subsequent CP tests completed over the next 3 days. The order of CP trials was randomized for each participant, with 1 trial completed on the second day, 2 trials completed on the third day, and the final trial completed on the fourth day. Participants were instructed to cease any strenuous exercise 24 hours before each testing session, along with caffeine and alcohol for at least 12 hours before each testing session. Participants were also instructed not to consume food, nutrient supplements, or water at least 3 hours before each testing session.

Familiarization Trial

Participants attended an initial introductory session for familiarization with the testing protocols and equipment, along with the collection of objective data: age (years), height (cm), weight (kg), resting heart rate (beats.min^{-1}), and completion of a VO_2max test. Initially, participants were fitted to an electronically braked cycle ergometer (Excalibur Sport, Corval Lode B.V., Lode Medical Technology, Groningen, the Netherlands) and asked to cycle for 5 minutes at 100 Watts. Bike adjustments were recorded for each participant and used in subsequent trials. Adjustments were recorded by seat height (cm), handlebar positioning (cm), and preferred cycling cadence (rpm).

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Demographics

Each participant's height was measured using a wall-mounted stadiometer (Seca, Hamburg, Germany) after they maintained an upright position and performed a full expiration breath. Weight was measured using a digital electronic scale (Seca, Hamburg, Germany). Age and sex of the participants were also taken and recorded in an Excel spreadsheet based on anonymous (de-identified) subject codes. Self-reported fitness levels were also recorded as low, moderate, or high.

Cycle ergometry

VO_2max Testing

Participants were asked to perform a ramp-based exercise protocol to volitional fatigue, ranging from 25 - 40 Watts·min^{-1} between participants (determined prior based on self-reported fitness levels and with the intent to reach volitional exhaustion within 8 - 12 minutes). Participants were fitted with a 5-lead electrocardiography (ECG) configuration (Custo-Med^{TM}, Ottobrunn, Germany) to collect heart rate data throughout the trial and to monitor for any adverse cardiovascular events. The initial workload was determined from double the predetermined ramp protocol, and participants were asked to cycle at their preferred cadence (within ± 5 rev.min^{-1}). Testing was ceased upon reaching volitional exhaustion, which was defined as the participant's inability to maintain cadence within ± 20 rev.min^{-1} of their predetermined target ramp cadence and/or volitional termination by the participant.

Critical Power

On the second day of testing, participants were asked to return to the laboratory 24 hours following their VO_2max test to complete the first CP trial with gas-exchange data collected throughout. The order of CP testing was randomized for each participant for bouts of 110, 125, 145, or 160% of each participant's calculated watts at the ventilation threshold (%Watts@VT) determined from their VO_2max test, and tests were administered across 3 days as follows: day 2, one trial; day 3, two trials; day 4, one trial. For day 2, participants lay supine for 15 minutes between trials to mitigate carryover effects from fatigue. Trial termination for all CP trials was determined by the inability of the participant to maintain cadence within 10 rev.min^{-1} of their predetermined target cadence for a 10 s period despite verbal encouragement.[21] In all trials, subjects were blinded to the work rate and elapsed time but received visual feedback of their cadence.

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Critical power was calculated (Equation 1) based on applying a one-phase exponential decay (hyperbolic response) to the time (x-axis) and power (Watts, y-axis), where critical power is denoted as the plateau of Watts with increasing time to failure. To calculate the curvature constant (W'), exercise time was transformed to reciprocal data, and a linear regression was applied to the Watts to 1/time data where the slope equaled W'.

$$Y = Span\ .\, e^{-k.x} + Plateau$$

Muscle electromyography

Surface electromyography (sEMG) was employed to record muscle activity throughout all exercise bouts. Muscle activity was recorded from the gluteus maximus, vastus lateralis, biceps femoris, and medial gastrocnemius. Data were collected using a Trigno Avanti wireless biofeedback system (Sensor model SP-W06; Base station model SP-W02,7,8; System model DS-T03Delsys, Boston, MA, USA,) which was sampled at 2000 Hz using LabChart software (AD Instruments, Colorado Springs, CO, USA).

Prior to the application of the sEMG sensors, participants were prepared by shaving the hair covering applicable locations (if required), rubbing the sensor locations with the use of fine sandpaper for skin abrasion, and finally wiping and cleaning the site with an alcohol wipe. Sensors were placed with direction from the SENIAM (sEMG for Non-Invasive Assessment of Muscles) guidelines over skin locations for the vastus lateralis, biceps femoris, gastrocnemius, and gluteus maximus muscles.

Electromyographic activity was collected from 4 muscles (as detailed previously) during the VO_2max test and 4 critical power tests. Due to complex and weak sEMG signals from the gastrocnemius and biceps femoris that prevented capture of individual contractions, data were only processed for the vastus lateralis and gluteus maximus muscles.

Muscle sEMG activity was processed using custom software (LabVIEW™, National Instruments, Austin, TX, USA). A baseline signal was acquired, and sEMG signals greater than at least 110% of baseline (varied within the program for different participants due to variation in the signal-to-noise ratio of the sEMG signal, as well as the shape of the sEMG signal after differentiation to assist in detection of the start and end of a contraction) were captured for each contraction. Signal captures were also dependent on a time factor for the cycling cadence (muscle contractions) for each muscle to ensure the prevention of sEMG noise from falsely being detected as a contraction. Captured contraction segments of data were

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mathematically processed for root mean square (rms) activity, and each of the mean and median frequency of the signals from spectral analysis.

The sEMG rms of each trial (VO_2max and 4 critical power trials) was plotted separately. As the purpose of this study focused on the sEMG responses near the end of each trial, the sEMG rms data were fitted with two linear segments spanning the last 30 s of the trial (segment 2) and the best-fit linear segment preceding this (segment 1). The range of segment 2 was predetermined, and the range of segment 1 was defined by the data segment having the least residual error.

Determination of VO_2max and constant non-steady state bout peak VO_2

Data from the VO_2max test were imported into custom software (LabVIEW™, V2017, National Instruments, Austin, TX, USA) for 7-breath averaging, where the highest processed data point was accepted as the maximal rate of VO_2 (VO_2max). For the non-steady state exercise bouts used for the determination of critical power, the same 7-breath averaging occurred, and the highest processed VO_2 data point was used to detect peak VO_2. The test durations and peak Watts for all exercise bouts were also recorded.

Other measures taken from the indirect calorimetry data included peak values for ventilation, tidal volume, breathing frequency, carbon dioxide production (VCO_2), and the respiratory exchange ratio (RER).

Determination of the ventilation threshold

For detection of the VT via the ventilatory equivalents method, custom software (LabVIEW™, V2017, National Instruments, Austin, TX, USA) was used to apply three linear segments to the data. Linear segments were adjusted to the lowest residual error, and the VT was determined as the time of the intersection between segment 1 (baseline response, slope ~ 0) and segment 2 (initial deviation from baseline) with detection requiring agreement (within ± 10 s) between two investigators.

Statistics

Data for the variables RER, peakVI, peakFbr, peakVt, and exercise time were all analyzed by repeated measures one-way ANOVA. Post-hoc analyses of the differences between the four means for each variable were tested using the Tukey test.

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For the sEMG data from the VO_2max test, slopes for sEMG rms over time for the two segments (SEGMENT) of the VL and Gmax muscles (MUSCLE) were analyzed by two-way (2 x 2) general linear model repeated measures ANOVA. Statistical significance was accepted at $p \leq 0.05$, and sphericity was assumed to be equal across all levels of each factor. One subject did not have quality sEMG signals for the Gmax, requiring data to be analyzed statistically for a sample size of 13 subjects.

For the sEMG data for all CP trials, quality data for the Gmax was only evident for 8 of the 14 subjects. A three-way (INTENSITY (4) x MUSCLE (2) x INTENSITY (2)) general linear model repeated measures ANOVA was not performed due to poor statistical power for the two levels of MUSCLE. Consequently, slopes for sEMG rms over time for the four non-steady state CP intensities (INTENSITY) for the two segments (SEGMENT) of the VL muscle were analyzed by two-way (2 x 2) general linear model repeated measures ANOVA. All statistical analyses were performed using SPSS (IBM SPSS Statistics, version 25, 2017, Armonk, New York, USA). Statistical significance was accepted at $p \leq 0.05$, and sphericity was assumed to be equal across all levels of each factor. All 14 subjects' data were used in this analysis.

Finally, statistical analysis was not conducted on the peak VO_2 data gathered from the VO_2max and CP trials, as this data will be presented within an additional manuscript.

Results

To further inform the Methods, raw data from one representative subject (#11) is presented in Figure 1 for the sEMGrms data from the five different exercise bouts, with added data examples for the raw sEMG signals of isolated contractions. The scales of the x- and y-axes are consistent between figures 1a-e to allow direct comparison. The increasing or sustained sEMG activity across all exercise conditions is evident.

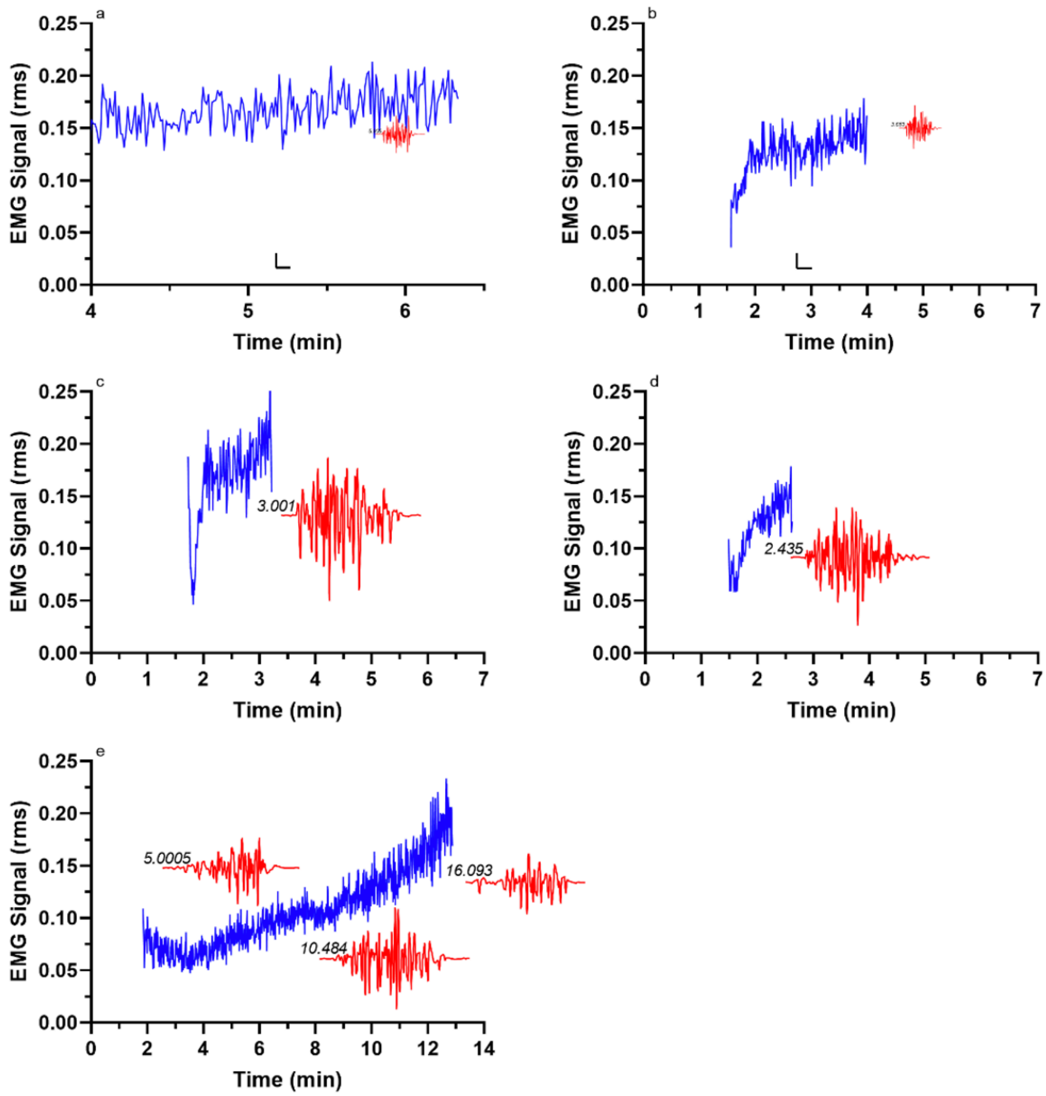

Figure 1. The raw data for subject 11 (see Table 1) for sEMG-rms and select examples of the raw sEMG rms signals for a) 110% VT CP trial (365 watts), b) 125% VT CP trial (415 watts), c) 145% VT CP trial (481 watts), d) 160% VT CP trial (531 watts), e) VO2max trial (peak = 425 watts).

The descriptive characteristics of the subjects are presented in Table 1, along with pertinent variables from the exercise testing. Similar gas exchange variables from the CP trials are presented in Table 2. For the repeated trial mean data of Table 2, one-way repeated measures ANOVA analyses for RER, peakVI, peakFbr, and peakVt were non-significant across 110-160 %Watts @VT. For exercise time, all mean data were significantly different from each other: $F=20.474(3)$, $p<0.0001$.

Subject No.	Demographics			Incremental				Critical Power
	Age (yrs)	Height (cm)	Body Weight (Kg)	VO_2max ($L{\cdot}min^{-1}$)	VO_2max ($ml{\cdot}min^{-1}{\cdot}kg^{-1}$)	RER	Ex time (min)	CP (Watts)
1	42	174.10	87.45	5.90	67.41	1.09	13.14	374
2	29	187.00	73.85	5.69	76.84	1.13	11.28	389
3	43	175.00	69.60	5.09	73.14	1.08	11.42	231
4	43	184.90	85.40	5.41	63.38	1.2	14.13	317
5	46	172.40	69.45	3.80	54.71	1.15	10.12	254
6	34	190.60	97.30	5.91	60.74	1.24	12.10	326
7	35	182.10	82.05	4.97	60.55	1.59	7.42	273
8	47	159.20	51.40	2.24	43.38	1.29	12.35	146
9	41	192.20	88.80	4.94	55.60	1.4	13.16	322
10	30	173.50	71.15	4.24	59.56	1.2	10.15	276
11	34	183.20	76.80	4.42	57.57	1.48	10.63	352
12	36	177.90	68.10	4.87	71.54	1.37	11.79	358
13	40	188.80	83.65	4.33	51.70	1.31	11.10	229
14	43	188.80	91.80	4.85	52.88	1.2	10.57	260
Mean	38.79	180.69	78.34	4.76	60.64	1.27	11.38	243.4
SD	5.75	9.17	12.04	0.96	9.20	0.15	1.65	67.4

Table 1. Descriptive data for all subjects, with collated data for means and standard deviations (SD).

CP Trial	VO_2peak ($L·min^{-1}$)	RER	Peak V_I ($L.min^{-1}$)	Peak F_{br} ($br.min^{-1}$)	Peak Vt (L)	Ex. Time* (min)
110%	4.52±0.86	1.13 ±0.11	123.46±20.45	49.13±9.44	2.59±0.60	10.40±7.59
125%	4.28±0.83	1.15±0.09	129.78±22.71	51.39±11.05	2.62±0.66	4.46±3.05
145%	4.57±0.79	1.21±0.19	127.63±19.7	50.80±11.23	2.63±0.74	2.19±1.08
160%	4.3±0.82	1.27±0.22	122.39±28.42	48.77±11.24	2.61±0.73	1.38±0.59

Table 2. Data (mean±SD) for pertinent gas exchange data from the critical power trials.

**All CP trials significantly different from each other (p<0.0001)*

VL and Gmax Peak sEMGrms

The results for the VL and Gmax peak sEMGrms data are presented in Figure 2. Post-hoc analyses revealed that compared to data for 110 %Watts@VT, sEMGrms was significantly larger for 145 %Watts@VT (p=0.026). Despite the lower sample size for Gmax, and the related non-significance, the data were presented to document the similar trend in sEMG responses across both muscles. Note the large difference between the absolute voltage of the VL vs. Gmax.

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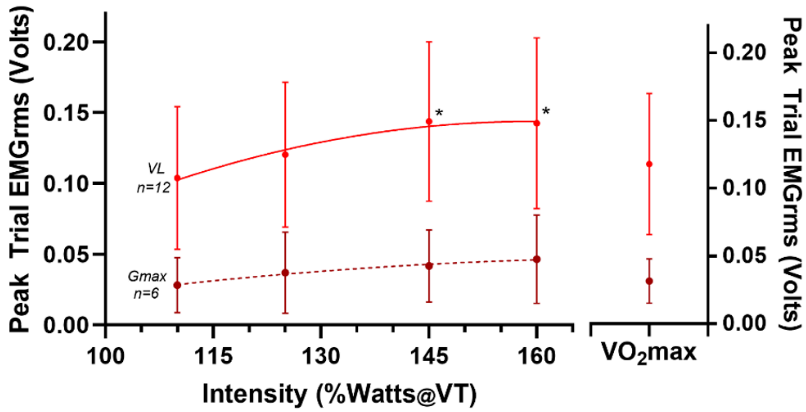

Figure 2. The peak sEMG rms data for the vastus lateralis (VL) and gluteus maximus (Gmax) during the CP and VO2max trials. *p<0.05 from 110 %Watts@VT

VL and Gmax sEMG rms Slope Profile during VO_2max Trial

Figure 3 presents the sEMG rms slope responses for the VL and Gmax results during the VO_2max trial. There was a significant main effect for SEGMENT where segment 2 was significantly greater than segment 1 (F=6.741, P=.023). There was no significant difference for the main effect of MUSCLE (F=2.115, P=.172). Given the non-significant interaction effect (F=1.865, P=.197), this revealed that for both the VL and Gmax, the sEMG rms activity continued to increase to contractile failure during incremental exercise to volitional exhaustion.

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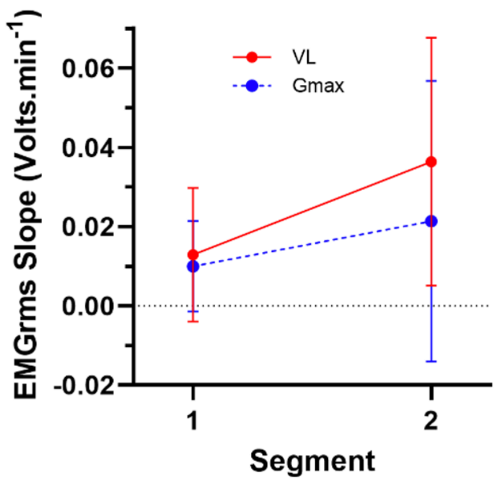

Figure 3. The slope profile of the vastus lateralis (VL) and gluteus maximus (Gmax) between segment 2 (final 30s of the trial) and segment 1 (best fit linear segment preceding the final 30s) during the VO_2max trial.

VL sEMG rms Slope Profile During CP Trials

Due to a combination of low signal-to-noise ratio for sEMG data collection and missing data in subjects, the Gmax muscle was unable to be analysed from the CP trials. Data were collected on only 8 subjects, which was not enough to provide sufficient statistical power to run a 3-way ANOVA that included the 2 muscles. Figure 4, therefore, presents sEMG rms data from the VL during the CP trials.

There were significant differences for each of INTENSITY (F=9.349, P= <.001), SEGMENT (F=5.443, P=.036), and the interaction effect (F=2.837, P=.05). Paired comparisons with Bonferroni correction were performed across the different levels of intensity for each segment. As shown in Figure 4, there were no significant differences between any of the four exercise intensities for segment 1. Significant differences existed between the 110% and 145%, and 110% and 160% intensities for segment 2.

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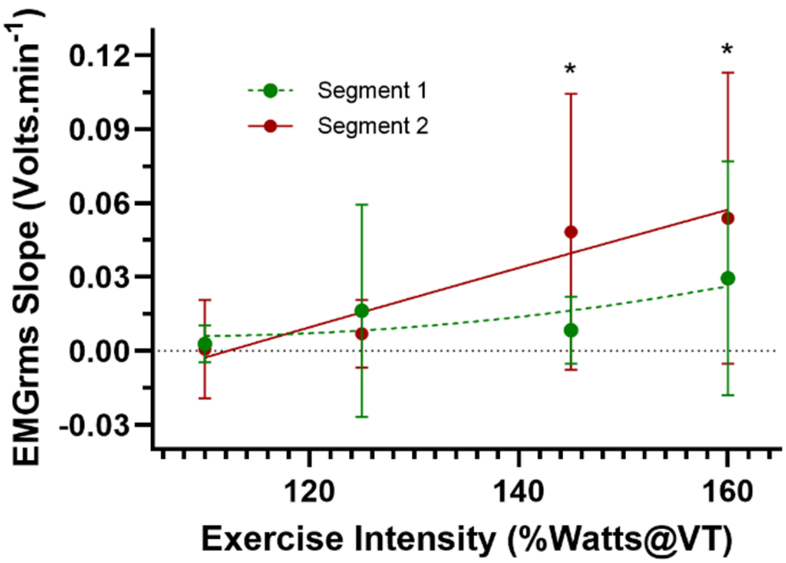

Figure 4. The slope profile of segment 2 (final 30s of the trial) and segment 1 (best fit linear segment preceding the final 30s) during the 4 critical power (CP) trials (110%, 125%, 145%, 160% of ventilation threshold) for the vastus lateralis (VL). * = significantly different from 110% bout.

Discussion

Summary of results and their overall implications

The purpose of this study was to investigate whether sEMG activity revealed sustained increases in rms sEMG signal intensity to volitional fatigue during constant load non-steady state exercise (CP trials) and the implications for the CGM. The VL during the 4 CP trials (Figure 2) revealed an increase in sEMG rms signal strength during the final 30 seconds of exercise across all participants. This is also consistent with the findings during the VO_2max trial, which also revealed increases in sEMG rms signal strength when exercising to volitional exhaustion. This data suggests that neural output from the brain to innervate motor units continues to increase to the point of volitional exhaustion.

Cardio-pulmonary data to reveal the high exercise intensities attained for each bout

Evidence shown in Tables 1 and 2 demonstrates that the VO_2max and CP trials were all performed at a very high intensity with participants who were highly trained. The VO_2max trial revealed a mean

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VO_2max of 60.64±9.2 ml·kg^{-1}·min^{-1} with the highest subject VO_2max value of 76.84 ml·kg^{-1}·min^{-1}. A study by Lamberts[22] gathered data on 82 trained to elite male cyclists and 20 trained to elite female cyclists from previous studies involving an incremental protocol. This data presented mean VO_2max values of 57.5±6.4 ml· kg^{-1}·min^{-1} for males, and 50.5±3.4 ml· kg^{-1}·min^{-1} for females, which further demonstrates the highly endurance-trained status of the subjects from this study.

Further values, such as RER and peak F_{br}, are strong indicators of cardiopulmonary demands during an exercise bout to exhaustion.[23][24][25] It is evident from the data available that the participants performed maximal efforts and are comparable to other studies that have measured RER and peak F_{br} during maximal efforts on a cycle ergometer.[23][24][25] This further strengthens the sEMG rms data collected to suggest maximal efforts to volitional fatigue were performed by the participants.

sEMG results during the VO_2max trial in relation to the CGM

The findings within this study provide significant results that were inconsistent with predictions made from the CGM. These findings indicate continued increases in sEMG rms activity throughout the duration of a maximal exercise bout to volitional fatigue. This can be observed within Figure 2, which displays sustained increases in the raw sEMG rms data for the VL and Gmax muscles. These results were also evident in the slope data for segment 1 and segment 2 of the sEMG rms signals during the VO_2max incremental protocol (Figure 3). For example, the sEMG rms data within the final 30 seconds of the protocol (segment 2) revealed significantly greater sEMG rms output in comparison to the data captured prior to the final 30 seconds (segment 1). This was observed in both the VL and Gmax muscles with no significant interaction between the two muscles, suggesting similar patterns in muscle sEMG activity between these muscles and involvement within maximal cycling bouts. Scheuermann et al.[9][26] also demonstrated continual increases in sEMG rms data through both slow and fast ramp protocols, in addition to extended bouts of intense constant load exercise. At exhaustion, no difference in the sEMG rms/work rate ratio between both protocols was observed, and sEMG rms relative to the increase in work rate was discovered to increase linearly or curvilinearly above a participant's lactate threshold.[9][26] This can be coupled with another study by Camata et al.[12] who also demonstrated significantly greater sEMG rms data from the VL, VM, and RF muscles at the end stage of an incremental protocol.

sEMG results during the CP trials in relation to the CGM

sEMG rms data acquired during the CP exercise trials were also inconsistent with predictions made from the CGM, where the segment 2 sEMG rms slope for the VL was significantly higher than the 110% bout for the 145 and 160 %Watts@VT bouts. These results suggest that there is no subconscious central inhibition to muscle activation as proposed by the CGM. The fact that these results were consistent across incremental exercise and multiple bouts of different duration intense exercise provides added emphasis on the repeatability of this response.

A recent study investigated the link between the degree of peripheral fatigue (change of maximal voluntary contraction and potentiated twitch force post-exercise), watts, and muscle activation (sEMG rms) during severe exercise bouts above a participant's CP.[27] Results found no correlation between the change in maximal voluntary contraction (MVC) and the total watts of an exercise bout but noted a faster rate of change in potentiated twitch force with increases in sEMG rms during higher intensity bouts.[27] Suggestions can be made that a greater degree of peripheral fatigue from increases in muscle activation and decreases in potentiated twitch force are consistent with higher recruitment and fatigue of motor units.[27] Regardless, considerable research and commentary[7][13][28][29][30] on the difficulty of how to interpret surface electrode EMG (sEMG) necessitate concern for interpreting changes in motor unit recruitment from this method. For this reason, we simply refer to muscle sEMG activity.

The prior results of Ducrocq and Blain[27] are similar to the significantly greater slopes displayed in Figures 3 and 4 between segment 2 and segment 1 and suggest that the more intense an exercise bout is, the greater the degree of peripheral fatigue and the need for increased neural drive to maintain power output. It is likely that with shorter duration, higher intensity exercise bouts, a greater/faster recruitment of fast-twitch muscle fibers is required to maintain power output. Due to the nature of these muscle fibers, higher rates of ATP hydrolysis lead to a more rapid accumulation of metabolites (Pi, La^-, K^+, H^+) associated with peripheral fatigue and as such lead to increases in muscle activation[27] (sEMG rms). Another recent study investigated peripheral components of muscular fatigue during constant load severe intensity exercise[31] (above CP). This study found that regardless of exercise duration and work rate, exercise limitation was associated with low values of muscle PCr, ATP, and pH, and high values of [La-], [Pi], and [H^+].[31] To continue, a strong correlation between sEMG rms data and the changes in muscle metabolites was observed and was consistent with the concept that greater central mechanisms are required to compensate for the development of peripheral fatigue.[31] Therefore, the data acquired during this study are consistent with previous findings of increased sEMG rms amplitude during constant load maximal intensity exercise, which collectively refute predictions based on the CGM.

Limitations

Whilst analysis of the amplitude (rms) of sEMG signal data represents a global picture of all active motor units, there are inherent limitations in using this method of analysis. Prior research has shown poor correlations between the patterns of motor unit recruitment and the amplitude (rms) of sEMG signals.[32] sEMG signals are widely used to assess the neural drive of contracting muscles, but it remains unclear as to the underlying contribution of changes in motor unit recruitment to the signal amplitude measured from sEMG signals.[30][32] This therefore suggests that using sEMG sensors remains an invalid tool for interpreting individual motor unit recruitment; however, this methodology remains the best tool to quantify global changes in the neural activation/electrical activity of skeletal muscle during dynamic exercise.

The data collected and used within this study were collected from only the VL and Gmax muscles, and data were unable to be used from the bicep femoris and medial gastrocnemius muscles due to poor signal resolution. Further research should investigate the contribution of the other quadricep muscles (vastus medialis, vastus intermedius, rectus femoris) to provide a greater understanding of the neural drive from all the quadricep muscles during exhaustive rides to volitional fatigue. Furthermore, even though the medial gastrocnemius data were complex and unable to be used in this data analysis, more research should be conducted to improve the understanding of the involvement of this muscle in intense cycling exercises.

Finally, given the study was confined to moderately to highly trained cyclists, data can only be generalized to this specific cycling population. However, it can be inferred, due to the nature of the study, that the results are likely to be consistent within multiple other populations.

Conclusions

The muscle sEMG rms data from this study revealed sustained increases in muscle activity in all bouts of intense exercise to volitional exhaustion. Regardless of the difficulty in using sEMG rms data as a reflection of increases in motor unit recruitment, if the CGM were valid, you would expect to see lowered sEMG rms due to constrained CNS neural output to the contracting skeletal muscles.

An explanation of contractile failure that may best fit the findings observed within this study is that through the different recruitment profiles of motor units in human muscle, the progressive increase in fast twitch motor unit recruitment induces intracellular metabolic conditions (e.g., increased intramuscular Pi) that directly contribute to contractile failure. In other words, the need to use fast twitch motor units during intense muscle contractions, and their related fatigability, means that such neuromuscular function is a built-in failure mechanism that prevents our capabilities from inducing structural and systemic damage during intense exercise.

Statements and Declarations

Submission Statement – All authors have read and agree with the manuscript content. In addition, while this manuscript is being reviewed for this journal, the manuscript will not be submitted elsewhere for review and publication.

Ethical Approval Statement – All research procedures were approved by the QUT University Human Research Ethics Committee (Ethics approval number 4252).

Authors Contribution Statement – Robergs conceived the idea and assisted in most data collection and data processing. O'Malley performed all data collection and assisted in data processing. Holmans performed data processing of critical power data and related EMG data, as well as assisted Robergs in the writing of the manuscript. All authors were involved in the proofreading and editing of the manuscript.

Conflict of Interest Statement – None of the authors have direct or indirect interests that are in direct conflict with the content of this manuscript.

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Declarations

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