



Effect of gender, age, fatigue and contraction level on electromechanical delay

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ABSTRACT

Objective: The aim of this study was to determine electromechanical delay (EMD) using supramaximal stimuli and to investigate its variation with gender, age, contraction level and fatigue.

Methods: Fifteen male and 15 female healthy subjects (aged between 18 and 60) participated in our study. Electromyogram (EMG) recordings were taken from triceps surae muscle. While subjects contracted their muscles voluntarily at specified percentages of maximum voluntary contraction, 10 supra-maximal stimuli were applied to the tibial nerve. The time lag between the onset of the EMG response (M-wave) and the onset of force generation was calculated as EMD.

Results: EMD was found to be 8.5 ± 1.3 ms (at rest condition), which is much shorter than those reported in previous studies. Although EMD did not significantly vary with gender ($P > 0.05$), it decreased significantly with escalating muscle contraction level ($P < 0.05$) and increased significantly with advancing age and with fatigue ($P < 0.05$).

Conclusions: EMD was found to be considerably shorter than those reported in previous studies, and hence we discuss the possible reasons underlying this difference. We suggest that supramaximal nerve stimulation and high resolution EMG and force recording may have generated this difference.

Significance: Current findings suggest that EMD is very sensitive to the method used to determine it. We discuss the reasons for the short EMD value that we have found in the present study.

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1. Introduction

Electromechanical delay (EMD) is defined as the latency between the onset of electrical activity in a muscle and the onset of force generation by that muscle's contraction. EMD is investigated as the difference between these two distinct time periods (Komi and Viitasalo, 1980; Bell and Jacobs, 1986; Moore et al., 2002; Zhou et al., 1995). When muscle action potentials initiate contractile phenomenon, the elements in the sarcomeres begin to contract but this contraction does not generate simultaneous movement on the joint. The main reason for this delay is the existence of serial and parallel elastic components (SEC and PEC, respectively) within the muscle, which are represented as springs in the Hill and Kelvin's viscoelastic muscle-tendon model (Palladino and Davis, 2005; Enoka, 2008). There are also two kinds of SEC in the muscle system; one is the active SEC (myofilaments) and the other is the passive SEC (tendon and aponeuroses) (Hof, 1998). Recently a

new technique has been used to identify the contribution of each of the SEC components to EMD using high frame rate ultrasound (Nordez et al., 2009).

After the contraction in the sarcomeres, time is needed to stretch SEC to eventually initiate the movement on the joint (Cavanagh and Komi, 1979; Bell and Jacobs, 1986; Muraoka et al., 2004; Moore et al., 2002). Any level of muscle contraction is, therefore, expected to reduce EMD by taking up the 'slack' as the background muscle contraction level stretches the SEC (Zhou et al., 1995). Contribution of SEC to EMD can therefore be studied at different levels of isometric muscle contraction.

It is well known that ligament and tendon injuries are seen more frequently in women than in men during sports activities (Arendt and Dick, 1995; Ireland et al., 1997; Kannus, 1997). It is possible that gender specific muscle/tendon elasticity may play a role in the higher incidence of such injuries in women. Despite its importance, there are only a few studies that investigated the difference of EMD between genders. In some of these studies, EMD has been found to be longer in women than men (Bell and Jacobs, 1986; Winter and Brookes, 1991; Zhou et al., 1995). However, Moore et al. have reported that pre-fatigue EMD was shorter in women compared with men (Moore et al., 2002). Recently, a

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study of Blackburn et al. have found that there was no difference in EMD between genders (Blackburn et al., 2009).

Muscle fatigue has also been thought as one of the influential factors on EMD. There are varying findings regarding the effect of fatigue on EMD. For example, it has been observed that the more intense the muscular fatigue, the longer the EMD (Zhou et al., 1996). It has also been reported that, there was an increase in EMD in women after fatiguing protocol, while no difference was observed in men (Moore et al., 2002). Moreover, in a study of Minshull et al., it was indicated that EMD increased at post-fatigue in women and did not change in men when subjects voluntarily contracted their muscles, but it decreased at post-fatigue for both women and men when muscles were stimulated magnetically (Minshull et al., 2007). Prolongation of EMD during fatigue is explained by the impairment of action potential propagation along the muscle membrane due to the K^+ efflux into the t-tubules during long-standing contraction (Zhou et al., 1996; Latash, 2008). In this case, action potentials cannot reach the depth of the muscle and hence Ca^{++} release is reduced, which then slows down the force of contraction (Allen et al., 1989).

What we notice in most of the previous EMD studies is that each group uses a different approach for determining EMD, varying from reaction time to reflex methods (summarized in Table 1). The first aim of the current study therefore was to discuss the methodologies used in recent studies that resulted in varying EMD values. The second aim of this study was to test the influence of four factors on EMD: gender, fatigue, age and contraction level. Our hypotheses are (1) women have longer EMD since tendon injury statistics indicate that they occur more frequently in women. (2) Muscular fatigue prolongs EMD, probably because action potential propagation impairs due to K^+ efflux in t-tubules after fatiguing exercise. (3) EMD prolongs with increasing age since muscle-tendon system has viscoelastic properties, and viscoelastic structures are exposed to deformation with age. (4) EMD decreases with increasing contraction level since the tendon loses its slackness with rising contraction level. In this case, force developed by the sarcomeres is transmitted to the joint in a shorter time.

2. Methods

2.1. Subjects

Fifteen male and 15 female subjects between the ages of 18 and 60 were selected among a population of healthy, sedentary adults that were not actively involved in sports. The subjects signed informed consent forms prior to participation in experiments.

Approval for the study protocol was obtained from Ege University Research Ethics Committee. Weight and height measurements of all subjects were taken. None of the subjects had used any muscle relaxants within previous 6 months. The subjects were also asked not to consume caffeine or nicotine 12 h before the experiments, since both these substances were shown to affect fatigue and muscular activities (Lopes et al., 1983). All experiments were performed approximately at the same time of the day, assuming all the subjects had performed similar muscle activities within the day.

2.2. Electrodes and sensors

Surface EMG recordings were taken from the triceps surae muscle group of the left leg while the subjects were lying prone. Two Ag/AgCl bipolar surface electrodes were used. On the triceps surae muscle, one electrode was placed between the two heads of the gastrocnemius muscle, and the other was placed 6 cm distal to the first one in order to record the activities coming from both the soleus and the gastrocnemius muscles. Subjects were grounded using lip-clip electrodes (Türker et al., 1988). In order to stimulate the tibial nerve, anode was placed over patella, and the cathode was placed on poplitea fossa. The force measurements were taken by a load cell that has 300 Hz natural frequency (Fig. 1).

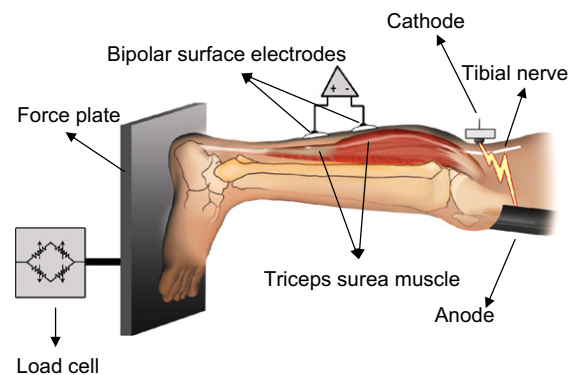


Fig. 1. Experiment setup. This figure illustrates the location of electrodes and force recording setup. There was 6 cm distance between bipolar electrodes. One of the electrodes was placed between the two heads of gastrocnemius; the other was placed on the soleus. Cathode stimulation electrode was placed on the poplitea fossa; anode stimulation electrode was placed just over patella.

Table 1
Summary of previous studies.

Authors	No. of subjects	Protocol	Muscle	Contr.	EMD (ms)
Nordez et al. (2009)	9 (M)	Electric	Gastroc. med.	Isometric	11.63 ± 1.51
Grosset et al. (2009)	30	Maximal electric	Triceps surae	Isometric	8 ± 0.2
Blackburn et al. (2009)	20 (M) + 20 (F)	Light (reaction experiment)	Biceps femoris	Isometric	^M 125.43 ± 21.51 ^F 127.49 ± 25.87 ~ ^F 25 ms, ~ ^M 30 ~ ^{M,F} 50
Minshull et al. (2007)	7 (M) + 9 (F)	Supramaximal magnetic Reaction experiment	Biceps femoris	Isometric	
Hopkins et al. (2007)	15	Reaction experiment Electric	Gastrocnemius	Isometric	22.8 ± 8.2 9.7 ± 3.1
Moore et al. (2002)	15 (M) + 15 (F)	Tap	Vastus lateralis	Isokinetic	Prefat. ^F 22.1 ± 5.8, ^M 28.4 ± 7.9 In fat. ^F 33.6 ± 11.2, ^M 27.1 ± 9.3
Yang and Türker (1999)	5 (M) + 4 (F)	Tap	Massater	Isometric	~15 ms
Zhou et al. (1995)	16 (M) + 5 (F)	Electric Reaction experiment	Vastus lateralis Rectus femoris	Isometric	22.1 (SEM 1.32) 38.7 (SEM 1.18)
Winter and Brookes (1991)	11 (M) + 11 (F)	Voice (reaction experiment)	Soleus	Isometric	^M 39.6 ± 1.2 ms, ^F 44.9 ± 2.0
Viitasalo and Komi (1981)	29	Voice (reaction experiment)	Quadriceps	Isometric	38.3 ± 1.7

EMD values that were reported in some previous studies (M = male, F = female).

2.3. Recording

The recording system was a CED 1902 quad-system amplifier, and a CED power 1401 analog to digital converter. To amplify the EMG signals, a home built preamplifier with artifact clamp was used in order to remove the interference of the stimulus artifacts. The surface EMG recordings were taken with a total of 500 amplification gain, 2 kHz sampling rate, and were filtered with a 20–500 Hz band-pass filter. The force recordings were taken with a total of 1000 amplification factor and 2 kHz sampling rate, without any filtering.

2.4. Stimulation protocol

Digitimer constant current stimulator model DS7A was used to stimulate the tibial nerve. The pulse width was 500 μ s and V_{max} was set to 200 V. To establish the maximal stimulus intensity for each subject, the stimulus was increased until the M-wave on triceps surae EMG increased no longer (Stim-Max). The suprathreshold level of stimulus used in the current study was $1.5 \times$ Stim-Max (the range of stimulus intensities used was 31.5–57 mA).

2.5. Experimental protocol

The experimental procedure was performed in three stages (fresh1, fresh2 and fatigue). In the fresh stages, the recordings were taken at different percentages of the maximum voluntary contraction (MVC). The subject had rested for 10 min between these stages (Hunter et al., 2006). The first and the second fresh stages were used to control the effect of time. The third phase was called the “fatigue” stage, in which the recording was obtained only during the rest condition (0% MVC) after the fatiguing protocol. The subjects were given rectified smoothed EMG as feedback to control the level of contraction of their muscles.

In order to determine the MVC, the subjects were asked to contract their muscles with their maximum effort in plantar flexion three times for a period of 5 s, with a rest of 10 s between the contractions. The highest level was determined as the MVC from the

rectified and smoothed EMG signals collected during these MVC attempts. In each of the fresh stages of the experiment, 0%, 10%, 20%, 30%, 40% and 50% of this MVC value was marked as lines on the feedback monitor in random order, and the subjects were asked to plantar flex to keep their smoothed triceps surae EMG on these lines. In each contraction level, 10 supramaximal stimuli were given randomly every 1–2 s. Using the supramaximal value, it was assumed that all the motor axons innervating the triceps surae were activated (Gandevia et al., 1999).

2.6. Fatigue protocol

To induce fatigue, the subjects were asked to plantar flex their left legs with their highest effort continuously. The muscles were assumed to have fatigued when the maximum force exerted on the load cell dropped to 40% of its initial value.

2.7. Analysis

In analysis, we accepted the point where the M-wave starts as the onset of electrical activity, and the point where the signal from the force sensors started to increase as the onset of force development. The time difference between these two points was calculated as the EMD (Fig. 2). In order to determine these points where M-wave and force starts, EMG and force data were averaged around the stimulus (–100 ms to +100 ms) in each contraction level.

Similar to our previously established method for identifying initiation of reflex deflection in the EMG (Türker et al., 1997; Brinkworth and Türker, 2003), we have used the maximal prestimulus deflection levels for both the EMG and the force for determining the very first poststimulus datum point that was above that level (Fig. 2). We determined the maximal deflections within 100 ms preceding the stimulus and placed horizontal cursors on them to identify the first significant poststimulus deflection. Both EMG and force signals were sampled at 2 kHz.

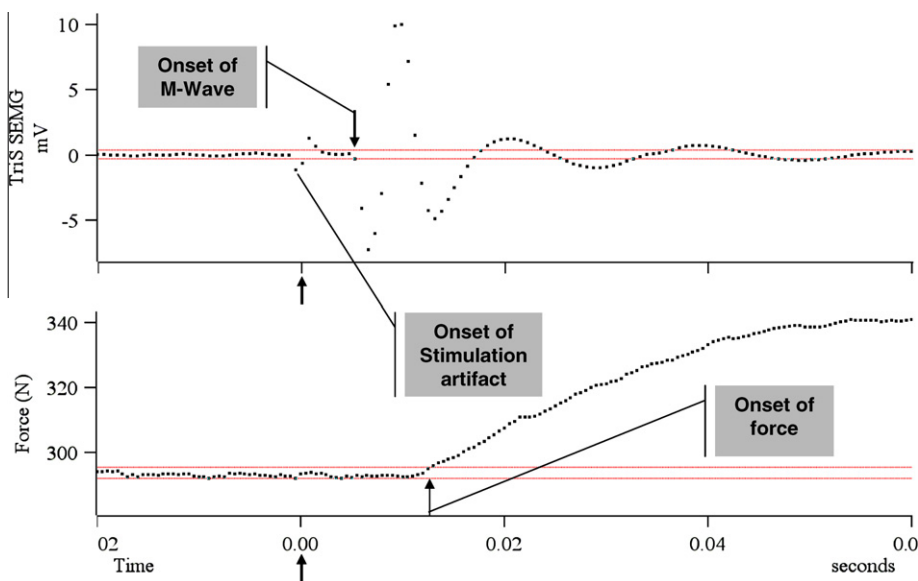


Fig. 2. An example of determining EMD. This figure shows the average of EMG and force around time of the stimuli ($n = 10$). Arrows indicate onset of electrical/force activity and time of the stimulus. EMD was determined as the time lag between the onset of M-wave and the onset of force. To determine the exact time, dot point graphs (each dot representing one individual sample at 2000 samples per second) were drawn instead of continuous lines in order to pinpoint the very first datum point which deviated from the prestimulus background level. Maximal prestimulus variability from 100 ms preceding the stimulus to time of the stimulus is indicated using horizontal lines. These lines are used to determine the first poststimulus datum point that is larger than the limits of these lines.

2.8. Statistics

SPSS® package program was used to analyse our results statistically. We used the general linear model with repeated measurements ANOVA to investigate the interaction between factors within subjects (contraction, stages) and between subjects (gender, age). In addition, the difference between genders, stages and contraction levels were investigated by repeated measurements ANOVA and the inter group relations was looked by Fisher's PSLD post hoc analysis. Before using ANOVA statistical test, the normality of distribution was proofed by Q-Q Plot test. Statistical significance was set to 0.05.

3. Results

3.1. EMD value

One of the most significant results of this study was the calculation of a considerably shorter EMD compared to previously reported values. The average EMD of all subjects at the rest condition was 8.5 ± 1.3 ms.

3.2. Gender difference

No statistically significant difference was observed in EMD between men ($n_m = 15$) and women ($n_w = 15$) in any of the conditions or in any of the contraction levels (Fig. 3). However, interestingly, EMD values of women were shorter than men in all of the situations, even though differences were not statistically significant. Gender difference was also examined within each age group, and no significant difference was found within any group either ($P > 0.05$).

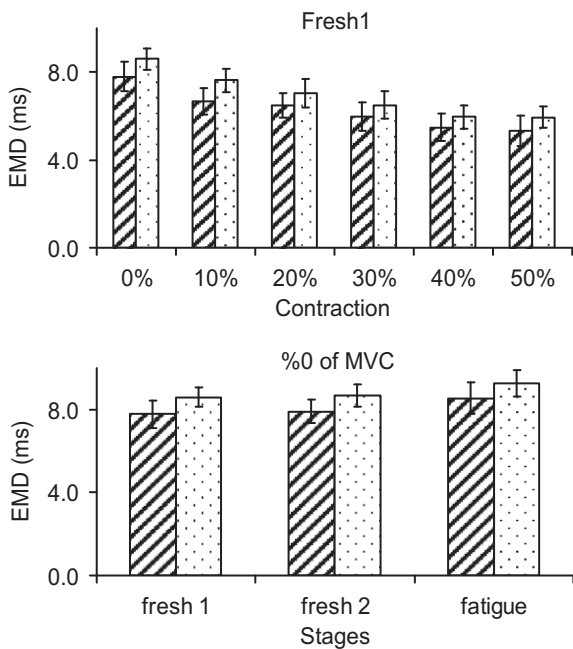


Fig. 3. Gender difference. Top histogram: the difference between women and men in six different contraction levels (0%, 10%, 20%, 30%, 40% and 50% of MVC). Bottom histogram: EMD values categorized by the stages (fresh1, fresh2 and fatigue). Diagonal lined bars indicate mean EMD values in women and dotted bars indicate mean values for men. Error bars indicate 95% confidence interval.

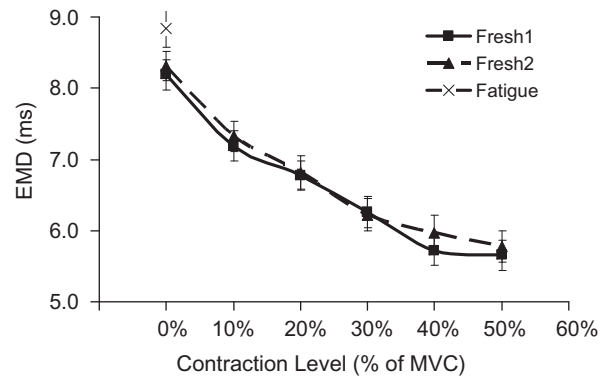


Fig. 4. The effect of contraction level on EMD. EMD values of fresh1 and fresh2 are indicated as lines and the EMD value for the post-fatigue recording is indicated as a star. EMD value decreases with increasing contraction level until 40% of MVC. Serial elastic component (SEC) is likely to have the main influence on the EMD reduction to the level of 40% of MVC (also see Nordez et al., 2009). Events that take place from the initiation to the completion of the force development in the contractile component (CC) and delay in the joint may account for the rest of the EMD.

3.3. Difference between contraction levels

Experiments comparing the EMD values during contraction levels of 0%, 10%, 20%, 30%, 40% and 50% of the MVC for each subject showed that the EMD decreased with increasing contraction level (Fig. 4). It was also observed that the EMD decreased until 40% contraction, and stayed approximately the same afterwards. Since there was no difference between men and women, the difference between contraction levels was investigated between all subjects ($n = 30$) by repeated measures ANOVA. Fisher's PSLD post hoc analysis showed that there was significant difference between all pairs ($P < 0.05$) except between 40% and 50% contraction levels ($P > 0.05$).

3.4. Effect of fatigue

Repeated measures ANOVA was applied in order to compare the EMD values obtained after each stage and the difference between stages. All possible pairs were compared by Fisher's PSLD post hoc analysis. The EMD values found at fresh1 and fresh2 stages were similar and there were no statistical significances in any of the contraction levels between these two fresh stages. However, the EMD values of the fresh stages determined at 0% MVC level

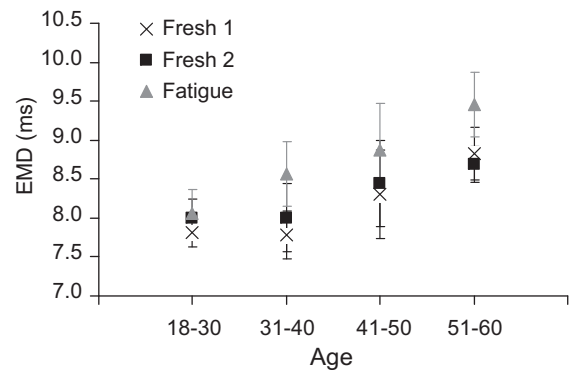


Fig. 5. The relationship between age and EMD. Each point indicates average EMD value in all subjects at 0% of MVC (at rest). Regression coefficients: ($R^2_{fresh1} = 0.87$, $R^2_{fresh2} = 0.88$, $R^2_{fatigue} = 0.96$). Age ranges for each group were as follows: Group 1 (18–30 years), Group 2 (31–40 years), Group 3 (41–50 years) and Group 4 (51–61 years). These groups were made up of 8, 7, 7 and 8 subjects, respectively.

were significantly shorter than the EMD value determined after the fatiguing contraction at the rest level ($P < 0.001$).

3.5. Effect of age

The subjects that were between the ages of 18 and 60 were grouped into four age intervals of 18–30, 31–40, 41–50 and 51–60. These groups were made up of 8, 7, 7 and 8 subjects, respectively. The graph showing the changes of EMD according to age groups revealed that EMD increased with age (Fig. 5). The best fit line drawn through the data points had significant regression coefficients ($R_{\text{fresh1}}^2 = 0.87$, $R_{\text{fresh2}}^2 = 0.88$, $R_{\text{fatigue}}^2 = 0.96$). Each point in Fig. 5 indicates EMD in all subjects at 0% of MVC.

4. Discussion

The primary finding of the present study is that the EMD value that was determined in the current study was shorter than in all previous studies. Secondly, we found that there was no difference between EMD values of males and females. Thirdly, we found that EMD increased with fatigue. Fourthly, EMD decreased with the increase in the contraction level. Finally our last novel finding was that an increase in EMD was observed with increasing age.

4.1. EMD and method

In the current study, other than a very recent study (Grosset et al., 2009), EMD values were found to be shorter than in previously reported values (Viitasalo and Komi, 1981; Miles and Wilkinson, 1982; Winter and Brookes, 1991; Zhou et al., 1995; Yang and Türker, 1999; Moore et al., 2002; Hopkins et al., 2007; Minshull et al., 2007; Blackburn et al., 2009; Nordez et al., 2009), which are summarized in Table 1. These differences are most likely to be due to the differences in methodologies used by researchers. One of the major differences between the present method and the others is the use of supramaximal electrical stimulation that activates all motor fibres in the tibial nerve (Gandevia et al., 1999). Since the entire muscle mass is activated by this kind of stimulation, determination of the onset of the M-wave and the resultant twitch becomes easy and accurate.

Some previous electrical stimulation EMD studies used stimulus intensity levels that just generated maximum M-wave amplitude (e.g., Hopkins et al., 2007). This level of stimulus intensity, however, may not be sufficient to activate all motor axons innervating the muscle, especially, when the relative position of the stimulating electrodes to the nerve changes slightly during an experiment. All underlying motor units may also not be activated when the EMD is determined using the reflex approach. Limited activation of a muscle during a reflex contraction depends not only upon the background level of excitation (Miles and Türker, 1986; Miles et al., 1989) but also the distribution of the synaptic input to the motoneuron pool (Heckman and Binder, 1990).

EMD determination using reaction time methods can also be criticised similarly as the timing of activation of the muscle depends upon the earliest recruited units. Since these units will be recruited asynchronously, determination of the initial set off point would be difficult to assess not only for the electrical record on the muscle and but also for the force record of the movement. Compared to this, both EMG and force records are much easier to pinpoint when the muscle is activated by generating action potentials in all motor units synchronously (Blijham et al., 2006).

Another important point in the method used is the position and distance between the bipolar EMG electrodes. Longer distance between electrodes (as long as one is placed on the innervations zone) allows us to record EMG from a larger area of the muscle

so that the initiation of the muscle action potential is recorded without any delay and without signal cancellation (Tucker and Türker, 2005). The distance between electrodes was arranged to be 6 cm in this study to ensure that the initiation of the M-wave was recorded more precisely by EMG.

Summary of the method used in the current study:

- Supramaximal stimulus strengths were used to make sure that all motor fibres are activated.
- EMG and force were recorded at high resolution and average responses were built around the time of the stimulus (using 10 stimuli).
- Each of the datum points (without connecting the data points to give a smooth image) were displayed to illustrate the exact timing of the changes on the average EMG and force records.
- Maximal prestimulus variations in the average force and EMG records were used to set thresholds for determining initiation of significant poststimulus events that are larger than the limits of the prestimulus variations.
- Long inter-electrode distances (one on the motor point and one several centimetres away from the first and along the direction of the muscle fibres) were used to allow the recording of the earliest arriving muscle action potentials.

4.2. Gender difference

Our results regarding the gender effect on EMD are contrary to some of the previous studies (Bell and Jacobs, 1986; Winter and Brookes, 1991; Zhou et al., 1995). However, as we have discussed above and summarized in Table 1, the EMD values in those studies were determined using methods that may not have included the activation of all motor units in those muscles and hence precise duration of the EMD may have been over estimated.

In this study, we hypothesized that women have longer EMD than men. We put forward this hypothesis based on some reports indicating more frequent tendon injuries in women than in men (Arendt and Dick, 1995; Ireland et al., 1997; Kannus, 1997). We have therefore tested this hypothesis to see whether the longer EMD in women may be one of the reasons for the high incidence of injuries. However, this hypothesis was not supported in this study. No significant difference was found between the EMD values of women and men. This may mean that the transfer of muscle activation to the movement of the foot is similar in both genders. The effect of oestrogen hormone on collagen synthesis which directly effect the quality of tendon structure is well known (Burgess et al., 2009a), and accordingly, the stiffness and the Young's modulus of the synthesized collagen are lower in women (Kubo et al., 2003). Similar EMD values that we found on both genders suggest that there are other factors that compensate the differences in the tendon structures.

4.3. Effects of fatigue

Our second hypothesis was that muscular fatigue elongated EMD, and results of this study confirmed this hypothesis. When we examined the relation between EMD and fatigue, we observed that EMD was lengthened by fatigue. There are a large number of studies regarding the effect of fatigue on skeletal muscles where different muscle fatigue definitions were proposed (Kallenberg et al., 2007; Enoka and Duchateau, 2008). In this study, fatigue was defined as a force decline to 40% of MVC. Of the two fatigue types that are known in literature (central and peripheral; Lopes et al., 1983), it is more likely that the peripheral fatigue is more effective on the EMD. Since the action potential propagation along T-tubules is impaired in the peripheral fatigue because of the K^+ efflux during maximum contraction (Sjøgaard et al., 1985; Allen

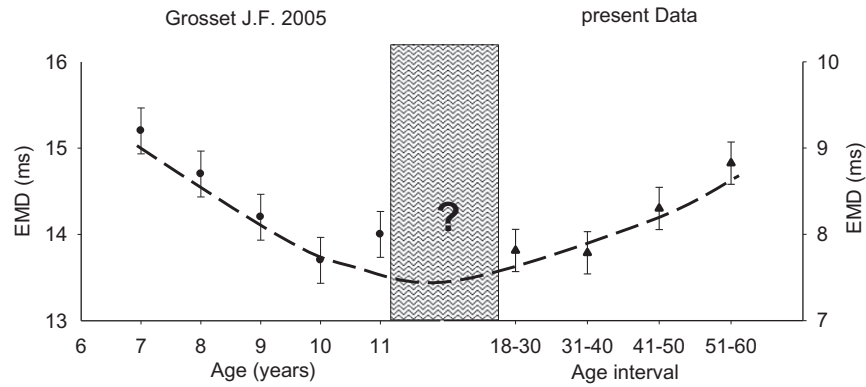


Fig. 6. Combination of Grosset's (left) and our findings (right). Our data shown in this figure consist of mean of all subjects at fresh1 stage at 0% of MVC. Note that these two graphs do not have the same scale, but drawing the two graphs together provided us to put forward a hypothesis to be tested later that the EMD is lowest during adolescent ages. Dashed line indicates tendency of changing of EMD with age roughly. Shaded area indicates the age group with unknown EMD.

et al., 2008), this may slow down the development of twitch force (Zhou et al., 1996). This suggestion may be strengthened by the findings that most of the tendon injuries are seen in fatigued individuals. More interestingly, some researchers have indicated that influence of fatigue on EMD differs between genders (Moore et al., 2002; Minshull et al., 2007) because the resistance of women and men to fatigue differs during different contraction types and levels (Russ and Kent-Braun, 2003; Enoka and Duchateau, 2008). Our findings support the influence of fatigue on the EMD but not the gender difference.

4.4. Contraction vs. EMD relationship

We hypothesized that the SEC behaves like a spring during voluntary contraction, and that the EMD will shorten with increasing level of contraction (Zhou et al., 1995; Muraoka et al., 2004). This hypothesis was also confirmed by our results. Our findings show that the muscle contraction level helps take up the slack on the muscle and its tendon and hence reducing the duration of the EMD. Tendon and muscle slack must be stretched to transfer the force developed in the muscle to the joint (Kawakami and Lieber, 2000).

Changing of EMD with contraction levels has been examined for 30%, 60%, 80% of MVC by Zhou et al. and they have reported that EMD was longer at 30% than other contraction levels (Zhou et al., 1995). Similarly, in our study EMD declined with the increase in the contraction levels until 40% MVC. EMD did not change between 40% and 50% of MVC. It can be assumed, therefore, that after 40% MVC level, the muscle arrives maximal stretching point and begins to behave like a rigid structure. In this case, the rest of EMD might originate from excitation/contraction coupling, parallel elastic component and delay within the joint. The EMD values obtained at high contraction levels in the current study (~6 ms) can be favourably compared with the findings of Nordez et al. (2009) who have illustrated that passive component of the SEC contributes to nearly half of the EMD measured in that study (47.5% of 11.63 ms).

4.5. Change in EMD with age

Except in one study on children (Grosset et al., 2005), there have been no studies in the literature that investigated the influence of the age on EMD. In the current study, EMD was investigated in women and men participants whose ages were between 18 and 60. Our hypothesis was that EMD prolongs with increasing in age. Since the system consists of muscle and tendon, which has viscoelastic properties as mentioned in the Hill's model (Enoka, 2008),

it is possible that this system may be exposed to deformation over the years (Johnson et al., 1994; Karamanidis and Arampatzis, 2005). Accordingly, as viscoelastic structures deform with age, slackness in the tendon may take place and hence EMD may increase, which may then increase that person's likelihood to have tendon injuries. The evidence has come from a recent study published by Burgess et al. They indicated that Young's modulus of tendon in elderly people is lower than in youngster (Burgess et al., 2009b). This means that the tendon becomes slacker in elderly people. On the other hand, the impairment of the movement of joint in elderly people may also be a factor in the elongation of EMD. In the only other study regarding the age related change (Grosset et al., 2005), EMD was examined in children and found that EMD decreases with age between 7 and 11 years old. Since our results show that EMD increases with age between 18 and 60 years old subjects, combining these two results indicate that EMD changes may generate a reverse bell shape curve that has a minimum at adolescent ages (Fig. 6).

5. Conclusion

EMD was found to be considerably shorter than those reported in previous studies. We have discussed methodologies used in various laboratories for EMD determination. Using the supramaximal stimulation approach, we have found no EMD differences between genders. We have also shown that EMD increases with advancing age and with muscular fatigue. The effect of contraction on EMD was one of our most dramatic findings, and can be used to determine the effect of the SEC on the mechanical properties of the muscle-tendon system.

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