Relationship between fatigue and EMG activity in triceps surae during isometric plantar flexion

（等尺性足底屈における下腿三頭筋の疲労と筋電活動の関係）

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概要

背景： 最大あるいは亜最大下での筋力保持の筋線維動員パターンと疲労についての研究は多い。しかし、筋線維動員パターンと疲労との関係をみた研究は少ない。そこで、本研究の目的は、最大筋力（MVC）までのランプ収縮と持続的な MVC 発揮時の下腿三頭筋の運動単位(MU)動員パターンから、中枢性疲労と末梢性疲労の関係を筋電図学的に明らかにする。

方法： 対象は被験者 14 名（男性 10 名、女性 4 名：34±13 歳, 169±9cm, 64±15kg; mean ±SD)とし、東邦大学倫理委員会（受付番号：23042）の承認を得て実施した。実験Ⅰと実験Ⅱの被験者は 14 人と 5 人で行った。実験Ⅰのプロトコルは、足底屈の等尺性収縮を用いて、15 秒間のランプ MVC 運動の力発揮と 60 秒間の最大等尺性収縮（MVC）を行った。実験Ⅱのプロトコルは、間欠的な MVC とし、5 秒収縮－5 秒弛緩で 24 回の反復運動を行った。実験ⅠとⅡの実験姿勢は、膝関節屈曲 90° 位、足関節底背屈 0° 位とした。筋電図は、ヒラメ筋（Sol）と腓腹筋（MG と LG）から、双極誘導とした。解析は、2 乗平均平方根（rmsEMG)、平均パワー周波数(MPF)、M 応答、H 反射、V 応答、silent period、voluntary activation (VA)、静脈血乳酸を測定した。また、実験Ⅰのランプ力発揮課題では、力発揮と rmsEMG の関係から変局点を（Break Point;BP）算出した。

結果： 間欠的な最大筋力時の V 応答は有意に減少し($p < 0.01$)、MVC 保持の VA と張力減少には関係がなかった。間欠的 MVC の VA の力積量には有意な関係が認められた($p < 0.05$)。60 秒間の MVC 後 potentiated twitch と静脈血乳酸値が有意に増加した($p < 0.0001$;$p < 0.01$)。また 60 秒間の MVC 保持における力発揮の減少率と BP の rmsEMG は MG にのみ相関傾向が認められた($p = 0.056$)。

考察: 個々人の中枢性ドライブと MU の動員は BP に関係していることが示唆された。また MU の動員と調節（rate coding）は BP と関係していると考えられる。60 秒間の

MVC 保持課題で、V 応答の振幅の減少と twitch potentiation （superimposed and resting twitch force）の増加から、中枢性疲労の早期発現が認められた。この中枢性疲労は、筋代謝性の酸性化による求心性入力の影響を受けた可能性が示唆された。

結論： 本実験から、60 秒保持した MVC と間欠的の MVC の張力減少は、筋性要因よりも神経性要因の機序に関係していることが明らかとなった。また MVC 保持の下腿三頭筋の疲労とランプ力発揮は、MG の MU の動員パターンに相互依存性のあることが示された。

ABSTRACT

Introduction: Muscle recruitment patterns and fatigue during sustained maximal voluntary contractions (MVCs) are well documented however the number of studies investigating the relationship between these two factors are few. Therefore, the purpose of this study was to investigate the relationship between central and peripheral fatigue during sustained maximal voluntary contraction (MVC) and the EMG activity of muscles of the triceps surae during ramp contraction to MVC.

Methods: Fourteen subjects (10 male and 4 female: 34 ± 13 years, 169 ± 9 cm, 64 ± 15 kg; mean ± SD) participated in 2 experiments approved by the local ethics committee (No. 23042). Experiment I comprised a 60 s maximal voluntary isometric plantar flexion of the triceps surae, a 60 s 50% MVC and a 15 s ramp contraction to MVC. Five male subjects from Experiment I performed 24 intermittent 5 s MVCs each followed by 5 s rest in Experiment II. Knee and ankle joints were fixed at 90 and 0 degrees flexion, respectively. Surface electromyographic (EMG) activity was recorded from the soleus (Sol) and medial and lateral heads of the gastrocnemius muscle (MG and LG). rmsEMG, MPF, H-reflex and M and V responses, silent period, voluntary activation (VA), blood lactate concentration and rmsEMG break points (BPs), defined as the point of change in the force slope versus rmsEMG, of the Sol, MG and LG during ramp contraction, were analysed in each subject.

Results: The amplitude of the V response declined during the sustained MVC task and significantly declined during the intermittent MVC task ($p < 0.01$). There was no relationship between VA and force decline during the sustained MVC however there was a significant relationship between VA and decline in impulse during the intermittent MVC task ($p < 0.05$). Potentiated twitch significantly increased after the 60 s MVC ($p < 0.0001$) along with blood lactate concentration ($p < 0.01$). A near significant correlation between the average rate of force decline, during sustained MVC, and the BP, only in the MG, during

ramp contraction, was found ($p = 0.056$).

Discussion: BPs represent individual neural drive patterns and a unique capacity to innervate motorneurones and recruit motor units (MUs) and may reflect a change in MU recruitment and/or rate coding. Central fatigue was observed during the sustained MVC task, highlighted by decreases in V response amplitude and voluntary activation, lengthening of the silent period and increases in superimposed and resting twitch force (twitch potentiation). This central component of fatigue was also likely to have been influenced by group III and IV afferents via spinal and supraspinal pathways.

Conclusion: These results suggest that neurogenic rather than myogenic factors are responsible for the decline in force during a sustained maximal plantar flexion and intermittent MVCs and that the fatiguability of the triceps surae, during sustained maximal isometric contraction, and the recruitment pattern of the MG, during isometric ramp plantar flexion to MVC, are interdependent.

CONTENTS

INTRODUCTION

Ever since Angelo Mosso's famous work entitled 'Fatigue' was published in 1904 [1], scientific researchers have debated whether the origin of fatigue is peripheral or central in nature. In his work, Mosso [1] talks on areas as diverse as "The migration of birds" and "The history of the study of the movements of animals to "The methods of intellectual work" and "Overpressure". The one theme that prevails and links all of his discourses is the phenomenon of fatigue and its various manifestations. Chapter IV of Mosso's book, 'Upon the General and Special Characteristics of Fatigue', describes his various investigations into fatigue, in particular his experiments looking at fatigue of the flexors of the middle finger and the 'ergograph' which he designed and built to record his findings. Mosso [1] discovered differences in the fatigue curves of humans, in vivo, compared to those in frogs, in vitro, explained in previous experiments by Hugo Kronecker [1]. He also found a difference in fatigue curves between subjects, and differences in the same subject, depending on factors such as levels of exercise and age. Mosso [1] alluded to central and peripheral components of fatigue by conducting experiments which "eliminated the mental element which might alter the fatigue curve of the muscle…by stimulating the nerve of the arm, or rather the flexor muscles of the finger." In this way "fatigue of both brain and nerves was excluded". Over 100 years have passed since Mosso [1] invented the ergograph and our understanding of fatigue and the methodologies we employ to investigate the phenomenon have come a long way. We know a lot more about the intricacies of central and peripheral fatigue but researchers all over the world are still trying to determine the causes of fatigue and the relative roles central and peripheral factors play in fatigue.

So what is 'fatigue' and what does 'peripheral' and 'central' fatigue mean? The concept of fatigue is complex because the perception of fatigue is subjective and there is no one all encompassing definition of fatigue. One clinical definition of fatigue is: "difficulty

in initiation of or sustaining voluntary activities" [2]. According to neuromuscular physiologists, fatigue, and this is by no means the only definition but probably one of the most accepted, is "any exercise-induced reduction in the ability of a muscle to generate force or power that has central and peripheral causes" [3~4]. Peripheral fatigue refers to "exercise-induced processes that lead to a reduction of force production and that occur at or distal to the neuromuscular junction" [3), 5)]. Central fatigue refers to processes proximal to the neuromuscular junction and can be defined as "a progressive exercise-induced failure of voluntary activation of the muscle" [3), 5)]. Central fatigue can be responsible for 20-25% of the loss of force under certain conditions [6)]. Supraspinal fatigue is now also recognised as a component of central fatigue. It can be defined as "a loss of force caused by suboptimal output from the motor cortex" [6)].

The recruitment of motor units (MUs) in the contraction of muscles has been proven time and again to follow the laws of Henneman's Size Principle, especially in isometric contractions [7~10)]. Henneman's theory suggests that MUs are recruited in the order of those with motor nerves of small diameter to those of larger diameter. Motor nerves with small diameters innervate slow, Type I, muscle fibres and nerves with larger diameters innervate fast, type IIA and IIB, muscle fibres. Therefore, motor units are recruited in order from those with a low activation threshold to those with a higher activation threshold. Exceptions to this rule have been shown to occur in certain nonisometric contrations [3), 11~16)] for example, in the case of a cat paw swipe [17)] where there is a reversal in the order of MU recruitment.

Previous studies have shown that the root mean square of the surface electromyography (rmsEMG) and mean power frequency (MPF) decline in conjunction with force during sustained isometric contractions [18~21)] and these results coincided with declines in intramuscular spike amplitude and frequency [18~23)]. However, during submaximal MVCs, significant declines in MPF were accompanied by increases in rmsEMG [4), 18~21), 24~26)]. During isometric muscle contractions, rmsEMG has been shown to increase both linearly [27)]

and non-linearly [28~29]. In examples where there is a sudden increase in rmsEMG as force increases, this point of change can be calculated as a deviation in linearity in the regression curve. We have defined this point as the rmsEMG Break Point (BP). This BP has also been described as the EMG Threshold ($EMG_{T(h)}$) in the literature, especially in studies investigating the phenomenon in cycling ergometer tests, and has been shown to be a reliable indicator of neuromuscular fatigue in the vastus lateralis muscle [30~32]. As mentioned above, the relationship between surface and intramuscular EMG and the increase in force during ramp contractions to MVC and the decline in force during sustained fatiguing contractions is well known. However, the relationship between the individual recruitment patterns, represented by the BP in muscles of the triceps surae, during voluntary isometric ramp contraction to MVC and the individual fatigue patterns, represented by the average rate of force decline during sustained maximal voluntary isometric contraction, has yet to be investigated. Therefore, the purpose of this study was to investigate the relationship between these two factors with the hypothesis that a relationship would be found between the ramp BPs of the gastrocnemius, medial (MG) and lateral (LG) heads, but not the soleus (Sol), and sustained MVC force rate decline, due to the anatomical and physiological differences, such as size, makeup and neural control, of these muscles.

METHODS

Setup

Fourteen healthy subjects, 10 male and 4 female (34 ± 13 years, 169 ± 9 cm, 64 ± 15 kg; mean ± SD), took part in experiments to examine EMG activity and force output, along with responses to electrical stimulation of the tibial nerve, during ramp and fatiguing voluntary isometric contractions of the triceps surae. All procedures were approved by the local ethics committee (No. 23042) and carried out in accordance with the Declaration of Helsinki with the informed consent of the subject which was gained after a short orientation session held before the experiment.

Subjects sat in a custom built chair with the right leg and ankle flexed at 90 degrees and 0 degrees, respectively. The knee was fixed in position from above and the foot was strapped, across the ankle, to a plate that was connected to a force transducer (Shinkoh - Type U3B1-100K-B) that measured plantar flexion torque. To limit upper body movement, a 'seatbelt' was strapped across the subjects' waists and their shoulders were fixed in position with purpose built apparatuses. Feedback of plantar flexion torque was provided to subjects by way of an X-Y pen recorder (San-ei, 8U16).

Cutaneous electrical stimuli (1 ms square pulse) were delivered to the tibial nerve using a stimulator (Dia Medical System, DPS-1300) via an isolator (Dia Medial System, DPS-105D). The anode electrode was attached to the surface of the skin in the popliteal fossa and held in place securely with surgical tape after the optimal stimulation site was located by a hand held probe [33]. The cathode was placed about 10 cm proximal to the anode on the outer thigh. It was also ensured, visually and by palpation, that the tibialis anterior muscle was not simultaneously activated.

Protocol

In Experiment I each subject (n=14) took part in two sessions, performed on a different day one or two days apart. In each session, subjects initially performed two or three brief, 2 or 3 second, maximal voluntary isometric plantar flexions with the right leg. Each MVC was separated by a rest period of at least 1 minute. In one session, a 50% MVC lasting for 60 seconds was then performed followed by a 60 second MVC after 30 minutes of rest. This rest period was chosen to allow for maximal recovery from the 50% MVC without extending the experimental period too long, reducing the chance of discomfort for the subjects. The duration of contraction was chosen to enable examination of the time course of the changes in EMG and EMG responses to tibial nerve stimulation, along with the changes in force output.

During each 60 second contraction, four supramaximal cutaneous electrical stimuli, 135 volt intensity, were given at 15 second intervals after the start of contraction to elicit corticospinal, maximal M, V and superimposed twitch responses. In the other session, a 15 second ramp contraction from 0% MVC to MVC was performed with supramaximal cutaneous electrical stimuli being given every 3 seconds after the start of contraction (5 times total during contraction). Five resting maximal H-reflex and M responses (control twitch) were elicited before each contraction and 5 maximal M responses (potentiated twitch) were elicited after each 60 second sustained MVC and 1 maximal M response (potentiated twitch) was elicited after the 15 second ramp contraction to MVC (Figures 1 and 2).

Voluntary activation (VA), defined as “the level of voluntary drive during an effort” [3], was measured using a twitch interpolation technique [3, 34~38]. The methods used to determine interpolated twitch amplitude and the formula used to calculate VA are shown in Figure 2.

Blood lactate concentration was also measured before (control) and 2 minutes after each contraction in both sessions using a blood lactate test meter and test strips (Arkray, Inc. Lactate Pro LT-1710 and Arkray, Inc. Lactate Pro Test Strip, 78101, respectively).

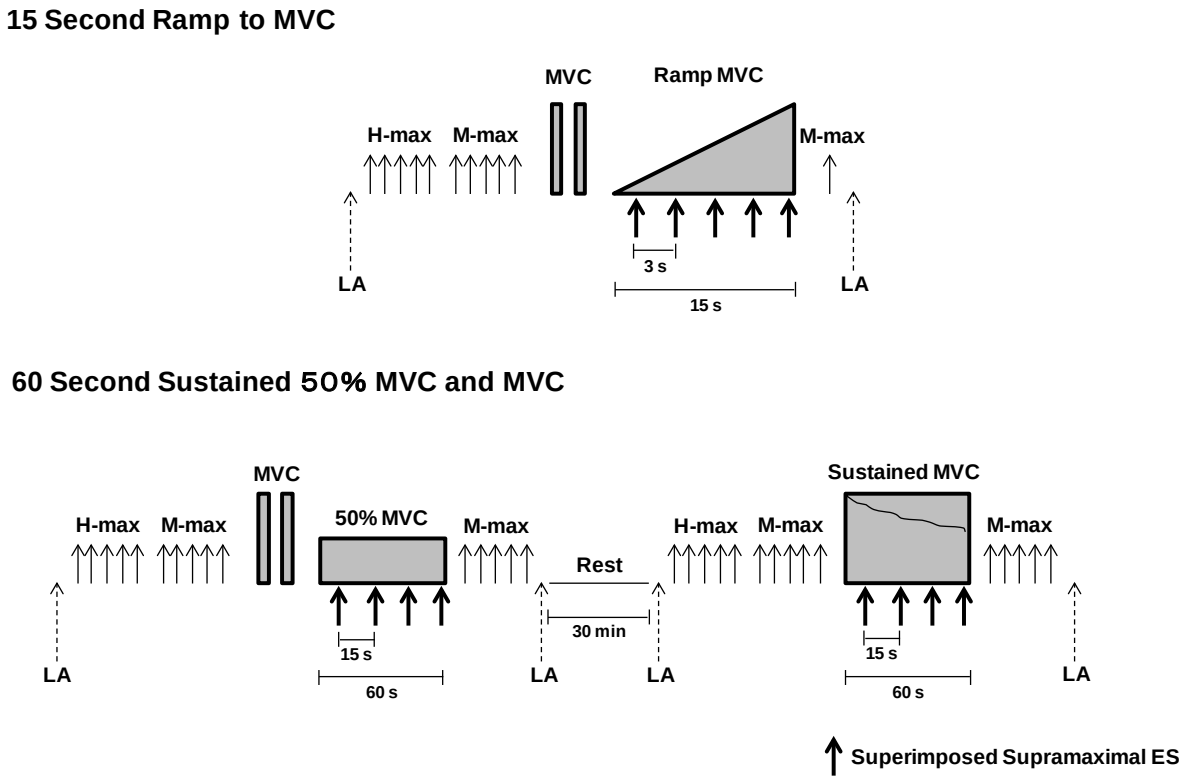
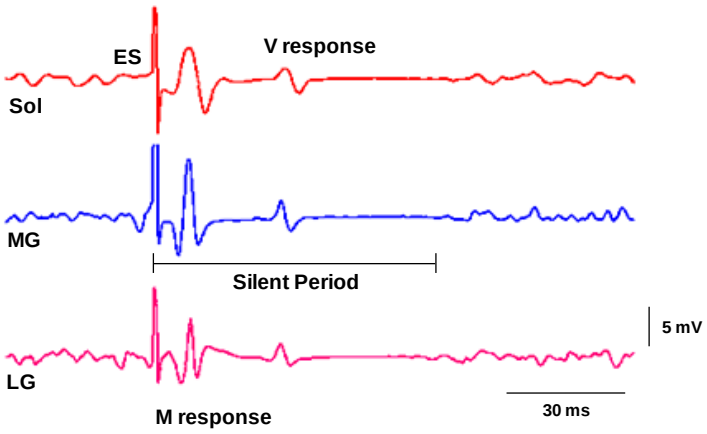
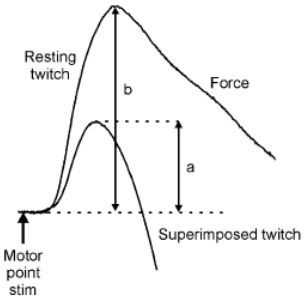


Figure 1. A schematic presentation of the protocols performed in Experiment I. The upper diagram represents the 15 second ramp to MVC and the bottom diagram represents the 60 second sustained 50% MVC and the 60 second sustained MVC. Arrows represent electrical stimuli to evoke maximal resting H-reflexes, M responses and twitch force. The bold arrows represent superimposed supramaximal electrical stimuli (ES) to evoke corticospinal (M and V responses) and superimposed twitch responses and the dotted arrows represent blood lactate (LA) measurements.





voluntary activation (%) =
[1 – (superimposed twitch/control twitch)] × 100

average of 5 control twitches

Adapted from Todd, Taylor and Gandevia, *J Physiol* (2003)

Figure 2. Typical corticospinal responses from the Sol, MG and LG in one subject (top). Interpolated twitch method (bottom left) and formula used to calculate voluntary activation (bottom right) in this study. Note that the control twitch was the average of five control twitches elicited before the 60 second sustained MVC.

In Experiment II (n = 5) the setup was more or less the same as Experiment I however the protocol was changed and subjects were asked to perform 24 intermittent MVCs. Subjects performed a 5 second MVC, followed by 5 seconds rest, 24 times continually. At the point where MVC was reached and maintained during the 5 second MVC, a supramaximal electric stimulus was applied to the tibial nerve to elicit M and V responses, along with a superimposed twitch, as in Experiment I (Figure 3). During the 5 second rest period, after each MVC, another electrical stimulus of the same intensity was delivered to elicit a maximal M response and twitch force (potentiated twitch). Impulse (Ns) was determined as the area under the force curve during each 5 second MVC (Figure 4).

Another factor that was different in Experiment II was the formula used to calculate voluntary activation, which used the potentiated twitch amplitude as the denominator (Figure 3). Blood lactate levels were not recorded in Experiment II. In both Experiment I and II strong verbal encouragement was given to all subjects, especially toward the end of the respective fatigue tasks.

$$\text{Formula of two VA}: \quad VA_P = 1 - \frac{(\text{superimposed twitch amplitude})}{(\text{potentiated twitch amplitude})} \times 100$$

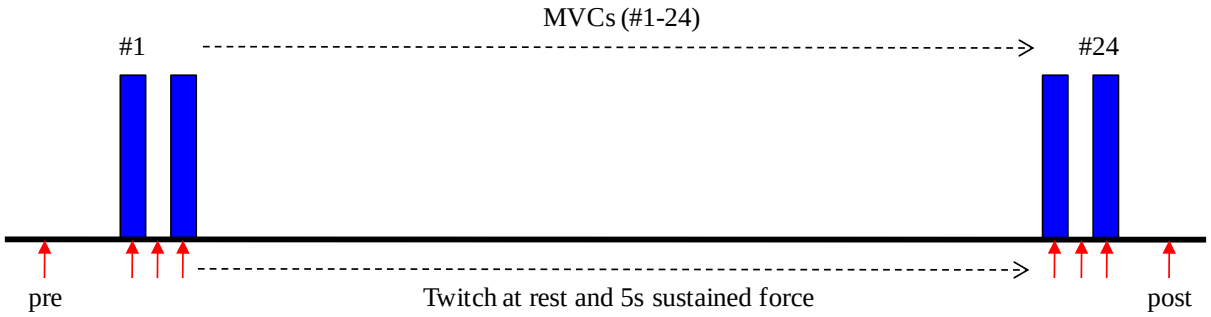


Figure 3. The formula used to calculate voluntary activation during the intermittent MVC fatigue task in Experiment II (top). Schematic presentation of the intermittent MVC fatigue task (bottom). Arrows represent supramaximal eletrical stimuli to evoke maximal M and resting, interpolated and potentiated twitch responses. Filled columns represent 5 second sustained MVCs.

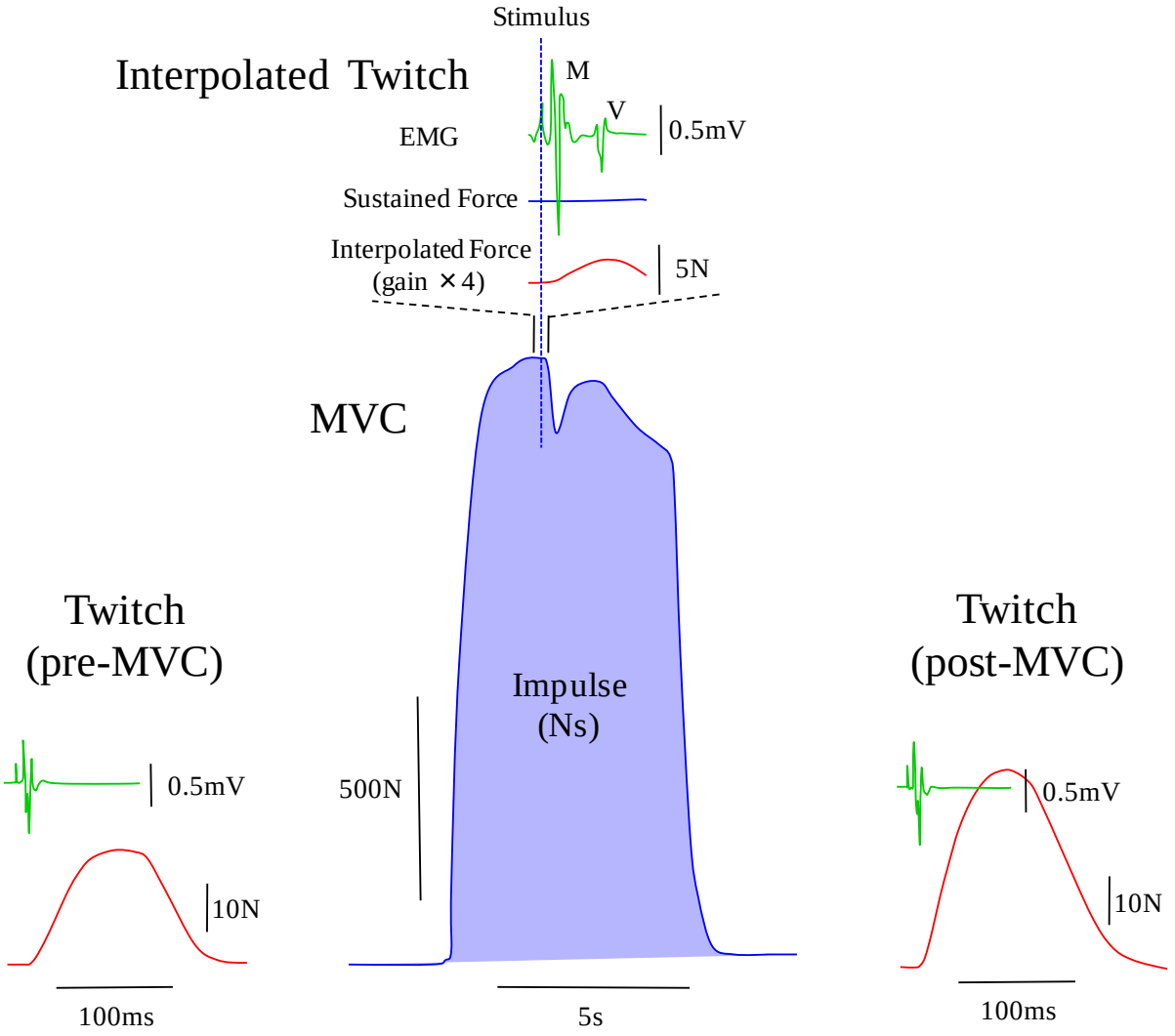


Figure 4. Recordings of twitch force, EMG, and sustained MVC force from one subject at the beginning of the intermittent MVC fatigue task. The insets (upper) present the M and V responses and interpolated twitch force (gain x4) during one 5 second MVC.

Surface Electromyography

EMG was recorded from Sol, MG and LG by means of surface electrodes [10mm diameter Ag/Ag, (n=14) and 5 mm diameter Ag/Ag miniature electrodes (n=4) (Unique Medical)] in Experment I. In Experiment II only miniature electrodes were used. The skin was abraded with fine sandpaper to reduce impedance and the electrodes were fixed in position over the belly of the respective muscles with surgical tape. The electrode wires were also taped to the body to reduce the possibility of noise interference. EMG and torque signals were amplified (NEC San-ei, 1253A and NEC San-ei, NQ224, respectively) and digitised at 10K/s (ADInstruments, Powerlab) and recorded onto a hard disk for off-line analysis using ADInstruments Chart 7, a computer software package.

Data Analysis

All analysis, including peak to peak amplitude of the M, H-reflex and V responses, rmsEMG and MPF (Fast Fourier transform with the Hamming window), silent period, force and interpolated twitch force, and regression analysis to determine ramp BPs and correlation coefficients, were calculated using ADInstuments, Labchart 7 and Microsoft Excel for Windows 2007. The force signal was also digitally low pass filtered, with a cut-off frequency of 50 Hz, when extrapolating twitch amplitude in Labchart 7.

Statistical Analysis

Computer software, StatView-J 5.0, was used to determine p values (regression analysis, one-way repeated measures ANOVA and paired t-tests). Statistical significance was set at $p < 0.05$.

RESULTS

Experiment I

Figure 5 is the typical data of one subject showing the force output (upper panel) and the rectified EMG for the Sol, MG and LG muscles (lower 3 panels) during the 15 second ramp contraction to MVC (left) and the 60 second sustained MVC (right). Rectified EMG increases non-linearly with force in all three muscles during the ramp contraction and decreases along with force in all three muscles during the fatiguing contraction.

The normalised force, in relation to each subject's MVC, and normalised rmsEMG, in relation to the maximal rmsEMG during the ramp contraction and sustained MVC, are represented as the average ± SD for all subjects in Figure 6. Again, as a whole, a gradual increase in normalised force during the 15 second ramp to MVC was accompanied by gradual increases in the normalised rmsEMG in the muscles of the triceps surae in relation to time. A gradual decline in normalised force during the 60 second sustained MVC was accompanied by gradual decreases in normalised rmsEMG in the muscles of the triceps surae in relation to time. Force and rmsEMG during ramp and sustained contractions were normalized to account for differences in the total force output among subjects in order to enable accurate comparison of data sets.

The time course of changes of MPF during the 15 second ramp to MVC (left panel) and the 60 second sustained MVC (right panel) are shown in Figure 7 (n = 14). During the ramp contraction MPF gradually increased for the first half of the contraction then declined during the second half of the contraction. This decline ended in levels below those at the beginning of the contraction in Sol and LG. MG levels were slightly higher at the end of contraction compared to the beginning of contraction. On the other hand, MPF levels declined from the beginning to the end of the sustained MVC (right panel).

Figure 8 represents the individual recruitment and fatigue patterns of subjects, showing the force output curves and rmsEMG curves in relation to time during the 15 second

ramp to MVC and the 60 second sustained MVC in 2 subjects. Subject A was relatively 'strong' among the 14 subjects and the maximum force output of Subject B was much lower than Subject A. The maximal force output, measured in a number of short 2-3 s MVCs before the ramp and sustained MVC protocols, in Subject A was 200 kg (1,960 N) and in Subject B was 144 kg (1,411 N). The maximal force output reached by Subject A during the 60 second sustained MVC was 200 kg (1,960 N) and in subject B was 140kg (1,372 N).

Figure 9 shows the % rmsEMG BPs in relation to % MVC for the Sol, MG and LG muscles during the 15 second ramp to MVC in the same two subjects as the previous figure (Subject A and Subject B). The Sol % rmsEMG BP in Subject A was 34% rmsEMG and in Subject B was 53.7% rmsEMG. In MG, the % rmsEMG BPs were 52.3% and 31%, and in LG, 14.3% and 20.6%, in Subjet A and Subject B, respectively. The % rmsEMG BPs were not consistent between muscles of the triceps surae (Sol, MG and LG) in each subject, nor were they consistent between subjects. This phenomenon represents the individuality of the recruitment pattern of MUs among subjects during the 15 second ramp contraction to MVC.

The most important results in this study are presented in Figure 10. This data set shows the relationship between the average force rate decline, during the 60 second sustained MVC, and the % rmsEMG BPs of the Sol, MG and LG, during the 15 second ramp to MVC, in a total of 14 subjects, but for 13 subjects in each muscle (Sol, MG and LG, respectively). No relationship was found between average force rate decline and % rmsEMG BPs in Sol and LG. However, a trend toward a significant relationship between these two factors was found in MG ($r = -0.537$, $p = 0.056$; $p < 0.05$). There are only 13 data points in this data set as no ramp rmsEMG BP was observed in Sol in one subject and in another subject no BPs were observed in the two heads of the gastrocnemius muscle (MG and LG).

Data related to corticospinal responses during the 15 second ramp to MVC and 60 second sustained MVC are summarised in Figure 11 which shows the relationship between normalised force and the ratio between V response and superimposed M response (V/M_{max}) in

the 14 subjects. There is a significant relationship between the increase in the amplitude of the V response and the increase in force during the 15 second ramp contraction to MVC in Sol, MG and LG ($r = 0.681$, $r = 0.668$ and $r = 0.642$, respectively). During the 60 second sustained MVC there is a relationship, however not significant, between the amplitude of the V response and force, both of which decline in conjunction with the other as the contraction progresses.

Figure 12 shows the relationship between the rate of increase in V response (V/M_{max}) amplitude during the 15 second ramp to MVC and the rate of decrease/increase in V response (V/M_{max}) amplitude during the 60 second sustained MVC. A significant relationship was observed in the Sol ($r = 0.669$, $p < 0.01$) and the MG ($r = 0.650$, $p < 0.05$), where subjects with a higher rate of V response amplitude increase during the ramp protocol had a higher rate of V response amplitude decline during the sustained MVC task, however no relationship was observed in the LG. On the other hand, subjects who had a lower rate of V response incline during the ramp protocol had a lower rate in decline of V response amplitude, or an increase in V response amplitude, during the sustained MVC task.

No relationship was found between VA and silent period duration (left panel) or force and silent period duration (right panel) during the 60 second sustained MVC (Figure 13). Figure 14 shows the relationship between force decline and voluntary activation during the 60 second sustained MVC ($n = 14$). No relationship was found between these two factors either. The insert in Figure 14 shows the difference between the control twitch, the average of 5 resting twitches elicited before the 60 second sustained MVC, and the potentiated twitch, the average of 5 resting twitches elicited immediately after the end of the 60 second sustained MVC, in one subject.

The relationship between the control twitch, the average of 5 maximal twitches elicited at rest before the 60 second sustained MVC, and the potentiated twitch, the average of 5 maximal resting twitches elicited after the 60 second sustained MVC, and the average

twitch force is shown in Figure 15. The upper graph in this figure shows the difference between the control twitch and the potentiated twitch in each subject. The lower graph shows the average control twitch and average potentiated twitch in the 14 subjects (mean ± SD; $p < 0.0001$).

During the sustained 50% MVC, when the data was pooled for all 14 subjects, there was a significant correlation between time and normalised rmsEMG in Sol ($r = 0.664$), MG ($r = 0.937$) and LG ($r = 0.695$) with normalised EMG increasing from the beginning of the contraction to the end of the contraction. There was also a significant correlation between time and MPF with MPF declining from the beginning of the contraction to the end of contraction in Sol ($r = 0.973$), MG ($r = 0.933$) and LG ($r = 0.948$) (Figure 16).

Results relating to the analysis of central drive during the 60 second sustained 50% MVC protocol are presented in Figure 17. No significant changes in V response amplitude and silent period duration were observed over the time course of changes during the sustained submaximal contraction in the Sol, MG and LG. The data sets are for all 14 subjects and all data are mean ± SD.

The results of blood lactate measurements are shown in Table 1. There was no significant difference between blood lactate levels before and after the 15 second ramp to MVC and the 60 second sustained 50% MVC. However, a significant increase in blood lactate was observed 2 minutes after the 60 second sustained MVC compared to levels recorded before the fatiguing contraction ($p < 0.01$). The average blood lactate level before the 60 second sustained MVC was 1.94 ± 0.54 mmol/L and 2 minutes after the fatiguing contraction it was 5.26 ± 3.32 mmol/L ($n = 14$).

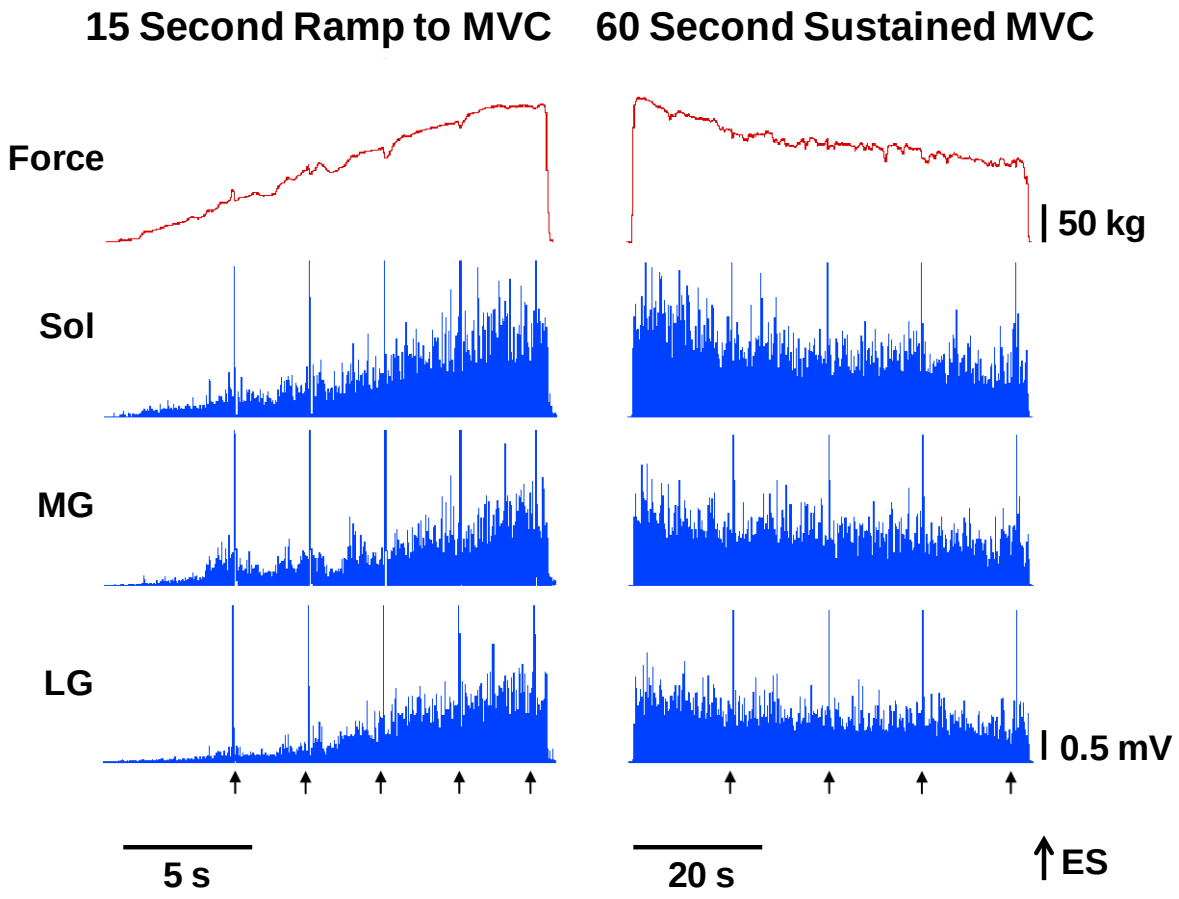


Figure 5. Typical data of one subject from Experiment I. The left panel shows the force trace and rectified EMG for Sol, MG and LG during the 15 second ramp to MVC and the right panel shows the same data during the 60 second sustained MVC. Arrows represent supramaximal electrical stimuli to evoke corticospinal and superimposed twitch responses.

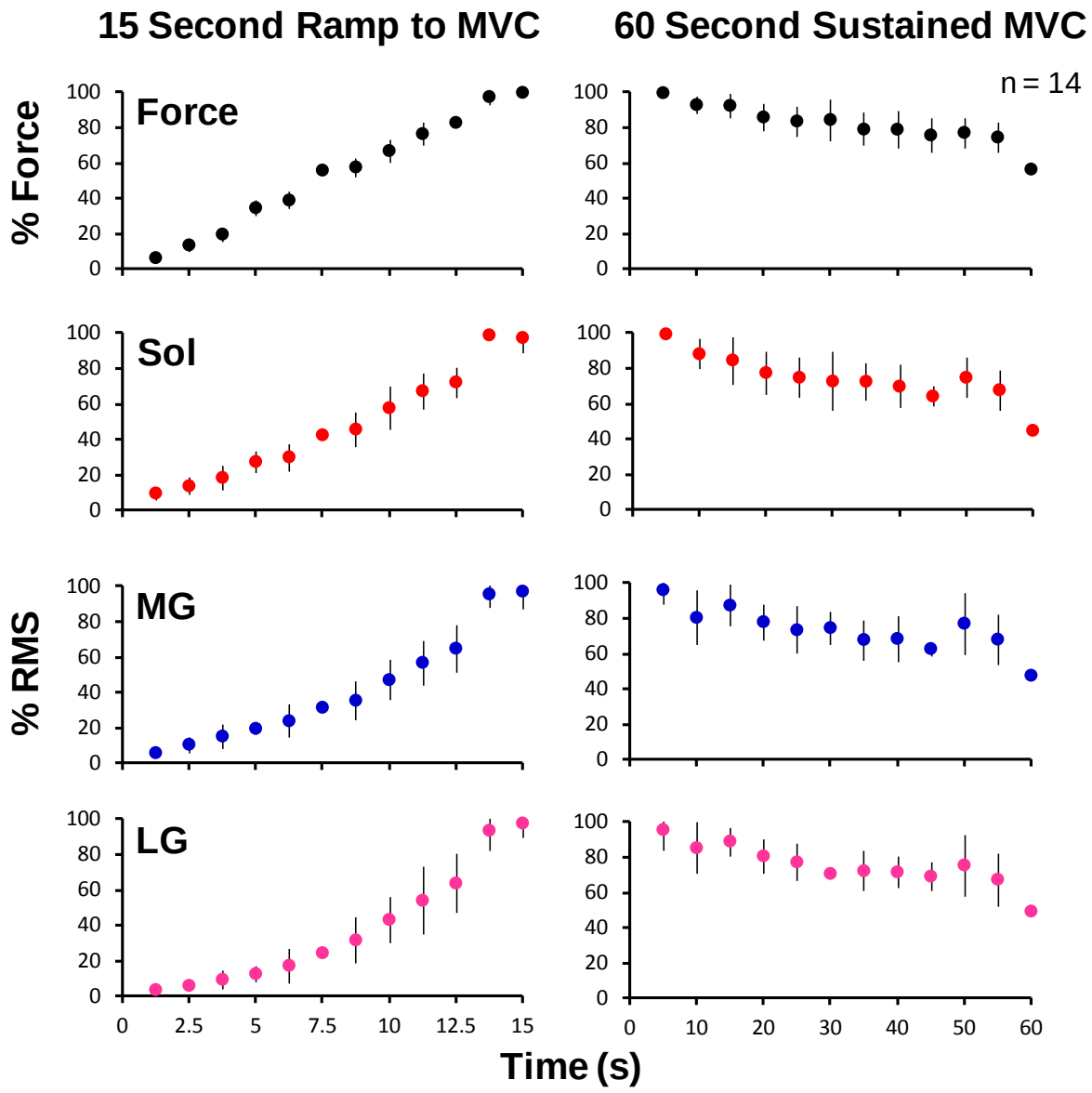


Figure 6. Normalised force and rmsEMG of Sol, MG and LG during ramp (left panel) and sustained MVC (right panel) in 14 subjects. All data are mean ± SD.

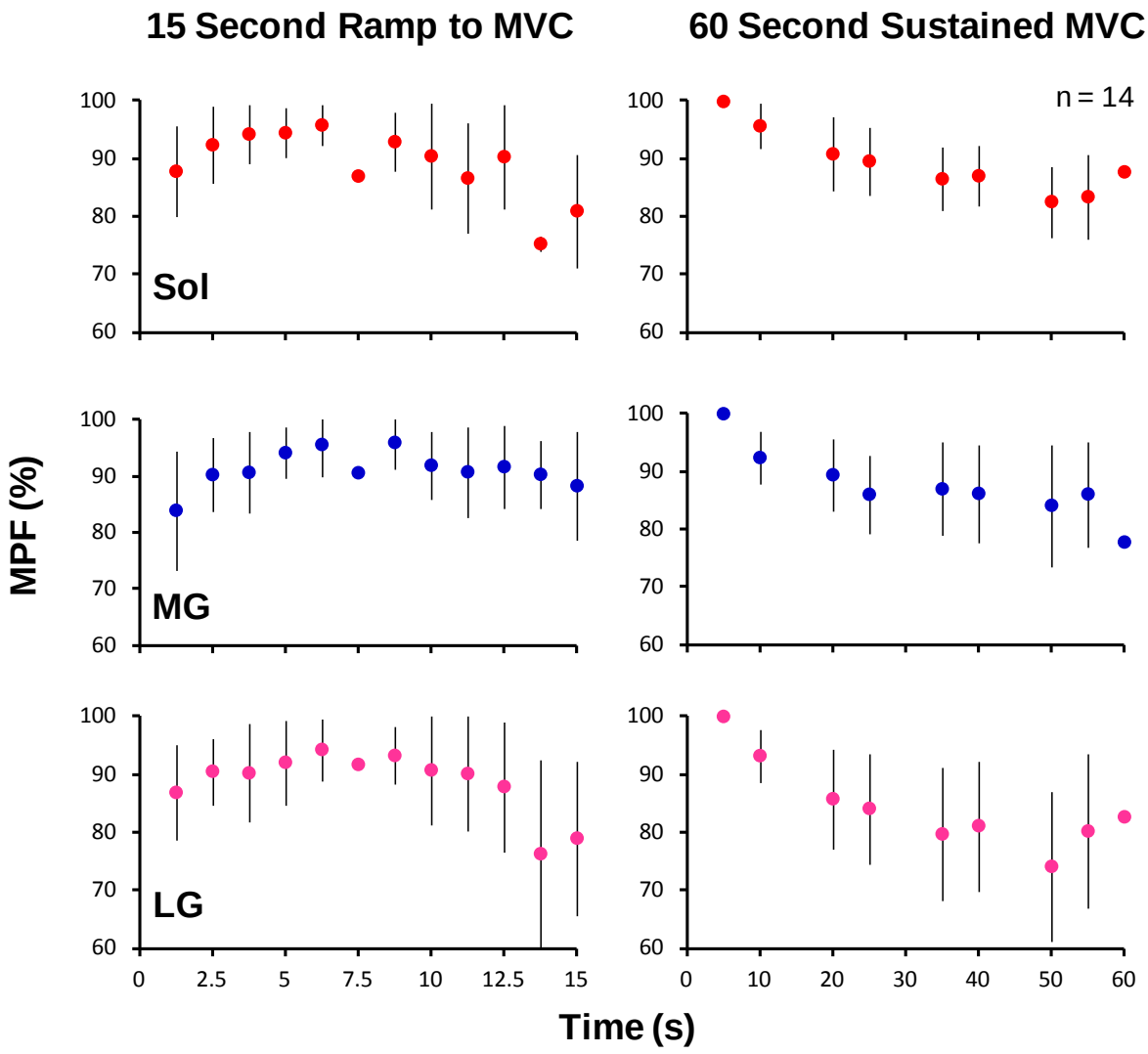


Figure 7. Normalised MPF of Sol, MG and LG during the 15 second ramp to MVC (left panel) and the 60 second sustained MVC (right panel) in 14 subjects. All data are mean ± SD.

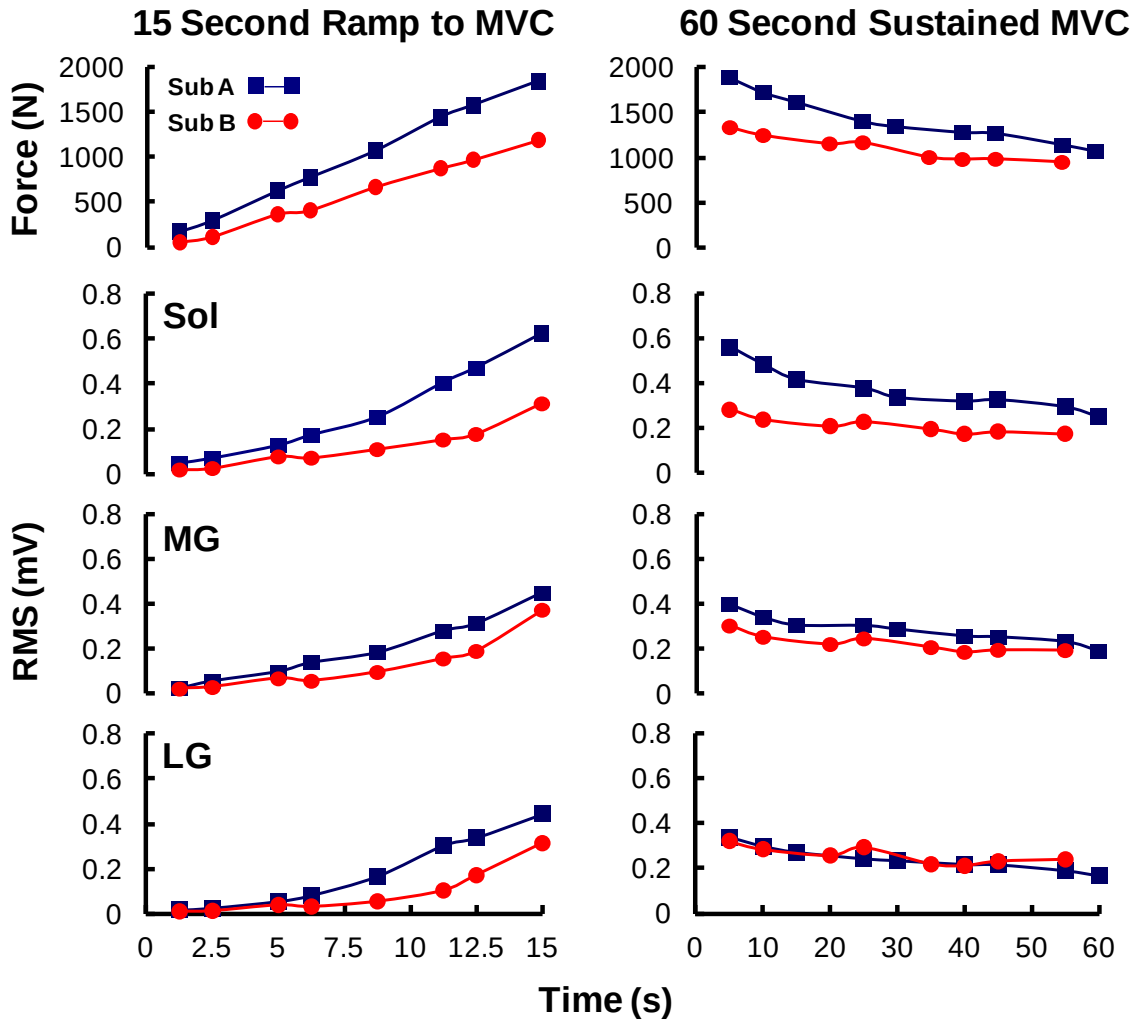


Figure 8. Comparison of force (N) and rmsEMG (mV) of Sol, MG and LG in 2 subjects (Subject A and Subject B) during the 15 second ramp to MVC (left panel) and the 60 second sustained MVC (right panel).

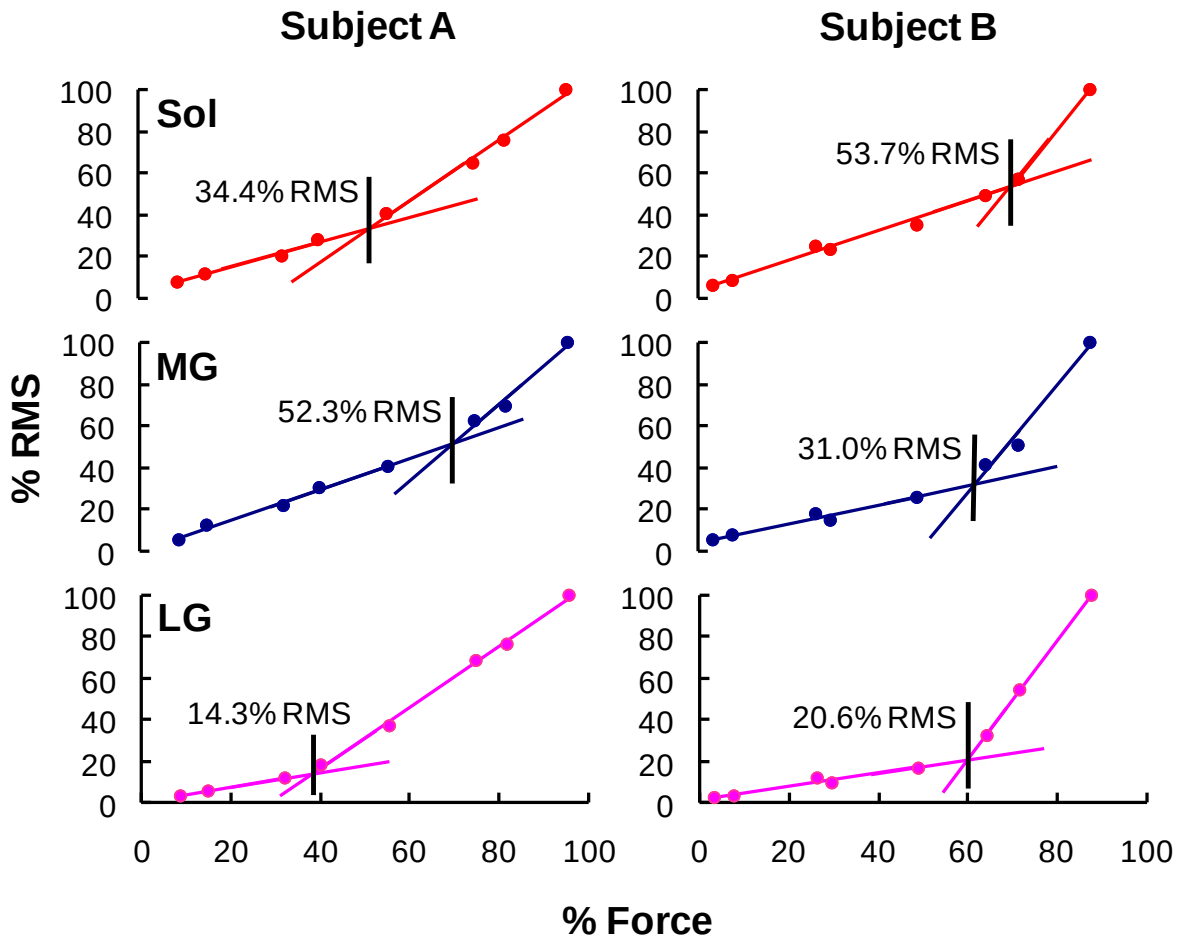


Figure 9. Comparison of rmsEMG BPs of Sol, MG and LG in Subject A and Subject B. On the x-axis is normalised MVC force (%) and on the y-axis is normalised rmsEMG (%).

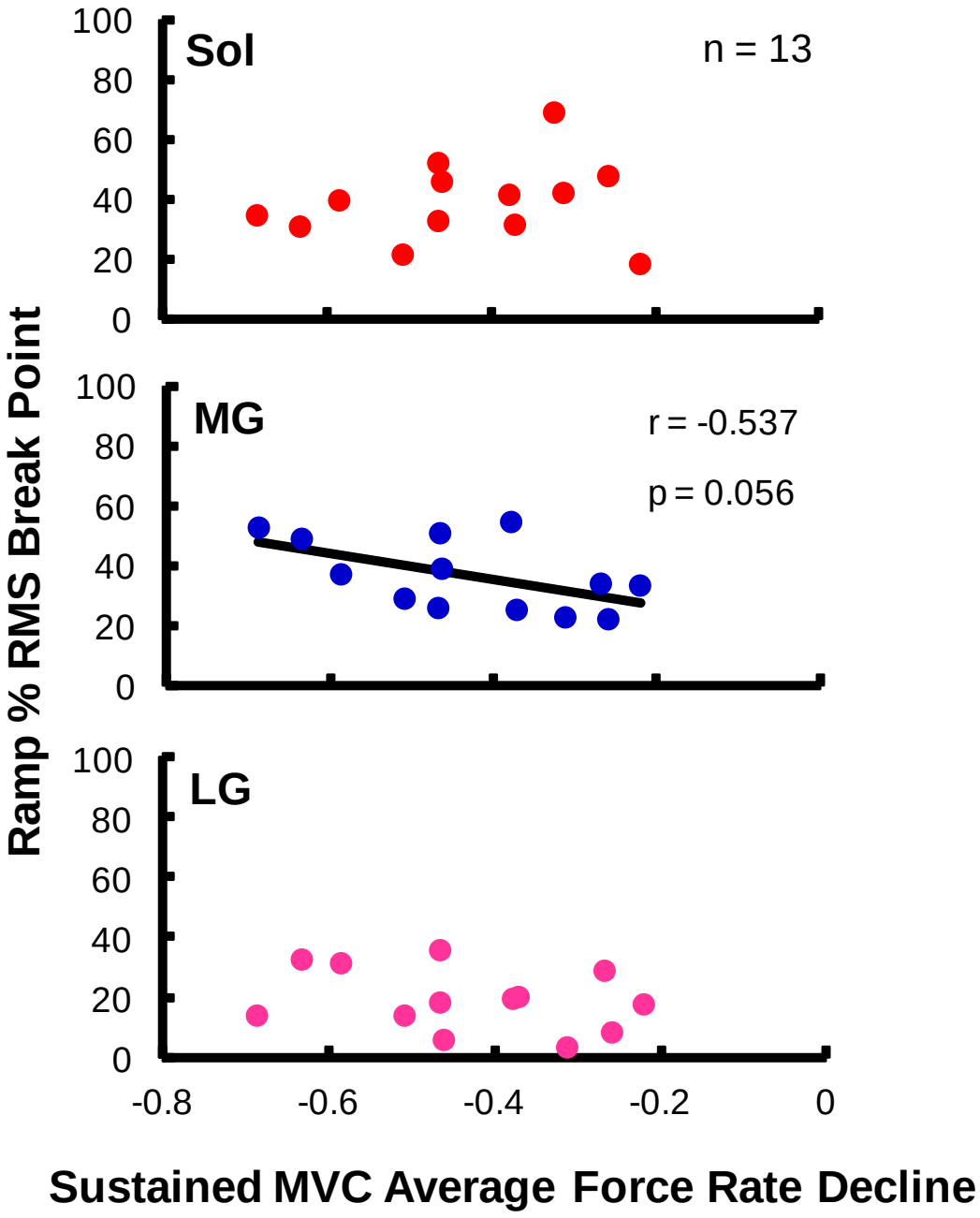


Figure 10. Relationship between 60 second sustained MVC force rate decline and 15 second ramp to MVC rmsEMG BPs in 13 subjects for each muscle. A trend toward significance was observed in only the MG ($r = -0.537$; $p = 0.056$).

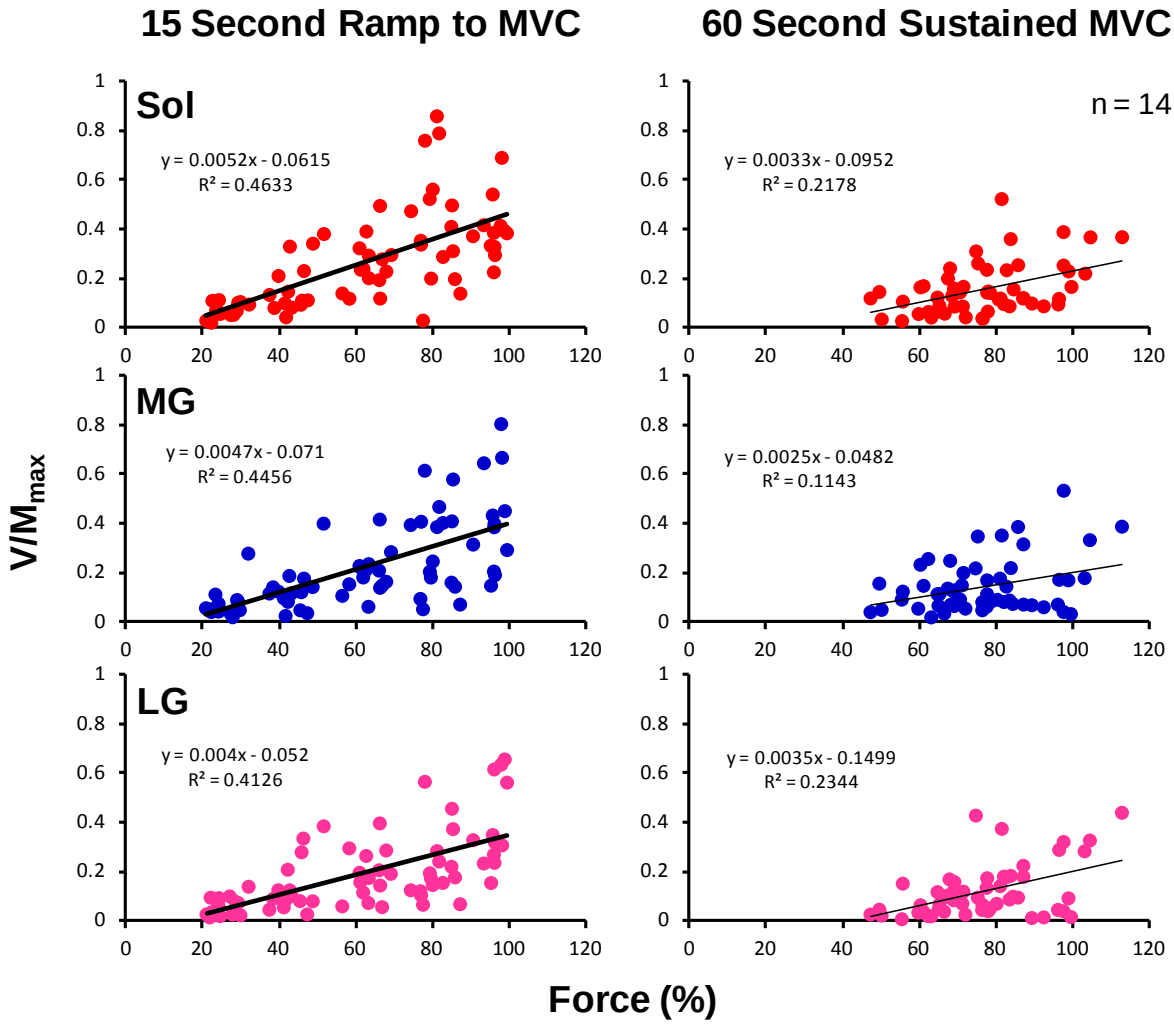


Figure 11. Relationship between normalised force (%MVC) and normalised V response amplitude (V/M_{max}) during the 15 second ramp to MVC (left panel) and the 60 second sustained MVC (right panel). A significant increase in V response amplitude was observed in Sol (r = 0.681), MG (r = 0.668) and LG (r = 0.642) during ramp and a trend toward a significant decrease in V response amplitude was observed in Sol, MG and LG during sustained MVC.

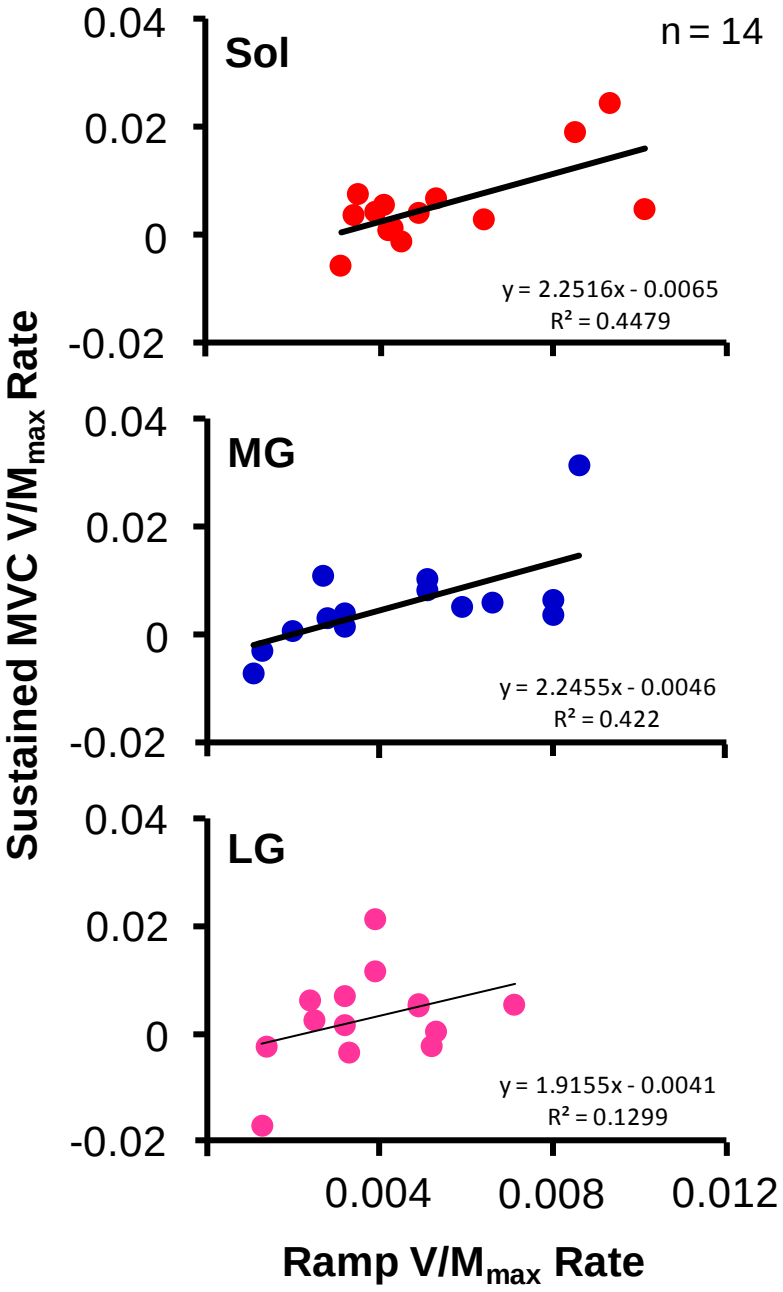


Figure 12. Relationship between the rate of increase in V response amplitude during the 15 second ramp to MVC and the rate of decrease/increase in V response amplitude during the 60 second sustained MVC. A significant relationship was observed in Sol ($r = 0.669$, $p < 0.01$) and MG ($r = 0.650$, $p < 0.05$). No relationship was observed in LG.

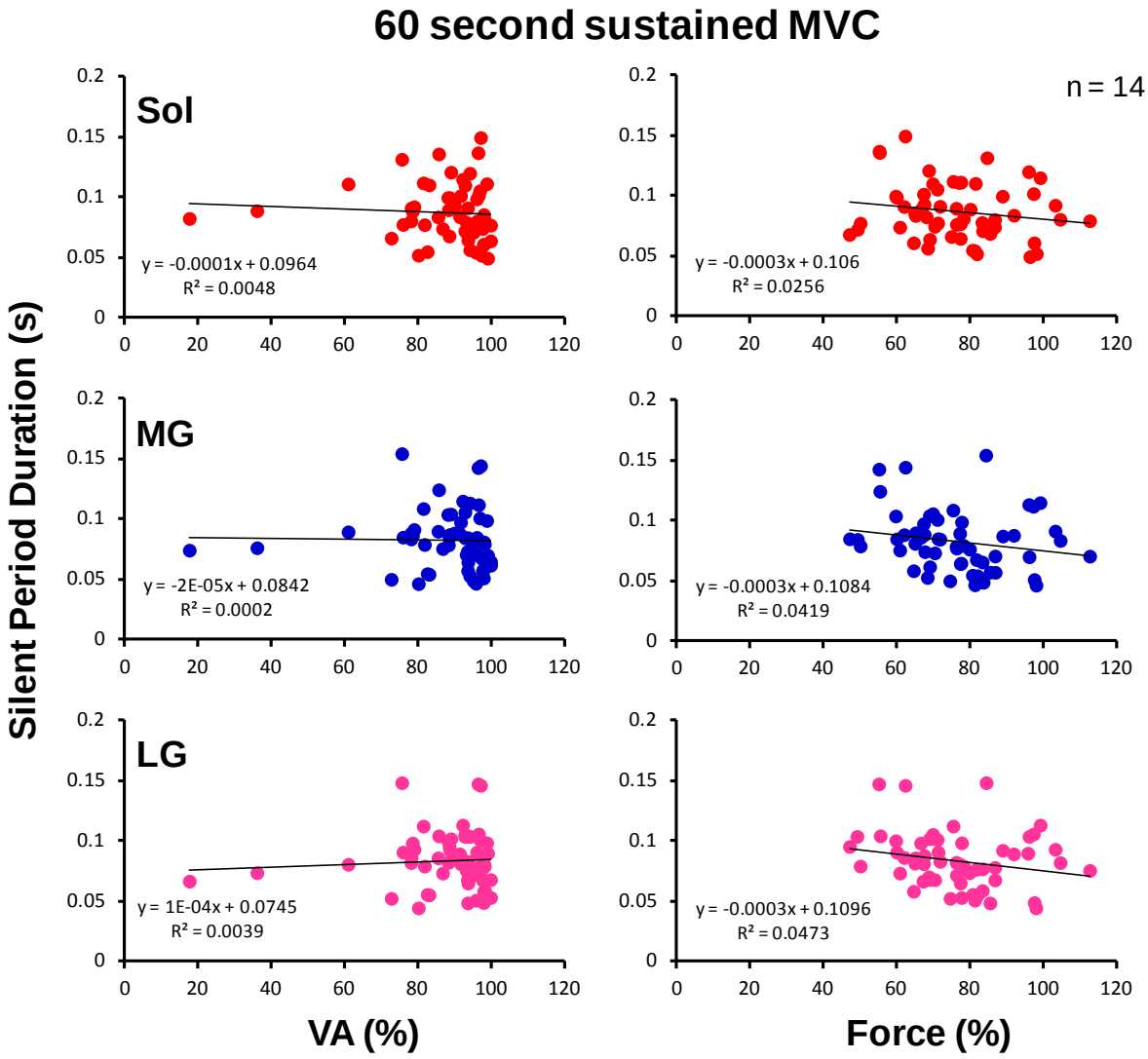


Figure 13. Relationship between Sol, MG and LG silent period duration (ms) and VA (%) and normalised force (%) during the 60 second sustained MVC. No significant relationship was observed between these factors.

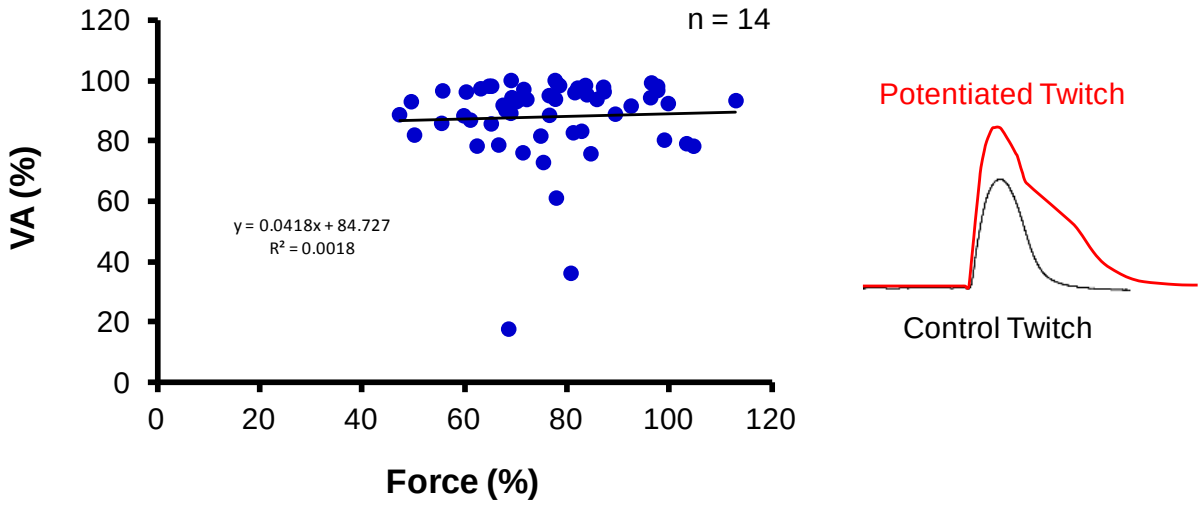


Figure 14. Relationship between force decline (%) and VA (%) during the 60 second sustained MVC. No significant relationship was observed between these two factors. The insert (right) shows the difference between the control twitch and potentiated twitch (average of 5 resting twitches) in one subject during the 60 second sustained MVC protocol.

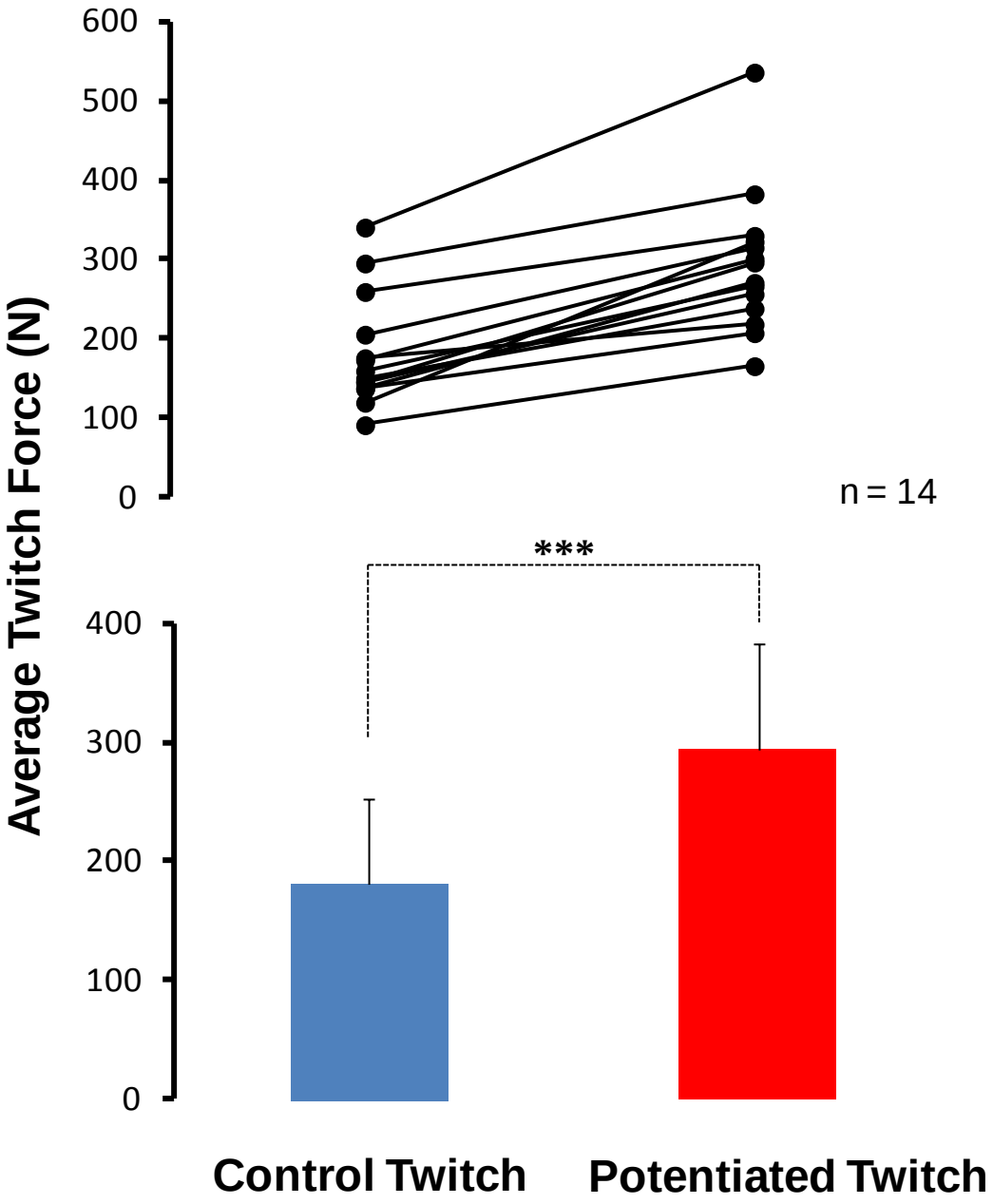


Figure 15. Average force (N) of 5 resting (before the 60 second sustained MVC) and 5 potentiated twitches (after the 60 second sustained MVC) in 14 subjects. The upper panel shows individual data (n = 14) and the lower panel shows the average data for all 14 subjects (mean ± SD). A significant increase (***) was observed in potentiated twitch compared to control twitch ($p < 0.0001$).

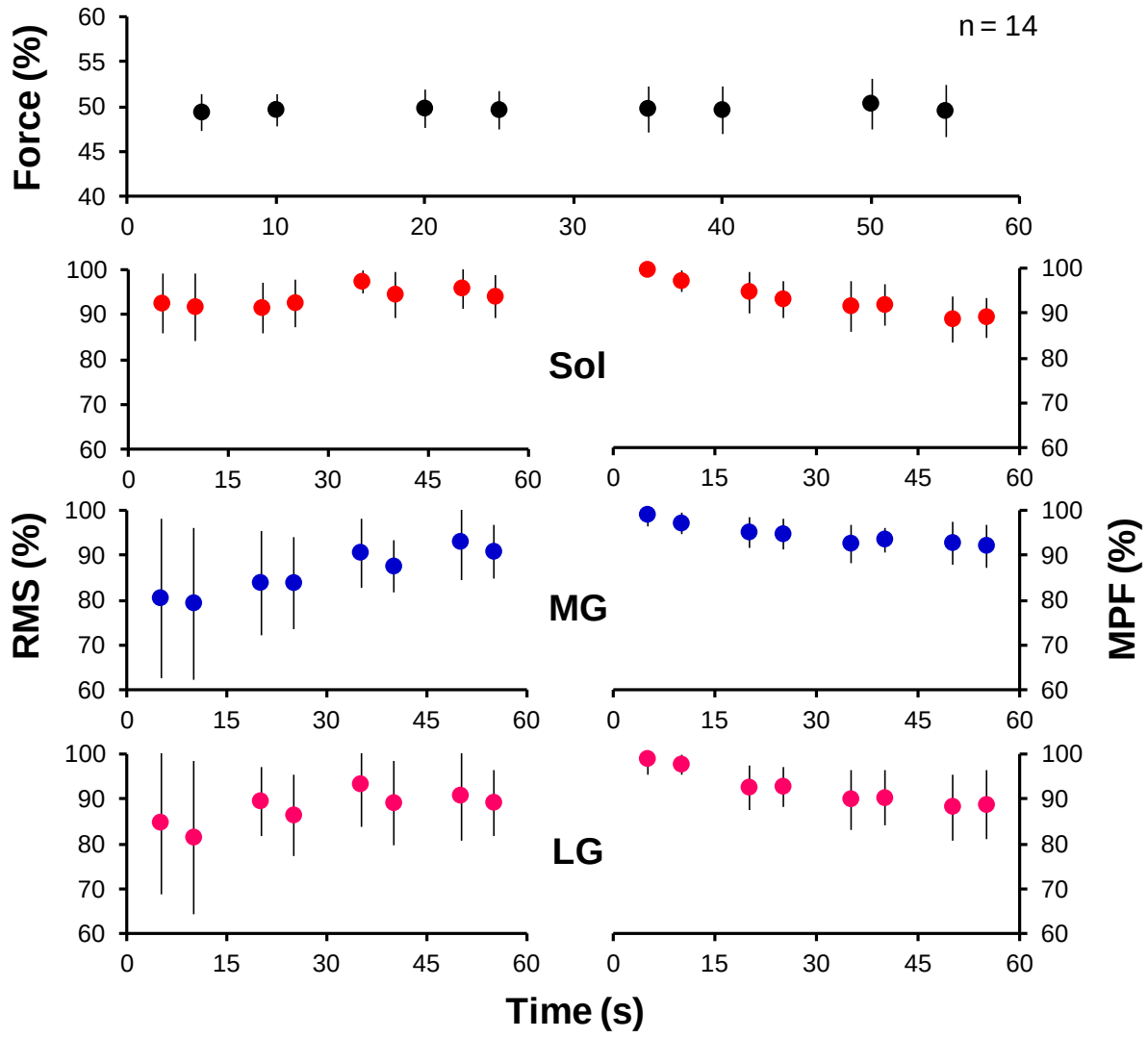


Figure 16. Normalised force (upper panel), rmsEMG (left panel) and MPF (right panel) of Sol, MG and LG during the 60 second sustained 50% MVC in 14 subjects. A significant increase in rmsEMG was observed in Sol (r = 0.664), MG (r = 0.937) and LG (r = 0.695) and a significant decrease in MPF was observed in Sol (r = 0.973), MG (r = 0.933) and LG (r = 0.948). All data are mean ± SD.

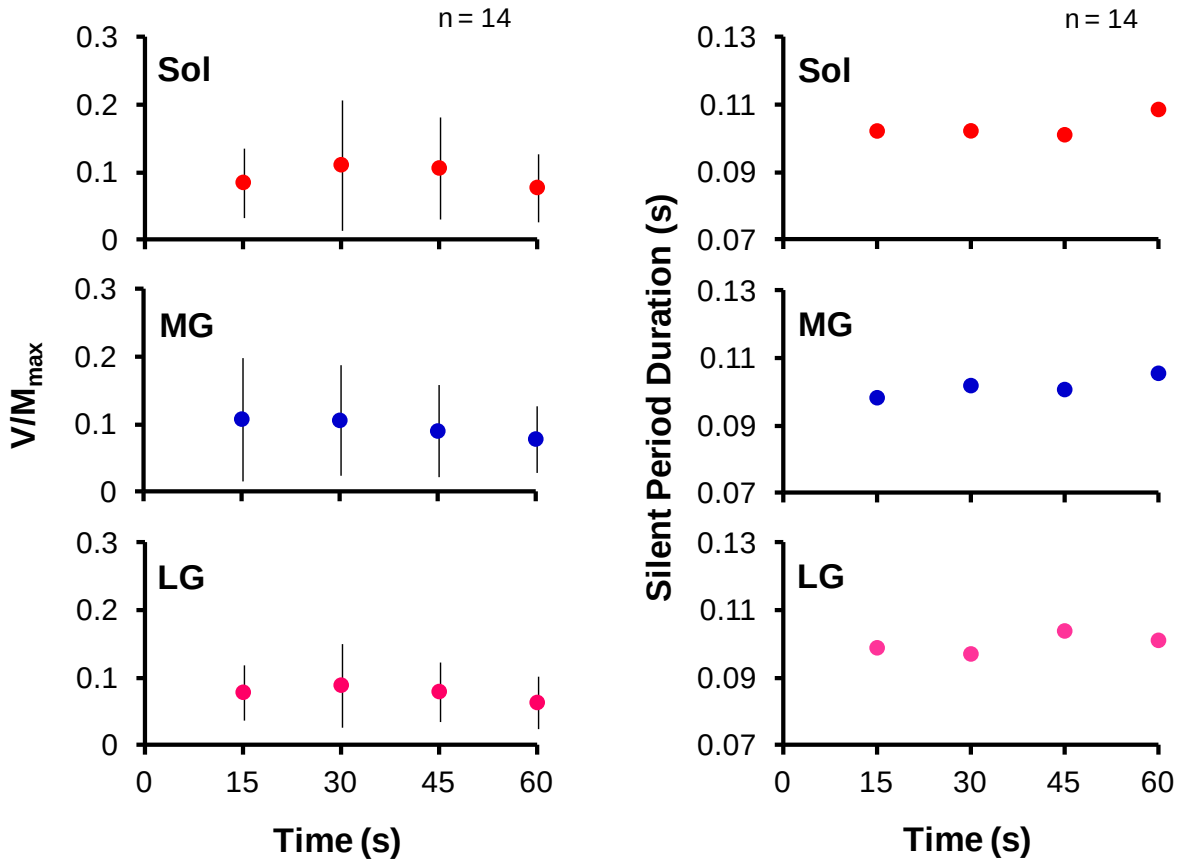


Figure 17. Time course of changes in V response amplitude (V/M_{max}) and silent period duration during the 60 second sustained 50% MVC protocol. No significant changes in V response amplitude and silent period duration were observed in Sol, MG and LG. All data are mean ± SD.

Table 1. Blood lactate measurements in 14 subjects from Experiment I. No differences in blood lactate concentrations were observed before and after the 60 second sustained 50% MVC and the 15 second ramp to MVC. A significant difference was observed in blood lactate concentration after the 60 second sustained MVC compared to before ($p < 0.01$). All data are mmol/L ± SD.

	15 s Ramp to MVC	60 s Sustained % MVC	
		50% MVC	MVC
Before (mmol/L)	**1.98 ± 0.67**	**2.24 ± 0.64**	**1.94 ± 0.54**
After (mmol/L)	**2.78 ± 4.03**	**2.09 ± 0.62**	**5.26 ± 3.32** **

** $p < 0.01$ Mean ± SD

Experiment II

Typical twitch force recordings from before and after one 5 second MVC, and EMG and sustained MVC force traces from one 5 second MVC, from 1 subject are shown in Figure 4. The upper insets show examples of the elicited M and V responses and interpolated twitch force (gain x4) during the same 5 second MVC.

Figure 18 shows the average impulse and VA ± SD in triceps surae during each of the 24 intermittent 5 second MVCs. Data has been normalised to that of the first intermittent MVC. MVC impulse significantly declines, as a percentage of the first MVC impulse, as the number of MVCs progresses. VA slightly increases, as a percentage of that of the first intermittent MVC, until the 10th intermittent MVC. From the 11th intermittent MVC onwards, VA tends to decrease as a percentage of VA in the first intermittent MVC.

A significant relationship was found between the impulse of intermittent MVCs and VA ($r = 0.559$, $p < 0.05$). As the intermittent MVCs progressed, impulse declined and, with this decline, VA also declined (Figure 19).

Regarding corticospinal responses during the intermittent MVC protocol, significant relationships were found between intermittent MVC impulse and V/M_{max} (left panel) and silent period (right panel) in Sol, MG and LG ($p < 0.01$). V response amplitude declined ($r = 0.471$, $r = 0.556$ and $r = 0.471$ for Sol, MG and LG, respectively) and the silent period lengthened ($r = 0.781$, $r = 0.731$ and $r = 0.707$ for Sol, MG and LG, respectively) with the progression of intermittent MVCs (Figure 20).

In Figure 21, data showing the normalised (in relation to the data from the first twitch recorded after the first intermittent MVC) impulse, peak force and half relaxation time (left panel) and M responses in triceps surae (Sol - top, MG - middle and LG - bottom) (right panel) are presented in relation to the progression of twitches elicited after intermittent MVCs. For the first 6 twitches after the corresponding intermittent MVC, the impulse of the twitch increases. Then, for the remaining twitches after the following MVCs, the impulse gradually

declines to levels about that of the first twitch after the first MVC, although slightly higher. The peak force of twitch after MVC increases until the sixth MVC and then decreases gradually, although slightly, for the following MVCs but finishes higher than the first twitch after the first MVC in the twitch elicited after the twenty fourth MVC. Half relaxation time in the twitches after the intermittent MVCs increases until the sixth MVC, then decreases from the seventh to the sixteenth MVC before leveling off at about 95% of the first twitch after the first MVC for the final 8 twitches after the corresponding MVCs.

The amplitude of the M response in twitches after each intermittent MVC slightly decreases as MVCs progress to finish at about 90% of the first twitch after the first MVC in Sol. However, in both MG and LG, M response amplitude gradually increases, with the progression of MVCs, to finish higher in the twenty fourth twitch (about 105% in both MG and LG) compared to the first twitch.

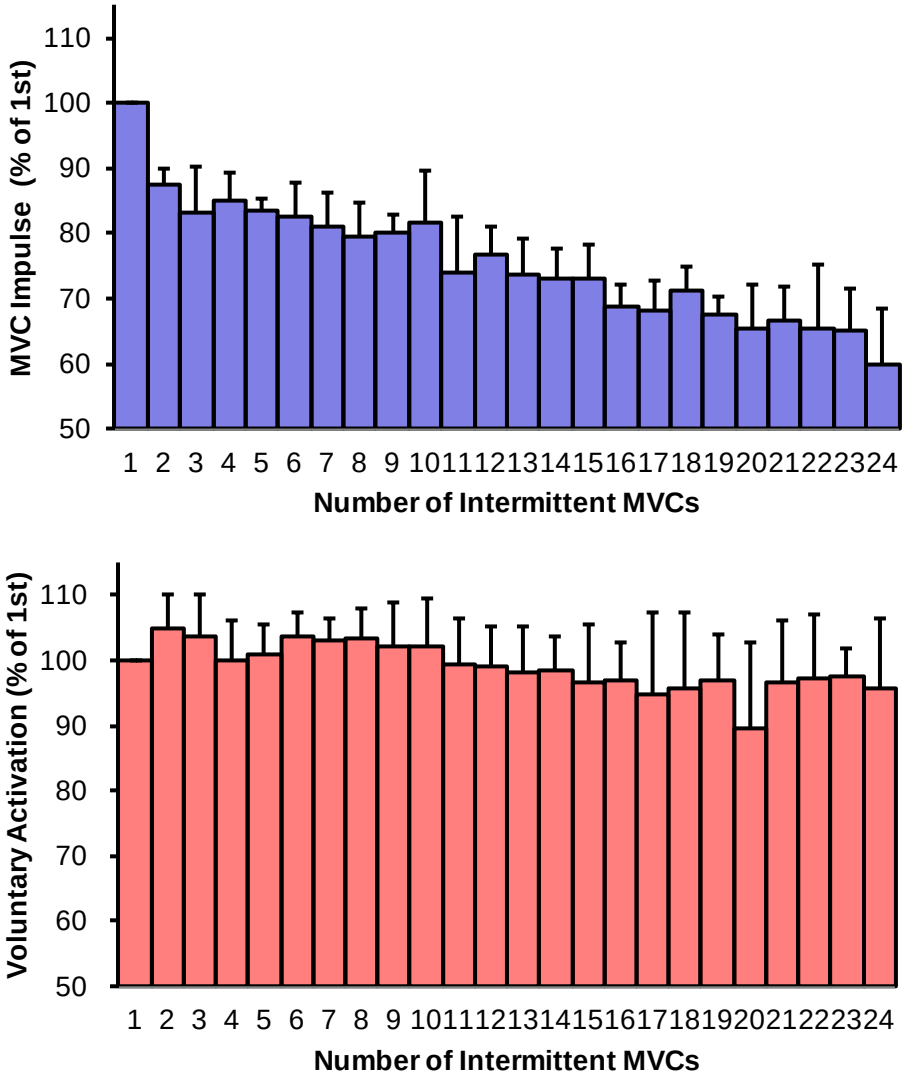


Figure 18. Normalised impulse and voluntary activation (% of 1st intermittent MVC) in triceps surae during 5 second sustained intermittent MVCs. All data are mean ± SD.

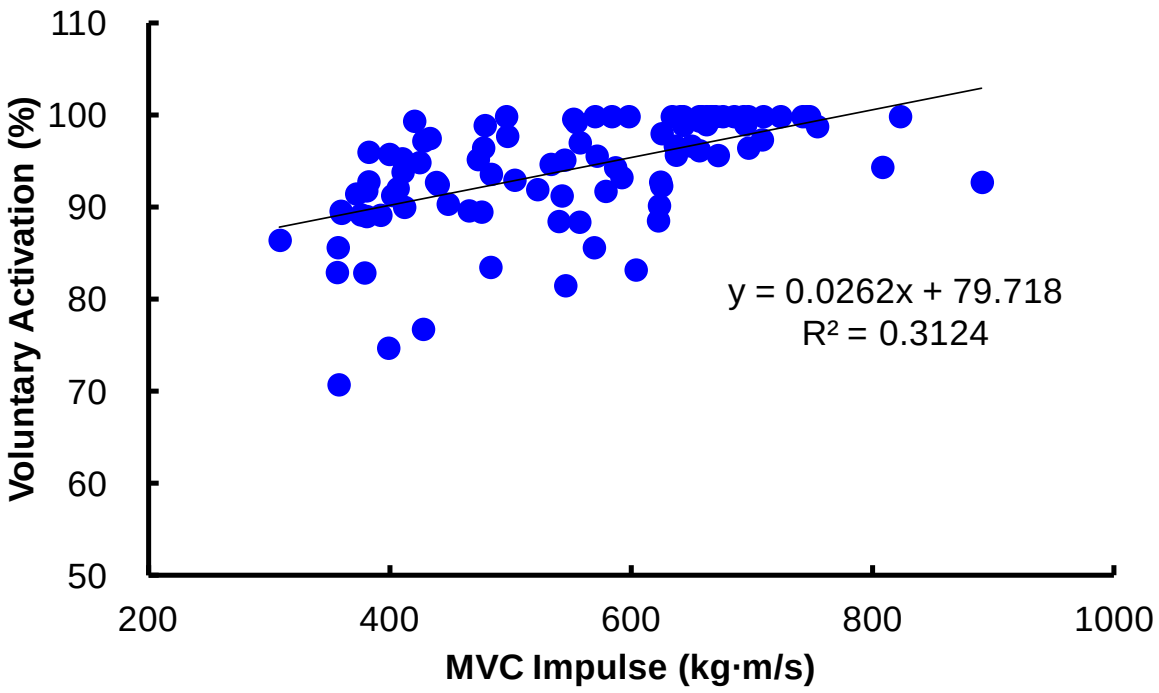


Figure 19. Relationship between voluntary activation (VA) and intermittent MVC impulse. Significance ($p < 0.05$).

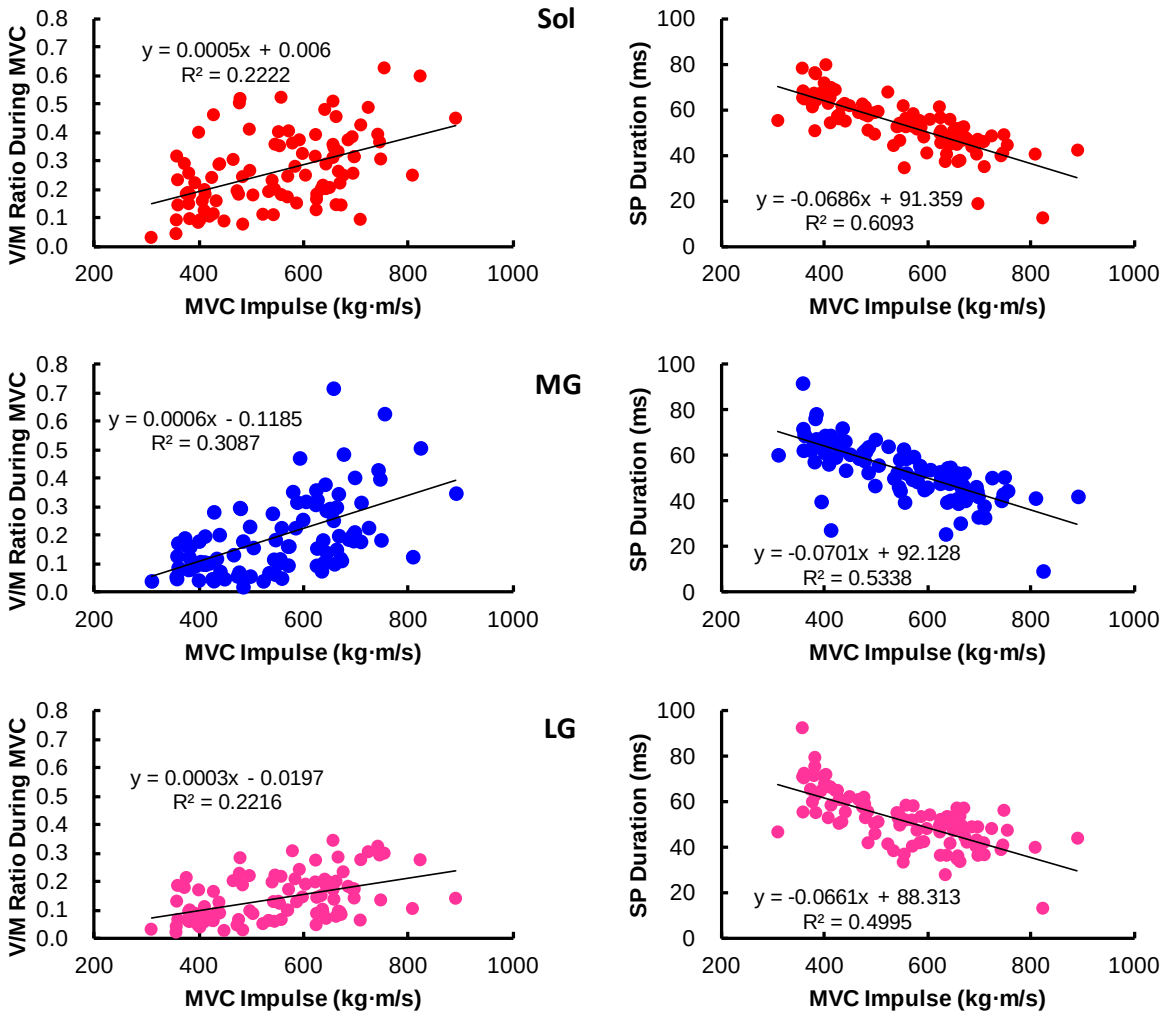


Figure 20. Relationship between impulse and V/M ratio (left side), and silent period duration (right side) during intermittent MVCs. Significance ($p < 0.01$).

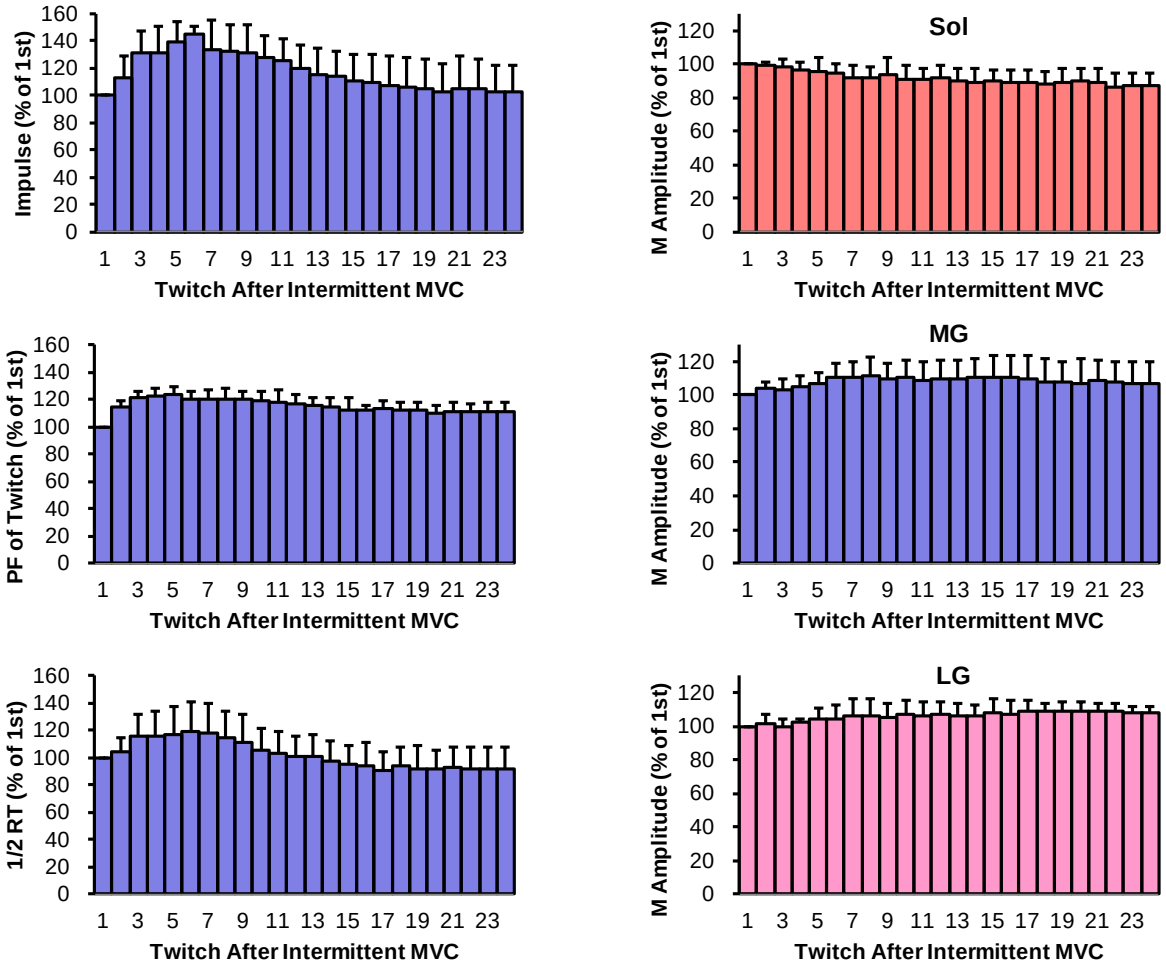


Figure 21. Normalised impulse, peak force and ½ relaxation time (left side) and M responses in triceps surae during twitch immediately after intermittent MVCs (right side). All data are mean ± SD.

DISCUSSION

Corticospinal Responses and Neural Drive

It has been shown that central factors play a significant role in the decline in force during sustained MVC and %MVC [1), 3), 5~6), 18), 24), 38~57)]. Our results have supported these findings with declines in V response amplitude in both sustained and intermittent MVC protocols, lengthening of the silent period in the intermittent MVC fatigue task and a reduction in voluntary activation in both sustained and intermittent fatiguing protocols. Moreover, potentiation of the maximal twitch force during and after the 60 second sustained MVC (superimposed and resting elicited twitches) and in twitches elicited after each intermittent MVC, highlight the fact that peripheral components of the muscles investigated remain capable of contributing to the production of force after a 60 second MVC [3), 19), 25), 38), 51), 57)].

The volitional wave, or V response, has been shown to be of reflex origin [58)] and is understood on the basis that when a supramaximal stimulus is applied, in the case of the present study, to the tibial nerve, action potentials are elicited in all Ia afferents and alpha-motor axons. The action potentials travelling orthodromically along alpha-motor axons result in a maximal M response or M_{max} while the action potentials travelling antidromically collide with descending motor action potentials of spinal and supraspinal origin, cancelling the two signals and clearing the motorneuron axons allowing the 'H reflex' volley of action potentials to proceed to the muscle resulting in the V response [33), 58~63)] Upton et al. [58)] suggested that the "the peak-to-peak amplitude of the V-wave expressed relative to that of the M response (V/M_{max}) reflects the amount and frequency of efferent nerve impulses travelling in α-motorneuron axons." Therefore, Aagaard et al. [33)] have suggested that "V/M_{max} may be taken to reflect the magnitude of the efferent motorneuronal output during voluntary muscle activation." It is well known that neural drive increases with training and increases in the V response and H-reflex have been shown to occur after training,

lending further support to the theory that V response amplitude may be used as an indicator of central drive to the muscle [33) 58~63)]. In the present study, V/M_{max} significantly increased during the 15 second ramp to MVC and decreased during the time course of the 60 second sustained MVC (Figure 11) and intermittent MVCs (Figure 20). These results represent an increase in central drive during the ramp protocol, leading to an increase in force output, and a decline in central drive during the sustained MVC and intermittent MVCs, leading to a decline in force output in these protocols. Therefore, it seems clear that central fatigue is present during sustained and intermittent fatiguing MVCs of the triceps surae because of a decline in central drive to the plantar flexor muscles during these tasks.

The silent period in the EMG can also be used as an indicator of central fatigue. The length of the silent period in the EMG after cortical stimulation increases during a sustained MVC of the elbow flexors as well as the silent period (peripheral) that is produced after motor nerve stimulation during a sustained MVC of the same muscles [3), 45), 64)]. Our results fail to show a significant lengthening of the silent period during the 60 second sustained MVC in relation to VA and force (Figure 13). The reasons for this may be numerous. It has been suggested that muscles are not driven maximally during a maximal effort [3)]. However, in the present study relatively high levels of VA were observed in most subjects. Changes in EMG recording conditions may also explain why the silent period did not lengthen during the sustained MVC protocol in this experiment. However, it is difficult to conceive that the current results were affected by this phenomenon because of the significant shortening of silent period observed during the intermittent MVC protocol, which was performed in an almost identical manner to the 60 second sustained MVC task. The complex nature of EMG responses and timing of the supramaximal stimulus in relation to timing of the firing patterns of central drive is another possible explanation. On the other hand, a significant lengthening of the silent period was observed in the EMG of the Sol, MG and LG after supramaximal stimulation of the tibial nerve during each 5 second MVC over

the time course of intermittent MVCs in the fatiguing protocol of Experiment II, lending further support to the idea that central factors play a significant role in the reduction of force during fatiguing voluntary contractions of the triceps surae.

Voluntary Activation

Another indicator of central fatigue is voluntary activation. As outlined earlier, voluntary activation is defined as "the level of voluntary drive during an effort" [3]. Voluntary activation can be measured using twitch interpolation during a maximal voluntary contraction [3), 19), 34~38), 64~66)]. In this study, VA declined only a little during the 60 second sustained MVC. Some subjects were even able to increase the voluntary drive to the triceps surae toward the end of the sustained contraction and one subject managed to drive the muscles of the triceps surae maximally at 45 seconds and 60 seconds into the contraction. When VA was compared to the decline in force, no relationship was found during the 60 second sustained MVC (Figure 14). However, a significant relationship was observed between the decline in impulse and the decline in VA as the intermittent MVCs progressed in Experiment II (Figure 19). The reason for the difference in these two results is most likely due to the methods used to calculate VA. During the intermittent MVC protocol, the potentiated resting twitch (the twitch elicited during the 5 second rest after each 5 second MVC) was used as the denominator in the equation to calculate VA. For the 60 second sustained MVC, VA was calculated by using the average of 5 resting twitches (elicited before the onset of the fatigue task) as the denominator in the equation to calculate VA. Therefore, the factor of twitch potentiation could not be utilised in analysis of the results. Twitch potentiation, an indicator that peripheral fatigue was not the main factor contributing to the decline in force during the sustained MVC due to the fact that the muscle has the ability to produce extra force even though the muscle is being driven 'maximally' but force output is in decline, was observed in this experiment during and after the 60 second sustained MVC (Figure 15). It was, partly,

for this reason, and the fact that the silent period did not lengthen during the sustained MVC, that it was decided to conduct Experiment II (intermittent MVC fatigue task), in five of the subjects from Experiment I, in order to investigate these factors further because it is thought that they play an important role in decline in force during sustained MVCs. One other possible reason why no relationship was found between VA and force decline during the 60 second sustained MVC may be due to the methods used. In this experiment a single pulse was applied to the motor nerve to elicit a twitch response, as this method has been shown to be able to measure voluntary activation [3)]. However, it has been argued that double, or trains of, pulses are the preferred method to be used when conducting twitch interpolation because the first pulse clears the efferent path so that the second (and subsequent) pulse(s) can elicit a truly maximal M response [3), 27), 51), 67)]. Therefore, it is possible that a maximal twitch was not produced in this experiment perhaps affecting the results related to VA. Even if a maximal twitch was not produced during the intermittent MVC protocol, the effect was sufficient to show a significant relationship between a decline in VA and force. Therefore the double pulse method may not be necessary in experiments involving intermittent plantar flexion MVC tasks, however may be a necessity in sustained MVC protocols.

Group III and Group IV Afferents

A reduction in EMG and the firing rates of motorneurones has been shown to occur in conjunction with the loss of force during sustained MVCs [18~23)]. The same phenomena were also observed in the present study. It has been suggested that the reduction in motorneurone firing rates is not responsible for the loss of force but motorneurone firing is controlled in order to optimise force generation as the muscle properties change [4), 68)]. The control of motorneurone firing may come from within the nervous system alone but it has been suggested [68)], and more recently shown to be most likely [3), 69~71)], that group III and group IV afferents play a role in this process. Group III and group IV nerve endings respond

to metabolic products that accumulate in the muscle during fatigue [68~69), 72~74)] and were originally thought to act in an inhibitory manner on the motorneurone pool. More recent studies have shown that group III and group IV afferents also facilitate motorneurones while inhibiting the motor cortex [3), 69)]. Blood lactate levels significantly increased after the 60 second sustained MVC in the present study (Table 1). Therefore the response of group III and group IV afferents to metabolites in the plantar flexor muscles cannot be dismissed when explaining possible mechanisms for the loss of force during a sustained maximal plantar flexion.

The rmsEMG BPs during ramp contraction to MVC represent individual neural drive patterns and a unique capacity to recruit motorneurones and/or MUs and may represent a change in MU recruitment and/or firing rate. The individual nature of BPs in the triceps surae (Figure 9) also point toward individual changes in the metabolic state of the Sol, MG and LG as the ramp progresses. One must also not neglect the possible influence of fibre type in the determination of BPs. Although the buildup of metabolic by products of muscle contraction are negligible in a 15 second ramp contraction to MVC (blood lactate concentration rarely changed from baseline during the ramp protocol in the current study), based on the discussion above, it may be possible to suggest that group III and group IV afferents play a role in the regulation of MU firing during an isometric ramp contraction. Lending further support to this theory, it has been suggested that the excitation of motorneurones by group III and group IV inputs appears differentially directed to higher threshold motorneurones [69)], those most likely to be modulated during ramp contraction to MVC to account for necessary increases in torque.

Further evidence to suggest that central rather than peripheral components play a greater role in the decline in force during sustained MVCs is that the ATP cost of contraction is not related to fatigue level in stimulated rat gastrocnemius muscle. In fact, it has been shown that there is a reduction in ATP cost during fatiguing contractions suggesting

systematic optimisation of ATP utilisation which may be due to mechanisms such as shift in fibre type recruitment and alternation of activation-relaxation processes [75].

A significant relationship between the rate of increase in V response amplitude during the 15 second ramp to MVC and the rate of decrease/increase in V response amplitude during the 60 second sustained MVC was observed in the Sol and MG (Figure 12). These results may suggest that subjects with 'stronger' central drive, represented by a greater increase in V response amplitude, during ramp contraction are more susceptible to the effects of central fatigue during sustained MVCs because they showed a greater decline in central drive, represented by a higher rate of decline in the amplitude of the V response, during the 60 second sustained MVC task. The fact that these results were significant in the Sol and MG ($p < 0.01$ and $p < 0.05$, respectively) may be no coincidence because of the apparent influence of group III and group IV afferents, discussed above and below, on these two muscles.

Morphological Factors

It is conceivable that muscle fibre type also plays a role in the current results, especially in the relationship between ramp rmsEMG BPs in MG and the decline in tricpes surae force during sustained MVC. The Sol muscle is generally made up of about 70% slow, type I, muscle fibres but has been shown to contain up to 90% slow type fibres in humans, the remaining portion being made up by fast, type IIA and IIB, fibres [74~75]. The gastrocnemius muscle on the other hand is made up of about 50% slow and 50% fast type fibres [76~78]. Because fast twitch fibres are less fatigue resistant and make up a higher proportion of the gastrocnemius muscle, we hypothesised that there would be a relationship between the ramp rmsEMG BPs of both heads, medial and lateral, of the gastrocnemius muscle and force rate decline of the triceps surae during the sustained MVC. However, a relationship was only observed in the MG. One reason for this may be due to relative contributions of the soleus and both heads of the gastrocnemius during plantar flexion at the knee angle used in this

experiment. Knee and ankle flexion at 90 degrees and 0 degress, respectively, was chosen for this experiment in order to isolate central drive to the triceps surae during contraction. However, under these conditions the contribution of the gastrocnemius to plantar flexion force is less than if the leg was extended [79~83]. This may explain some of the results in the intermittent MVC protocol suggesting slightly more peripheral involvement, perhaps via group III and group IV afferent pathways, as the soleus contributes more to plantar flexion force in the current set-up [49), 83~85)]. Chemoreceptors are more prevalent in slow type fibres and the soleus is predominantly made up of type I fibres, therefore making the soleus muscle more prone to modulation induced by group III and group IV afferent feedback leading to a reduction in M response amplitude during the intermittent MVC task. Also, the total workload in the intermittent MVC protocol was higher than that in the sustained MVC which may have allowed for more factors to come into play as time progressed.

Joint angle may also play a role in the fact that a relationship was observed between MG ramp BPs and sustained MVC force decline but not between LG ramp BPs and sustained MVC force decline. It is possible that the relative muscle volume of the two heads of the gastrocnemius also played a role in these results. It has been shown in 12 healthy subjects that among the triceps surae, the soleus had the largest mean muscle volume (~490 cm^3), followed by the MG (~240 cm^3) and LG (~140 cm^3). Thus, the soleus and the gastrocnemius comprised 46 and 36% of the plantar flexor muscle volume, respectively [86~87)]. Therefore, although at 90 degrees flexion the gastrocnemius contribution to plantar flexion torque is reduced, the relative contribution of the MG, compared with LG, is increased due to its larger muscle volume. It has also been suggested that the MG has a predisposition for producing force whereas the Sol is designed for producing tension at the expense of velocity and the LG's priority is velocity [86)]. Another factor that may have influenced the present results is that a reduction in MG EMG is often observed when the knee is in flexed positions. However, this reduction has been shown to be influenced by factors other than just the MG

fascicle length and force potential relationship and the MG at rest has been shown to be not actively insufficient between 80 and 165 degrees flexion [81].

Submaximal Voluntary Contractions

Various researchers have shown that during submaximal fatiguing isometric contractions new unfatigued motor units are recruited to compensate for a reduction in the ability of the working muscle to produce force. Some studies focused on the data from single MU recordings [20] while others have looked at changes in the surface EMG [51), 68]. Particularly, by employing an H-reflex technique, twitch interpolation and analysing changes in EMG and force tremor, Löscher et al. [51] concluded that there is elevation of excitatory drive to the alpha-motorneurone pool during fatiguing submaximal contractions and found that central fatigue is also present because of the termination of contraction when there is still muscular capacity for extra force production by muscles of the triceps surae. The results of this study lead to similar conclusions because there was a steady increase in rmsEMG in Sol, MG and LG throughout the sustained 50% MVC, suggesting recruitment of MUs in order to maintain force. The rate of increase in rmsEMG was higher in MG and LG than in Sol, perhaps due to the greater number of fast twitch MUs present in the gastrocnemius muscle compared to the Sol. Following the laws of Henneman's Size Principle, more fatigue resistant slow twitch MUs are recruited early in the contraction then in order to maintain the target force, more unfatigued, possibly fast type, MUs are recruited as the contraction progresses. MPF declined throughout the sustained 50% MVC suggesting modulation of rate coding and perhaps syncronisation of MU firing. The rate of decline of MPF in Sol was higher than in MG and LG, perhaps because of the smaller number of slow twitch fibres innervated by low threshold motorneurones, about 100 in cat hindlimb muscle and as low as 5 in human muscle [88], meaning that syncronisation occurs quicker than among fast twitch fibres. The fact that fast twitch fibres are more prevalent in the gastrocnemius muscle expounds this

phenomenon, especially as one motor unit can innervate up to 2,000 muscle fibres in the human MG [88].

From the H reflex technique used in this study, observing changes in the V response and silent period in order to evaluate central drive to the triceps surae, there were no significant changes in V response amplitude and silent period length in the Sol, MG and LG as the fatiguing submaximal contraction progressed (Figure 17). This suggests that central drive was maintained throughout the contraction. As alluded to earlier, Martin et al. [69] have suggested that group III and group IV afferents inhibit the motor cortex but both inhibit and excite the motorneurone pool, excitation being preferentially directed toward high threshold motorneurones [69]. No doubt these mechanisms play a role in the current results, however interpretation of the results in submaximal contractions is a much more complex endeavour [3, 55].

Limitations

Some of the limitations of this study have been touched upon in the body of the text but to outline some of them briefly here is appropriate. As early as 1988 Brenda Bigland-Ritchie came to the conclusion that "no fixed relation is to be expected between force and EMG" [41]. Recent studies have further highlighted the difficulties in interpreting surface EMG [89~93].

Regarding twitch interpolation, it was not possible to use the potentiated twitch when calculating VA in the sustained MVC task. This may have led to no relationship being observed between VA and force decline. This led to the follow up intermittent MVC study being performed in order to investigate the phenomenon of twitch potentiation more closely. Also, we only used a single stimulus to elicit maximal twitch responses at rest and during sustained and intermittent MVCs. Therefore, it is possible that the twitches produced were submaximal because the efferent pathways had not been cleared of voluntary orthodromic

activity which would allow a second stimulus, or following train of stimuli, to evoke a maximal response if they were employed [3), 36), 51), 72)].

It has also been suggested that VA declines more in the second half of a 2 minute sustained MVC. The present study made use of a 60 second sustained MVC to investigate fatigue of the triceps surae, therefore the time course may have been too short to have accommodated significant changes in VA. These results may add to the literature suggesting that there is little reduction in VA in the first 60 seconds of maximal isometric plantar flexions of the triceps surae.

Regarding blood lactate concentrations, there may have been some inconsistencies in the recordings made because in some subjects samples were taken from subjects' fingers. Subjects were free to use their hands to grip the sides of the seat they were sitting in when performing the plantar flexion tasks. It is possible that the blood lactate recordings in these instances were not a true representation of total body blood lactate levels because of the extra work the finger muscles performed causing a spike in the lactate concentration in vessels close to the point of blood extraction. However, inconsistencies are considered to be minimal because of the similarity in the readings obtained from both the fingertips and earlobes of subjects.

Only 4 female subjects took part in the present study. We believe that the near significant result regarding the relationship between ramp rmsEMG BPs in the MG and sustained MVC force decline rate should become significant if the number of participants, especially, for the purpose of a balanced data set, the number of female participants, was increased. Therefore, in order to confirm the current findings, more female subjects must be recruited to take part in this and future experiments. Ramp rmsEMG break points were also not observed in the Sol in one subject and in the MG and LG in another subject. This reduced the number of data points for analysis which may have affected the results negatively.

CONCLUSION

The results of the present study indicate that the decline in force during a 60 second sustained maximal voluntary contraction of the triceps surae is due to neurogenic rather than myogenic factors and that, under the present conditions, neural drive to the medial head of the gastrocnemius muscle plays an important role in the regulation of force output during voluntary plantar flexion of the triceps surae, highlighted by the relationship between the rmsEMG BP of the MG, during a 15 second ramp to MVC, and the decline in force of the triceps surae, during a 60 second sustained MVC. One may also suggest that human muscle has a high resistance to fatigue and can maintain its capacity to perform contractions, even during sustained maximal voluntary contractions.

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